2015-05-06

# The Effect of Disclosure of HIV Status and Sexual Orientation on HIV Prognosis over Four Years

Calvin J. Fitch
*University of Miami*, calvin.fitch.dlp@gmail.com

UNIVERSITY OF MIAMI

# THE EFFECT OF DISCLOSURE OF HIV STATUS AND SEXUAL ORIENTATION ON HIV PROGNOSIS OVER FOUR YEARS

By

Calvin J. Fitch

A THESIS

Submitted to the Faculty
of the University of Miami
in partial fulfillment of the requirements for
the degree of Master of Science

Coral Gables, Florida

May 2015

UNIVERSITY OF MIAMI

A thesis submitted in partial fulfillment of
the requirements for the degree of
Master of Science

# THE EFFECT OF DISCLOSURE OF HIV STATUS AND SEXUAL ORIENTATION ON HIV PROGNOSIS OVER FOUR YEARS

Calvin J. Fitch

Approved:

_____
Gail Ironson, M.D., Ph.D.
Professor of Biology

_____
Michael Antoni, Ph.D.
Professor of Psychology

_____
Rick Stuetzle, Ph.D.
Lecturer, Psychology

_____
M. Brian Blake, Ph.D.
Dean of the Graduate School

_____
Julie Barroso, Ph.D., ANP-BC, RN, FAAN
Professor of Nursing and Health Studies

FITCH, CALVIN J. (M.S., Psychology)
(May 2015)

The Effect of Disclosure of HIV Status and
Sexual Orientation on HIV Prognosis Over Four Years

Abstract of a thesis at the University of Miami.

Thesis supervised by Professor Gail Ironson.
No. of pages in text. (60)

Several studies have demonstrated the deleterious impact that psychological inhibition can have on psychological and immunological well-being. In the field of HIV, this psychological inhibition has often been operationalized as (a) lack of disclosure of HIV status or, for gay men, (b) disclosure of sexual orientation. However, research on the effect of disclosure on HIV disease status is limited, with only one study having examined both forms of disclosure simultaneously (Strachan et al., 2007). The present study seeks to replicate these findings by investigating whether or not disclosure of HIV status and disclosure of sexual orientation are related to HIV disease progression. The study seeks to extend the prior findings by Strachan et al. (2007) by employing a longer follow-up period (4 years) and using a more generalizable sample. Participants included 177 HIV-infected men and women recruited from hospitals and specialty clinics. At baseline, participants completed self-report measures of disclosure of both HIV status and sexual orientation. Participants also underwent a blood draw to assess CD4 cell count. Questionnaires and blood draws were repeated every 6 months for 4 years. Hierarchical Linear Modeling (HLM) analysis revealed that increased disclosure worries as measured by the HAT-QoL were significantly related to lower CD4 cell counts over a

period of four years. Results did not suggest that this relationship was mediated by depressive symptoms or social support. No significant results were found for any other HIV disclosure measures or sexual orientation disclosure measures. Possible reasons for non-significant findings are discussed. Significant findings may warrant close attention to disclosure worries as a person living with HIV begins to disclose their status.

## TABLE OF CONTENTS


Page

LIST OF TABLES ..... iv

LIST OF APPENDICES ..... v

Chapter

1 INTRODUCTION ..... 1
HIV Disclosure ..... 6
Sexual Orientation Disclosure ..... 13
Aims and Hypotheses ..... 19

2 METHOD ..... 21
Statistical Analysis ..... 25

3 RESULTS ..... 32

4 DISCUSSION ..... 37

REFERENCES ..... 45
TABLES ..... 51
APPENDICES ..... 58

LIST OF TABLES

TABLE 1:……..…… 51

TABLE 2……... 52

TABLE 3……... 53

TABLE 4……... 54

TABLE 5……... 55

TABLE 6…… 56

TABLE 7…… 57

LIST OF APPENDICES

APPENDIX A:…..……..…… 58

APPENDIX B…….... 59

APPENDIX C…….... 60

*Chapter 1:*

## Introduction

Health psychology researchers have tried to understand the connection between psychosocial variables and progression of human immunodeficiency virus (HIV). Specifically, researchers have been interested in examining how psychosocial variables might predict changes in CD4+ cell counts and viral load. For example, Ironson et al. (2005) found that, among 177 HIV-infected men and women, depression, hopelessness and lack of education predicted changes in CD4 cell counts and viral load over a period of two years. Furthermore, they found that life event stress and avoidant coping were positively related to viral load such that increased life event stress and avoidant coping predicted higher viral load, controlling for clinical features, substance use, and sociodemographic variables. An earlier study done by Ickovics et al. (2001) among 765 HIV-infected women showed significant differences in mortality between women with little or no depression, those with intermittent depression, and those with chronic depression. Over a period of 7 years, 8% of the women with little or no depression died, 16% of the women with intermittent depression died, and 23% of the women with chronic depression died. Among women with CD4+ cell counts of less than 200, intermittent and chronic depression was associated with twice the risk of mortality. Life stress (Evans et al., 1997; Leserman et al., 2002), religiosity/spirituality (Ironson, Stuetzle, & Fletcher, 2006), avoidant coping (Ironson et al., 2005), substance use (Carrico, 2011) and psychological inhibition (Eisenberger, Kemeny, & Wyatt, 2003; Ironson et al., 2013; Petrie et al. 2004; Strachan, Bennett, Russo, and Roy-Byrne, 2007;

Ironson et al., 2013) are other variables that have been shown to have an effect on HIV disease progression.

Before being applied to HIV, the concept of psychological inhibition had been used to demonstrate the effect of psychosocial factors on general health-related outcomes. The overarching theory which guided this work was based on the idea that non-disclosure about stressful or traumatic topics is a form of psychological inhibition. Several studies uncovered the physiological effects of psychological inhibition/disinhibition (Esterling, Antoni, Kumar, and Schneiderman, 1990, Fowles, 1980; Pennebaker, 1985; Pennebaker, Barger & Tiebout 1989; Pennebaker & Beall, 1986; Pennebaker, Hughes, & O'Heeron, 1987; Pennebaker, Kiecolt-Glaser, & Glaser, 1988). Much of the earlier work on psychological inhibition and health-related outcomes came from Pennebaker and colleagues. For example, Pennebaker and Beall (1986) randomized 46 undergraduate students to one of three different trauma writing groups or to a control writing group. The trauma writing groups were instructed to write a narrative about the facts of the trauma, to write only about their emotions related to the traumatic event, or to write about both the facts and their emotions related to the traumatic event. The control condition was instructed to write about a neutral topic. Writing occurred for 15 minutes on four separate days. While those in the trauma writing groups showed more short-term blood pressure changes, long-term effects were less deleterious for the trauma writing condition as they had fewer visits to the health center over a period of six months. This study provided promising evidence in support of the impact of psychological inhibition on health-related outcomes. In another study, Pennebaker, Barger & Tiebout (1989) conducted videotaped interviews with 33 Holocaust survivors about their experiences before and during World

War II while simultaneously monitoring their heart rate and skin conductance level. These autonomic measures were correlated with the degree to which the participant's experience was rated as traumatic to come up with a measure of personal disclosure. Positive correlations indicated a higher degree of disclosure. Controlling for health one week before the interview, the degree of disclosure was positively associated with health 14 months after the interview. Similar to the Pennebaker and Beall (1986) study, Pennebaker, Kiecolt-Glaser, and Glaser (1988) used undergraduates to demonstrate the effects of disclosure on buffering the negative effects of inhibition. Fifty healthy undergraduate students were assigned to write about a traumatic experience or about a control topic on four separate occasions. Those who wrote about the traumatic experience not only had fewer health center visits and improved mood relative to the control condition, they also showed better immune functioning as evidenced by the blastogenic response of T-lymphocytes to two mitogens. Furthermore, controlling for relevant medical and lifestyle variables, Esterling, Antoni, Kumar, and Schneiderman (1990) found that inhibition predicted poorer control of Epstein-Barr Virus regardless of whether one had a repressive or sensitive interpersonal style. Early work examining the psychosomatic effect of psychological inhibition provided strong evidence that inhibition has negative effects on general health and immune functioning.

Effects of psychological inhibition on immune functioning are particularly important for those who are HIV-infected, given the nature of HIV. HIV attacks the immune system's T-helper (CD4) cells, reducing the host's ability to fight off infection by foreign pathogens (Fahey et al., 1984). Severity of HIV infection is often quantified by the amount of virus present in the blood (i.e., viral load) and/or the degree to which T-

helper cells (CD4 cells) have been depleted (i.e., CD4 cell counts). A few other cells are also important in helping to fight off the infection. Natural killer cells (NKC) and cytotoxic (CD8) T-cells are instrumental in the immune system's defense against infection (Fehniger et al., 1998; Walker et al., 1991). NKCs are unique as they have a more aggressive immune response capable of killing virus-infected cells (and the virus within them) without having an antigen present on the cell surface (Fehniger et al., 1998). CD8 cells, however, require that virus-infected cells be marked with an antigen before they can be destroyed (Walker et al., 1991). Decreases in NKC number or function can hinder the body's ability to fight off an HIV infection. Increases in CD8 cells can be a signal of a higher viral load and worsened disease progression.

Research has provided evidence that psychological inhibition of concealable stigmatized status can promote psychological distress since the inhibition is typically driven by a fear of negative evaluation and an effort to avoid rejection (Segerstrom & Miller, 2004). This type of distress can have negative repercussions on the immune system. In 2004, Segerstrom and Miller (2004) conducted a meta-analysis of over 300 studies investigating the relationship between psychological distress and immune functioning. They found that while brief naturalistic stressors (such as taking an exam) were associated with suppression of cellular immunity but intact humoral immunity, more chronic stressors were associated with global deterioration of cellular and humoral immunity. Furthermore, in those with HIV, major and minor life events were significantly associated with lower numbers of natural killer cells and marginally associated with lower numbers of T-cytotoxic lymphocytes in peripheral blood. Greeson et al. (2008) applied these findings to a population of HIV-infected men and women by

using structural equation modeling to examine whether diminished killer lymphocyte number and function mediated the relationship between psychological distress (HIV-related anxiety, depression, and life stress) and HIV disease severity (as measured by viral load and CD4 cell count). In this cross-sectional study, not only was psychological distress associated with disease severity but this relationship was mediated by diminished innate immunity (i.e., reduced number and cytotoxic function of natural killer cells) and also increased T-cytotoxic lymphocyte activation. The psychological distress that often results from being psychologically inhibited has been reliably linked to poorer immune functioning which is particularly adverse for those living with HIV.

Psychological inhibition in the context of HIV is often operationalized as non-disclosure of one's HIV status and/or, in the case of men who have sex with men (MSM), it may be operationalized as non-disclosure of one's sexual orientation. Self-disclosure in these domains involves revealing personal information about oneself and is a complex process with a vast array of potential consequences. For example, the target of disclosure may find the information useful or they may find it harmful, which may result in either greater acceptance or greater rejection toward the person doing the disclosing (Zea et al., 2005). Potentiating the difficulty of self-disclosure is disclosure about one's seropositive HIV status, since the stigma associated with HIV leads many to think that a person who has contracted HIV has done so as a result of either homosexual contact, injection drug use, or prostitution, activities that are all stigmatized in and of themselves (Comer, Henker, Kemeny & Wyatt, 2000; Swendeman, Rotheram-Borus, Comulada, Weiss, & Ramos, 2006). Additionally, HIV is sometimes thought of as something that is avoidable which causes the infected person to bear complete responsibility for transmission

(Swendeman et al., 2006). A relatively large proportion of HIV infections are happening among gay and bisexual men (Prejean et al., 2011); thus, an additional stressor becomes whether or not to disclose one's sexual orientation and all of the possible consequences of doing so.

