Child Abuse as a Determinant of Barriers to HIV Status Disclosure Among Women in Canada

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Graduate Program in Epidemiology and Biostatistics
A thesis submitted in partial fulfillment of the requirements for the degree in Master of Science
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Abstract

Introduction: In Canada, people with HIV are legally required to disclose their HIV status to their sexual partners prior to having sex that presents a “realistic possibility of HIV transmission”. This policy has been criticized for failing to consider the safety and autonomy of women with HIV, which can be compromised following disclosure.

Objectives: To investigate whether experiences of childhood physical or sexual abuse (CPSA) affect barriers to HIV disclosure to partners in adulthood, which include: later physical or sexual abuse, sexual agency, HIV stigma, and perceived social support.

Methods: Propensity scores and inverse probability of treatment weights were used to estimate the difference in barriers to HIV disclosure attributable to CPSA among n=1307 women with HIV. Effects were reported for the total sample, and within ethnoracial groups (Indigenous, Black African, Black Caribbean, white, and “other”).

Results: CPSA increased prevalence of both physical and sexual abuse in adulthood in the total sample and within ethnoracial groups, while effects for other barriers were subgroup specific.

Conclusion: This study illustrates that the environment in which women are legally expected to disclose their HIV status is often characterized by abuse, with high risk of revictimization from childhood to adulthood. While further research should investigate mediating pathways between CPSA and disclosure barriers across ethnoracial groups, this study provides the first targeted evidence suggesting that the causes of HIV disclosure may be more distal and more complex than presumed under Canadian non-disclosure policy.

Keywords

Women with HIV; child abuse; violence; HIV disclosure; intersectionality; propensity scores; generalized boosted modelling
Co-Authorship Statement

All chapters of this thesis were written by Siobhan Churchill as partial fulfillment of requirements for her Master of Science degree from the Department of Epidemiology and Biostatistics. Ms. Churchill incorporated feedback on this thesis from her supervisory committee which included Dr. Greta Bauer and Dr. Igor Karp. Secondary data for the present analysis came from the Canadian HIV Women's Sexual & Reproductive Health Cohort Study (CHIWOS), to which Ms. Churchill was granted access by the CHIWOS National Management Team.

The research question was initially identified through conversations between Ms. Churchill and Dr. Greta Bauer, and refined through consultation with researchers (Dr. Mona Loutfy, Dr. Angela Kaida) and community partners (Shazia Islam, and Mary Ndung’u) from CHIWOS. All background research, including the adaptation of a novel theoretical model, was conducted by Ms. Churchill with input from Dr. Greta Bauer, Shazia Islam, and Mary Ndung’u.

The statistical methods for the present analysis were initially planned by Ms. Churchill and Dr. Greta Bauer, with further input from the Dr. Igor Karp. All data cleaning and coding was conducted by Ms. Churchill, with some aspects in consultation with Dr. Greta Bauer, Dr. Igor Karp, Dr. Guangyong Zou, and Dr. Dan Lizotte. Results were interpreted by Ms. Churchill in consultation with Dr. Greta Bauer and community partner Shazia Islam.
Dedication

“It is a terrible irony that we have come to a place where the medications we fought for will allow us to live a relatively normal quality of life, and now we are going to jail for doing so.”

- Louise Binder, Canadian human rights activist and woman living with HIV

This thesis is dedicated to the CHIWOS peers and participants who made this research possible, including the 69 participants who have since passed away.
Acknowledgments

I would first like to thank my supervisor, Dr. Greta Bauer for her steadfast support of myself and my work for these past two years. Dr. Bauer has gone out of her way to give me learning opportunities and skills that I will continue to build upon for the rest of my career. However, her most valuable gift to me has been her trust and confidence in my work, which has inspired me to have confidence in myself.

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This thesis would not have been possible without the support of the CHIWOS team, including Shazia Islam and Mary Ndung’u, whose vision and community knowledge have grounded this project from the very beginning. Thank you to Dr. Mona Loutfy, Dr. Angela Kaida, Mina Kazemi, and Dr. Ashley Lacombe-Duncan for your valuable comments and support. Furthermore, I would like to thank the CHIWOS participants and the National Management Team for allowing me to access this data and tell this story.

Finally, I would like say thank you to my family. To my mom, who showed me that women can do anything, and my dad who taught me how to achieve balance in life. To my grandparents who have encouraged me every step of the way, and to my sisters who motivate me to close my laptop and enjoy my time at home. Lastly, thank you to Anna, whose love and support is always there to greet me at the end of a long day.
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<table>
<thead>
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<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>AEE</td>
<td>Average effect of the exposure among the exposed</td>
</tr>
<tr>
<td>AIDS</td>
<td>Acquired immune deficiency syndrome</td>
</tr>
<tr>
<td>ART</td>
<td>Antiretroviral therapy</td>
</tr>
<tr>
<td>ATE</td>
<td>Average treatment effect</td>
</tr>
<tr>
<td>ATT</td>
<td>Average treatment effect among the treated</td>
</tr>
<tr>
<td>BC</td>
<td>British Columbia</td>
</tr>
<tr>
<td>CBR</td>
<td>Community based research</td>
</tr>
<tr>
<td>CHIWOS</td>
<td>Canadian HIV Women’s Sexual and Reproductive Health Cohort Study</td>
</tr>
<tr>
<td>CIC/OOC</td>
<td>Citizenship and Immigration Canada/Out of Country</td>
</tr>
<tr>
<td>CIHR</td>
<td>Canadian Institutes of Health Research</td>
</tr>
<tr>
<td>CPA</td>
<td>Childhood physical abuse</td>
</tr>
<tr>
<td>CPSA</td>
<td>Childhood physical or sexual abuse</td>
</tr>
<tr>
<td>CSA</td>
<td>Childhood sexual abuse</td>
</tr>
<tr>
<td>GBM</td>
<td>Generalized boosted modelling</td>
</tr>
<tr>
<td>GIPA</td>
<td>Greater involvement of people with HIV/AIDS</td>
</tr>
<tr>
<td>HALCO</td>
<td>HIV &amp; AIDS Legal Clinic Ontario</td>
</tr>
<tr>
<td>HIV</td>
<td>Human immunodeficiency virus</td>
</tr>
<tr>
<td>IDU</td>
<td>Injection drug use</td>
</tr>
<tr>
<td>IPTW</td>
<td>Inverse probability of treatment weighting</td>
</tr>
</tbody>
</table>
KS – Kolmogorov-Smirnov

MIPA – Meaningful involvement of people with HIV/AIDS

MSM – Men who have sex with men

NIR – No identified risk

ON – Ontario

PR – Prevalence ratio

PRA – Peer research associate

PrEP – Pre-exposure prophylaxis

PS – Propensity score

QC – Quebec

SRP – Sexual relationship power

SRPS – Sexual relationship power scale

TWANG – Toolkit for weighting and analysis of non-equivalent groups
Chapter 1

1 Introduction and Objectives

This chapter will provide background information to contextualize the study rationale, discuss the data source, identify study objectives, and specify my role in the project.

1.1 Study Rationale

The Canadian legal system (and in some provinces, the Public Health system) enforces regulations requiring the sexual and/or needle-sharing partners of people living with HIV to be notified that they may be at risk for HIV. The legal precedent set by the Supreme Court of Canada in 2012 requires people living with HIV to disclose their HIV status to their sexual partners prior to having sex that presents a “realistic possibility of HIV transmission”, with potential legal consequences including criminal charges of aggravated sexual assault. Canada’s position on HIV criminalization has been criticized for failing to account for scientific advances in HIV treatment, and for diminishing the safety and autonomy of people with HIV. This precedent-setting case law purports to enhance public safety, but does not acknowledge what all people with HIV know: disclosure of one’s HIV status can be dangerous, sometimes leading to discrimination, loss of resources, and life-threatening violence. For women with HIV, disclosure is often inextricably tied to gendered power imbalances which intersect with other social positions including race, class, and Indigeneity. However, the criminal justice system frames HIV disclosure as a fairly simplistic choice, with limited understanding of how violence against women and girls influences decision making. Thus, the overarching goal of this thesis is to complicate how the system understands “choice” as related to HIV non-disclosure.

Among women in the general population, childhood physical or sexual abuse (CPSA) is a salient risk factor for adverse outcomes in adulthood. Women who experience CPSA are at greater risk for further violence in the future (“revictimization”) and may be more vulnerable to power-imbalanced sexual relationships than women who did not experience CPSA. Importantly, CPSA is also known to increase HIV risk.
pathways between violent victimization and HIV infection have been studied widely, less attention has been given to how experiences of violence may produce further vulnerability among women with HIV. Of particular interest is whether experiences of childhood violence have an impact on factors which enable or disenable women to disclose their HIV status to sexual partners in adulthood. These include experiencing physical or sexual abuse in adulthood, having low sexual relationship power, experiencing high HIV stigma, and perceiving oneself to have low social support. If CPSA has a causal effect on these outcomes (as has sometimes been found among the general population), the implication is that adverse childhood experiences not only increase risk for HIV, but for further vulnerability among women with HIV. In the Canadian context of HIV criminalization, this vulnerability to non-disclosure translates into socio-legal risk. Thus, the goal of this research is to assess whether women with HIV who experienced abuse during childhood experience barriers to partner disclosure differently from the larger population of women with HIV. This question will be explored using intersectionality theory in an eco-social framework in order to account for aspects of CPSA which may be unique to women with HIV at particular intersections of gender, HIV, and ethnoracial identity.

This project is positioned to advocate for women living with HIV by highlighting potential inequities that are not addressed by Canadian HIV criminalization. Additionally, this research has the potential to reach women with HIV who have experienced child abuse and assure them that their experiences with HIV disclosure are valid, shared, and not their own fault.

1.2 Thesis Objectives and Hypotheses

1. **Objective:** To adapt a theoretical model which incorporates an eco-social intersectionality perspective with the goal of appropriately contextualizing the causes and consequences of CPSA among women with HIV in Canada. This objective will be addressed as part of the literature review.
2. **Objective:** To assess whether women with HIV who experienced CPSA experience greater barriers to disclosing their HIV status to partners [(a) current experiences of physical or (b) sexual violence, (c) low sexual relationship power, and (d) high levels of HIV stigma, and (e) reduced perceived social support] compared to women with HIV who have not experienced child abuse.

**Hypothesis:** It is expected that among women with HIV, as for other women not in the study, exposure to CPSA will impact later experiences of (a) physical or (b) sexual violence, (c) sexual relationship power, and (d) perceived social support, but with additional HIV-specific consequences such as (e) heightened feelings of HIV stigma. On average, it is expected that women with HIV who have experienced child abuse will face greater barriers to partner disclosure during adulthood.

3. **Objective:** To evaluate the aforementioned causal relationships across strata of ethnoracial groups, specifically: Indigenous, Black African, Black Caribbean, and white women, as well as a group for other ethnoracial groups limited by sample size (e.g. Latin American, South Asian women).

**Hypothesis:** It is expected that the causal relationships between child abuse and barriers to HIV disclosure will be somewhat distinct across these groups. Lasting familial effects of institutionalized abuse within residential schools, as well as current experiences of colonialism will impact Indigenous women in causal pathways not experienced by non-Indigenous women. At a structural level, racialization may also be a determinant of access to treatment and services following child abuse and into adulthood. Furthermore, cultural perceptions and acceptance of child abuse can be highly heterogeneous, potentially affecting the nature and sequelae of child abuse.
1.3 Canadian HIV Women’s Sexual and Reproductive Health Cohort Study

The Canadian HIV Women’s Sexual and Reproductive Health Cohort Study (CHIWOS) is a national prospective study developed in response to a lack of research on women with HIV outside of studies on reproductive health. The overarching objective of this study is to identify the barriers and facilitators to accessing women-centred HIV care experienced by HIV positive women in Canada. Here, women-centred HIV care is briefly defined as “care that supports women living with HIV to achieve the best health and wellbeing as defined by them.” In order to establish a basis for women-centred HIV care in Canada, the CHIWOS team operates under several guiding frameworks/principles, including: community based research (CBR), Greater (and Meaningful) Involvement of People Living with HIV (GIPA, MIPA), as well as intersectionality, critical feminism, and anti-oppression. CBR is an increasingly common research method which prioritizes community needs and ideas by placing decision-making power into the hands of community members working alongside researchers. The involvement of the community under study not only maximizes the relevance of research results, but ensures that results are properly contextualized as well as communicated to all relevant stakeholders. In CHIWOS, CBR has been operationalized by involving women with HIV in identifying study objectives, as well as developing and testing the survey. Most notably, the CHIWOS team hired and trained a national team of 39 women with HIV as Peer Research Associates (PRA) responsible for data collection, among other research activities. CHIWOS is one among few studies which have successfully implemented CBR on a national scale, especially within quantitative research. The heavy involvement of women with HIV in study design through to knowledge translation and exchange also speaks to the principles of GIPA and MIPA. Finally, CHIWOS applies several feminist and social justice frameworks such as intersectionality and anti-oppression in order to create a study that acknowledges the diverse experiences of women with HIV while shedding light on social privilege/oppression, and striving for meaningful change.
In addition to peer research associates, the CHIWOS research team consists of a national, multi-disciplinary team of principal investigators, led by nominated principal investigator Mona Loutfy (MD, FRCPC, MPH) at the University of Toronto. Each province where CHIWOS collects data has a research coordinator, as well as a Community Advisory Board. Along with the Community Advisory Boards, the study is led by the National Steering Committee. Women with HIV are active decision makers at all levels of the CHIWOS team. The study is also supported by an extended team of co-investigators, collaborators, and students. As of June 2018, knowledge and data from CHIWOS has supported the publication of 25 papers along with multiple webinars and conference presentations.

1.3.1 My Role in the Project

Under its Project and Data Request Policy, CHIWOS allows researchers (including principal investigators, co-investigators, students, and collaborators) access to CHIWOS data under several conditions. Students, in particular, must be under the supervision of a CHIWOS-affiliated researcher, and anyone who accesses data must be added to the regional ethics board statement and submit a Data Request Form for acceptance by the National Management Team. My thesis supervisor, Dr. Greta Bauer, is a CHIWOS co-investigator who participated in survey development and provides ongoing support to the study team, especially concerning studies of transgender health. The idea for the current study was formulated by Dr. Bauer and myself, born from my interest in the effects of trauma from a life course perspective, a mutual interest in quantitative intersectionality, and Dr. Bauer’s knowledge of community perspectives of HIV non-disclosure policy in Canada.