**HIV Disclosure**

Deciding whether to tell and who to tell about a positive HIV-status can be a complicated stressor, the results of which can have an effect on oneself and others. For instance, not informing sexual partners about a HIV diagnosis may lead to inaccurate estimation of risk for the seronegative partner and, consequently, increased risky sexual behaviors (Zea, Reisen, Poppen & Diaz, 2003). This increase in risky sexual behavior may leave unknowing sexual partners vulnerable to becoming infected with HIV. If the seronegative partner then becomes infected with HIV and conceals their new HIV positive status from sexual partners, the cycle can then repeat itself. Understanding what factors are associated with disclosure may help us to better understand the nature of HIV disclosure.

**Who discloses and why?** In order to better understand the relationship between HIV disclosure and disease progression, it is helpful to know who discloses and under what circumstances they disclose. One's cultural background has been shown to influence whether one discloses one's HIV status, (Bird, Fingerhut, and McKirnan, 2010; Petrak, Dorle, Smith, Skinner, & Hedge, 2001). For example, in a study by Bird et al. (2010), African American MSM were less likely than their Latino and White counterparts to disclose their HIV status to their sexual partners. Bird et al. (2010) note that one thing that has been thought to influence this difference in disclosure is stigma. Greater

endorsement of HIV-related stigma has been reliably associated with fewer disclosures of one's HIV status, though with modest effect size ($r$ = -0.189; Smith, Rossetto, and Peterson, 2008). Although HIV-infected individuals experience HIV-related stigma irrespective of their race, Blacks are more likely to report overt discrimination based on their HIV-status than their white counterparts (Overstreet, Earnshaw, Kalichman, & Quinn, 2013). For HIV-infected black women, both experienced stigma and perceived stigma go into their calculation of risk for disclosing their HIV-status (Black & Miles, 2002). Selective disclosure by these women in many cases is adaptive by allowing them to tell only specific trusted others, thus increasing their chances for social support. Furthermore, selective disclosure is occasionally used as a method by the women to protect vulnerable others from negative emotion (e.g., children or parents; Black & Miles, 2002). This concern for others is also seen in Asian American men. In one study, three issues related to non-disclosure of status among HIV-infected gay Asian men were protection of the family from shame, protection of family from feeling obligated to help, and avoidance of disclosing highly personal negative information (Yoshioka & Schustack, 2001). Increased actual or perceived stigma may play a role in the effect that culture has on likelihood of disclosure.

Disclosure may also depend on the target. For example, for mothers living with HIV, several factors may influence their decision regarding whether or not to tell their children such as stage of illness, inquiries from the child, child age and gender, and fears of stigma. In the workplace, it has been shown that only a minority of individuals might disclose (Simoni, Mason, and Marks, 1997). Qualitatively, reasons for disclosure might include concerns about performance or the need for accommodations, whereas factors

inhibiting disclosure might include privacy, the nature of the work environment, and fear of consequences (Fesko, 2001). Among sexual partners, it has been documented that greater disclosure of HIV status occurs with primary partners relative to casual partners (Sullivan, 2005). When we examine HIV disclosure in the context of romantic relationships, we can see gender differences in disclosure due, in part, to differing consequences for men versus women.

Mixed findings have been reported regarding gender differences in disclosure patterns (Obermeyer, Baijal, and Pegurri, 2012). However, research suggests that, simply due to their gender identification, women experience a distinct intensification of HIV-related stigma (Obermeyer et al. 2012; Sandelowski, Lambe, & Barroso, 2004). The ability to give birth to HIV-infected children is a contributing factor to the exacerbation of HIV-related stigma in women. In addition to the potentially positive effects of disclosure of HIV status, disclosure in light of the exacerbated stigma could have negative effects for women such as social isolation or violence (Sandelowski et al., 2004). For example, in a sample of over 250 middle-aged African American women from low SES backgrounds, 4% indicated that they were physically or sexually assaulted as a result of their status, and 16% reported having no one to count on for money or housing (Gielen, Fogarty, O'Campo et al., 2000). This suggests that for women, inhibition may be beneficial at times since disclosure of HIV status may have potentially negative physical or financial consequences and must be more carefully considered.

Serovich (2001) discusses theories relating to why people might disclose their HIV status. According to Serovich, the disease progression theory is one such theory and states that people may disclose as a result of the severity of the symptoms they are

experiencing. For example, Hays et al. (1993) found that disclosure among HIV-infected men who were asymptomatic was less frequent when compared to those who were symptomatic. It may be that the difficulty of hiding the diagnosis becomes much harder when one is experiencing symptoms, since one must then find ways to explain these symptoms to others without disclosing the true reason why the symptoms are occurring. Serovich (2001) mentions that the disease progression theory may not currently be as applicable due, in part, to the advances that have been made in regard to treating HIV. Advances in medical care for those living with HIV (e.g., HAART therapy) have allowed people to be asymptomatic for a longer time and thus, disease progression or increased symptomology may no longer be a compelling reason to disclose because fewer people are getting to the symptomatic stage.

The consequence theory of disclosure was proposed as another alternative (Serovich, 2001). This suggests that those fighting with the decision of whether or not to disclose are constantly weighing the potential costs and benefits of disclosing their status. This theory has had support in ethnic minority populations of MSM (Zea et al., 2005) and women (Black & Miles, 2002). Some of the potential costs that may be weighed are financial (e.g., loss of housing, employment), social (ostracism, degradation), and sometimes even physical in nature. Some of the benefits of disclosing HIV status may be increased social support, acceptance, assistance, and access to resources (Sandelowski, Lambe, & Barroso, 2004). When the benefits outweigh the potential costs, disclosure is more likely to occur (Serovich, 2001). Chaudoir, Fisher, and Simoni (2011) noted that the aforementioned theories commonly look at disclosure as the endpoint and do not take into account the potential consequences of disclosure.

The Disclosure Processes Model (DPM; Chaudoir et al., 2011) suggests that the decision to disclose is influenced by antecedent goals which can be in the form of approach goals (those in which a person is pursuing positive outcomes) or avoidance goals (in which the person is trying to avoid negative outcomes). These goals, then, go on to affect the disclosure event itself, where those with more approach goals will disclose more fully and in a more detailed manner than those with more avoidance goals which may then result in more positive responses from the targets of disclosure. Through multiple mediating processes (e.g., inhibition alleviation, changes in social support and social information), the disclosure event can go on to affect long-term individual, dyadic, and social/contextual outcomes. Thus, if a person has a good experience, they may feel alleviated inhibition, and may have increased social support, which may change the way that they feel they are perceived socially. Chaudoir et al. (2011) note that this model of disclosure maintains itself via a feedback loop where each disclosure event can affect subsequent disclosure events, such that those with more positive disclosure events will be increasingly more open about their HIV status and those with more negative disclosure events may be more secretive about their HIV status.

**HIV Disclosure and Disease Progression.** In knowing some of the reasons why people might disclose their HIV status in addition to knowing possible reasons for differences in disclosure rates across different types of people, only one study (Strachan et al., 2007) has examined what effect HIV status disclosure may have on HIV disease progression. Other studies have looked at disclosure about emotional or traumatic situations and related disclosure to HIV-related clinical outcomes such as CD4 and viral load. For example, Petrie, Fontanilla, Thomas, Booth, and Pennebaker (2004) looked at

emotional disclosure through writing as a means to improve health among HIV-infected individuals. Thirty-seven HIV-infected participants were asked to write for thirty minutes on four separate days about either the most traumatic or emotional experience of their lives or about how they used their time. Petrie et al. (2004) found that those who were instructed to write about their most traumatic/emotional experiences for four consecutive days had higher CD4 cell counts at the end of the intervention than did those who wrote about the neutral control topic. This study lends evidence to the notion that psychological disinhibition (i.e., not suppressing expression of thoughts/feelings and not being psychologically inhibited) may have advantageous effects on health outcomes, at least in the acute phase (Petrie et al., 2004). One of the major limitations, besides the small sample size of only 37 participants, is the lack of analysis of whether emotional writing has similar effects for men vs. women, and how long the therapeutic effects of written disclosure last. From the results, it is difficult to ascertain whether or not the effects of the writing intervention only last a short while and diminish over time. Petrie et al. (2004) suggested that the health benefits experienced by writing may be due to not inhibiting one's thoughts or feelings (i.e., disinhibition) rather than the expression of one's thoughts or feelings per se.

This study expands on prior work examining the effects of psychological inhibition on disease progression. Eisenberger et al. (2003) conducted interviews which cross-sectionally assessed HIV-specific emotional support, emotional support, and emotional expression/inhibition (measured by percentage of expressive vs. inhibitive words). When comparing these data to data collected on CD4 cell counts, they found that inhibition was associated with lower CD4 cell counts.

Ironson et al. (2013) expanded on some of the questions left unanswered regarding gender differences in the effect of written emotional disclosure on HIV-related outcomes. Two hundred forty-four men and women were instructed to write for 30 minutes on four separate days either about the most traumatic or upsetting experiences in their life, or about what they did the previous day, avoiding expression of emotion or opinion. Participants were followed over the course of one year. Although the researchers found no main effect of the intervention on self-reported HIV-related symptoms, there was a gender x group interaction, where women in the trauma-writing group saw a decrease in HIV-related symptoms from baseline to 12-month follow-up, while women in the control group experienced non-significant decreases. Among the men, there were no differences between the trauma-writing group and the control group in terms of HIV-related symptoms from baseline to 12 month follow-up. Interestingly, however, the writing intervention had no effect on objective measures of HIV disease progression (i.e., CD4 cell counts and viral load) for either men or women. The fact that HIV-related symptoms were subjectively measured may suggest some discrepancy between self-reported experience of disease progression and what may actually be happening. Perhaps, for women, the beneficial experience of emotional disclosure made symptoms easier to deal with and thus, less noticeable or pervasive. This work expands on Petrie et al. (2004), by including more women (96 women compared to only 2 women), having a longer follow-up period (12 months compared to six months), and by taking into account gender x group effects. Overall, results of Ironson et al. (2013) indicate that written disclosure may be more beneficial for women than men.

Strachan et al. (2007) was the only study to look specifically at how HIV disclosure predicted disease progression in HIV-infected individuals. A sample of 373 psychiatric outpatients was assessed for level of disclosure about both HIV status and sexual orientation (sexual orientation will be discussed in the next section) as well as CD4 cell counts. Degree of disclosure of HIV-status was measured on a five-point Likert-type scale. Participants were asked, in general, how open they are about their HIV status. They found that disclosure of one's HIV status was associated with increased CD4 cell counts over one year. A primary limitation to this study was the sample, which consisted entirely of psychiatric outpatients, 82% of whom had a diagnosis of major depression. Since the individuals in that sample already suffer from negative psychological states, it may be difficult to generalize findings to a non-psychiatric sample. The sample also consisted largely of men (almost 90%), which limits generalizability to women, a population in which we also see high rates of HIV infection (particularly among black women; Prejean et al., 2011) and unique disclosure patterns. The study also only examined effects on CD4 but did not examine effects on viral load. This study seeks to offer more generalizable evidence that concealment of one's HIV status may lead to a hastened progression of HIV virus by way of increasing viral load and decreasing CD4 cell counts. The current study seeks to increase generalizability by using a non-psychiatric sample with a higher concentration of women. Additionally, the current study will have a longer follow-up period (4 years).