Under Dr. Bauer’s supervision and with the support of two CHIWOS peer research associates (Shazia Islam, Mary Ndung’u), my thesis supervisory committee (Dr. Greta Bauer, Dr. Igor Karp), other CHIWOS students (Mostafa Shokoohi, Ashley Lacombe-Duncan), research objectives and methodology were developed and submitted to the National Management Team for review. The form involved identifying a manuscript preparation team, a plan for meaningful involvement of people with HIV, study rationale and objectives, and detailed statistical methods. While the form was being refined for
submission, a novel theoretical model to support the research question was adapted by myself along with Dr. Bauer, Shazia Islam, and Mary Ndung’u. The form was accepted with minor revisions in December 2017, with data cleaning and analysis conducted by myself (supported by Dr. Greta Bauer, Dr. Igor Karp, Dr. Guangyong Zou, and Dr. Dan Lizotte) over the next several months. Results were interpreted with the use of personal and community knowledge along with quantitative expertise by my CHIWOS study team (myself, Dr. Bauer, Shazia Islam and Mary Ndung’u). During the preparation of this thesis, a corresponding manuscript for submission to a scientific journal was developed. While the entirety of this thesis was written by myself, the journal manuscript will be co-authored by myself, Dr. Bauer, Shazia Islam, Mary Ndung’u, Angela Kaida, Alexandra de Pokomandy, and Mona Loutfy on behalf of the CHIWOS Research Team. Prior to publication, the journal manuscript (though not the body of this thesis) will be reviewed for acceptance by the CHIWOS National Management Team.
Chapter 2

2 Literature Review

This chapter introduces the medical and social history of HIV, and describes the population of women with HIV in Canada, contextualizing their experience using intersectionality theory. Canada’s current legal policy of criminalizing HIV non-disclosure to sexual partners is discussed, alongside scientific and ethical objections to this practice. Literature on selected barriers to partner disclosure among women with HIV is presented, with barriers chosen being those identified from previous research, and which are also available in the CHIWOS dataset. Child abuse is defined and its causes are discussed using eco-social theory and intersectionality. Finally, a novel theoretical model integrating the aforementioned theories is presented, with the objective of outlining the causes of child abuse, as well as how child abuse may cause barriers to HIV disclosure later in life.

2.1 HIV in Canada

Human immunodeficiency virus (HIV) is a viral infection affecting an estimated 63,400 to 87,600 Canadians as of 2014.19 Symptomatology of HIV was first documented in 1981 among patients with late stage HIV infection which would come to be known as acquired immune deficiency syndrome (AIDS).20 From the beginning, AIDS was a disease steeped in stigma and exceptionalism, stemming from prejudicial fear of the populations affected by the virus, and fear of the death sentence that it carried.21,22 In particular, AIDS was first documented among American gay men and intravenous drug users.21 The symptoms included skin lesions, enlargement of the lymph nodes, pneumonia, and apparent immunosuppression leading to opportunistic infection.20 By 1984, it was established that AIDS was likely a sexually transmitted disease also transmitted by exposure to blood products,23 the retrovirus implicated as the cause of AIDS was isolated,24 and the biological mechanisms driving the shift from HIV infection (“pre-AIDS”) to symptomatic AIDS were under investigation.25 It is now known that HIV can be transmitted by exchange of specific body fluids from an infected person via contact with the blood, a mucous membrane, or damaged tissue.26 Furthermore, untreated HIV
infection begins with a latent period of an estimated 4.2 to 15 years\(^\text{27}\) characterized by a progressive decline in CD4 lymphocytes\(^\text{28}\) and increase in plasma viral load.\(^\text{29}\) The latent period is followed by onset of symptoms (wasting, fatigue, pain, pyrexia, and coughing)\(^\text{30}\) often attributable to immunosuppression by way of CD4 lymphocyte deficiency.\(^\text{31}\) Without treatment, the current estimated survival time after a diagnosis with AIDS is two years, but this estimate can vary greatly based on age at diagnosis and comorbid conditions.\(^\text{32,33}\)

Elucidation of the HIV mechanism has led to the development of anti-retroviral therapy (ART).\(^\text{34,35}\) Where such treatment is accessible, HIV can be treated as a communicable chronic condition rather than a deadly infectious disease.\(^\text{36}\) ART, established as the standard of care for HIV in 1996,\(^\text{37}\) refers to a given combination of antiretroviral medications which can render plasma viral load undetectable, increase CD4 cell count, and increase lifespan to match that of those without HIV.\(^\text{34,35,38}\) Consequently, more people than ever are living with HIV.\(^\text{39}\) Since the uptake of ART in the 1990s, cases of AIDS in Canada have dropped 90% to 188 reported cases in 2014,\(^\text{19}\) an indicator of people with HIV living longer on treatment. Additionally, because ART can reduce plasma viral load, transmission rates among couples where one partner is HIV-positive have been estimated at 0 events per 100 couple-years, with some variability based on sexual behaviour.\(^\text{40}\) As of 2016, Health Canada has also approved the use of pre-exposure prophylaxis (PrEP), an antiretroviral medication aimed at reducing transmission risk by treating those at risk for HIV.\(^\text{41}\)

While medical advances since 1981 have succeeded in allowing people with HIV who have access to treatment to live long and healthy lives, the social stigma surrounding HIV infection remains pervasive in Canadian society. Since the onset of the epidemic, widespread homophobia and misinformation about HIV led many members of the public to hold prejudicial beliefs about HIV and the people affected by it, so much so that HIV has been theorized to occupy a dual status as both an infectious disease and social phenomenon.\(^\text{42}\) Today, public and institutionalized HIV stigma is less overt but remains an issue affecting people with HIV every day. As of 2012, 29% of Canadians still held medium or high levels of stigmatizing beliefs about those with HIV (e.g. fear, belief that
people with HIV are to blame for their HIV status) and 34% held medium or high levels discriminatory beliefs (e.g. the rights to employment, sexual activity, and privacy of people with HIV should be restricted). The discrimination experienced by those with HIV and the internalized stigma that results from it can have widespread and devastating effects including lack of access to healthcare as well as loss of income or housing. HIV stigma is also known to affect a person’s ability to disclose their HIV status to family, friends, and sexual partners. While many campaigns and programs have been implemented in Canada with the goal of reducing HIV related discrimination, it remains a pervasive determinant of health and wellness for people with HIV.

### 2.2 Women Living with HIV

An estimated 15,219 women in Canada are have been diagnosed with HIV since the onset of the epidemic, with adult women accounting for 23.3% of incident cases nationally in 2016. This proportion has remained stable since 2004. While the incidence of HIV is lower among women compared to men (3.0 per 100,000 and 9.8 per 100,000, respectively), women with HIV face unique challenges that make them a key population in the study of HIV. Until very recently women in Canada tended to be, on average, diagnosed with HIV and AIDS at younger ages than men, though it was unclear whether this difference was attributable to differences in age of infection or diagnosis itself. As of 2016 these age differences are negligible, with the majority of HIV diagnoses for both men and women (27.7% and 31.8%, respectively) occurring between ages 30 to 39. The majority of women with HIV in Canada, since the start of the epidemic, have been diagnosed in the most populous provinces: Ontario (ON), Quebec (QC), and British Columbia (BC) (Table 1). However, the 2016 HIV diagnosis rate per 100,000 population indicates that incidence of HIV among women is highest in Saskatchewan, Yukon, and Manitoba. CHIWOS data reports that 81.7% of women with HIV in Canada (ON, QC, and BC) live in a large city as opposed to a small or medium city, a factor which likely impacts access to HIV care. According to the same study, 3.7% of Canadian women with HIV have not engaged in HIV care in the past year while 2.8% have never accessed HIV care.
Table 1: All-ages Canadian HIV cases by sex and province/territory, 1985-2016

<table>
<thead>
<tr>
<th>Province/territory</th>
<th>Total number of reported cases, 1985-2016</th>
<th>Diagnosis rate per 100,000 population, 2016</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Females</td>
<td>Males</td>
</tr>
<tr>
<td>British Columbia</td>
<td>2,270</td>
<td>12,913</td>
</tr>
<tr>
<td>Yukon</td>
<td>19</td>
<td>44</td>
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<tr>
<td>Alberta</td>
<td>1,617</td>
<td>5,362</td>
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<tr>
<td>Northwest Territories</td>
<td>12</td>
<td>45</td>
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<tr>
<td>Nunavut</td>
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<td>4</td>
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<tr>
<td>Saskatchewan</td>
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<td>Ontario</td>
<td>5,690</td>
<td>29,590</td>
</tr>
<tr>
<td>Quebec</td>
<td>3,745</td>
<td>14,155</td>
</tr>
<tr>
<td>New Brunswick</td>
<td>70</td>
<td>374</td>
</tr>
<tr>
<td>Nova Scotia</td>
<td>114</td>
<td>729</td>
</tr>
<tr>
<td>Prince Edward Island</td>
<td>9</td>
<td>32</td>
</tr>
<tr>
<td>Newfoundland and Labrador</td>
<td>62</td>
<td>250</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td>15,219</td>
<td>66,568</td>
</tr>
</tbody>
</table>

Data source: Canadian Community Disease Reports, HIV in Canada – Supplementary tables, 2016.\(^{37}\)
Data on sex were submitted for 99.6% of reported HIV cases in 2016. DS: indicates cases where data has been suppressed per the request of the province or territory. Reporting of HIV cases for individuals younger than two years of age varies among provinces and territories. For Quebec, the number of HIV cases is based on the minimum number of HIV-positive individuals.

HIV exposure patterns are also distinct among women compared to men; while men who have sex with men (MSM) remains the predominant HIV exposure category among men, three times as many cases among women are attributable to heterosexual contact (63.5% versus 21.6%) and approximately twice the proportion of cases are attributable to injection drug use (IDU) (27.3% versus 10.9% IDU and 5.1% MSM/IDU) (Table 2).\(^{46}\)

Other exposure categories commonly applied in public health surveillance are perinatal transmission, receipt of blood or blood products, no identified risk (NIR), and other, which may include less common exposures such as receipt of semen from an HIV-positive donor.\(^{19}\) Furthermore, the heterosexual contact category is broken down into exposure from heterosexual contact with a person at risk (Het-risk, i.e. heterosexual contact with someone who has HIV, who injects drugs, is a bisexual male, or is a person from an HIV-endemic country), heterosexual contact with no identified risk factors (Het-
NIR), and origin from an HIV-endemic country (Het-endemic).\textsuperscript{19} HIV-endemic countries are those with an HIV prevalence greater than 1% and either (a) 50% or more HIV cases attributable to heterosexual transmission, (b) a male to female ratio of 2:1 or less, or (c) HIV prevalence greater than or equal to 2% among women receiving prenatal care.\textsuperscript{19}

While cases originating from HIV-endemic countries may not necessarily have acquired HIV in the HIV-endemic country or be attributable to heterosexual contact, they are presumed to be so based on the above conditions. In 2016, origin from an HIV-endemic country was the most common heterosexual exposure for incident HIV cases among women, followed by heterosexual contact with no identified risk and heterosexual contact with a person at risk (Table 2). Data on exposures from HIV-endemic countries may be more complete than domestic exposures as Citizenship and Immigration Canada has conducted mandatory HIV screening for permanent resident applicants over the age of 15 (or under the age of 15 with given risk factors) since 2002.\textsuperscript{19,49}

Table 2: Number and percentage distribution of Canadian HIV diagnoses among adults (\(\geq 15\) years old) by sex and exposure category, 2016

<table>
<thead>
<tr>
<th>Exposure category</th>
<th>Females</th>
<th></th>
<th>Males</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>%</td>
<td>n</td>
<td>%</td>
</tr>
<tr>
<td>MSM</td>
<td>-</td>
<td>-</td>
<td>633</td>
<td>59.0</td>
</tr>
<tr>
<td>MSM/IDU</td>
<td>-</td>
<td>-</td>
<td>55</td>
<td>5.1</td>
</tr>
<tr>
<td>IDU</td>
<td>99</td>
<td>27.3</td>
<td>117</td>
<td>10.9</td>
</tr>
<tr>
<td>Blood/blood products</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Recipient of blood/clotting factor</td>
<td>0</td>
<td>0.0</td>
<td>1</td>
<td>0.1</td>
</tr>
<tr>
<td>Recipient of blood</td>
<td>0</td>
<td>0.0</td>
<td>1</td>
<td>0.1</td>
</tr>
<tr>
<td>Recipient of clotting factor</td>
<td>0</td>
<td>0.0</td>
<td>0</td>
<td>0.0</td>
</tr>
<tr>
<td>Heterosexual contact</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Origin from HIV-endemic country</td>
<td>85</td>
<td>23.5</td>
<td>66</td>
<td>6.2</td>
</tr>
<tr>
<td>Sexual contact with person at risk</td>
<td>67</td>
<td>18.5</td>
<td>60</td>
<td>5.6</td>
</tr>
<tr>
<td>No identified risk, heterosexual</td>
<td>78</td>
<td>21.5</td>
<td>106</td>
<td>9.9</td>
</tr>
<tr>
<td>Other*</td>
<td>33</td>
<td>9.1</td>
<td>33</td>
<td>3.1</td>
</tr>
<tr>
<td><strong>Subtotal</strong></td>
<td>362</td>
<td>100.0</td>
<td>1,072</td>
<td>100.0</td>
</tr>
<tr>
<td>No identified risk</td>
<td>18</td>
<td>N/A</td>
<td>69</td>
<td>N/A</td>
</tr>
<tr>
<td>Not reported</td>
<td>160</td>
<td>N/A</td>
<td>640</td>
<td>N/A</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td>540</td>
<td>N/A</td>
<td>1,781</td>
<td>N/A</td>
</tr>
</tbody>
</table>
Data on exposure category were submitted for 61.6% of reported HIV cases in 2016. Exposure categories are mutually exclusive and are meant to identify an individual’s most likely transmission route. Where multiple potential transmission routes are reported, a hierarchy is used to assign the case to a single category (see Appendix 1).