**Sexual Orientation Disclosure**

Although HIV status disclosure has been shown to predict HIV disease progression, much of the research on disclosure has focused on the effect of sexual

orientation disclosure on disease progression in gay men. In these studies, those who conceal their sexual orientation are conceptualized as psychologically inhibited (Cole et al., 1996; Strachan et al., 2007; Ullrich, Lutgendorf, & Stapleton, 2003). A number of different variables including comfort with sexuality, acculturation, and satisfaction with social support may help to predict whether someone discloses their sexual orientation (Garcia, Lechuga, & Zea, 2012). Disclosure of sexual orientation has been shown to have a beneficial effect on the progression of HIV disease (Cole et al., 1996; Strachan et al., 2007; Ullrich, Lutgendorf, & Stapleton, 2003). Furthermore, disclosure of sexual orientation may buffer against negative effects of chronic stress (i.e., increased allostatic load, psychiatric symptoms, and altered stress hormone profiles; Juster, Smith, Ouellet, Sindi, & Lupien, 2013). However, much like disclosure of HIV status, disclosure of sexual orientation may produce negative consequences by increasing the risk of victimization. For example, in a sample of 416 LGB individuals, three out of four reported being victimized or physically threatened as a result of their sexual orientation (D'Augelli & Grossman, 2001). In order to better understand the effect that disclosure of one's sexual orientation may have, we must first investigate what makes people disclose sexual orientation in the first place.

**Predictors of Sexual Orientation Disclosure.** There are quite a few factors that have been shown to be predictive of one choosing to accept, and present to others, a homosexual identity. In general, one important predictor has been comfort level with their sexual orientation (Garcia, Lechuga, & Zea, 2012). This predictor may operate in a bidirectional fashion where the more someone is comfortable with their sexual orientation, the more likely it is that they may disclose and the more one discloses, the

more they can become comfortable with their sexual orientation. Satisfaction with social support as well as involvement in the gay community (e.g., attending events) were also shown to be significant predictors (Garcia et al., 2012).

However, likelihood of disclosure may change depending on the target of disclosure. For example, in the workplace, fear of disclosure has been associated with non-disclosure. Sexual orientation of co-workers and supervisors, and also perceived supportiveness of the workplace and opportunities to for promotion have been associated with increased likelihood of disclosure (Ragins, Singh, & Cornwell, 2007). In an environment of individuals with similar sexual orientation or even a supportive environment with plenty of opportunities for advancement, lesbian, gay, and bisexual (LGB) individuals were more likely to disclose their sexual orientation. One study showed that those who were more fearful about disclosure of sexual orientation and did disclose received fewer promotions and bonus incentives at their job. This suggests that non-disclosure may also be beneficial, especially when perceived social support and acceptance is low and perceived stigma is high.

There are also ethnic group differences in who discloses their sexual orientation. In talking about both an ethnic and sexual minority identity simultaneously, we introduce the idea of intersectionality – the overlapping and interaction of multiple marginalized identities and the experiences of subordination and dejection that may result. This concept was originally termed by Crenshaw (1989), to describe the struggles of black women in navigating experiences of both racism and anti-feminism. When applied to the literature on disclosure of sexual orientation, we do see, for example, African-American and Latino MSM may disclose their sexual orientation less often than their Caucasian

counterparts (Kennamer, Honnold, Bradford, & Hendricks, 2000; Rosario, Schrimshaw, & Hunter, 2004). Kennamer et al. (2000) also mention that part of this may be due to different social networks where Caucasians may have social support groups that include more gay/bisexuals but, whereas, African-Americans may have fewer gay/bisexual members in their social networks. This has been supported by research showing that Black men may experience more stress when interacting in gay social scenes than White men (Siegel & Epstein, 1996). In addition, Kennamer et al. (2000) note that members of ethnic minority groups may face more stringent ideas of masculinity and stigma associated with being gay or bisexual. Research suggests that these factors may not necessarily impact identity formation per se but may increase the time needed to integrate the various aspects of and individual's identity. Various predictors might influence whether or not one chooses to disclose their sexual orientation, and ethnic minority status can fundamentally change the experience of disclosure of sexual orientation.

**Sexual Orientation and Disease Progression.** A few studies (Cole et al., 1996; Strachan et al. 2007; Ullrich et al., 2003) have investigated the link between disclosure of one's sexual orientation and its effect on HIV disease progression. These results consistently show that disclosing one's sexual orientation is positively associated with CD4 cell counts such that the more open someone is about their sexual orientation, the more CD4 cells they have to fight off HIV infection, relative to those who conceal their sexual orientation (Cole et al., 1996; Ullrich et al., 2003; Strachan et al. 2007). Cole et al. (1996), instead of looking at disclosure of one's sexual orientation, examined the effects of concealing one's homosexual identity. They found that, among 80 seropositive gay men, those who concealed their sexual orientation progressed faster to critically low CD4

cell counts, AIDS diagnoses, and AIDS mortality over a period of nine years when compared to those who did not conceal their sexual orientation. This faster disease progression was not mediated by psychiatric diagnoses, social support, or repressive coping. As the researchers note, the sample was relatively homogeneous in terms of demographics and also fairly affluent so perhaps there was not enough variability in social support for it to function as a mediator. Although this study had one of the longest follow-up periods of any of the other studies of its kind, the data were collected between 1987 and 1993 which was the pre-HAART era so the data may not be generalizable to the present time since HAART has now dramatically increased the amount of time a person may live with HIV and not progress into AIDS.

Ullrich et al. (2003) expanded on the work of Cole et al. (1996) and examined the effects of concealing one's homosexual identity on HIV disease progression. They found, once again, that concealing one's sexual orientation was associated with a lower CD4 cell count, but only for those who rated high satisfaction with social support. Ullrich et al. (2003) reasoned that the positive effects of disclosure may be most salient under conditions of high social support satisfaction. It may also be that having high social support increases the amount of attention that is paid to a person. This increased attention, though good in nature, may be a cause of increased social arousal in people who are attempting to conceal their sexuality. Someone not satisfied with their social support network may not be receiving as much attention and may not experience as much arousal in trying to conceal their sexual orientation. As Ullrich et al. (2003) point out, although this study was not a true replication of Cole et al. (1996), it did add more evidence to the link between sexual orientation disclosure and HIV disease progression.

Strachan et al. (2007), in addition to examining HIV disclosure and its effect on disease progression, also examined the effects of sexual orientation disclosure on disease progression. Much like the other studies, the results of this study showed that disclosure of sexual orientation predicted increased CD4 cell counts over time. In relation to other studies examining the relationship between sexual orientation disclosure and HIV disease progression, a strength of this study was the use of a more heterogeneous sample in terms of demographics. The sample was both multiethnic and had representation from low SES individuals. Additionally, Strachan et al. (2007) were able to capture a more accurate portrayal of the effects today since there have been numerous advances in HAART therapy since the Cole et al. (1996) study was conducted. As discussed previously, the sample consisted of psychiatric outpatients versus the general population, which may limit generalizable to those not struggling with psychiatric conditions.

The present study seeks to investigate whether disclosure of HIV status (among HIV-infected men and women) and disclosure of sexual orientation (among HIV-infected gay men) predict HIV disease progression as evidenced by CD4 cell counts and viral load. It will use Hierarchical Linear Modeling (HLM) techniques to model change in CD4 cell counts over a period of 4 years, controlling for relevant sociodemographic and medical confounds. We will also investigate, among gay men, whether there is an incremental effect of disclosing in more than one area; that is, we will attempt to address the question of whether or not there are differences in CD4 change when one discloses neither HIV status or sexual orientation versus discloses one or the other versus whether one discloses both. Furthermore, we will investigate whether or not any significant effects of disclosure on disease progression might be mediated by reductions in

depressive symptoms or increases in social support. The study will expand upon prior work done by evaluating HIV disclosure and sexual orientation disclosure over a longer follow-up period and among a more generalizable sample. This study will potentially provide evidence in support of the hypothesis that psychological inhibition has a deleterious impact on HIV prognosis.

## Aims and Hypotheses

**Aim 1:** To determine whether disclosure of HIV status has an effect on CD4 cell counts over a period of 4 years.

Hypothesis 1: Higher levels of disclosure of HIV status at baseline will predict higher CD4 cell counts over a period of 4 years.

Hypothesis 2: Higher levels of disclosure of HIV status at baseline will predict lower viral load over a period of 4 years.

**Aim 2:** To determine whether disclosure of sexual orientation has an effect on CD4 cell counts and viral load over a period of 4 years.

Hypothesis 1: Higher levels of disclosure of sexual orientation at baseline will predict higher CD4 cell counts over a period of 4 years.

Hypothesis 2: Higher levels of disclosure of sexual orientation at baseline will predict lower viral load over a period of 4 years.

**Aim 3:** To determine whether social support and depression mediate the relationship between disclosure and HIV disease progression

Hypothesis 1: When depression and social support are controlled, the relationship between disclosure and disease progression will no longer be significant.

**Exploratory Aim #1:** To investigate patterns of disclosure among males and females and determine whether disclosure to each target differs as a function of gender.

**Exploratory Aim #2:** To investigate, among the gay men whether there is a synergistic effect of disclosing both HIV status and sexual orientation.

*Chapter 2:*

# Method

## Participants

One hundred seventy-seven participants were recruited from hospitals, specialty clinics, service organizations, and physician offices through study flyers and advertisements. Participants were HIV-positive and were classified in the midrange of illness (having CD4 cells between 150 and 500 at study entry). This was done in order to ensure that the effects of disclosure on CD4 cell counts could be captured in either direction. Exclusion criteria included having an AIDS-defining opportunistic infection (Category C) symptom, having lifetime CD4 cell count below 75, being under the age of 18, having active suicidality or psychosis, having a current substance dependence issue, or having another life-threatening illness.

Participants in the sample were mostly male (70.1%) and were about middle aged (M = 37.49, SD = 8.88). The sample was fairly heterogeneous in terms of ethnicity with the slim majority identifying as African American (36.2%) followed by non-Hispanic white (30.5%), Hispanic white (28.2%), and other (5.1%).

## Procedures

Data were collected between the years of 1997 to 2002. Participants were assessed every six months for a period of 4 years. At baseline, participants completed informed consent, demographic questionnaires, and a battery of psychosocial questionnaires. Participants also had blood drawn for CD4 assay and viral RNA quantification. Medication use and adherence were assessed using the AIDS Clinical Trials Group (ACTG; Chesney et al., 2000) adherence measure. Psychosocial questionnaires, CD4/HIV RNA assay,

and medication adherence information were assessed every six months at each follow-up period.

**Measures**

**Disease Progression**. CD4 cell counts (CD3+CD4) were assessed using whole-blood 4-color direct immunofluorescence using a coulter XL-MCL flow cytometer. To measure viral load (i.e., the amount of viral RNA contained in blood plasma), the Roche Amplicor assay sensitive to 400 copies/mL was used.

**Medication Adherence**. Ninety percent of participants were taking antiretroviral medicationsduring at least one study time point and thus, medication adherence information is only available for this portion of the sample ($n$ = 160). Medication adherence was assessed using the AIDS Clinical Trials Group Adherence Measure (Chesney et al., 2000). Adherence was operationalized as the proportion of missed doses in the preceding three days before their study visit. Analyses were re-run to control for medication adherence in an effort to separate any significant effects of inhibition from effects of pharmacological treatment.

**Psychosocial measures**. Depression was assessed at each time point (i.e., every six months) using the Beck Depression Inventory-II (BDI-II; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961). This 21-item questionnaire asks participants to rate the severity of different cognitive, affective, and behavioral symptoms of depression for the previous two weeks. The scores are added together to yield a total score. Higher scores indicate more severe depressive symptoms. Scores above 13 indicate mild depressive symptoms, scores above 19 indicate moderate depressive symptoms, and scores above 28 indicate severe depressive symptoms. The BDI is composed of both affective and somatic

symptoms. Given that somatic symptoms might be confounded by HIV or side effects from the medications used to treat it, the affective subscale will be used. The cumulative average for this subscale over the first four time points (2 years) was used to provide a more reliable measure of depressive symptoms versus using a measure from a single time point.