*For Alberta, cases identified as Citizenship and Immigration Canada/Out of Country (CIC/OOC) were classified in the exposure category of “Other”

**Excludes 2,612 cases where sex was not reported or reported as transsexual or transgender.

Due to inter-provincial and territorial heterogeneity in case reporting practices, some surveillance data may not completely represent the current state of the HIV epidemic. In particular, surveillance data on race/ethnicity and exposure category is often incomplete or is not submitted by some provinces. At the national level, only 48.6% of reported cases among adults included information on race/ethnicity and 61.6% specified an exposure category in 2016. With the understanding that this limitation severely impacts the interpretability of this data, existing data indicates that there may be greater racial diversity among women compared to men with HIV in Canada. Black women made up 36.5% of incident diagnoses among women in 2016, followed by Indigenous First Nations women (32.7%) and white women (21.0%) (Table 3). Comparatively, half (47.8%) of 2016 male cases were among white men, followed by Indigenous men and Black men. While greater sex-stratified proportions of new cases are among Black and Indigenous women than Black and Indigenous men, the proportion of new cases among white, South Asian/West Asian, Arab, Asian, and Latin American women is lower than that of males of the same racial/ethnic group.
Table 3: Number and percentage distribution of all-ages Canadian HIV cases by sex and race/ethnicity, 2014, 1998-2014

<table>
<thead>
<tr>
<th></th>
<th></th>
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<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Indigenous</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>First Nations</td>
<td>103</td>
<td>32.7</td>
<td>1,082</td>
<td>30.2</td>
<td>113</td>
<td>13.7</td>
<td>1,149</td>
<td>13.0</td>
</tr>
<tr>
<td>Métis</td>
<td>7</td>
<td>2.2</td>
<td>117</td>
<td>3.3</td>
<td>11</td>
<td>1.3</td>
<td>166</td>
<td>1.9</td>
</tr>
<tr>
<td>Inuit</td>
<td>1</td>
<td>0.3</td>
<td>5</td>
<td>0.1</td>
<td>2</td>
<td>0.2</td>
<td>13</td>
<td>0.1</td>
</tr>
<tr>
<td>Indigenous, unspecified</td>
<td>3</td>
<td>1.0</td>
<td>256</td>
<td>7.2</td>
<td>1</td>
<td>0.1</td>
<td>287</td>
<td>3.2</td>
</tr>
<tr>
<td>South Asian/West Asian/Arab</td>
<td>7</td>
<td>2.2</td>
<td>38</td>
<td>1.1</td>
<td>37</td>
<td>4.5</td>
<td>249</td>
<td>2.8</td>
</tr>
<tr>
<td>Asian</td>
<td>4</td>
<td>1.3</td>
<td>69</td>
<td>1.9</td>
<td>62</td>
<td>7.5</td>
<td>458</td>
<td>5.2</td>
</tr>
<tr>
<td>Black</td>
<td>115</td>
<td>36.5</td>
<td>1,220</td>
<td>34.1</td>
<td>135</td>
<td>16.4</td>
<td>1,226</td>
<td>13.8</td>
</tr>
<tr>
<td>Latin American</td>
<td>3</td>
<td>1.0</td>
<td>36</td>
<td>1.0</td>
<td>52</td>
<td>6.3</td>
<td>386</td>
<td>4.4</td>
</tr>
<tr>
<td>White</td>
<td>66</td>
<td>21.0</td>
<td>730</td>
<td>20.4</td>
<td>394</td>
<td>47.8</td>
<td>4,792</td>
<td>54.1</td>
</tr>
<tr>
<td>Other</td>
<td>6</td>
<td>1.9</td>
<td>27</td>
<td>0.8</td>
<td>17</td>
<td>2.1</td>
<td>136</td>
<td>1.5</td>
</tr>
<tr>
<td><strong>Subtotal</strong></td>
<td>315</td>
<td>100</td>
<td>3,580</td>
<td>100</td>
<td>824</td>
<td>100</td>
<td>8,862</td>
<td>100</td>
</tr>
<tr>
<td>Race/ethnicity not reported</td>
<td>232</td>
<td>N/A</td>
<td>7083</td>
<td>N/A</td>
<td>964</td>
<td>N/A</td>
<td>23,782</td>
<td>N/A</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td>547</td>
<td>N/A</td>
<td>10,663</td>
<td>N/A</td>
<td>1,788</td>
<td>N/A</td>
<td>32,644</td>
<td>N/A</td>
</tr>
</tbody>
</table>

Data source: Canadian Community Disease Reports, HIV in Canada – Supplementary tables, 2016.\(^47\) Data on race were submitted for 48.6% of reported HIV cases in 2016. For all provinces and territories, race/ethnicity information is not available before 1998. Race/ethnicity information is not submitted by Quebec or British Columbia, and is not available for Ontario before 2009. Reporting of HIV cases for individuals younger than two years of age varies among provinces and territories. *Excludes 291 cases where sex was not reported or reported as transgender.

Presumably due to the aforementioned reporting limitations, Public Health Agency of Canada surveillance reports do not provide sex-segregated rates of exposure by category and race/ethnicity simultaneously. However, based on cross-classification of exposures by category and race/ethnicity for males and females combined (Figure 1) as well as some regional sex-stratified data,\(^50\) there is reason to believe that exposure category varies by race and sex. Where race and exposure category were reported, IDU was the primary exposure category among Indigenous populations in 2016.\(^46\) 53.2% of people with HIV who are Black were classified as Het-Endemic, though, as previously noted, this does not necessarily mean that exposure was actually heterosexual, or took place in
the HIV-endemic country. Among people with HIV who are South Asian, West Asian, or Arab, as well as white, Asian, Latin American, or another ethnicity, exposures were primarily MSM followed by heterosexual contact. Some provinces provide a more nuanced breakdown of incident HIV cases by sex, race, and exposure category. Looking to Saskatchewan, where the 2016 HIV diagnosis rate for women was the highest in Canada, 88% of newly diagnosed women were Indigenous and 58% of newly diagnosed Indigenous women reported IDU while 38% reported heterosexual activity as their primary HIV risk factor. This speaks to a larger trend elucidated by Roy and colleagues, whereby IDU in general is becoming less common, however, specific drugs and/or drug use practices may contribute to increased risk of blood-borne infection. Roy specifically points to the growing opioid epidemic, and a recent study of people who inject drugs in Montreal which found that people who injected opioids exhibited more behaviours related to transmission risk (syringe sharing, injecting more frequently and in public places), and greater Hepatitis C risk than people who inject drugs other than opioids. In this way, increased injection of opioids may drive HIV incidence, even in the context of reduced IDU in the general population. This illustrative example points to the need for further research on HIV transmission patterns in Canada, while reporting quality of surveillance data indicates the need for such data at the intersection of race and sex.
Figure 1: Percentage distribution of all-ages Canadian HIV cases by race/ethnicity and exposure category, 2016 [n=2,344]

<table>
<thead>
<tr>
<th>Race/Ethnicity</th>
<th>MSM</th>
<th>MSM/IDU</th>
<th>IDU</th>
<th>Blood</th>
<th>Het-Endemic</th>
<th>Het-Risk</th>
<th>Het-NIR</th>
<th>Other</th>
<th>NIR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Indigenous</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Black</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>South Asian / West Asian / Arab</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asian</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Latin American</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Data source: Canadian Community Disease Reports, HIV in Canada – Supplementary tables, 2016. Data on race were submitted for 48.6% of reported HIV cases, while data on exposure category were submitted for 61.6% cases in 2016. Reporting of HIV cases for individuals younger than two years of age varies across provinces and territories. Abbreviations: MSM=Men who have sex with men, MSM/IDU= men who have sex with men and use injection drugs, IDU=injection drug use, Blood= receipt of blood or blood products, Het-risk= heterosexual contact with risk factor, Het-NIR= heterosexual contact with no identified risk factors, Het-Endemic= origin from HIV-endemic country, NIR= no identified risk.

The social position occupied by women with HIV can be articulated within the framework of intersectionality, a term coined by Black feminist legal scholar Kimberlé Crenshaw. Intersectional perspectives posit that embodied social positions and identities interact in the context of structural power inequities, resulting in inequities across population groups. In this context, intersectionality theory suggests that population characteristics of women with HIV are determined by the systematic factors such as sexism, racism, colonialism, transphobia, and homophobia which interact with gender.
(and one another) to make women vulnerable to HIV. The result of intersecting power inequities such as racism and transphobia impacting vulnerability to HIV is that the population of women with HIV tends to be multiply-marginalized, further affecting health and access to care, as well as other aspects of life such as stigma and discrimination. Furthermore, the HIV care women receive is often not population-specific, but rather tailored to men with HIV, posing a systematic obstacle to women achieving optimal health and wellness. Systematic intersectional disadvantages for women with HIV are also visible in fields outside of health, one notable example being that the majority of women convicted in HIV non-disclosure cases occupy intersectionally marginalized positions which likely influence their ability to disclose. Indigenous women are particularly over-represented, accounting for an estimated 42% of women charged. An intersectional framework will be used to contextualize the experiences of women with HIV throughout this thesis.

2.3 Criminalization of HIV Non-Disclosure in Canada

Under Canadian case law (R v Mabior), criminal charges may be laid against people with HIV who do not disclose their HIV status to sexual partners prior to having sex that poses a “realistic possibility of HIV transmission”. Charges can be laid regardless of whether transmission actually occurred, and can range from common nuisance to murder, with the most common charge being aggravated sexual assault. However, the precedent set in the 2012 R. v. Mabior case does not specify which sexual acts under which circumstances constitute a realistic possibility of transmission. Guidelines created by the Canadian HIV/AIDS Legal Network interpret the R. v. Mabior ruling such that people with HIV have a legal obligation to disclose prior to engaging in vaginal sex with a condom if their plasma viral load is not “low,” or prior to condomless vaginal sex regardless of viral load. Based on risk of transmission, the guidelines estimate that disclosure requirements for anal sex would be at least as strict, and requirements for oral sex may be more lenient. Notably, in 2017, the Ontario Attorney General and Ontario Minister of Health and Long Term Care announced that cases of HIV non-disclosure in Ontario would not be prosecuted where the person with HIV has had a suppressed viral load for six months. However, this decision is limited to Ontario and (similarly to the Supreme
Court decision) does not specifically address other situational factors which may make HIV transmission risk negligible. As of 2016, at least 184 individuals in Canada have been charged in cases of HIV non-disclosure, 101 have been convicted, and 93% of those convicted have received a prison sentence.\textsuperscript{57} As of 2014, Canada was second to only the United States, a population ten times its size, in the number of arrests and prosecutions in cases of HIV non-disclosure.\textsuperscript{1,59}

Another consequence of criminalization is that it renders the empirical study of HIV disclosure to sexual partners unethical. Because information exchanged between researchers and participants may be subject to subpoena in a court of law investigating claims of non-disclosure, researchers often deliberately avoid asking participants whether they have disclosed their HIV status to their sexual partners. One way to circumvent this obstacle is to study barriers to HIV disclosure rather than disclosure itself.

\subsection*{2.3.1 Evidence-Based Objections to HIV Criminalization}

While HIV status disclosure prior to sex represents a “best-case scenario” in some circumstances, research does not support the use of criminal law to encourage this behaviour. Canada’s legal enforcement of HIV disclosure in particular has been criticized for failing to take into account scientific evidence regarding transmission risk; the Supreme Court of Canada’s vague definition (“realistic possibility of HIV transmission”) has allowed for charges to be laid where evidence shows the risk of transmission to be insignificant.\textsuperscript{60} To reiterate the evidence supporting use of ART, risk of HIV transmission when viral load is suppressed is estimated to be near zero for all sex acts, even without the use of condoms.\textsuperscript{40} If prosecution was consistently carried out with attention to such evidence, condomless sex with suppressed viral load would no longer be considered to pose realistic possibility of HIV transmission.

From a policy perspective, public health professionals have criticized the Canadian legal system for undermining HIV prevention strategies.\textsuperscript{60} In particular, criminalization may serve as a disincentive for HIV testing: it is theorized that those who suspect that they have HIV may knowingly avoid HIV testing in order to claim inculpability should they expose someone else.\textsuperscript{61} This concern is significant, as an estimated 21% of people with
HIV in Canada do not know their HIV status, and this population is more likely to engage in condomless sex than their HIV status-aware counterparts. In addition to the potential for unintended consequences due to HIV criminalization, there remains uncertainty regarding whether the law actually prevents HIV transmission in the population. Canadian policy research accounting for the coverage and efficacy of HIV criminalization as an intervention concluded that non-disclosure law is likely a poor tool to promote population change. Because HIV criminalization only targets those who are aware of their serostatus (and who transmit HIV at rates far lower than those who are unaware), the intervention does not apply to the population with the highest potential for impact. In fact, if criminalization does serve as a disincentive for HIV testing, the intervention may actually increase the rate of HIV transmission among people with HIV who do not know their serostatus. The inconsistent use of scientific evidence in convictions related to HIV non-disclosure, and the potentially dysfunctional mechanism of HIV criminalization call to question the practical value of this approach.

### 2.3.2 Ethical Objections to HIV Criminalization

From an ethical perspective, the use of criminal law to enforce HIV disclosure has been a highly contested topic in Canada and around the world. Beyond the efficacy of such laws, many have argued that HIV non-disclosure should be criminalized based on two main legal philosophies: the harm principle and legal moralism. The harm principle, proposed by John Stuart Mill, argues that an action should only be criminalized if it causes harm to another person, regardless of whether the action was moral or was done for the good of the person committing it. However, Mill states that if the harm is consented to by the person being harmed, or if a person harms themselves, there should be no legal consequences. By his definition, “harm” is inclusive of not only intentional harms but also negligent harm such as failure to meet an obligation; the majority of HIV non-disclosure cases would meet this definition. However, when applied to cases of HIV non-disclosure the harm principle raises several issues, primarily, what constitutes harm? Some scholars argue that only transmission of HIV without disclosure constitutes harm, while others argue that exposure to HIV without disclosure can also potentially represent harm, analogous to endangerment. Non-disclosure itself, regardless of whether
transmission occurs, has also been deemed by some to be a moral wrong.\textsuperscript{65,67} Moral wrongs are actions which violate some ethical standard, and under the principle of legal moralism may be punished by criminal law.\textsuperscript{68} Criminalization of moral wrongs functions to hold society to a given moral standard and disincentivize behaviour that the society decides is immoral, including acts that do not harm others. This principle has been applied to HIV criminalization, arguing that failing to disclose information that might otherwise cause someone to withdraw consent to sex invalidates that consent.\textsuperscript{67} However, simply because an act causes harm or is judged to be morally wrong does not mean that this act has sufficient cause to be criminalized. Both the harm principle and legal moralism warn that if criminalization causes more harm than it prevents, application of criminal law is not justified.\textsuperscript{69}

In order to judge whether HIV non-disclosure should be criminalized, attention must be given to practical factors including the efficacy (or inefficacy) of the current law, definitions of harm in the context of improved HIV treatment, potential disclosure-related harm to people with HIV, and criminalization as it relates to HIV stigma. Without minimizing the gravity of an HIV diagnosis, it is important to note that since the Supreme Court of Canada first addressed HIV non-disclosure in 1998,\textsuperscript{70} significant advances in HIV research have rendered HIV a largely treatable chronic condition for those with access to care.\textsuperscript{36} Considered alongside the dearth of cases prosecuting instances of non-disclosure for other treatable but serious communicable diseases (herpes, hepatitis B and C),\textsuperscript{1} one must ask whether harms resulting from HIV exposure today are truly significant or the result of longstanding HIV stigma and exceptionalism. The drastic and exceptional treatment of HIV underpins other aspects of HIV criminalization as well; criminalization purports to protect the general population (especially heterosexual women) from contracting HIV but fails to consider that legal obligations to disclose place people with HIV (especially women) in vulnerable situations. In fact, it is not uncommon for ethical analyses of HIV disclosure to explore exclusively the potential harms to HIV negative partners without so much as mentioning the harms that disclosure can inflict on people living with HIV.\textsuperscript{65,66} For some, disclosure of one’s HIV status can result in financial instability, loss of relationships, loss of social support, social defamation, and physical, sexual, or emotional abuse.\textsuperscript{71} In the context of financial dependence or intimate partner
violence, non-disclosure can be an act of survival. Non-disclosure law in Canada does not adequately address any of these factors, and in-fact allows for estranged or abusive partners of people with HIV to file or threaten to file a non-disclosure lawsuit. In this way, the law not only holds people with HIV accountable for disclosing but gives the people who make disclosure impossible the power to enforce that accountability.

At the societal level, criminalization has been criticized for contributing to the HIV stigma and exceptionalism that drives the epidemic. Attribution of social stigma to a behaviour deemed to be immoral is at the heart of legal moralism’s mechanism, and existing stigma is reinforced when legitimized by Canadian law. The fact that the legal system most often treats cases of non-disclosure as aggravated sexual assault further problematizes this issue. Sexual assault charges imply that, as with sexual assault, responsibility for the act lies only with the actor. However, the responsibility for engaging in safer sex lies with both partners and cannot be wholly attributed to people with HIV. Non-disclosure law tells the public otherwise, generating additional stigma, the effects of which are not insignificant. The UN Secretary-General has famously stated that “[stigma] helps make AIDS the silent killer, because people fear the social disgrace of speaking about it, or taking easily available precautions. Stigma is a chief reason why the AIDS epidemic continues to devastate societies around the world.” Reflecting on the justification of applying the harm principle and legal moralism in cases of HIV non-disclosure given the surrounding context, it is difficult to argue that criminalization prevents more harm than it incites. This position has been upheld by expert groups including the Joint United Nations Programme on HIV/AIDS and an international team of twenty HIV scientists who strongly condemn non-disclosure laws for reasons related to lack of evidence based justification and respect for the autonomy and safety of people with HIV.

2.4 Barriers to Partner Disclosure

A woman’s decision to disclose (or not to disclose) her HIV status to a sexual partner is known to be informed by several factors related to herself, her relationships, and her support system. These factors may serve as barriers to, or facilitators of partner disclosure. Despite differing cultural perceptions of HIV, studies across the world report
that fear of rejection and abandonment serves as a major barrier to partner disclosure among women with HIV. Fear of rejection in an intimate relationship is tied to the relationship’s power dynamics, including whether the relationship is abusive. The threat of violence, whether physical, sexual, or emotional, can make partner disclosure dangerous. Factors outside of the relationship may also have bearing on disclosure; at the level of the individual, people who feel internalized stigma about their own HIV status are known to struggle with disclosure. Furthermore, those without adequate social support may be hesitant to disclose due to the possibility of rejection or abandonment, especially in the presence of societal HIV stigma.

The following paragraphs on the barriers to partner disclosure are cognizant of the varying theoretical models and corresponding measures of disclosure, which have changed over the years. A common practice has been to consider disclosure a unidimensional process whereby an individual progressively informs their social network of their HIV status one-by-one, with the same barriers and facilitators informing each decision. The most common measure that maps onto this approach enquires about the members of an individual’s social network, and which of those members are aware of the individual’s HIV status. Using this information, the proportion of social network disclosed to is calculated (e.g. ). The creation of unidimensional disclosure indices (e.g. ) is a similar method in that neither allows the researcher to examine whether disclosure practices vary based on the target of disclosure. Such measures may be a threat to research validity, as research has demonstrated that disclosure rates as well as reasons for disclosing vary by target. A recent study of 158 people with HIV (70% women) in Tanzania investigated the dimensionality of voluntary disclosure to a comprehensive list of 21 potential targets, finding that disclosure to children, close family, larger community, and partners represented four independent factors. Moreover, disclosure to targets across these categories was differentially associated with two of the main determinants of disclosure: stigma and social support. These results indicate that traditional conceptions of disclosure as a unidimensional process may be insufficient to capture the true determinants of disclosure. Accordingly, while the following barriers to partner disclosure may also apply to other disclosure targets, studies specific to partner disclosure have been prioritized in order to maximize construct validity.
2.4.1 Physical and Sexual Abuse

Physical abuse refers to acts of physical violence against another person, while sexual abuse refers to sexual acts which are forced or coerced. Often these types of abuse are grouped together under the term intimate partner violence (IPV), which refers more broadly to the perpetration of controlling behaviours, as well as physical, sexual, or emotional abuse against an intimate partner. However, physical and sexual abuse do not only occur within the context of intimate relationships, and IPV is not the only type of abuse relevant to HIV disclosure. Physical and sexual abuse in any context may undermine the personal agency of those who experience it and thus affect the perceived costs of partner disclosure. Furthermore, physical and sexual abuse may be perpetrated by and experienced by people of any gender. However, the majority of abuse against women is perpetrated by men, a factor reflected in the literature and thus the following paragraphs.

Women who experience physical or sexual violence, or who expect that their partner may become abusive, cite fear of such abuse as a barrier to disclosing their HIV status to sexual partners. This barrier is significant given that women with HIV experience a high rate of violence, and that experiencing violence following disclosure of one’s HIV status is not uncommon. The same survey of American medical and mental health care providers that identified fear of abandonment as a prevalent concern for women with HIV also found that 29% of their female patients feared physical abuse, and 56% feared emotional abuse upon disclosing their HIV status to their partners. A qualitative study of 50 mostly African-American women found similar results; 12% feared that people would react violently to them disclosing their HIV status, and two women described consciously choosing not to disclose because they feared that their partner would react violently. Notably, one of these women described that her partner had not been physically violent towards her in the past, but that the anger he had displayed in other situations made her question whether communicating with him about safer sex was worthwhile. This experience was echoed by women from a different American disclosure study; one woman who experienced life-threatening IPV from a previous partner would not disclose her HIV status to her current partner, stating that “…
sometimes the sweetest men on earth will just turn on you … You just can’t tell”. Thus, fear of violence even without current experiences of IPV, may be a barrier to disclosure.

HIV disclosure may also incite violence against women with HIV, even among those who did not experience violence prior to disclosure. In a clinic sample of 310 HIV+ women, 4% reported physical abuse as a direct result of disclosing their HIV status to somebody (i.e. not partner-specific). However, upon examining the prevalence of physical and sexual abuse experienced before and after their HIV diagnosis (and presumably some degree of disclosure), a larger gap was discovered: 45% of women had experienced abuse after diagnosis, with 13% of that abuse being new, i.e. occurring only after diagnosis. Among those who experience abuse prior to disclosure, disclosure may also affect the frequency and severity of abuse as articulated by one participant from this study: “He was abusive before I told him I was HIV-positive, and afterwards, well, the beatings got worse and more . . . they happened more regularly.” Indeed, the majority of abuse experienced after diagnosis is among those who also experienced abuse prior to diagnosis. Because current abuse provokes fear of further abuse upon disclosure, and this fear is shown to be valid, abuse represents a significant barrier to partner disclosure.

### 2.4.2 Sexual Relationship Power

According to Social Exchange Theory and feminist literature, power is defined as the potential one has to influence the actions of another (“power to”) as well as one’s ability to do something against another’s wishes (“power over”). Sexual relationship power (SRP) can be defined as the balance or imbalance of interpersonal power across partners in an intimate relationship. Just as intersectional power differentials cause health inequities at the societal level, an imbalance of interpersonal power can affect the health of those within a sexual relationship. SRP dynamics often come to light in the context of partnered decision making; a more dominant partner may take control of joint decisions, a tendency that is especially relevant to HIV research in the context of sexual communication and decision making. In fact, reduced sexual relationship power has been repeatedly implicated as a risk factor for sexually transmitted infections (STIs) including HIV, under the theory that gendered power imbalances disenable heterosexual women from negotiating safer sex practices with their male partners. Since
the advent of this theory in the 1990s, scholars have hypothesized that such power imbalances are not only gendered but intersectional, mutually constructed by gender, race, class, and sexual orientation. Furthermore, it is hypothesized that in addition to being a barrier to safer sex practices, reduced SRP may be a barrier to HIV status disclosure.

However, the construct of SRP is more often applied explicitly in research on HIV risk than HIV disclosure, where SRP is evident in discussions of fear of abandonment, loss of economic support, rejection, abuse and suspicions of infidelity. Each of these fears is indicative of reduced power under Social Exchange Theory, which argues that power is based on the balance/imbalance of resources (e.g. emotional, economic) across partners and any resulting interpersonal dependence. Fear of abandonment, an indicator that the partner with this fear is in some way dependent on the other partner, was reported by women with HIV in the Africa and North America. A qualitative study of recently tested people with HIV in Tanzania found that fear of their partner’s reaction was the most influential barrier to HIV disclosure, and among women, abandonment was the most-feared reaction. This result was replicated in the partnered quantitative study, where among a sample of 245 women who underwent HIV testing, 52% of those who had not disclosed their HIV status to their sexual partner after three months cited fear of their partner’s reaction as their reason for non-disclosure. In addition to fearing abandonment, these women feared loss of economic support, another indicator of reduced SRP. North American studies also demonstrate women with HIV fearing abandonment as a result of disclosure; Moneyham et al. conducted a series of focus groups on disclosure with women in the southeastern United States, where participants feared that disclosure would put an end to romantic relationships, resulting in a loss of social support. Similarly, an American survey of 136 medical and mental health care providers with experience treating women with HIV found that an estimated 35% of their female HIV patients had expressed fear of abandonment (defined as withdrawal of resources) as a result of disclosure. While “abandonment” can refer to withdrawal of both tangible resources and emotional support, “rejection” encompasses the emotional aspects of abandonment. Fear of rejection was similarly reported by women. Anticipated abuse and loss of social support as a result of disclosure, whether
actualized or not, also represent aspects of SRP which may disenable women from communicating with their partners about HIV.

2.4.3 HIV Stigma

HIV stigmatization has been conceptualized as a dynamic social process that marks differences between groups of people, especially those experiencing social inequalities pertaining to class, race, gender, and sexuality.\textsuperscript{102} Stigma is produced by these inequalities, and reproduces them,\textsuperscript{102} affecting health outcomes for those who are stigmatized.\textsuperscript{103} Furthermore, HIV stigma has been theorized to operate at multiple levels: interpersonal, in the form of enacted stigma or discrimination, as well as intrapersonal in the form of felt-normative or perceived stigma (the subjective awareness of societal stigma) and internalized stigma (the degree to which individuals accept stigmatized beliefs to be true).\textsuperscript{104} These types of stigma have the potential to enact different outcomes for people with HIV, including in the context of partner disclosure.\textsuperscript{105}

A 2016 systematic review gathered studies on the partner disclosure-specific effects of enacted (n=1), felt-normative (n=6), and internalized stigma (n=7), as well as studies where the type of stigma measured was multidimensional or not specified (n=14).\textsuperscript{105} This review found that enacted stigma was often presented as a barrier to partner disclosure in qualitative research, with two studies documenting abuse perpetrated by the partners of seropositive individuals following disclosure,\textsuperscript{106,107} findings which have been mirrored in other studies where enacted stigma is framed as acts of discrimination.\textsuperscript{90,108} Felt-normative stigma had a variable effect on disclosure to sexual partners, with three studies reporting higher levels of perceived stigma to be associated with reduced partner disclosure\textsuperscript{109-111} and two studies reporting no association between the two.\textsuperscript{84,112} These studies come exclusively from Africa and the United States, with sample composition ranging from 27% to 100% women. The largest sample (n=1552, 59% women) comes from a longitudinal study of ever-married women and their husbands in Malawi.\textsuperscript{111} This study provided HIV testing and counselling to each spouse in private, and two years later investigated the factors associated with partner non-disclosure. Among women only, higher levels of felt-normative HIV stigma were associated with non-disclosure to their spouse (OR=1.22 [95% CI 1.05, 1.42]). While data on disclosure to extra-marital sex
partners was also collected, the role of perceived stigma in this context was not reported and thus this inference is limited to married heterosexual couples. An American study of 341 people with HIV (34% heterosexual women, 26% heterosexual men, 40% MSM) investigated the role of relationship type further, finding that increased perceived HIV stigma was associated with decreased odds of disclosing to all sex partners (OR=0.89 [95% CI 0.81, 0.93]), and that this relationship was not moderated by sexual behaviour subgroup, the nature of sexual relationships (primary partner, casual partner(s), both) or the HIV serostatus of sexual partners.\textsuperscript{109} However, perceived stigma in this study was measured using an HIV stigma subscale specific to disclosure concerns (see Berger and colleagues\textsuperscript{113}), which may not be synonymous with felt-normative stigma, and theoretically should be associated with disclosure. A similar study in South Africa (n=630, 67\% women) found the same association between perceived stigma and disclosure, however, the effect was found to be significant only among those in steady relationships as opposed to casual partners.\textsuperscript{110} Other studies reported no relationship between perceived stigma and disclosure: the Dima study that investigated the multidimensionality of disclosure among people with HIV in Tanzania (detailed in 2.4) found that perceived stigma was unrelated to both cumulative disclosure and partner specific disclosure, but was positively associated with level of disclosure to the larger community.\textsuperscript{84} An American study found similar results; perceived stigma was unrelated to partner disclosure, but negatively associated with disclosure to parents.\textsuperscript{112} Taken together, these results indicate that felt-normative stigma may be a barrier to HIV disclosure, however, further analyses should be mindful of gender differences and the potential effect of partner relationship type.

While the effects of enacted and felt-normative stigma have not fully been established, there is compelling evidence pointing towards internalized stigma as a barrier to partner disclosure among women. However, the only identified North American study on this topic is specific to middle-aged homeless and unstably housed people with HIV, the majority (70.7\%) of whom were male.\textsuperscript{114} This cross-sectional study found that higher levels of internal HIV stigma were associated with 1.47 greater odds of non-disclosure to at least one recent sex partner prior to their first sexual encounter, adjusted for variables associated with general HIV stigma (including gender). While women reported higher
levels of internalized stigma than men, the effect of gender on this relationship was not reported. Similarly, in a sample of 862 people with HIV (56.3% women) in Ethiopia, Mozambique, and Africa, Geary and colleagues found that women reported higher internalized stigma compared to men, and that disclosure to one’s sexual partner was significantly associated with lower levels of internalized stigma in bivariate analysis. Again, the effect of gender on the relationship between stigma and partner disclosure was not reported, however gender was found to moderate associations between internalized stigma and perceived wellbeing, indicating the potential importance of addressing gendered aspects of internalized stigma. Partially filling this gap, a longitudinal study of 293 pregnant women recently diagnosed with HIV in South Africa measured internalized HIV stigma during and post-pregnancy, finding that while stigma had no effect on partner disclosure during pregnancy, higher internalized stigma predicted continuing partner non-disclosure at 3 months post-pregnancy among those who had not yet disclosed at baseline. This result indicates that internalized stigma may develop over time, or be a characteristic of women who chronically struggle with partner disclosure. Further longitudinal research from Uganda (n=259, 67% women) found similar results; internalized stigma measured at baseline was negatively associated with subsequent partner disclosure in both univariable and multivariable models (ARR=0.94 [95% CI 0.90, 0.99]). Expressed as an average marginal risk, each additional point on the study’s internalized stigma scale was associated with a 4.0% [95% CI 1.0%, 7.0%] lower probability of disclosure to a primary sexual partner over the median 1.8 years of observation. Interestingly, the magnitude of the relationship between internalized stigma and disclosure increased with social distance from the participant, such that stigma was a greater barrier to disclosure to neighbours and religious leaders, and the general public than it was to sexual partners. Other studies have found no relationship between internalized stigma and partner disclosure; a mixed-methods analysis found no association between internalized stigma and partner disclosure in univariate or multivariable models. However, a two-item measure was used to capture internalized stigma, which may be insufficient compared to the psychometrically evaluated measures applied in the other studies in question. Furthermore, outcome data on disclosure to sexual partners was missing for 17.8% of respondents who knew their HIV status and
were married/cohabitating with a partner, calling to question the validity of these results. Interestingly, the methodologically sound Dima study of 158 people with HIV (70% women) detailed prior found that internalized stigma was associated with cumulative disclosure, as well as disclosure to close family, children, and the larger community, but not sexual partners. The results of this analysis appear to support the social distance hypothesis posited in the Tsai study. Taken together, current evidence does not discount the presence of a relationship between internalized stigma and partner disclosure, though this relationship may be more important to disclosure targets at greater social distance.