Social support was also evaluated at each time point using the ENRICHD Social Support Instrument (ESSI; Mitchell et al., 2003). The ESSI is a 7-item self-report measure which examines social support over the past month. Scores from each of the 7 questions will be added to yield a total score. Higher scores indicate more social support. We will also use the cumulative average for this scale to provide for a more reliable measure and to account for the possibility that changes in disease state might affect a person's social support network.

**HIV Disclosure.** To assess disclosure about HIV status, a scale was created which asks about degree of disclosure to several different targets in a person's social circle (i.e., mother, father, siblings, other relatives, fellow workers, employer, best male heterosexual friend, and best female heterosexual friend). For each target, the participants indicated whether they had 'fully disclosed' their orientation, 'disclosed in general terms,' 'said nothing on the topic,' 'misrepresented' their orientation or whether the target of disclosure was 'not applicable' to them (e.g., a participant who does not have a brother or sister). Full disclosure corresponded to a score of 2, disclosure in general terms corresponded to a score of 1, no disclosure corresponded to a score of -1, misrepresentation corresponded to a score of -2, and not applicable corresponded to a score of 0. Disclosure scores were averaged across all applicable targets of disclosure to

yield a score denoting a participant's average level of disclosure about their sexual orientation. Total level of disclosure was also calculated. Both scores were used separately in the analysis to avoid negatively affecting those with smaller social networks but also to avoid overlooking the possibility that disclosure to more people might also have an effect on disease progression. Thus, with this scale, we can examine total amount of disclosure or average level of disclosure across all targets and both have been included in the analysis.

HIV disclosure was also measured using the HIV disclosure worries subscale on the HAT-QOL (Holmes & Shea, 1998). The subscale contains five items which assess the degree to which a person has limited what they tell others. Each question is rated on a 5-point Likert-type scale with 5 denoting 'None of the time' and 1 denoting 'All of the time.' Thus, higher scores for the five items on this scale reflect fewer disclosure worries and lower scores reflect higher disclosure worries.

**Sexual Orientation Disclosure.** All of the women in the sample identified as heterosexual; however, a subset of the men from the sample identified as "homosexual" ($n$ = 96). Thus, information was collected from these men at baseline about their degree of disclosure about their sexuality to a variety of targets (i.e. mother, father, siblings, other relatives, fellow workers, employer, best male heterosexual friend, and best female heterosexual friend). For each target, the participants indicated whether they had 'fully disclosed' their orientation, 'disclosed in general terms,' 'said nothing on the topic,' 'misrepresented' their orientation or whether the target of disclosure was 'not applicable' to them (e.g., a participant who does not have a brother or sister). Full disclosure corresponded to a score of 2, disclosure in general terms corresponded to a score of 1, no

disclosure corresponded to a score of -1, misrepresentation corresponded to a score of -2, and not applicable corresponded to a score of 0. Much like with HIV disclosure, we chose to use both the total and average levels of disclosure in our analysis.

**Statistical Analysis**

SPSS Version 20 was used for preparation and descriptive analysis of data. Hierarchical Linear Modeling software (HLM 7) was used to investigate longitudinal changes in CD4 cell counts and viral load and the effect of psychosocial and medical variables on these changes. Hierarchical Linear Modeling was chosen as the method of analysis due to its ability to examine slope of CD4 cell counts and expected changes in slope as a result of psychosocial variables. Furthermore, it allows for statistical control of medication adherence at each time point. While latent growth modeling could have been applied from a structural equation modeling perspective, HLM is better equipped to handle smaller sample sizes (<200) in the level 2 data set (Huta, 2014). The variance in outcome variables over time is partitioned into two levels where Level 1 corresponds to within-person change in repeated measures of CD4 cell counts and Level 2 examines individual change in CD4 cell counts as a function of psychosocial variables. Means and standard deviations for psychosocial and demographic variables were calculated. Based on past research done with these data (Ironson et al., 2005) we anticipated that the values for viral load would be skewed; thus, we used a logarithmic transformation in SPSS and $\log_{10}$ values for viral load were used.

To accomplish the first aim, we used HLM to investigate the impact of HIV disclosure on HIV disease progression as measured by CD4 cell counts. This analysis included two levels. Level 1 covariates included use of antiretroviral medication and time

since baseline (measured in months) as time-varying covariates, and the interactions between these terms. Time since baseline corresponds to the time at which each of the 9 repeated measures was done (including the baseline measure). Antiretroviral medication was dummy-coded into three levels: no medication, combination therapy and HAART with the 'no medication' group serving as the reference group. Combination therapy excluded protease inhibitors or other HAART medications.

Level 2 focuses on individual differences in change in level 1 as a function of disclosure, controlling for a priori covariates relevant to HIV disease progression. Demographic variables such as gender (male vs. female), race (white, black, Hispanic, other), age, and education level (some high school or less, high school graduate, trade-school or some college, college graduate, graduate degree) were added to level 2 as covariates. Education level was used as a proxy for SES as employment might become less of a reliable indicator as participants advance in terms of HIV disease severity. Initial CD4 cell count at baseline was also added as a covariate to control for the possibility that starting CD4 cell count may influence HIV disease progression throughout the study. All continuous variables were centered and categorical variables were coded such that the lowest level of the variable is assigned a value of 0. The equations for Level 1 and Level 2 which will examine the effect of HIV disclosure on HIV disease progression are shown below:

Level 1 (repeated measures):

$$Y_{ti} = \beta_{0i} + \beta_{1i}\,(\text{time})_{ti} + \beta_{2i}\,(\text{antiretroviral1}) + \beta_{3i}\,(\text{antiretroviral2})_{ti} + \beta_{4i}\,(\text{antiretroviral1 x time})_{ti} + \beta_{5i}\,(\text{antiretroviral2 x time})_{ti} + e_{ti}$$

Level 2 (individuals):

$\beta_{01}$ (intercept) = $\gamma_{00} + u_0$

$\beta_{1i}$ (slope) = $\gamma_{10} + \gamma_{11}$ (Ethnicity) + $\gamma_{12}$ (Education)$_i$ + $\gamma_{13}$ (Age)$_i$ + $\gamma_{14}$ (Gender)$_i$ + $\gamma_{15}$ (Initial CD4)$_i$ + $\gamma_{16}$ (HIV Disclosure)$_i$ + $u_{1i}$

$\beta_{2j} = \gamma_{20}$

$\beta_{3j} = \gamma_{30}$

$\beta_{4j} = \gamma_{40}$

$\beta_{5j} = \gamma_{50}$

At Level 1 changes in repeated measures are examined for each individual. $Y_{ti}$ is the dependent variable and refers to CD4 cell counts for an individual (i) at a given time point (t). The equation is composed of an intercept, slope terms, and an error term which, when put together, are believed to account for an individual's variability from the regression line. $\beta_{0i}$ refers to the intercept which is the CD4 cell count at study entry for a given participant. $\beta_{1i}$ is a slope term which captures linear change in CD4 for a given participant. $B_{2i}$ - $\beta_{5i}$ represent slope terms for the main effect of type of antiretroviral medication with the latter two terms representing the interaction of antiretroviral medication and time. The addition of these terms accounts for use of a particular type of antiretroviral therapy and a given time point. Lastly, $e_{ti}$ captures the unexplained variance in CD4 cell count for a given participant at a given time point.

At Level 2 we investigated the effect of covariates and HIV disclosure on the change captured in Level 1. The intercept in level 2 refers to the average CD4 cell count across participants. The slope term is comprised of several terms. $\gamma_{10}$ refers to the average linear change in CD4 cell counts each month. $\gamma_{11}$ - $\gamma_{15}$ refer to the effect of a priori covariates on change in CD4 cell counts. $\gamma_{16}$ will represent the effect of individual differences in CD4

cell counts that is attributable to HIV disclosure. The u term will represent unexplained variance resulting from the estimation of the γ coefficients. The analysis was repeated using the total score from the HAT-QOL in lieu of values from the HIV disclosure questionnaire. The HLM model and equations will be identical to those above; however, the score that is used for HIV Disclosure will come from the total HAT-QOL score rather than the primary HIV disclosure measure.

The Level 1 and Level 2 analyses were repeated using $\log_{10}$ viral load as the outcome measure (equations are listed below). For Level 1, $Y_{ti}$ will be the dependent variable and refers to the viral load for an individual (i) at a given time point (t). $\beta_{0i}$ refers to the intercept or viral load at study entry for a given participant. $\beta_{1i}$ is a slope term which captures linear change in viral load for a given participant. $B_{2i}$ - $\beta_{5i}$ represent slope terms for the main effect of type of antiretroviral medication with the latter two terms representing the interaction of antiretroviral medication and time. The addition of these terms accounts for use of a particular type of antiretroviral therapy and a given time point. Lastly, $e_{ti}$ captures the unexplained variance in viral load for a given participant at a given time point.

At Level 2 we investigated the effect of covariates and HIV disclosure on the change in viral load captured in Level 1. The intercept in level 2 refers to the average or grand mean of viral load for all participants. The slope term is comprised of several terms. $\gamma_{10}$ refers to the average linear change in viral load each month. $\gamma_{11}$ - $\gamma_{15}$ refer to the effect of a priori covariates on change in viral load. $\gamma_{16}$ will represent the effect of individual differences in viral load that is attributable to HIV disclosure.

Level 1 (repeated measures):

$Y_{ti} = \beta_{0i} + \beta_{1i}\,(time)_{ti} + \beta_{2i}\,(antiretroviral1) + \beta_{3i}\,(antiretroviral2)_{ti} + \beta_{4i}\,(antiretroviral1 \times time)_{ti} + \beta_{5i}\,(antiretroviral2 \times time)_{ti} + e_{ti}$

Level 2 (individuals):

$\beta_{01}$ (intercept) $= \gamma_{00} + u_0$

$\beta_{1i}$ (slope) $= \gamma_{10} + \gamma_{11}\,(Ethnicity) + \gamma_{12}\,(Education)_i + \gamma_{13}\,(Age)_i + \gamma_{14}\,(Gender)_i + \gamma_{15}\,(Initial\ VL)_i + \gamma_{16}\,(HIV\ Disclosure)_i + u_{1i}$

$\beta_{2j} = \gamma_{20}$

$\beta_{3j} = \gamma_{30}$

$\beta_{4j} = \gamma_{40}$

$\beta_{5j} = \gamma_{50}$

The HLM models used to investigate whether disclosure of sexual orientation impacts HIV disease progression were similar in many aspects to the one which examines the impact of HIV disclosure. However, in the model examining the effect of disclosure of sexual orientation on CD4 cell counts and the model examining the effect on viral load, gender was not added as a covariate (since all of the participants in this analysis will be males). Additionally, the level 2 slope equation does not have a $\gamma$ coefficient corresponding to HIV disclosure; this was replaced with a score for sexual orientation disclosure. When examining the effect of sexual orientation on viral load, the $\gamma_{15}$ coefficient term will be multiplied by initial viral load and not initial CD4 cell count. Sample size will be reduced from 177 to 96 participants as this analysis will be restricted to the gay men in the sample.