2.4.4 Social Support

Social support, or the security that we gain from positive interpersonal relationships, is a known facilitator of positive health outcomes. Such support may come in the form of tangible support (for example, someone who will take care of you when you’re sick), emotional support, affection, and social companionship. In the context of HIV disclosure, social support has been cited as both a reason for disclosing (i.e. desire for support) and not disclosing (i.e. fear of losing social support). A meta-analysis (n=2253) of the relationship between social support and HIV disclosure (inclusive of all targets) revealed a weak positive correlation between the two (r=0.159), though the confidence interval included zero. Furthermore, the analysis does not specify the temporal relationship between the two variables, making it impossible to draw conclusions about social support as a facilitator or barrier to HIV disclosure. Some quantitative studies support the hypothesis that social support is associated with higher cumulative and target-specific rates of disclosure, while others found no association between cumulative disclosure and social support. Within these studies, it is important to identify which types of social support are being measured. Social support measures often tap into several dimensions (tangible support, emotional support, affection, and social companionship, from any of the following perspectives: received support, perceived support, reciprocated support, need for support, or satisfaction with support. Additionally, measures may be HIV-specific or target-specific. Some domains and perspectives appear to be more salient to disclosure than others; both Simoni and Petrak found no association between satisfaction with support and
cumulative disclosure. Conversely, perceived social support has been associated with cumulative disclosure,\textsuperscript{84} and target-specific disclosure,\textsuperscript{120} including disclosure to intimate partners.\textsuperscript{84} Among a sample of 158 people with HIV (70% women) in Tanzania, perceived instrumental support (specific to HIV) was positively correlated with voluntary disclosure to intimate partners (r=0.14, p<0.05).\textsuperscript{84} Interestingly, neither HIV-specific emotional/informational support nor adherence support were related to partner disclosure in this study, indicating that the dimensionality of social support in addition to disclosure target is of importance.

However, quantitative studies of disclosure and social support specific to intimate relationships have been largely cross-sectional, raising the question of whether given levels of social support preclude disclosure, or result from it. Qualitative research can aid in nuancing this relationship; while some women report fear of losing social support from their partners as a result of disclosing,\textsuperscript{101} others disclose because they desire additional social support from their partners.\textsuperscript{100,101,123} It seems that social support can function as a facilitator to disclosure, if it is perceived to be present already. One woman from Sowell et al.’s study of American women with HIV spoke to this dynamic: “I tell people who I know will be there for me no matter what. The people I have chosen has been there for me with other problems”.\textsuperscript{124} This relationship has also been documented in the context of HIV testing; one of the strongest predictors of partner disclosure is the degree of awareness and involvement the partner has in the HIV testing experience.\textsuperscript{74,100} Additionally, women who chose not to involve their partners in the testing procedure were more likely to experience abuse as a result of disclosure,\textsuperscript{100} indicating that their partner’s negative reaction was likely anticipated. It appears that social support may drive disclosure similarly to involvement with HIV testing such that women who experience low social support anticipate low social support as a result of disclosure, and vice versa. Further quantitative longitudinal studies of perceived social support and partner disclosure would aid in substantiating this hypothesis.
2.5  Childhood Physical and Sexual Abuse

2.5.1  Definition

Child abuse or maltreatment, as defined by the WHO, refers to “all forms of physical and/or emotional ill-treatment, sexual abuse, neglect or negligent treatment or commercial or other exploitation, resulting in actual or potential harm to the child’s health, survival, or development of dignity in the context of a relationship of responsibility, trust, or power”.\textsuperscript{125} This definition frames abuse based on the impact of abusive behaviour on the child rather than the intention of the abuser, as the child is the subject of interest for this study. Furthermore, this definition captures the main forms of child abuse covered under the Criminal Code of Canada, including physical abuse, sexual abuse, exposure to family violence, and neglect.\textsuperscript{126} According to retrospective self-report from the 2001 wave of the Ontario Child Health Study, before the age of 16, 28.2% of females had experienced physical abuse and 22.1% experienced had sexual abuse.\textsuperscript{127} The combined proportion of females who experienced childhood physical and/or sexual abuse was 38.8%.\textsuperscript{127} While these figures come from a representative population-based study, the prevalence of child abuse tends to be elevated among community samples of women with HIV.\textsuperscript{128}

2.5.2  Child Abuse and HIV-Related Outcomes

The frequent co-occurrence of HIV and histories of CPSA among women has been the subject of a great deal of research. Multiple meta-analyses have indicated significant, positive relationships between both childhood physical\textsuperscript{11} and sexual abuse\textsuperscript{12,13} and HIV vulnerability in adulthood. Factors increasing HIV vulnerability in these studies include engaging in condomless sex, sex work, early first sexual experience, having multiple sex partners, and experiencing sexual abuse in adulthood.\textsuperscript{11–13} Many theoretical models exist to explain the relationship between CPSA and HIV, tending to focus on intrapersonal/psychological factors such as learned sexual scripts.\textsuperscript{129} However, any such model must be contextualized by accounting for social power hierarchies, particularly with respect to gender. Gendered power imbalances have been articulated in several models of HIV risk,\textsuperscript{86,130,131} however, Rosenthal and Levy explicitly extend the definition
of gender-based violence to include violence against young girls. Acts such as rape and abuse (termed “force”) are said to undermine the personal agency of women and girls, contributing to HIV vulnerability via reduced sexual relationship power. Recalling that reduced sexual relationship power is also a barrier to HIV disclosure, CPSA is revealed to not only be a risk factor for HIV, but may also impact a woman’s ability to disclose her HIV status once diagnosed. In this way CPSA acts as a sort of “double jeopardy,” increasing risk for HIV as well as for further vulnerability among those who are affected.

The relationship between CPSA and reduced sexual relationship power has been investigated implicitly via factors contributing to relationship power including low self-esteem, distrust, lack of meaningful communication with partners, having a partner who is controlling and/or uncaring and relationship dissatisfaction. However, sexual relationship power is not the only HIV-related outcome of CPSA. Studies among women in the general population have shown CPSA to be a risk factor for physical/sexual abuse in adulthood (i.e. revictimization), as well as reduced perceptions of social support. If these relationships hold among women with HIV, CPSA will be further implicated as a cause of barriers to HIV status disclosure among women. There is also the potential for experiences of CPSA to impact barriers to HIV disclosure that are not investigable among the general population, such as HIV stigma. While theoretical bases for these relationships have not been formally articulated in the literature, the effects of CPSA are diverse and can reach far into adulthood. Women in the general population who have experienced CPSA have reported stigma related to the abuse as well as distrust and perceived powerlessness in adulthood. These outcomes, while not specific to HIV, document connections between CPSA and later stigma, as well as relationships between CPSA and factors known to contribute to HIV disclosure concerns. Whether the HIV-specific analogues of these outcomes are related to CPSA among women with HIV should be the subject of further inquiry, including the current study.

2.5.3 Eco-Social Theory

Given that CPSA has been implicated as a cause of both HIV vulnerability and barriers to HIV disclosure, it is of interest, from a causal modelling perspective, to understand the causes of CPSA (i.e. the “causes of the cause”). While many theoretical models exist to
explain the etiology of child maltreatment, the currently favoured model is Belsky’s ecological or eco-social model.\textsuperscript{136} This model posits that CPSA is caused by social-psychological forces acting and interacting across multiple domains: the developmental experiences of the caregiver (termed “ontogenic environment”), the family (“microsystem”), the community (“exosystem”) and the larger social culture (“macrosystem”). Belsky identified several risk factors and potential mechanisms at each level. Notably, risk factors stemming from colonization of Indigenous land are not addressed in this theory, and are thus further discussed in section 2.6. Belsky’s analysis of the ontogenic environment suggests that the child-caregiver relationship is likely influenced by the caregiver’s childhood relationship with their own caregivers. Intergenerational transmission of abuse is a widespread theory, though the relationship between experience of abuse and perpetration of abuse is complex, depending on parental and contextual factors.\textsuperscript{137–139} The family microsystem risk factors include characteristics of the child that, in interacting with caregiver characteristics, may make the child more vulnerable to abuse. These include low birthweight or premature birth, as well as temperamental characteristics such as hyperactivity, lethargy, or disobedience. Furthermore, family attributes such as having many family members and small living spaces have been theorized to cause stress on the family, and may interact with caregiver factors to increase the risk of CPSA. While caregiver factors are important, it must be stated that parents/caregivers are not the only potential perpetrators of child maltreatment. However, the Belsky model focuses on CPSA within families, stating that “the parent-child system is the crucible of child maltreatment”.\textsuperscript{136}

In this eco-social model the family is nested within larger social units within the community, or the exosystem.\textsuperscript{136} At this level, social isolation of the family may play a role in potentiating abuse; families without support may lack friends or extended family to aid with child care, and lack of close connections may lead to fewer “outsiders” in the home observing or monitoring caregiving practices. The workplace represents another major connection to the outside world, and workplace factors such as unemployment or lack of job satisfaction are theorized to have bearing on a caregiver’s propensity to maltreat children under their care. At the macrosystem, or societal level, national or even global events can influence factors at other levels; an example is given of a global
financial crisis causing mass unemployment, an exosystem factor relevant to CPSA. Also existing at this level are cultural attitudes towards violence and child rearing; Belsky presents cross-cultural descriptive evidence suggesting that societies where corporal punishment is not accepted have lower rates of child maltreatment.\textsuperscript{136} It is important to consider that such societal attitudes can shift dramatically over time. At the time that Belsky’s theory was published in 1980 most forms of corporal punishment were legal in North America, while as of 2004 corporal punishment was significantly sanctioned under Canadian law,\textsuperscript{140} and mass social campaigns exist with the intent of criminalizing all forms of corporal punishment.\textsuperscript{141} Furthermore, while Belsky’s ecological model represents a significant advancement in the history of CPSA research, the theories presented in this model should be further substantiated with more contemporary research.

Since the publication of Belsky’s theory, the four ecological levels have been re-conceptualized and additional risk factors have been identified. In 2002 the World Health Organization published the World Report on Violence and Health, undertaking a review of the literature on the causes and consequences of child maltreatment that have been implicated repeatedly in studies across the world.\textsuperscript{142} Rather than the first level focusing on the child-caregiver relationship, or ontogenic environment, this model focuses on factors increasing a child’s vulnerability as the initial level nested within the other three. Risk factors identified at this level include the child’s age (cases of physical abuse occur more commonly among pre-pubescent children while rates of sexual abuse generally increase after puberty),\textsuperscript{142} the child’s sex (female children are at greater risk for most forms of abuse other than harsh physical punishment)\textsuperscript{142–144}, and special characteristics of the child. Special characteristics increasing children’s vulnerability to abuse, like low birth weight or disobedience as identified in Belsky’s theory, have been expanded to include children who are twins,\textsuperscript{142,145–147} and children who have a disability or illness.\textsuperscript{142,148,149} When considering such risk factors, it is important to reiterate Belsky’s condition stating that a child cannot sufficiently cause their own abuse; it is only through interaction with risk factors at other levels that these child-level risk factors may have an effect.\textsuperscript{136} These may include attributes of the child’s caregiver and family, as well as others. In particular, the type of abuse perpetrated may be related to the sex of an abusive caregiver. For example, while physical discipline is more common among female
caregivers, sexual abuse and severe physical abuse are committed largely by male caregivers. As in Belsky’s original theory, the WHO model notes the potential importance of the caregiver’s history of abuse, family and household size, social isolation of the family, and stress. Furthering these theories, recent research has conceptualized family risk factors as resources, or lack thereof, that the family may draw on to care for the child and the family as a whole. These include caregiver education/income, age, employment status, and the number of caregivers in a household. Additionally, a lack of stability in the household (i.e. the environment changes often or people come and go frequently) is known to be related to CPSA. This risk factor speaks to the fact that CPSA is not exclusive to parents or caregivers, but may be perpetrated by others present in a child’s household and life more broadly. Finally, children have been found to be at increased risk of experiencing CPSA in households where a caregiver perpetrates intimate partner violence. Taken together, many of these caregiver and family characteristics are indicative of the reserves a family draws upon to raise and protect children, and the ways that these resources may become depleted.

In both Belsky’s theory and the updated WHO model, the child and family are nested within their community. While Belsky placed the family’s access to social support and workplace satisfaction at this level, the WHO model focuses more explicitly on poverty and social capital. Communities where many people are living in poverty, and experiencing unemployment, overcrowding, and population instability are known to be high-risk sites for CPSA. Social capital was not directly addressed in Belsky’s theory but refers to the interpersonal connectivity drawn on by a community in order to live and work together. Research has shown that social capital, as measured by neighbourhood instability (e.g. high turnover, vacancy) has been predictive of neighbourhood-level maltreatment rates. Norms, a component of a network’s social capital may also be determinants of CPSA. According to both the Belsky and WHO models, societal norms regarding violence as well as value of children can affect child maltreatment rates by positioning violence and abuse as more or less socially acceptable. While risk factors at this level have not been adequately investigated, the WHO identifies several potential social forces and resulting norms that may influence
rates of child maltreatment, including: gendered income inequality, gender roles, public policy related to children and families, the role of preventative health care and social services in identifying and preventing cases of CPSA, and social or political conflicts including war.¹⁴²

2.5.4 Intersectionality Theory

Expanding further upon the eco-social model of CPSA, Nadan et al. provided insight on how intersectionality can be incorporated into this framework.¹⁶⁸ Applied to the ecological model, intersectionality emphasizes that while children and families are nested in communities and culture, they also exist at the intersection of multiple social positions such as gender, race, ethnicity, and socioeconomic status. While children and to some extent families occupy intersectional positions, at a higher level interlocking social and structural forces affect these children and families at all eco-social levels. These forces are not limited to sexism and ageism as identified in the WHO model, but can include racism, ableism, colonialism, and others. Race has sometimes been erroneous treated as a potential risk factor in the child maltreatment literature, incorporated at the child/family levels often without addressing the mechanisms by which race may impact experiences of CPSA. Intersectionality asks researchers to consider the higher-level social and structural impacts of racism and other axes of oppression given that society systematically advantages people who are white and disadvantages those who are not. For example, while cases of CPSA involving Indigenous children are disproportionately reported to and substantiated by child protection services,¹⁶⁹ this effect has been attributed to the poverty, family disruption, and lack of access to government services often experienced by Indigenous families.¹⁷⁰,¹⁷¹ While the systematic and ongoing disenfranchisement of Indigenous peoples in Canada and other countries has not been addressed specifically in the Belsky or WHO models, multilevel intersectional thinking allows for the incorporation of these and other factors.
2.6 Theoretical Model: Eco-Social Intersectionality for Women with HIV

Building on the foundational models of eco-social theory and intersectionality, a new theoretical model is proposed to contextualize the relationship between childhood maltreatment and HIV disclosure among women with HIV in Canada. The adaptation of this model, as follows, fulfils Objective 1 of this thesis. Belsky and WHO provide us with the nested levels at which risk and protective factors for CPSA exist: child/individual, family, community, and society. Nadan has tied eco-social theory of child maltreatment to feminist intersectionality by locating these four levels within intersectionality’s “matrices of domination”. In order to adapt these existing theories to the context of the study at hand it is of interest to (1) clarify the role of culture as related to CPSA; (2) understand how intersectionality and axes of oppression operate at the four eco-social levels; and (3) address colonialism as an axis of oppression affecting Indigenous women in Canada. Finally, this model will be used as a roadmap to understand the objectives and study design for the current project.

In the past, race and cultural factors have been wrongfully treated and studied as potential risk factors for CPSA. In more recent years, efforts have been made to unpack race and culture to gain a more nuanced understanding of how not only race and culture, but systematic racism affect health and social outcomes for people of colour. It has been shown that use of broad phenotypic racial categories is the most superficial means of studying race, as race and culture as constructs are undoubtedly multidimensional. In order to understand the dimensions of race and ethnicity that are relevant to studies of CPSA, these constructs must be unpacked. Unpacking culture refers to examining specific traits associated with given cultures which have the potential to impact child maltreatment, rather than bluntly assuming that the culture itself is the cause. Examples of such traits include familism (a value elevating the importance of family bonds, often attributed to Latinx families) and ‘ohana (a similar construct in Hawaiian American families), and machismo (valuing male pride and dominance, also an aspect of Latinx culture). These values have been found to predict CPSA such that familism and ‘ohana are protective factors, while machismo represents a risk factor. When
unpacking culture into values, it is imperative to remember that no culture is monolithic and that culture, values, and race are not synonymous. Another important attribute of cultural values is their potential to be protective against CPSA. Furthermore, culture does not exist in a vacuum; as posited by intersectionality, individuals exist within larger interlocking axes of oppression.54 Racism, colonialism, and xenophobia intersect with other axes including sexism/patriarchy and ableism to affect our lives, social structures, policies, and institutions. Paired with eco-social theory, these interlocking axes can be visualized as reaching over all levels of analysis, while individuals and to some extent families occupy intersectional social positions which exist within the larger social hierarchy (Figure 2). To illustrate, hypothetical examples of given intersections and their potential impact at each eco-social level are provided in Table 4.

Table 4: Mapping intersectionality onto eco-social theory levels with examples

<table>
<thead>
<tr>
<th>Eco-Social Theory Level</th>
<th>Level applied to contextualize child abuse</th>
<th>Intersectionality at this level (^{168})</th>
<th>Hypothetical example intersection and outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Macro-system</td>
<td>Societal level</td>
<td>Societal factors are impacted by larger intersecting power differentials in the context of that society</td>
<td>Sexism (male dominance) and ageism (the value a society places on children) intersect to produce societal norms, such as the normativity of physical punishment of children</td>
</tr>
<tr>
<td>Exo-system</td>
<td>Community level</td>
<td>Community level factors are impacted by larger intersecting power differentials in the context of that community</td>
<td>Racism and classism intersect resulting in discriminatory practices within local child welfare agencies, such as a system that removes racialized children without class privilege from the home, where while children lacking class privilege would not be removed</td>
</tr>
<tr>
<td>Meso-system</td>
<td>Family level</td>
<td>As a unit, families occupy intersectional positions within a larger system of intersectional social inequality</td>
<td>Discrimination based on immigration status and racism intersect resulting in a family being socially isolated and without access to childcare</td>
</tr>
<tr>
<td>Micro-system</td>
<td>Individual (child) level</td>
<td>Individuals occupy embodied intersectional positions existing within</td>
<td>Sexism and ableism intersect resulting in increased vulnerability to sexual abuse</td>
</tr>
</tbody>
</table>
The number of social positions and their intersections at which an individual exists is virtually unknowable, as the matrices of oppression that govern our society are vast, varying, and dynamic. Furthermore, some intersectional positions are present/relevant in given contexts but non-existent in others. In a Canadian context (as well as American, Australian, and others) theories of child maltreatment are incomplete without consideration of the intersecting axes of colonialism and sexism resulting in the historic and ongoing systematic oppression of Indigenous peoples, and Indigenous women. Colonialism here refers to “Indigenous peoples’ forced disconnection from land, culture and community by another group.” When European settlers colonized Indigenous land and imposed both patriarchal gender roles and racist cultural assimilation efforts upon Indigenous communities, the position of Indigenous women was transformed. While First Nations varied in structure, Indigenous women had traditionally occupied positions of power and leadership in their communities, such as that of the Clan Mother, working in non-hierarchical balance with men in the community. After colonization, Indigenous women were harshly punished for not conforming to patriarchal European expectations of womanhood: domesticity, perceived sexual virtue, and monogamy. Formal policy was used not only to punish those who did not conform to European standards, but to indoctrinate Indigenous children with such values, alienating them from their own culture. One of the largest efforts to eliminate Indigenous family structures and values was the government-funded residential school system, operating from 1870-1996. Attendance at off-reserve residential schools was mandatory for Indigenous children aged 7-16, and parents were forced to rescind legal custody of their children to the principals of these schools. The abuse perpetrated by employees of residential schools unto children under their care took many forms, including forced abandonment of Indigenous language and beliefs, unsafe/unsanitary living conditions, as well as physical, sexual, emotional, and medical abuse. If/when children returned home outside of the school terms, the cultural fabric that bonded families together was tarnished: parents and children spoke different languages and were taught different values. Forced cultural and familial disruption, including trauma inflicted by the residential school system, is
widely regarded as the root cause of health and social inequities among Indigenous people today. Furthermore, colonial practices continue to impact the lives of Indigenous women and children, one example being the “sixties scoop” (1960s-1980s) during which Canadian authorities forcibly placed thousands of Indigenous children into the foster care system, again disrupting families and placing Indigenous children at risk. To this day, 48% of children in the foster care system are Indigenous and 54% of Indigenous children in foster care live in homes without an Indigenous foster parent.

The causes of CPSA among Indigenous people are qualitatively different from determinants for people without a history of systematic colonialism and sexism. The proposed theoretical model acknowledges that causes of CPSA within Indigenous populations stem from this ongoing history of institutionalized child maltreatment and family disruption. The mechanisms by which European colonization have affected (and continue to affect) Indigenous people living in Canada are clearly traceable, and the effects of these actions are visible today. Survivors of this legacy of cultural disruption are remarkably resilient, but carry with them the burden of intergenerational trauma. Multiple generations of familial disruption can result in cyclical generations of parents who were raised in the absence of a healthy parenting model and thus are disadvantaged when attempting to provide such a model for their own children. In particular, this cycle can include intergenerational transmission of abuse. Illustrating the overarching impact of colonization on this outcome, it is worth noting that physical discipline of children was reportedly an uncommon practice prior to implementation of the residential school system. However, as of 2008, the Canadian Incidence Study of Reported Child Abuse and Neglect reports that the rate of substantiated maltreatment investigations among Indigenous children was 5.1 times that among non-Indigenous children (noting, of course, the potential for substantiation judgements to differ for Indigenous vs. non-Indigenous children). The lasting effects of this familial disruption, institutionalized abuse and systematic disenfranchisement of Indigenous women are expected to impact Indigenous women in causal pathways not experienced by non-Indigenous women. Thus, the proposed theoretical model will include risk factors from multiple eco-social levels that acknowledge the intersectional position and experiences of Indigenous women in Canada.
Figure 2: Intersectional/eco-social model of determinants of childhood physical or sexual abuse
2.7 Current Study

A final objective of the proposed theoretical model is to use intersectionality and eco-social frameworks to understand the relationship between CPSA and HIV. Briefly, CPSA has repeatedly been implicated as a risk factor for HIV. Additionally, child maltreatment is known to affect other outcomes in adulthood (e.g. sexual relationship power, experiences of abuse, perceived social support, etc.). Among women with HIV, these outcomes hold particular significance, as they may be barriers or facilitators of HIV disclosure to sexual partners. If CPSA is a cause of HIV, as well as a cause of barriers to partner disclosure among women with HIV, abuse during childhood may represent a “double jeopardy” of sorts. By acting as a risk factor for HIV and disenabling those women who acquire HIV from disclosing their HIV status, child abuse may substantially affect both the quality of life and the socio-legal vulnerability of women with HIV in Canada. Portions of this causal chain are already well described in the literature. These include the relationship between CPSA and HIV risk, CPSA and outcomes such as experiences of abuse/disempowerment in adulthood among the general population, and the relationship between HIV disclosure and legal risk. The causal relationship that has yet to be examined is that between CPSA and barriers to disclosure among a population of women with HIV (Figure 3).
Figure 3: Sequential model contextualizing hypothesis to be tested (*)
The eco-social intersectionality framework will be incorporated in investigating this causal question. In particular, the eco-social framework will be used to identify CHIWOS variables at the level of the individual, family, community, and society which may affect risk for CPSA. In contrast to past models, variables acknowledging the effects of colonial practices such as family attendance at residential school will be incorporated. Using advanced statistical methods, the eco-social risk factors will be balanced across the group of participants with and without histories of CPSA as a method of confounding control. Furthermore, an intersectional framework will be incorporated in calculating measures of effect for the given hypothesis. This involves allowing effects to vary across population groups defined by intersecting social positions/identities that exist within larger systems of power and oppression: here, defined by gender, HIV status, and ethnoracial group. Among women with HIV, it is expected that experiences of CPSA as well as its consequences will be qualitatively different based on ethnoracial group. Thus, measures of effect for CPSA on barriers for HIV disclosure will be reported separately for Indigenous, Black African, Black Caribbean, white, and other women.

The need for this study arises from not only the documented barriers to disclosure faced by women with HIV, but also from the Canadian legal precedents which fail to protect them. Investigating the causes of disclosure barriers can create space for future interventions, shed light on the disclosure experiences of women with HIV, and highlight the lack of understanding of these experiences implicit in Canadian HIV non-disclosure policies.
Chapter 3

3 Methodology

This chapter will describe the methodology used in the study, including: study design, recruitment procedures, statistical methods, variable selection rationale, measurement/coding, and approaches to missing data.

3.1 Canadian HIV Women’s Sexual and Reproductive Health Cohort Study

3.1.1 Ethics and Funding

Ethics approval for CHIWOS was provided by the research ethics boards for Women’s College Hospital, McGill University Health Centre, University of British Columbia/Providence Health, Simon Fraser University, and recruitment sites with independent REBs. CHIWOS is funded by the Canadian Institutes of Health Research (CIHR) and the Ontario HIV Treatment Network, with support from the CIHR Canadian HIV Trials Network. In addition to institutions already mentioned, CHIWOS is supported by Women’s College Research Institute, Women’s Health in Women’s Hands Community Health Centre, and the British Columbia Centre for Excellence in HIV/AIDS.

3.1.2 Recruitment and Sampling

CHIWOS is a multi-province prospective study, collecting data at three time-points: baseline (Wave 1), 18-months (Wave 2), and 36 months (Wave 3). Originally, three provinces were selected to participate based on the relatively high estimated prevalence of women with HIV in each area: ON, QC, and BC. Wave 1 data collection for these provinces occurred from August 2013 through May 2015, while Wave 2 data were collected from June 2015 to January 2017. Wave 3 data for ON, QC, and BC started in February 2017 and is scheduled to finish in August 2018. Data from Waves 1 and 2 are included in the present analysis. Since data collection began for these provinces, a decision was made to also collect data in Saskatchewan (SK) and Manitoba (MB), based on the relatively high HIV incidence in these regions. Alternative research methods
including arts-based data collection has been used to draw inference from these provinces, and thus these data were not included in the present analysis.\textsuperscript{14} Within the selected provinces, a non-random quota sampling method was applied, starting with overall provincial quotas of \( n=700 \) for ON, \( n=350 \) for BC, and \( n=350 \) for QC. The decision to use sampling quotas was informed by the objectives to compare outcomes across health service delivery areas, and to collect data on key subgroups of women with HIV who may not be captured by traditional sampling methods.\textsuperscript{14} Provinces identified “health regions” based on existing data on HIV prevalence among women in order to set regional sampling quotas; in ON, the Ontario HIV Epidemiologic Monitoring Unit health regions were used,\textsuperscript{182} collapsing some regions with low HIV prevalence. BC based their sampling frame on Drug Treatment Program data across Regional Health Authorities. In QC, surveillance data from Institut National de Santé Publique du Québec characterized the distribution of HIV cases among women across geographic regions. Based on community and clinician expertise, as well as data availability by province, sampling quotas were also created for priority populations within provinces and sometimes within health regions. In Ontario, sampling quotas of \( n=70 \) per strata were set for priority populations including: younger women, older women, trans people, lesbian, gay, bisexual, queer, questioning, and Two-Spirit women, Indigenous women, African and Caribbean Black women, other women of colour, those not accessing care, women with a history of IDU, and women with a history of sex work. BC and QC set similar quotas for recruiting women diverse in terms of age, ethnoracial group, language, and HIV exposure category.

Recruitment procedures for CHIWOS were driven by the PRAs alongside the research team.\textsuperscript{15} Potential participants were identified through the personal networks of PRAs, community advisory board members, and other women in the community, as well as AIDS service organizations, HIV clinics, social media promotion, and posters circulated in non-HIV specific organizations such as women’s shelters.\textsuperscript{14} Screening of potential participants was done by PRAs or provincial research coordinators either in person or by phone when required, and those who met the inclusion criteria and provided informed consent were enrolled. In order to be included in CHIWOS at baseline, participants were required to (1) identify as women (inclusive of cisgender, transgender, intersex, Two-
Spirit, and gender queer or questioning women); (2) be at or over the age of 16; (3) have been diagnosed with HIV; and (4) reside in one of the included provinces. In some cases, women who met these criteria were not enrolled in the study in order to comply with the quota sampling targets. Inclusion was not restricted by language, as surveys were administered in either English or French, or with the assistance of a translator for other languages. PRAs administered the survey in person or by phone/Skype using White Label FluidSurveys™, a web-based platform allowing for automatic skip-patterns. Median time to complete the survey was 120 minutes for Wave 1, while the mean time to completion at Wave 2 was 111 minutes. Participants were compensated $50 cash after completing an interview, while PRAs were compensated $75 for every interview they conducted. Follow-up contact from one wave to the next was made by the same PRA who conducted the previous interview, by means of contact indicated by the participant. PRAs attempted contact three times, and if all were unsuccessful attempted contact through community organizations or clinics associated with the participant. Where follow-up interviews could not be conducted and reasons for loss-to-follow-up were known, these reasons were recorded in the dataset.