Level 1 (repeated measures):

$Y_{ti} = \beta_{0i} + \beta_{1i}\,(time)_{ti} + \beta_{2i}\,(antiretroviral1) + \beta_{3i}\,(antiretroviral2)ti + \beta_{4i}\,(antiretroviral1 \times time)ti + \beta_{5i}\,(antiretroviral2 \times time) + e_{ti}$

Level 2 (individuals):

$\beta_{01}$ (intercept) $= \gamma_{00} + u_0$

$\beta_{1i}$ (slope) $= \gamma_{10} + \gamma_{11}\,(Ethnicity) + \gamma_{12}\,(Education)_i + \gamma_{13}\,(Age)_i + \gamma_{14}\,(Initial\ CD4)_i + \gamma_{16}\,(Gay\ Disclosure)_i + u_{1i}$

$\beta_{2j} = \gamma_{20}$

$\beta_{3j} = \gamma_{30}$

$\beta_{4j} = \gamma_{40}$

$\beta_{5j} = \gamma_{50}$

In the event of significant results for the impact of our disclosure variables on disease progression markers, we investigated whether or not the findings were mediated by depression or social support. Baron and Kenny's (1986) methods for testing mediation will be used as they may apply to multilevel models (Zhang, Zyphur, and Preacher, 2009). Zhang et al. (2009) would describe the mediation measure as a '2-2-1' model in which a level 2 predictor is hypothesized to influence a level 2 mediator which then influences a level 1 outcome variable. Per Zhang et al. (2009), the first step in the mediation process is to establish significance between the predictor and the outcome variable (path c). The next would be to establish significance between the predictor and the proposed mediator (path a). The next step would be to establish significance between the proposed mediator and the outcome (path b). Lastly, we must control for the proposed mediator and examine the relationship between the predictor and the outcome (c'). In looking at the relationship between c and c', if c' becomes non-significant, we will

consider the mediator a full mediator. If c' remains significant but lowers in absolute value, the mediating variable will be considered a partial mediator.

*Chapter 3:*

# Results

## Missing Data

Examination of the data revealed large proportions (> 20%) of missing data for HIV disclosure variables and HAT-Qol measures. The reason for the missing data was that these questionnaires were not administered until later in the study accrual period. People who were not given the questionnaires were excluded from the analysis. This was thought to be an appropriate way of dealing with the missing data since the people missing were thought to be a random subset of the sample. Thus, we did not hypothesize that there would be any systematic differences between observed values and missing values. Due to the amount of missing data and also the complexity of the model, it was thought that multiple imputation might lead to biased estimates (Hardt, Herke, Brian, & Laubach, 2013).

## Description of Sample

Statistics on participant demographics for our full sample as well as the reduced sample of $n = 143$ are listed in Table 1. Our overall sample ($n = 177$) was diverse in many demographic areas such as ethnicity, sexual orientation, and gender. Most of the participants had had an education level of some college/trade school or less and were of low SES (reported income of less than $10,000 per year). In terms of medical variables, participants reported an average CD4 cell count of approximately 297 and an average viral load of approximately 44,861. Our reduced sample ($n = 143$) appears to match the full sample in terms of demographics and no clinically meaningful differences exist in terms of our medical variables. Descriptive statistics for covariates and variables of interest in the main models are listed in Table 3.

Descriptive statistics for different patterns of disclosure are listed in Table 2. Among men, the most common targets of disclosure either in full or in general terms were best femaleheterosexual friend (64.9%), mothers (63.6%), and spouses (61.1%). The targets who were disclosed to the least were coworkers (32.6%), bosses (34.4%), and fathers (44.3%). Among, women, the most common targets of disclosure either in full or in general terms were mothers (81.0%), best female heterosexual friend (60.5%), and best male heterosexual friend (57.9%). Chi-square tests of independence (Table 2) revealed that disclosure in full or in general was dependent on gender when disclosure was to best male homosexual friend, best female homosexual friend and to spouse. Among gay men, the most common targets for either full disclosure or disclosure in general terms were best female heterosexual friend (45.8%), mother (43.5%), and best male heterosexual friend (39.0%). The least common targets were bosses (20.3%), coworkers (26.6%), and fathers (28.8%).

**Modeling Change in CD4 Cell Counts**

**Baseline Model.** Statistical significance tests for the baseline model examining change in CD4 over time as a function of a priori covariates are presented in Table 3. The tests show a non-significant linear decrease in CD4 cell count over a period of 4 years with a decline of about -3.14 CD4 cells per month. The baseline model also revealed significant individual differences in the trajectory of change over time, $\chi^2$ (168) = 543.97, $p < .001$. Use of combination therapy or HAART was associated with a higher CD4 cell count. In terms of level 2 predictors, baseline CD4 was a significant predictor of change in CD4 over time where higher baseline CD4 and more education were protective against

CD4 cell declines. Ethnicity, age, and gender did not significantly predict change in CD4 cell counts over time.

**HIV Disclosure variables and CD4 cell change over time.** We investigated whether or not HIV disclosure was related to change in CD4 cell count over time using three different measures of HIV disclosure. Results can be found in Table 5. Recall that we examined average level of disclosure across targets, total amount of disclosure across targets and total score on the HIV disclosure worries subscale of the HAT-QoL. Higher scores on the HAT-QoL predicted significantly greater increases in CD4 cell counts over the 4 year period, $\gamma_{16} = 0.199$, $t\ (91) = 2.52$, $p < .05$, controlling for a priori covariates. Average level of disclosure and total amount of HIV disclosure at baseline were not significantly related to change in CD4 cell counts. In controlling for medication adherence, these results still held, $\gamma_{16} = 0.200$, $t\ (91) = 2.16$, $p < .05$. We did not find any evidence that disclosure of either HIV status or sexual orientation versus disclosure of both versus disclosure of neither had an effect on change in CD4.

**Testing for Mediation**. In light of the significant effect of higher HAT-QoL scores on CD4 cell counts over time, per our a priori hypotheses, we investigated whether or not this relationship was mediated by depression or social support. *Path C* (the relationship between the independent variable and the outcome) was already established in the main analyses. The value of the coefficient for this path can be found in Table 7. The significance of *Path A* (the relationship between the independent variable and the proposed mediator, i.e., depression) was tested with hierarchical linear regression using the same a priori covariates. Path A was not significant, and thus we were unable to proceed with testing depression as a mediator. In testing cumulative social support (ESSI

scores over four time points), we were unable to show a significant relationship between social support and change in CD4 cell counts (path b). Thus, we were unable to proceed with testing social support as a mediator.

**Sexual Orientation Disclosure.** We did not find that the total amount of disclosure of sexual orientation across targets nor the average level of disclosure of sexual orientation across targets predicted changes in viral load.

**Modeling Change in Viral Load**

Statistical significance tests for the baseline model examining change in viral load over time as a function of a priori covariates can be found in Table 4. Results show that the average baseline viral load was 4.43 log units and there was a significant linear increase of about 0.013 log units per month. The baseline model also revealed significant differences among participants' viral load growth trajectories, ($\chi^2$ (168) = 284.34, $p < .001$). Use of combination therapy or HAART was associated with decreases in viral load. Ethnic minority status was marginally associated with viral load increases over time with ethnic minorities showing higher viral load than non-ethnic minority individuals. No other a priori covariates were significantly related to change in viral load.

**HIV disclosure and sexual orientation disclosure on change in viral load**. As seen in Table 5, we did not find that any of our HIV disclosure summary scores or HAT-QoL were significantly related to change in viral load over time. We also did not find any significant relationships between our sexual orientation disclosure variables and change in viral load over time. We also did not find any evidence that disclosure of either HIV

status or sexual orientation versus disclosure of both versus disclosure of neither had an effect on change in viral load.

## *Chapter 4:*

## Discussion

The current study sought to examine whether disclosure of HIV status and/or sexual orientation was related to HIV disease progression. We hypothesized that higher levels of disclosure of HIV status or sexual orientation would predict slower disease progression. In looking at general patterns of disclosure, we see that mothers and friends are often disclosed to most often in terms of both HIV-status and sexual orientation. We also see that disclosure of either status in the work place is less likely. We also found that disclosure to spouse was differentially affected by a participant's gender with less women disclosing to their spouses than men. This seems to be consistent with the idea that many women may choose not to disclose as a protective strategy (Sandelowski et al., 2004). We found partial support for our HIV disclosure hypothesis as the total score on the disclosure worries subscale significantly predicted higher CD4 cell counts over 4 years. This suggests that the less worried someone is regarding disclosing their HIV status and the less this worry impedes their disclosure, the better their immune functioning. However, we did not find that the link between disclosure worries and change in CD4 was mediated by depressive symptomology or social support. Results from the HIV disclosure worries analysis are consistent with the model proposed by Greeson et al. (2008). Although we did not examine natural killer cell activity or T-cell cytotoxicity, our analysis revealed that psychological distress resulting from disclosure worries was significantly related to deleterious changes in immune functioning over time.

Our significant findings for the HAT-QoL suggest disclosure worries predict disease progression over a period of 4 years. This finding offers support for Greeson's

(2008) model of the relationship between psychological distress and immune functioning. Since the HAT-QoL talks about worry which is a common trait of anxiety, it may be that anxiety is what primarily drives the relationship between non-disclosure and accelerated disease progression. Indeed among those living with HIV, we see higher rates of PTSD and GAD when compared to the general population and these rates may be even more pronounced in groups with higher prevalence of HIV infection (i.e. MSM and high-risk women; O'Cleirigh, Hart, & James, 2008). Among individuals with anxiety disorders, we see higher norepinephrine reactivity in response to stressors and abnormal cortisol release patterns (Brawman-Mintzer et al., 1997; Tancer et al., 1993). As mentioned earlier, these things may have deleterious effects of HIV disease progression as increased norepinephrine levels have been shown to predict accelerated disease progression, perhaps through facilitation of viral entry into cells and subsequent viral replication (Cole, Korin, Fahey, & Zack, 1998; Cole, Kemeny, Fahey, Zack, & Naliboff, 2003; Ironson et al., 2014). Those with higher levels of disclosure worries may show an accelerated disease progression in part due to dysregulation in the release of stress hormones, namely norepinephrine and cortisol; however, future research would be needed to say this conclusively.

We did not find any significant results when we looked at our HIV disclosure scales or our sexual orientation disclosure scales. One of the reasons for the non-significant results could have been the disclosure measures themselves. One potential reason is that the measure only captures the actual act of disclosing, not necessarily the phenomenon of psychological disinhibition. Psychological disinhibition refers to keeping something concealed that, in an ideal world, one would like to disclose. As Petrie et al.

(2004) noted in their study, it may not be the actual act of disclosing that is important, it may be not inhibiting. It may have been the case that many participants in the current study who were not disclosing were doing so for protective reasons (e.g., to avoid financial losses or to avoid physical harm). For these people, non-disclosure may have not equated to inhibition because they were not disclosing on their own volition. However, past studies (Cole et al., 1996; Strachan et al., 2007; Ullrich et al., 2003) have used a single-item rated on a 5-point Likert-type scale asking participants how open they are about either their HIV status or sexual orientation and have found significance. Another potential reason for the non-significant findings could have been the fact that the disclosure measures were only administered at baseline. It may have been that people disclosed later. Indeed, research does indicate that increased symptomology may be a reason for disclosure either voluntarily or involuntarily (Serovich & Mosack, 2003). Baseline CD4 cell counts from the current study do, in fact, reveal that the sample was fairly immunologically compromised. Having measures of disclosure at subsequent time points would have allowed later disclosure to be added as a time-varying covariate in the HLM model and may have increased the chances of finding an effect. Strachan et al. (2007) employed this method and found an effect; however, Cole et al. (1993) did not employ this method and found significant effects as well when examining sexual orientation disclosure. Lastly, we observed a non-significant linear trend in CD4 and viral load. This suggests that, although there was a lot of inter-individual variation in these disease markers, the intra-individual change over the study period was minimal. That is, each individual stayed relatively constant in terms of disease progression. Lower amounts of intra-individual change results in less intra-individual variation over time that can be

explained by predictors in the model and could make it difficult to find an effect. Lastly, we must consider the possibility that the relationship truly does not exist. While results may appear to suggest that HIV disclosure and sexual orientation disclosure are not significantly related to HIV disease progression, we cannot say this conclusively until these factors are addressed, especially in light of significant results from other studies.