The Wave 1 CHIWOS questionnaire, developed with CBR methods, contained 436 items and spanned nine general content areas (Table 5). Participants had the option to skip items (response options: “don’t know” or “prefer not to answer”) or subsections (e.g. questions on residential school) as they deemed appropriate. The sections on violence and abuse and women’s sexual health contained sensitive content with the potential to invite response bias in the presence of a survey administrator, thus, these sections could optionally be self-administered rather than administered by a PRA. The Wave 2 survey included a subset of sections/items from Wave 1, with the addition of some new sections/items. Most relevant to the present study, Wave 2 collected supplementary information on immigration experiences.
Table 5: CHIWOS Survey content areas by wave

<table>
<thead>
<tr>
<th>Section</th>
<th>Wave 1 Section Topic</th>
<th>Wave 2 Section Topic</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Demographics and Socio-economic Status</td>
<td>Demographics and Social Determinants of Health</td>
</tr>
<tr>
<td>2.</td>
<td>Medical and HIV Disease Information</td>
<td>Medical and HIV Disease Information</td>
</tr>
<tr>
<td>3.</td>
<td>Health Care and Support Service Utilization</td>
<td>Health Care and Support Service Utilization</td>
</tr>
<tr>
<td>4.</td>
<td>Women’s Reproductive Health</td>
<td>Emotional Wellbeing, Resiliency, and Health Related Quality of Life</td>
</tr>
<tr>
<td>5.</td>
<td>Stigma and Discrimination</td>
<td>Women’s Reproductive Health</td>
</tr>
<tr>
<td>6.</td>
<td>Substance Use</td>
<td>Stigma and Discrimination</td>
</tr>
<tr>
<td>7.</td>
<td>Violence and Abuse</td>
<td>Substance Use</td>
</tr>
<tr>
<td>8.</td>
<td>Women’s Sexual Health</td>
<td>Violence and Abuse</td>
</tr>
<tr>
<td>9.</td>
<td>Emotional Wellbeing, Resiliency, and Health Related Quality of Life</td>
<td>Women’s Sexual Health</td>
</tr>
<tr>
<td>10.</td>
<td>N/A</td>
<td>Resilience</td>
</tr>
</tbody>
</table>

3.2 Summary of Statistical Methods

Section 3.2 introduces theory, rationale, and statistical specifications for the analytic methods applied in this study. Counterfactual theory and the corresponding potential outcomes framework are outlined as the chosen approach for addressing confounding. Following this, propensity score methodology is described, along with the chosen propensity score estimation method (generalized boosted modelling), application method (inverse probability of treatment weighting by weighted regression), and counterfactual estimand (average treatment effect among the treated). Rationale based on causal modelling and counterfactual theory is provided for variable selection, and corresponding measurement/coding information is provided.

For the present study, the exposure variable was a measure of ever experiencing childhood physical or sexual abuse (CPSA), while the outcomes were barriers to HIV disclosure to sexual partners. These included ever experiencing physical abuse in
adulthood, ever experiencing sexual abuse in adulthood, sexual relationship power, HIV stigma, and perceived social support. The causes of the exposure to be balanced across groups, referred to hereinafter as propensity score covariates, were participant age, sexual orientation, gender identity, immigration to Canada before age 16, indicator for stress-related circumstances surrounding that immigration, being a survivor of the residential school system, and having one or more family members who attended residential school. For the sensitivity analyses, child physical abuse (CPA) and childhood sexual abuse (CSA) became the exposure variables.

### 3.2.1 Counterfactual Theory & Potential Outcomes Framework

Counterfactual theory, born in philosophy, conceptualizes causation using multiple realities: one where a potential causal factor exists, and another reality identical but for the potential cause and its subsequent effects.\textsuperscript{185,186} If a given outcome occurs in the first reality where the causal factor is present, but not in the second, the factor is indeed causal. When translating this philosophical concept into empirical research we are presented with several issues, most significantly the existence of a single reality. Aiding in the explanation of empirical counterfactual thinking is Rubin’s potential outcomes framework for binary exposures.\textsuperscript{187} Within this framework, an exposure and its counterfactual opposite (i.e. lack of exposure) are denoted $T=1$ and $T=0$, respectively (Table 6).\textsuperscript{187} Each study subject has the potential to be exposed or unexposed, as well as a potential outcome under each exposure [$Y_i(1)$ denoting the outcome of an exposed individual, and $Y_i(0)$ denoting the outcome of that same individual under the unexposed condition]. Clearly, only one of these situations (exposed vs. unexposed) represents an observable reality, while its opposite represents a counterfactual reality.\textsuperscript{188} At a population level, $Y(1)$ refers to the average outcome of a group of exposed individuals, while $Y(0)$ refers to the average outcome of the same group were they unexposed.
Table 6: Rubin’s potential outcomes framework for binary exposures

<table>
<thead>
<tr>
<th>Exposure</th>
<th>Individual level</th>
<th>Group level</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$T_i=1$ for exposed individual, $T_i=0$ for unexposed individual</td>
<td>$T=1$ for a group where all individuals were exposed, $T=0$ for a group where all individuals were unexposed</td>
</tr>
<tr>
<td>Outcome</td>
<td>$Y_i$ = the outcome level (i.e. mean, proportion) for an individual</td>
<td>$Y$ = the outcome level (i.e. mean, proportion) for a group of individuals</td>
</tr>
<tr>
<td>Outcome at given exposure level</td>
<td>$Y_i(1)$ = outcome level for individual $i$ if this individual were exposed, $Y_i(0)$ = outcome level for individual $i$ if this individual were unexposed.</td>
<td>$Y(1)$ = mean outcome level for a given group, if everyone in the group were exposed. $Y(0)$ = mean outcome level for a given group, if everyone in the group were unexposed.</td>
</tr>
</tbody>
</table>

Sources: Rubin 1974 and Hirano & Imbens 2001\textsuperscript{187,189}

If we could observe the same group of people in each reality, we could calculate the difference in average outcomes between the two conditions, and thus the causal effect. Because this is impossible, the next best solution is to observe the outcomes of two groups that are exchangeable, i.e. under the same exposure conditions, group A would experience the same outcome as group B. This exchangeability of groups can be achieved by randomization, which is why randomized control trials (RCTs) represent the gold standard for causal effect estimation. Causal effects that can be estimated directly from RCTs include the Average Treatment Effect (ATE=$E[Y(1) - Y(0)]$) or the effect attributable to the exposure among the population, and the Average Treatment Effect among the Treated (ATT=$E[Y(1) - Y(0)|T=1]$) or the effect attributable to the exposure among those actually exposed. Estimation of causal effects in observational research presents another issue: other factors (potential confounders) may select subjects non-randomly into the exposed and unexposed groups, rendering them un-exchangeable. Because these groups differ based on factors other than the exposure itself, the ATE and ATT cannot be estimated directly.

In different research settings, one of the ATE or the ATT may be more meaningful than the other. Austin gives the example of providing people who smoke with a brochure, or some other intervention easily applied to the total implicated population.\textsuperscript{190} In this situation where treatment of the total population is realistic, it is most useful to base inference on the total population sampled by calculating the ATE. Conversely, if the
intervention is an intensive cessation program, it may be unrealistic to provide the treatment to all people who smoke, and thus the effect of the program on those who actually received it (ATT) is more valuable. Likewise, in a non-experimental context, it is often of interest to estimate the effect of a factor that (1) can only be studied observationally and (2) does not warrant inference for the total population based on its lack of desirability. CPSA falls into this category, where inference based on the ATT allows us to estimate the effect of counterfactually eliminating CPSA experiences among the women who actually experienced them. Because the undesirable nature of CPSA also means it should not be called a “treatment”, the ATT will hereinafter be referred to as the AEE, or average effect of the exposure among the exposed.

3.2.2 Propensity Scores

Propensity scoring is a statistical method for controlling multiple confounders in observational studies, allowing them to mimic the exchangeability of an RCT (with the understanding that residual confounding is ubiquitous in observational research). First proposed by Rosenbaum and Rubin in 1983, a propensity score (PS) represents the probability that an individual experiences a given exposure, conditional on a set of covariates preceding that exposure (Formula 1). In the case of this study, a PS will be the probability from 0-1 (exclusive) that an individual experienced CPSA, as predicted by the potential causes of child abuse. PSs can be used to control for confounding in several ways, most generally: matching on the PS, creating PS strata, regression adjusted for the PS, and PS weighting (also called model-based direct adjustment, or inverse probability of treatment weighting). Inverse probability of treatment weighting (IPTW) was selected for the present study on the basis of its superior ability to achieve covariate balance (i.e. bias reduction), greater interpretability (as compared to stratification or adjustment for the PS), and minimization of data loss (as compared to certain types of matching).

IPTW, when implemented to calculate the AEE, creates a population that is representative of the exposed group in terms of distribution of potential confounders. As demonstrated in Table 7 and Formula 2, exposed individuals receive a weight of 1, while unexposed individuals receive a weight equal to their odds of
exposure. Thus, unexposed individuals with greater odds of exposure (i.e. greater similarity to the exposure group) are weighted heavier than those unexposed with reduced odds of exposure. In the weighted sample, membership in the exposed or unexposed group is independent of the measured causes of such membership, thereby mimicking the exchangeability generated by randomization.\textsuperscript{190} Another way of stating this is that the causes of the exposure (confounders) are balanced across the exposed and unexposed groups, blocking all measured non-causal pathways between the exposure and the outcome. Thus, such weighting allows for estimation of the AEE, controlled for measured confounders. The AEE, which represents the average effect of the exposure among those actually exposed (and thus, the level of outcome that can be reduced by eliminating the exposure) is calculated using Formula 3.

Table 7: AEE weighting notation

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>$x_i$</td>
<td>Represents a given model of the propensity score covariates for individual $i$</td>
</tr>
<tr>
<td>$\text{PS}_i$</td>
<td>Actual (theoretical) propensity score for individual $i$</td>
</tr>
<tr>
<td>$\hat{\delta}(x_i)$</td>
<td>Propensity score for individual $i$, estimated by model of propensity score covariates</td>
</tr>
<tr>
<td>$w_i$</td>
<td>Inverse probability of treatment weight for individual $i$</td>
</tr>
<tr>
<td>$\tau_t$</td>
<td>Average treatment effect among the treated (AEE)</td>
</tr>
<tr>
<td>$\tau_{t,w}$</td>
<td>Average treatment effect among the treated (AEE) calculated using IPTW</td>
</tr>
<tr>
<td>$N_t$</td>
<td>Number of individuals in the exposed group</td>
</tr>
<tr>
<td>$i \in T$</td>
<td>The $i$th observation in the exposed group</td>
</tr>
<tr>
<td>$i \in C$</td>
<td>The $i$th observation in the unexposed group</td>
</tr>
</tbody>
</table>

Sources: Hirano & Imbens 2001, Hirano, Imbens, & Ridder 2003, McCaffrey, Ridgeway, & Morral, 2004\textsuperscript{189,198,199}

\[
PS_i = \hat{\delta}(x_i) = P(T_i = 1|x_i = x) = \frac{\exp(x_i)}{1 + \exp(x_i)}
\]

Formula 1: Propensity score for individual $i$

\[
w_i = T_i + (1 - T_i) \frac{\hat{\delta}(x_i)}{1 - \hat{\delta}(x_i)}
\]

Formula 2: Inverse probability of treatment weight for the AEE, individual $i$
\[ \tau_{t,w} = \frac{\sum_{i \in T} y_i}{N_t} - \frac{\sum_{i \in C} w_i \cdot y_i}{\sum_{i \in C} w_i} \]

**Formula 3: AEE calculated using IPTW**

Estimates of treatment effects based on IPTW will be essentially unbiased upon meeting several assumptions: (1) each participant has the potential to experience either exposure; (2) the stable unit treatment value assumption holds; and (3) the outcome is independent of the exposure after accounting for differences in the pre-exposure PS variables (i.e. exchangeability is achieved). Assumption 1 is satisfied in that no child is exempt from vulnerability to maltreatment. With regard to item 2, we make the assumption that any given participant’s outcome is not influenced by another participant’s history of CPSA. This assumption seems reasonable, except in the situation that multiple participants were raised in the same household and/or are part of the same family. Given that CHIWOS does not collect household or family-level data, the actual risk of peer effects is not estimable, however is presumed to be negligible. Assumption 3, which maps onto counterfactual theory/exchangeability, can be addressed with extension to causal modelling. Briefly, conditioning the exposure on all of its potential causes blocks all non-causal paths which may lead to exposure-outcome confounding. It is this capability which makes PS analyses particularly well-suited to studies with a single exposure and multiple outcomes. While theoretically the causes of the exposure create a sufficient set, in practice, the ability to measure and operationalize a sufficient set of all causes becomes less realistic and the selection of variables which cause not only the exposure but also the outcome becomes more important. Inclusion of these “true confounders” has been shown to result in improved PS overlap and reduced mean squared error when compared to conditioning the PS only on variables related to treatment allocation. For this reason, PS variable selection was implemented by identifying the ecological causes of CPSA, and within these causes identifying variables available from the CHIWOS dataset with an eye towards non-mediating causes of study outcomes (see 3.3.3 Propensity Score Covariates).
3.2.3 Generalized Boosted Modelling for Propensity Score Estimation

Traditionally, PSs are estimated using a logistic regression model with the exposure as the dependent variable, and its causes as the independent variables. In creating the scores, researchers are encouraged to iteratively re-specify the model (adding interaction terms and/or higher order functions) until an optimal covariate balance is reached. Ideally, weighting participants using the given scores would result in the exposed and unexposed groups having effectively the same distribution of the covariates, thereby controlling their effects. However, considering that PS modelling is equipped and even intended to accommodate a large number of covariates, the re-specification process can be tedious. The traditional logistic regression method for PS estimation also specifies a linear relationship between covariates and the logit of the PS, an assumption which may or may not be satisfied. Furthermore, PS estimates based on logistic regression models can be unstable, generating extreme weights and imprecision.

Several PS modelling techniques have been developed in response to these issues, including Generalized Boosted Modelling (GBM), proposed in the context of PSs by McCaffrey, Ridgeway, and Morral in 2004. GBM is a nonparametric, machine-learning method that fits many multivariate models of a dependent variable which are combined to produce a probability estimate, such as a PS. The method proposed by McCaffrey and colleagues uses regression trees which algorithmically partition the dataset based on covariate splits which minimize prediction error for the PS. “Boosting” generally refers to an algorithm which adaptively combines weakly predictive models to produce a final model that is more predictive than those that it is derived from. GBM for PS modelling works by modelling the log-odds of treatment assignment (i.e. log-odds of actually experiencing CPSA) denoted g(x), then searching for an adjustment to the model, h(x), that improves model fit. H(x) is a regression tree where the residual error of current model fit is the dependent variable and independent variables that cause the exposure variable are the covariates. In the case that the regression tree improves model fit (i.e. that particular covariate split minimizes residuals), the model for the log-odds of treatment assignment becomes $g(x) + h(x)$. 
This process of selecting and incorporating sub-models that improve model fit happens iteratively, however, because the overall model changes with the addition of each sub-model \([g(x) + h(x) + \ldots h(x)]\), the dependent variable for each new sub-model (i.e. the residuals of the new overall model) is also different. In plain language, this means that with each iteration, the sub-model selection criteria adapt to improve overall model fit. The data-adaptive nature of this method means it is a sequential ensemble method that learns with each iteration, standing in contrast to parallel ensemble methods such as bootstrap-aggregating (“bagging”) which fit many sub-models at once to generate an overall average model.