However, the findings for our other disclosure variables for HIV and sexual orientation are contradictory to the literature. One point raised earlier was that our disclosure measures did not necessarily account for the phenomenon of psychological disinhibition per se and concealment for many of the participants may have been beneficial. However, what complicates this point is that other studies have used similar measures which do not quite get at disinhibition and have found an effect (Cole et al., 1993; Strachan et al., 2007; Ullrich et al., 2003). When we look closer at sample characteristics from other studies in comparison to the multiethnic, multi-gender, low SES sample in the current study, there are some noteworthy differences. For example, the sample for Cole et al. (1993) consisted of relatively affluent Caucasian gay men. With more resources at one's disposal and less reliance on others for these resources, disclosure may be beneficial; however, with less resources and more dependency on others for resources, disclosure can be harmful. The sample in Ullrich et al. (2003) was low SES like the sample for the current study but also consisted entirely of men and the overwhelming majority of the men were Caucasian. The most closely matched sample to the sample in the current study was that of Strachan et al. (2007) which was multiethnic and low SES; however, the sample had a lower amount of representation from women. As mentioned previously, the consequences of disclosure can be fundamentally different

for men and women. While our significant findings are consistent with what is documented in the existing literature, the non-significant findings in the current study may suggest that capturing the disinhibition component of disclosure may be particularly important when dealing with populations who are marginalized on multiple domains (e.g., gender, ethnicity, and SES) as non-disclosure may not necessarily equate to inhibition for these groups.

It would be premature to suggest that, based on the results, not being worried about disclosing one's HIV status is beneficial for health. It would also be premature to suggest that disclosure of HIV status or sexual orientation do not matter in terms of HIV disease progression. In many cases, it may be beneficial to either be worried about disclosing one's status or to keep it concealed as disclosing it may have worse consequences than keeping it concealed. The findings, however, do point to the influence that disclosure worries might have on immunological functioning. The stigma attached to being HIV-infected makes the decision of what to disclose and to whom very complicated and stressful and this complication and stress may be exacerbated in certain groups relative to others (e.g., women). Clinically, the findings may warrant close attention to patients as they navigate through the complicated nature of disclosure of their HIV status.

**Limitations**

This study comes with its limitations which have been mentioned in other parts of this section. The most obvious of these limitations would be the sample size. With the measures only being administered to a subset of the study sample reductions in sample size and statistical power becomes an issue. This may leave us vulnerable to Type II error

(i.e. failing to detect an effect when an effect really exists). Another limitation is the disclosure scales not assessing for disinhibition. While the scales ask about the degree of disclosure for various targets, they do not address the participant's satisfaction with that level of disclosure. Would they like to have disclosed more? Disclosed less? Two individuals may have said nothing on the topic of their HIV status or sexual orientation; however, for one individual that may be the desired level of disclosure whereas another person may desire to disclose more but chooses to stay inhibited. Additionally, these measures were only administered at one time point in the study in which limits the ability to control for later disclosure of HIV status or sexual orientation.

**Future Directions**

Future research might look into standardizing the way that disclosure is measured and creating a scale that not only captures the actual act of disclosure but also whether or not that act constitutes an act of psychological disinhibition. This study is the only one of its kind to capture disclosure information from multiple targets in a person's social network and integrate this into composite measures. Most of the other studies of this kind have used a general question that taps into how open people are about their HIV status or sexual orientation in general. A standardized way of measuring disclosure in the context of psychological inhibition would increase consistency across studies and also allow us to truly investigate the phenomenon of psychological disinhibition. Future work might also look at whether differential effects on disease progression occur when the objectivity of the measurement of disclosure changes. It could be argued that measures of disclosure in the current study were somewhat more objective since in past studies, participants were asked in general how open they are about HIV status or sexual orientation. In the current

study, this information was calculated for them based on their level of disclosure to various targets. Also, while we did not find that depression or social support mediated the relationship between disclosure worries and change in CD4, future work might look at investigating other potential mediators or moderators to the relationship. Earlier in the discussion, we speculated on the role of gender, ethnicity, and SES as potential moderators, Future work migh include interaction terms for these variables and disclosure and investigate the effects of these interactions on change in CD4 and viral load. In this study disclosure was measured at one time point and this did not allow us to account for the possibility that disclosure may have occurred later. In future studies, it may be beneficial to measure disclosure at multiple time points and add the subsequent disclosure measures as time-varying covariates to the model. Lastly, we found significant results for disclosure worries predicting change in CD4 over time, and given the link between anxiety and accelerated disease progression, future work may consider looking at hormonal mediators (e.g. norepinephrine and cortisol) to see if they may mediate the relationship between disclosure worries and accelerated disease progression.

The present study found that disclosure worries were significantly related to change in CD4 cell counts over a period of 4 years. The study was unable to provide support for the influence of sexual orientation disclosure on CD4 cell counts or viral load and also not able to show significance with a more comprehensive measure of HIV disclosure. This may have been attributable to low sample size or the disclosure measures themselves. Findings suggest that disclosure worries may be an important target for intervention, and considering the sample and the differential stigma associated with ethnic minority groups, it may be especially important for this population. However,

future work must be done in this area. Future work might look at standardizing measures of disclosure to aid in a more consistent portrayal of the nature of psychological disinhibition and its effects on immune functioning.

## References


Beck, A. T., Ward, C., & Mendelson, M. (1961). Beck depression inventory (BDI). *Arch Gen Psychiatry, 4*(6), 561-571.

Bird, J. D., Fingerhut, D. D., & McKirnan, D. J. (2011). Ethnic differences in HIV-disclosure and sexual risk. *AIDS Care, 23*(4), 444-448.

Black, B. P., & Miles, M. S. (2002). Calculating the risks and benefits of disclosure in african american women who have HIV. *Journal of Obstetric, Gynecologic, & Neonatal Nursing, 31*(6), 688-697.

Carrico, A. W. (2011). Substance use and HIV disease progression in the HAART era: Implications for the primary prevention of HIV. *Life Sciences, 88*(21), 940-947.

Chaudoir, S. R., & Fisher, J. D. (2010). The disclosure processes model: Understanding disclosure decision making and postdisclosure outcomes among people living with a concealable stigmatized identity. *Psychological Bulletin, 136*(2), 236.

Chaudoir, S. R., Fisher, J. D., & Simoni, J. M. (2011). Understanding HIV disclosure: A review and application of the disclosure processes model. *Social Science & Medicine, 72*(10), 1618-1629.

Chesney, M. A., Ickovics, J., Chambers, D., Gifford, A., Neidig, J., Zwickl, B., . . . Patient Care committee & adherence working group of the outcomes committee of the adult aids clinical trials group (AACTG). (2000). Self-reported adherence to antiretroviral medications among participants in HIV clinical trials: The AACTG adherence instruments. *AIDS Care, 12*(3), 255-266.

Cole, S. W., Kemeny, M. E., Taylor, S. E., Visscher, B. R., & Fahey, J. L. (1996). Accelerated course of human immunodeficiency virus infection in gay men who conceal their homosexual identity. *Psychosomatic Medicine, 58*(3), 219-231.

Comer, L. K., Henker, B., Kemeny, M., & Wyatt, G. (2000). Illness disclosure and mental health among women with HIV/AIDS. *Journal of Community & Applied Social Psychology, 10*(6), 449-464.

Crenshaw, K. (1989). Demarginalizing the intersection of race and sex: A black feminist critique of antidiscrimination doctrine, feminist theory and antiracist politics. *U.Chi.Legal F.,* , 139.

D'AUGELLI, A. R., & Grossman, A. H. (2001). Disclosure of sexual orientation, victimization, and mental health among lesbian, gay, and bisexual older adults. *Journal of Interpersonal Violence, 16*(10), 1008-1027.

Eisenberger, N. I., Kemeny, M. E., & Wyatt, G. E. (2003). Psychological inhibition and CD4 T-cell levels in HIV-seropositive women. *Journal of Psychosomatic Research, 54*(3), 213-224.

Esterling, B. A., Antoni, M. H., Kumar, M., & Schneiderman, N. (1990). Emotional repression, stress disclosure responses, and epstein-barr viral capsid antigen titers. *Psychosomatic Medicine, 52*(4), 397-410.

Evans, D. L., Leserman, J., Perkins, D. O., Stern, R. A., Murphy, C., Zheng, B., . . . Van der Horst, Charles M. (1997). Severe life stress as a predictor of early disease progression in HIV infection. *American Journal of Psychiatry, 154*(5), 630-634.

Fahey, J. L., Prince, H., Weaver, M., Groopman, J., Visscher, B., Schwartz, K., & Detels, R. (1984). Quantitative changes in T helper or T suppressor/cytotoxic lymphocyte subsets that distinguish acquired immune deficiency syndrome from other immune subset disorders. *The American Journal of Medicine, 76*(1), 95-100.

Fehniger, T. A., Herbein, G., Yu, H., Para, M. I., Bernstein, Z. P., O'Brien, W. A., & Caligiuri, M. A. (1998). Natural killer cells from HIV-1+ patients produce C-C chemokines and inhibit HIV-1 infection. *Journal of Immunology (Baltimore, Md.: 1950), 161*(11), 6433-6438.

Fesko, S. L. (2001). Disclosure of HIV status in the workplace: Considerations and strategies. *Health & Social Work, 26*(4), 235-244.

Fowles, D. C. (1980). The three arousal model: Implications of gray's two-factor learning theory for heart rate, electrodermal activity, and psychopathy. *Psychophysiology, 17*(2), 87-104.

García, L. I., Lechuga, J., & Zea, M. C. (2012). Testing comprehensive models of disclosure of sexual orientation in HIV-positive latino men who have sex with men (MSM). *AIDS Care, 24*(9), 1087-1091.

Gielen, A. C., Fogarty, L., O'Campo, P., Anderson, J., Keller, M. J., & Faden, R. (2000). Women living with HIV: Disclosure, violence, and social support. *Journal of Urban Health, 77*(3), 480-491.

Greeson, J. M., Hurwitz, B. E., Llabre, M. M., Schneiderman, N., Penedo, F. J., & Klimas, N. G. (2008). Psychological distress, killer lymphocytes and disease severity in HIV/AIDS. *Brain, Behavior, and Immunity, 22*(6), 901-911.

Hays, R. B., McKusick, L., Pollack, L., Hilliard, R., Hoff, C., & Coates, T. J. (1993). Disclosing HIV seropositivity to significant others. *Aids, 7*(3), 425-432.

Holmes, W. C., & Shea, J. A. (1998). A new HIV/AIDS-targeted quality of life (HAT-QoL) instrument: Development, reliability, and validity. *Medical Care, 36*(2), 138-154.

Ickovics, J. R., Hamburger, M. E., Vlahov, D., Schoenbaum, E. E., Schuman, P., Boland, R. J., & Moore, J. (2001). Mortality, CD4 cell count decline, and depressive symptoms among HIV-seropositive women. *JAMA: The Journal of the American Medical Association, 285*(11), 1466-1474.

Ironson, G., O'Cleirigh, C., Leserman, J., Stuetzle, R., Fordiani, J., Fletcher, M., & Schneiderman, N. (2013). Gender-specific effects of an augmented written emotional disclosure intervention on posttraumatic, depressive, and HIV-disease-related outcomes: A randomized, controlled trial. *Journal of Consulting and Clinical Psychology, 81*(2), 284.

Ironson, G., Stuetzle, R., & Fletcher, M. A. (2006). An increase in religiousness/spirituality occurs after HIV diagnosis and predicts slower disease progression over 4 years in people with HIV. *Journal of General Internal Medicine, 21*(S5), S62-S68.

Ironson, G., O'Cleirigh, C., Fletcher, M. A., Laurenceau, J. P., Balbin, E., Klimas, N., . . . Solomon, G. (2005). Psychosocial factors predict CD4 and viral load change in men and women with human immunodeficiency virus in the era of highly active antiretroviral treatment. *Psychosomatic Medicine, 67*(6), 1013-1021. doi:10.1097/01.psy.0000188569.58998.c8

Juster, R. P., Smith, N. G., Ouellet, E., Sindi, S., & Lupien, S. J. (2013). Sexual orientation and disclosure in relation to psychiatric symptoms, diurnal cortisol, and allostatic load. *Psychosomatic Medicine, 75*(2), 103-116. doi:10.1097/PSY.0b013e3182826881 [doi]

Kalichman, S. C., DiMarco, M., Austin, J., Luke, W., & DiFonzo, K. (2003). Stress, social support, and HIV-status disclosure to family and friends among HIV-positive men and women. *Journal of Behavioral Medicine, 26*(4), 315-332.

Kennamer, J. D., Honnold, J., Bradford, J., & Hendricks, M. (2000). Differences in disclosure of sexuality among african american and white gay/bisexual men: Implications for HIV/AIDS prevention. *AIDS Education and Prevention,*

Leserman, J., Petitto, J., Gu, H., Gaynes, B., Barroso, J., Golden, R., . . . Evans, D. (2002). Progression to AIDS, a clinical AIDS condition and mortality: Psychosocial and physiological predictors. *Psychological Medicine, 32*(06), 1059-1073.

Leserman, J. (2008). Role of depression, stress, and trauma in HIV disease progression. *Psychosomatic Medicine, 70*(5), 539-545.

Mitchell, P. H., Powell, L., Blumenthal, J., Norten, J., Ironson, G., Pitula, C. R., . . . Huber, M. (2003). A short social support measure for patients recovering from myocardial infarction: The ENRICHD social support inventory. *Journal of Cardiopulmonary Rehabilitation and Prevention, 23*(6), 398-403.

Obermeyer, C. M., Baijal, P., & Pegurri, E. (2011). Facilitating HIV disclosure across diverse settings: A review. *American Journal of Public Health, 101*(6), 1011-1023.

Overstreet, N. M., Earnshaw, V. A., Kalichman, S. C., & Quinn, D. M. (2013). Internalized stigma and HIV status disclosure among HIV-positive black men who have sex with men. *AIDS Care, 25*(4), 466-471.

Pennebaker, J. W. (1985). Traumatic experience and psychosomatic disease: Exploring the roles of behavioural inhibition, obsession, and confiding. *Canadian Psychology/Psychologie Canadienne, 26*(2), 82.

Pennebaker, J. W., & Beall, S. K. (1986). Confronting a traumatic event: Toward an understanding of inhibition and disease. *Journal of Abnormal Psychology, 95*(3), 274.

Pennebaker, J. W., Hughes, C. F., & O'Heeron, R. C. (1987). The psychophysiology of confession: Linking inhibitory and psychosomatic processes. *Journal of Personality and Social Psychology, 52*(4), 781.

Pennebaker, J. W., Kiecolt-Glaser, J. K., & Glaser, R. (1988). Disclosure of traumas and immune function: Health implications for psychotherapy. *Journal of Consulting and Clinical Psychology, 56*(2), 239.

Pennebaker, J. W., Barger, S. D., & Tiebout, J. (1989). Disclosure of traumas and health among holocaust survivors. *Psychosomatic Medicine, 51*(5), 577-589.

Petrak, J. A., Doyle, A., Smith, A., Skinner, C., & Hedge, B. (2001). Factors associated with self-disclosure of HIV serostatus to significant others. *British Journal of Health Psychology, 6*(1), 69-79.

Petrie, K. J., Fontanilla, I., Thomas, M. G., Booth, R. J., & Pennebaker, J. W. (2004). Effect of written emotional expression on immune function in patients with human immunodeficiency virus infection: A randomized trial. *Psychosomatic Medicine, 66*(2), 272-275.

Preacher, K. J., Zyphur, M. J., & Zhang, Z. (2010). A general multilevel SEM framework for assessing multilevel mediation. *Psychological Methods, 15*(3), 209.

Prejean, J., Song, R., Hernandez, A., Ziebell, R., Green, T., Walker, F., . . . Lansky, A. (2011). Estimated HIV incidence in the united states, 2006–2009. *PloS One, 6*(8), e17502.

Ragins, B. R., Singh, R., & Cornwell, J. M. (2007). Making the invisible visible: Fear and disclosure of sexual orientation at work. *Journal of Applied Psychology, 92*(4), 1103.

Raudenbush, S. W., & Bryk, A. S. (2002). *Hierarchical linear models: Applications and data analysis methods* Sage.

Rosario, M., Schrimshaw, E. W., & Hunter, J. (2004). Ethnic/racial differences in the coming-out process of lesbian, gay, and bisexual youths: A comparison of sexual identity development over time. *Cultural Diversity and Ethnic Minority Psychology, 10*(3), 215.

Sandelowski, M., Lambe, C., & Barroso, J. (2004). Stigma in HIV-Positive women. *Journal of Nursing Scholarship, 36*(2), 122-128.

Segerstrom, S. C., & Miller, G. E. (2004). Psychological stress and the human immune system: A meta-analytic study of 30 years of inquiry. *Psychological Bulletin, 130*(4), 601.

Serovich, J. M. (2001). A test of two HIV disclosure theories. *AIDS Education and Prevention : Official Publication of the International Society for AIDS Education, 13*(4), 355-364.

Serovich, J. M., & Mosack, K. E. (2003). Reasons for HIV disclosure or nondisclosure to casual sexual partners. *AIDS Education and Prevention : Official Publication of the International Society for AIDS Education, 15*(1), 70-80.

Siegel, K., & Epstein, J. A. (1996). Ethnic-racial differences in psychological stress related to gay lifestyle among HIV-positive men. *Psychological Reports, 79*(1), 303-312.

Simoni, J., Mason, H., & Marks, G. (1997). Disclosing HIV status and sexual orientation to employers. *AIDS Care, 9*(5), 589-599.

Smith, R., Rossetto, K., & Peterson, B. L. (2008). A meta-analysis of disclosure of one's HIV-positive status, stigma and social support. *AIDS Care, 20*(10), 1266-1275.

Strachan, E. D., Bennett, W. M., Russo, J., & Roy-Byrne, P. P. (2007). Disclosure of HIV status and sexual orientation independently predicts increased absolute CD4 cell counts over time for psychiatric patients. *Psychosomatic Medicine, 69*(1), 74-80.

Sullivan, K. M. (2005). Male self-disclosure of HIV-positive serostatus to sex partners: A review of the literature. *Journal of the Association of Nurses in AIDS Care, 16*(6), 33-47.

Swendeman, D., Rotheram-Borus, M. J., Comulada, S., Weiss, R., & Ramos, M. E. (2006). Predictors of HIV-related stigma among young people living with HIV. *Health Psychology, 25*(4), 501.

Ullrich, P. M., Lutgendorf, S. K., & Stapleton, J. T. (2003). Concealment of homosexual identity, social support and CD4 cell count among HIV-seropositive gay men. *Journal of Psychosomatic Research, 54*(3), 205-212.

Walker, C. M., Moody, D. J., Stites, D. P., & Levy, J. A. (1986). CD8+ lymphocytes can control HIV infection in vitro by suppressing virus replication. *Science (New York, N.Y.), 234*(4783), 1563-1566.

Yoshioka, M. R., & Schustack, A. (2001). Disclosure of HIV status: Cultural issues of asian patients. *AIDS Patient Care and STDs, 15*(2), 77-82.

Zea, M. C., Reisen, C. A., Poppen, P. J., Bianchi, F. T., & Echeverry, J. J. (2005). Disclosure of HIV status and psychological well-being among latino gay and bisexual men. *AIDS and Behavior, 9*(1), 15-26.

Zea, M. C., Reisen, C. A., Poppen, P. J., & Díaz, R. M. (2003). Asking and telling: Communication about HIV status among latino HIV-positive gay men. *AIDS and Behavior, 7*(2), 143-152.

TABLE 1: Demographic information for the full sample ($n$ = 177) and the reduced sample ($n$ = 143) due to missing data on disclosure variables.

| **Demographic Information** | | **Medical Information** | |
|---|---|---|---|
| Gender | | Baseline CD4 cell count | |
| Male | 70.1% / **69.9%** | M | 277.48 / **306.02** |
| Female | 29.9% / **30.1%** | SD | 190.79 / **99.16** |
| Age | | Viral Load | |
| M | 37.49 / **37.93** | M | 44,861.42 / **35,1881** |
| SD | 8.88 / **8.705** | SD | 120,118.81/ **110,532** |
| Ethnicity | | Antiretroviral Medication | |
| Caucasian | 30.5% / **30.1%** | None | 23.2 / **20.3** |
| African-American | 36.2% / **33.6%** | Combination Therapy | 20.3 / **31.5** |
| Hispanic | 28.2% / **30.8%** | HAART | 56.5 / **47.6** |
| Other | 5.1% / **5.6%** | | |
| Education | | | |
| Some HS or less | 18.1% / **17.5%** | | |
| HS graduate | 13.7% / **14.0%** | | |
| Some College | 40.7% / **36.4%** | | |
| College grad or above | 18.7% / **32.2%** | | |
| Sexual Orientation | | | |
| Homosexual/Bisexual | 54.8% / **55.9%** | | |

* Statistics for the reduced sample are shown in bold typeface.

TABLE 2: Percentage within each gender who have disclosed either fully or in general terms to various targets and chi-square significance tests for gender differences

| Target | Male (%) | Female (%) | $\chi^2$ | $p$ |
|---|---|---|---|---|
| Mom | 63.6 | 81.0 | 4.902 | .086 |
| Dad | 44.3 | 41.0 | 1.108 | .601 |
| Brother | 45.7 | 35.9 | 2.280 | .320 |
| Sister | 50.0 | 54.1 | 0.883 | .829 |
| Coworkers | 32.6 | 27.8 | 2.351 | .309 |
| Employer/Boss | 34.4 | 21.6 | 4.167 | .124 |
| Best male heterosexual friend | 58.5 | 57.9 | 0.185 | .912 |
| Best female heterosexual friend | 64.9 | 60.5 | 4.663 | .097 |
| Best male homosexual friend | 77.9 | 17.1 | 41.137 | <.001** |
| Best female homosexual friend | 52.3 | 27.0 | 8.328 | .016* |
| Spouse | 61.1 | 57.1 | 6.226 | .044* |

*$p < .05$, **$p < .001$

TABLE 3: Percentages of gay men who have disclosed in full or in general terms to various targets

| Target | Disclosure rate (%) |
|---|---|
| Mom | 43.5 |
| Dad | 28.8 |
| Brother | 31.6 |
| Sister | 29.9 |
| Coworkers | 26.6 |
| Employer/Boss | 20.3 |
| Best male heterosexual friend | 39.0 |
| Best female heterosexual friend | 45.8 |

TABLE 4: Descriptive statistics for disclosure measures and psychosocial measures.

| Variable | Mean | SD |
|---|---|---|
| Average HIV Disclosure | 0.87 | 0.94 |
| Total HIV Disclosure | 7.66 | 8.80 |
| HAT-QoL Total | 8.82 | 4.57 |
| Average S.O. Disclosure | 0.75 | 0.93 |
| Total S.O. Disclosure | 5.21 | 6.84 |
| Cumulative Depression | 10.05 | 7.08 |
| Cumulative Social Support | 8.81 | 5.31 |

TABLE 5: Basic HLM Model with coefficients and significance tests for level 1 and 2 covariates predicting CD4 over 4 years.

| | Coefficient | Std. Error | T-value | *df* | *p* |
|---|---|---|---|---|---|
| Fixed Effects | | | | | |
| CD4 intercept $\beta_0$ | | | | | |
| Average intitial CD4, $\gamma_{00}$ | -3.14 | 2.02 | -1.56 | 169 | .560 |
| CD4 slope ( per month), $\beta_1$ | | | | | |
| Average slope, $\gamma_{10}$ | -3.138 | 2.018 | -1.56 | 169 | .122 |
| Ethnicity, $\gamma_{11}$ | -0.229 | 0.393 | -0.584 | 169 | .560 |
| Education, $\gamma_{12}$ | 0.675 | 0.343 | 1.966 | 169 | .051 |
| Age, $\gamma_{13}$ | 0.021 | 0.041 | 0.529 | 169 | .597 |
| Gender, $\gamma_{14}$ | 0.447 | 0.876 | 0.511 | 169 | .610 |
| Baseline CD4, $\gamma_{15}$ | 0.010 | 0.003 | 2.937 | 169 | .004** |
| Antiretroviral 1 effect, $\beta_2$ | | | | | |
| Average effect, $\gamma_{20}$ | 63.206 | 19.015 | 3.324 | 995 | .001** |
| Antiretroviral 2 effect, $\beta_3$ | | | | | |
| Average effect, $\gamma_{30}$ | 33.593 | 15.236 | 2.205 | 995 | .028* |
| Antiretroviral1 x months since baseline, $\beta_4$ | | | | | |
| Average effect over time, $\gamma_{40}$ | 0.112 | 0.959 | 0.117 | 995 | .908 |
| Antiretroviral2 x months since baseline, $\beta_5$ | | | | | |
| Average effect over time, $\gamma_{50}$ | 1.349 | 0.680 | 1.984 | 995 | .047* |
| | | | | | |
| Random Effects | SD | Variance | *df* | $\chi^2$ | *p* |
| Intercept, $U_0$ | 97.590 | 9523.820 | 173 | 634.628 | <.001*** |
| Slope, $U_1$ | 3.863 | 14.926 | 168 | 543.973 | <.001*** |
| Error, *R* | 82.115 | 6742.854 | | | |

P < .05, ** p <.01, p < .001

TABLE 6: Basic HLM Model with Coefficients and Significant Tests for level 1 and 2 covariates predicting viral load over 4 years.

| | Coefficient | Std. Error | T-value | *df* | *p* |
|---|---|---|---|---|---|
| Fixed Effects | | | | | |
| CD4 intercept $\beta_0$ | | | | | |
| Average intitial VL, $\gamma_{00}$ | 4.428 | 0.111 | 39.877 | 174 | <.001 |
| CD4 slope ( per month), $\beta_1$ | | | | | |
| Average slope, $\gamma_{10}$ | 0.004 | 0.011 | 0.372 | 169 | .710 |
| Ethnicity, $\gamma_{11}$ | 0.003 | 0.002 | 1.813 | 169 | .071 |
| Education, $\gamma_{12}$ | -0.002 | 0.002 | -1.366 | 169 | .174 |
| Age, $\gamma_{13}$ | -0.00003 | 0.0002 | -0.113 | 169 | .910 |
| Gender, $\gamma_{14}$ | 0.00009 | 0.005 | 0.020 | 169 | .984 |
| Baseline VL, $\gamma_{15}$ | -0.00005 | 0.002 | -0.027 | 169 | .979 |
| Antiretroviral 1 effect, $\beta_2$ | | | | | |
| Average effect, $\gamma_{20}$ | -1.065 | 0.145 | -7.342 | 982 | <.001** |
| Antiretroviral 2 effect, $\beta_3$ | | | | | |
| Average effect, $\gamma_{30}$ | -1.097 | 0.142 | -7.715 | 982 | <.001** |
| Antiretroviral1 x months since baseline, $\beta_4$ | | | | | |
| Average effect over time, $\gamma_{40}$ | 0.005 | 0.006 | 0.722 | 982 | .470 |
| Antiretroviral2 x months since baseline, $\beta_5$ | | | | | |
| Average effect over time, $\gamma_{50}$ | -0.002 | 0.006 | -0.327 | 982 | .743 |
| | | | | | |
| Random Effects | SD | Variance | *df* | $\chi^2$ | *p* |
| Intercept, $U_0$ | 0.903 | 0.815 | 173 | 852.894 | <.001*** |
| Slope, $U_1$ | 0.016 | 0.0002 | 168 | 284.347 | <.001*** |
| Error, *R* | 0.638 | 0.406 | | | |

P < .05, ** p <.01, p < .001

TABLE 7: Gamma coefficients from HLM Model Examining Effect of Disclosure variables on both Change in CD4 cell count and change in viral load (log) over a period of 4 years.

| Predictor | Change in CD4 Cell Count | | | Change in Viral Load (Log) | | |
|---|---|---|---|---|---|---|
| | *γ* coefficient | *t* ratio | *p* | *γ* coefficient | *t* ratio | *p* |
| HIV Disclosure | | | | | | |
| Average | -0.298 | -0.444 | .503 | 0.002 | 1.007 | .316 |
| Total | -0.023 | -0.467 | .641 | 0.0001 | 0.513 | .609 |
| Sexual Orientation Disclosure | | | | | | |
| Average | -0.457 | -0.754 | .453 | 0.001 | 0.370 | .712 |
| Total | -0.082 | -1.023 | .309 | 0.0002 | 0.519 | .605 |
| Combination | -0.062 | -0.197 | .845 | .0003 | 0.255 | .799 |
| HAT-QoL Total | 0.199 | 2.518 | .014* | -0.0004 | -0.772 | .442 |

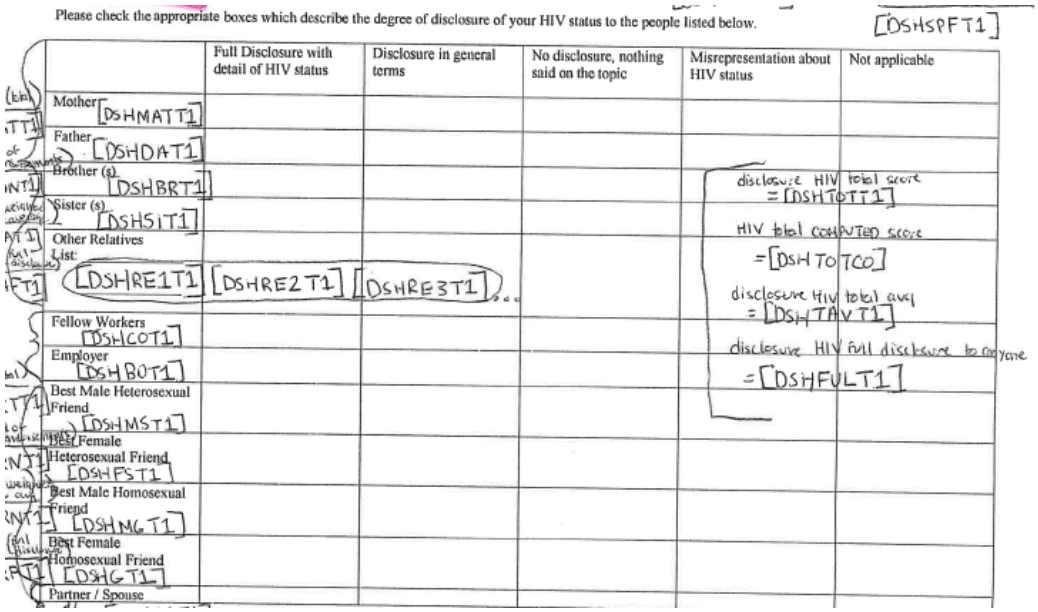
*$p < .05$

## Appendix A: HAT QOL Measure

3. The following questions ask about your disclosure worries in the past 4 weeks.

| | All of the time | A lot of the time | Some of the time | A little of the time | None of the time |
|---|---|---|---|---|---|
| a. In the past 4 weeks, I have limited what I tell others about myself | ☐ | ☐ | ☐ | ☐ | ☐ |
| b. In the past 4 weeks, I have been afraid to tell other people that I have HIV | ☐ | ☐ | ☐ | ☐ | ☐ |
| c. In the past 4 weeks, I have been worried about my family members finding out that I have HIV | ☐ | ☐ | ☐ | ☐ | ☐ |
| d. In the past 4 weeks, I have been worried about people at my job/routine daily activities finding out that I have HIV | ☐ | ☐ | ☐ | ☐ | ☐ |
| e. In the past 4 weeks, I have been worried that I will lose my source of income if other people find out that I have HIV | ☐ | ☐ | ☐ | ☐ | ☐ |

## Appendix B: HIV Disclosure Measure



Please check the appropriate boxes which describe the degree of disclosure of your HIV status to the people listed below. [DSHSPFT1]

| | Full Disclosure with detail of HIV status | Disclosure in general terms | No disclosure, nothing said on the topic | Misrepresentation about HIV status | Not applicable |
|---|---|---|---|---|---|
| Mother [DSHMATT1] | | | | | |
| Father [DSHDAT1] | | | | | |
| Brother (s) [DSHBRT1] | | | | | |
| Sister (s) [DSHSIT1] | | | | | |
| Other Relatives List: [DSHRE1T1] [DSHRE2T1] [DSHRE3T1] ... | | | | | |
| Fellow Workers [DSHCOT1] | | | | | |
| Employer [DSHBOT1] | | | | | |
| Best Male Heterosexual Friend [DSHMST1] | | | | | |
| Best Female Heterosexual Friend [DSHFST1] | | | | | |
| Best Male Homosexual Friend [DSHMGT1] | | | | | |
| Best Female Homosexual Friend [DSHGT1] | | | | | |
| Partner / Spouse | | | | | |

disclosure HIV total score = [DSHTOTT1]

HIV total computed score = [DSHTOTCO]

disclosure HIV total avg = [DSHTAVT1]

disclosure HIV full disclosure to anyone = [DSHFULT1]

## Appendix C: Sexual Orientation Disclosure Measure

**SEXUAL ORIENTATION DISCLOSURE**

Please check the appropriate boxes which describe the degree of disclosure of your sexual life to the people listed below.

| | Full Disclosure With Detail of Sexual Life | Disclosure in General Terms | No Disclosure, Nothing Said on Topic | Misrepresentation About Orientation | Not Applicable |
|---|---|---|---|---|---|
| Mother [dsgmat] | [DSGFIIT] | = disclosure gay | Immediate fam | Score T1 | |
| Father [dsgdat1] | [DSGFINT1] | = " | " | number of endorsements | |
| Brother(s) [dsgbrT] | [DSGFIAT1] | = " | " " | weighted average T1 | |
| [DSGSIT1] Sister(s) | [DSGFIFT1] = | " | " " | full disclosure at T1 | |
| [DSGRET1] Other Relatives | [DSGFATT] = discl. gay family total score T1 [DSGFAAT1] = Weighted average T1 | | [DSGFAFT1] = | ANT] = # of endorsements T1 full disclosure at T1 | |
| [DSGCOT1] Fellow Workers | [DSGWKTT1] = discl. gay at work total score T1 | | | [DSGWKNT1] = # of endorsements at T1 | |
| [DSGBOT1] Employer | [DSGWKAT1] = weighted average T1 | | [DSGWKFT1] = | full disclosure at T1 | |
| [DSGMST1] Best Male Heterosexual Friend | [DSGFRTT1] = disclosure to gay friends total score T1 [DSGFRAT1] = weighted average T1 | | [DSGFRFT1] = | full disclosure at T1 | [DSGFRNT1] = # of endorsements T1 |
| [DSGFST1] Best Female Heterosexual Friend | | | | | |