GBM has been demonstrated to outperform traditional logistic regression models, as well as other ensemble specification methods, especially when estimating the AEE by IPTW. In a comparative study by Harder et al., GBM with IPTW was the only method of nine competitors (logistic regression, logistic regression with iterative inclusion of covariate interactions, and GBM each applied using 1:1 matching, full matching, and IPTW) which achieved adequate covariate balance for estimation of the AEE. GBM has also outperformed other ensemble methods such as bagged classification and regression trees as well as random forests in terms of covariate balance, confidence interval coverage, and tendency to produce extreme weights. For these reasons, GBM was selected as the method of PS estimation. In the present analysis, GBM was operationalized using RAND Corporation’s toolkit for weighting and analysis of non-equivalent groups (“TWANG”) macro for SAS. Use of this macro required installation and background use of R software version 3.4.3. CPSA was specified as the binary exposure variable across which PS covariates would be balanced (Section 3.4.3). Per the recommendations of Ridgeway et al., the maximum number of iterations (i.e. classification trees) was specified as \(n=5000\) in order to allow ample space for model optimization. Also per recommendations, up to 3-way interactions were allowed within models. In order to avoid overfitting, the shrinkage parameter was specified at 0.01, striking a balance between an overly smooth model with many iterations (i.e. over-fit) and a more “jagged” model which does not adequately fit the data. Two types of balance criteria were applied to evaluate the optimal iteration; these are also called “stopping rules”. The iterations which minimized the absolute
standardized mean difference (or effect size, ES), and the mean Kolmogorov-Smirnov (KS) statistic for PS variables, respectively were identified. The standardized ES identifies differences in covariate balance across the CPSA+ and CPSA- groups while the KS test identifies differences in covariate distribution. Because the KS stopping point minimized the mean and maximum ES and KS statistics equally or in some cases better than the alternative stopping point, the KS mean stopping point was selected.

3.2.4 Propensity Score Overlap and Balance Diagnostics

In order for inferences based on PS weighting to be valid, the PS distributions for the exposed and unexposed groups must overlap, and the weighting must demonstrably balance measured covariates across these groups. In order to avoid potential off-support inference, PS distributions were evaluated by comparing box plots for the CPSA+ and CPSA- groups. Balance diagnostics, which show whether weighting successfully balanced covariates across exposure groups, were evaluated as suggested in Austin and Stuart’s best practice guidelines. Specifically, Wald Chi Square tests, t-tests and KS tests (where applicable) of the PS covariates across exposure groups were compared before and after weighting. These tests were conducted for the distribution of covariates across CPSA for the total analytic sample as well as within each ethnoracial group in order to identify potential stratum-specific confounding. The TWANG macro also creates indicator variables for the level of missingness on each PS covariate, allowing users to achieve and evaluate balance on covariate missingness. Balance of covariate missingness was evaluated in the same way as covariate balance.

3.2.5 Outcome Analysis

Several approaches exist for outcome analysis using IPTW with AEE weights, including direct estimation using weighted means, and regression modelling. There are also several different approaches for calculating the standard error and confidence intervals for these estimates. Ideally, calculation of outcome variance should account for variability in the original propensity score model. However, for GBM, as for other propensity score estimation methods, such a method has not been proposed. Solutions to account for this excess variance include estimating bootstrap or jackknife confidence intervals, or using
robust standard errors. Additionally, because outcome estimates are weighted, precision calculations must account for the potential increase in variance due to weighting. Joffe and colleagues suggest that regression models that accommodate weighting, along with robust variance estimators can accomplish this. This direction combined with the desire to estimate prevalence ratios rather than odds ratios for binary outcomes led to the decision to use modified Poisson regression with robust variance estimators, weighted for the AEE, implemented using PROC GENMOD as per Sato and Matsuyama. For continuous outcomes, PROC GENMOD with robust variance estimation and AEE weights was also implemented, but specifying a normal distribution. SAS code is available in Appendix 2.

In order to account for potential moderating effects differentiating key populations of women with HIV, the AEE for all outcomes was calculated for the total population (Objective 2), as well as within 5 ethnoracial strata: Indigenous, Black African, Black Caribbean, white, and “other” women. Then, interaction terms between CPSA and ethnoracial group were used to compare effects across subgroups, with white women as the reference group (Objective 3). When comparing effects across subgroups, confidence intervals that did not cross the null value were interpreted as evidence of effect modification. This subgroup analysis applied the propensity scores and corresponding weights calculated using the total sample, rather than generating unique propensity scores for each subgroup. Scores derived from this approach are termed cohort propensity scores, in contrast to subgroup propensity scores. This decision is justified based on prior studies which found that using cohort propensity scores for subgroup analysis resulted in effect estimates not significantly different from those obtained with subgroup-specific propensity scores, with some variation where subgroups were small. Cohort scores have also performed similarly to subgroup scores across multiple conditions when evaluated on covariate balance, bias, and precision. There is still debate as to the validity of cohort scores as compared to subgroup scores; however, the literature does not provide sufficient evidence to recommend one approach over the other. In order to ensure that the cohort propensity score weights balanced covariates across exposure groups in the overall sample and within ethnoracial groups, traditional balance diagnostics were supplemented by stratum-specific balance diagnostics.
3.2.6 Sensitivity Analysis

In order to test the assumption that CPA and CSA have sufficiently similar effects to use the composite outcome of CPSA, two alternative sets of propensity scores were generated for sensitivity analysis: one predicting CPA, and the other predicting CSA. The methods for generating and applying these scores were identical to the methods described for CPSA, however, scores for CPA included CSA as a covariate and vice-versa. Controlling the effect of each variable on the other allowed for the identification of CPSA effects that were potentially driven by CPA or CSA only.

3.2.7 Temporality

While the CHIWOS project has collected two waves of cohort data, it is important to note that the present analysis represented a cross-sectional approach with an aspect of nested temporality. Because women were asked retrospectively about their experiences of CPA and CSA including an age cut-off of 16, any reported abuse could be attributed to the time period prior to outcome measurement. Additionally, variables selected as PS covariates were those known to occur or be potentially identifiable prior to that same age cut-off. Thus, while it is known that covariates precede outcome measurement, it is possible that some covariates measured factors occurring after the exposure. Efforts to address this limitation are addressed in 3.3.3. While the present analysis applies a causal framework and epidemiological method, the inherent limitations of cross-sectional data should be considered when interpreting the results.

3.3 Study Variables

3.3.1 Outcomes

Outcomes were identified using a literature review of qualitative and quantitative research documenting determinants of partner HIV status disclosure among women. Briefly, these barriers included factors related to current or past relationships (trust, dependence, security, abuse), internal factors (HIV stigma) as well as expectations of the effects of partner disclosure (perceived social support, expected discrimination, abuse). These factors were then mapped onto similar variables available in the CHIWOS dataset:
sexual relationship power, history of physical abuse in adulthood, history of sexual abuse in adulthood, perceived HIV stigma, and perceived social support. Because sexual relationship power was only measured among those women who indicated they had been sexually active in the past month (Wave 1) or the past 6 months (Wave 2), the relationship between CPSA and sexual inactivity was examined. This outcome was intended to add to the interpretability of sexual relationship power, rather than to represent a barrier to HIV status disclosure.

3.3.2 Exposure Variable

The decision to examine the effect of childhood physical or sexual abuse rather than the independent effects of each was based on theoretical and practical considerations. Prior research suggests that psychological outcomes among adults with a history of abuse during childhood do not vary greatly by the nature of the abuse. Furthermore, physical and sexual abuse during childhood (CPA, CSA) often co-occur, making effects statistically difficult to tease apart. However, in the interest of testing this assumption to the best of our abilities, effects were reported for PS models predicting CPSA (main analysis), as well as models predicting CPA and CSA (sensitivity analyses).

3.3.3 Propensity Score Covariates

As specified by the statistical analysis plan, the PS estimation variables for this study were causes of CPSA, especially causes of CPSA which may also be causes of the outcomes. Furthermore, PS estimation variables should not be mediators along the causal pathway. A literature review of causal theories for CPSA was conducted, leading to the integration of eco-social and intersectional theoretical models for the context of Canadian women with HIV (2.6). Using this model, causes of CPSA at the level of society, community, family/caregiver, and child were identified. Within these domains, causes of vulnerability available from the CHIWOS dataset that could identifiably have an effect during childhood were identified. These variables included: age as an indicator for society’s shifting acceptability of child abuse over time, sexual orientation, gender identity, immigration to Canada at or before the age of 16, stress-related immigration circumstances among those who immigrated to Canada in childhood, unstable or refugee
housing prior to immigration in childhood, being adopted, spending time in foster care or a group home, being a survivor of residential school, and having family members who attended residential school. These factors span all eco-social domains identified in the theoretical model. Within this set of variables, it was necessary to remove any variable which could be caused by CPSA (i.e. potential mediators). Because foster care, group home residence, and adoption are so often consequences of CPSA, a decision was made to not include these variables in the PS model. Furthermore, the variable related to living in unstable or refugee housing prior to immigration in childhood was excluded based on low prevalence (0.15%) and thus low potential impact.

Based on the condition that all variables in a PS model should precede the exposure of interest, some may question the inclusion of sexual orientation and gender identity in this study. Arguments on the etiology of sexual orientation and gender identity aside, this decision was made based on evidence demonstrating that adults who identify as a sexual orientation other than heterosexual and/or a gender that doesn’t align with the sex assigned to them at birth retrospectively report elevated rates of CPSA compared to their heterosexual/cisgender peers. While opinions on the cause of this relationship vary, it is theorized that whether or not an individual is aware of their sexual orientation/gender identity during childhood, perceived atypicality with respect to gender/sexual norms during childhood can result in increased targetability for abuse. In fact, signs of gender atypical behaviour during childhood have been associated with onset of verbal and physical abuse from both parents and peers. This ties into the eco-social framework which identifies individual-level markers that may increase likelihood of maltreatment, though only in the presence of risk factors at other levels (i.e. children are not responsible for their own abuse).

3.4 Measures

3.4.1 Outcomes

All outcomes were measured at both Wave 1 and Wave 2, with the exception of social support which was measured at Wave 1 only. Because the measures for physical and sexual abuse in adulthood allowed for collapsibility across waves, single “ever-
experienced” variables are used for these outcomes. Where measures were available from both waves but not collapsible as with physical and sexual abuse, the Wave 1 measures were used on the basis of larger sample size. An exception to this rule was the sexual relationship power variable, which was only asked of women who indicated that they had been sexually active in the past month at Wave 1. An unexpectedly high prevalence of sexual inactivity at Wave 1 prompted the decision to push the referent time frame for sexual activity to the past 6 months in the Wave 2 questionnaire. In addition to potentially better validity (this measure can be answered hypothetically for those who are not currently sexually active) this decision resulted in a greater number of non-missing responses at Wave 2 rather than Wave 1 (n=486 and n=440, respectively). For these reasons the sexual relationship power measure (and the corresponding measure of sexual inactivity, for interpretability) were based on Wave 2 outcome data.

**Physical Abuse During Adulthood (Ever)**

Experience of physical abuse during adulthood was measured using two yes/no items: “As an adult, has someone ever physically hurt you?” (Wave 1) and “Since your last CHIWOS interview, has someone physically hurt you?” (Wave 2). Participants who reported abuse in either interview were coded “yes” while participants who reported no abuse in both interviews were coded “no”. Where Wave 1 was missing and no physical abuse was reported in Wave 2 (n=1), the participant was coded as missing as the Wave 2 item does not capture “ever” abuse, which is the construct of interest. Conversely, where Wave 1 was missing but physical abuse was reported in Wave 2 (n=1), the participant was coded “yes” since any abuse is “ever” abuse. When Wave 2 responses were missing, Wave 1 responses were coded, and if both waves were missing, the final variable was also missing. The amount of missing information for this variable in the analytic sample was 0.08%.

**Sexual Abuse During Adulthood (Ever)**

Experience of sexual abuse during adulthood was measured using two yes/no items: “As an adult, has someone ever sexually forced themselves on you, or forced you to have sex?” (Wave 1) and “Since your last CHIWOS interview, has someone sexually forced
themselves on you, or forced you to have sex?” (Wave 2). Participants who reported abuse in either interview were coded “yes” while participants who reported no abuse in both interviews were coded “no”. If Wave 1 was missing but no sexual abuse was reported in Wave 2 (n=11), the participants were coded as missing as the Wave 2 item does not capture “ever” abuse, which is the construct of interest. No participants who were missing Wave 1 reported experiencing sexual abuse at Wave 2. When Wave 2 responses were missing, Wave 1 responses were coded, and if both waves were missing, the final variable was also missing. The amount of missing information for this variable in the analytic sample was 0.92%.

Sexual Relationship Power

The Sexual Relationship Power Scale (SRPS) was developed by Pulerwitz, Gortmaker, and DeJong in 2000 in order to test the hypothesized relationship between low sexual relationship power and condom efficacy among women. The SRPS consists of two subscales: relationship control (Cronbach’s alpha=0.86) and decision-making dominance (Cronbach’s alpha=0.62), using metrics from the original validation study. The regularly weaker internal consistency as well as questionable construct validity of the decision-making dominance subscale has led some researchers (including the CHIWOS team) to use only the relationship control subscale. The relationship control subscale has 15 items, focusing on sexual violence and risk as well as other aspects of power including the amount of control a sexual partner has over the participant’s day-to-day life. In CHIWOS, the SRPS was preceded by the statement: “The following questions ask about your relationship with your partner. If you currently have more than one sexual partner, please think about the person you consider your primary sexual partner”. Furthermore, the partner pronouns from the original scale were changed to “s/he” in order to account for non-heterosexual relationships. A four-point Likert scale including “strongly agree,” “agree,” “disagree,” and “strongly agree” are the response options, with agreement to statements corresponding to lower scores and thus lower sexual relationship power in the summed score. Summed scores were divided by the number of non-missing items, with participants missing ≥ 20% of items (≥ 3/15) marked as missing (867/1307 missing at Wave 1, 821/1307 missing at Wave 2). While traditionally the SRPS has been
recoded into a categorical variable with three tertiles indicating low, medium, and high sexual relationship power. These are not validated cutpoints and thus may not represent meaningful differences. For this reason, the SRPS was treated as a continuous scale, with scores ranging from 1 to 4.

**Sexual Inactivity**

As previously discussed, sexual inactivity as a screening question for the SRPS was measured with different referent time frames from Wave 1 to Wave 2. At both waves, participants who indicated they would complete the Women’s Sexual Health section were asked “Have you had consensual sex in the past 6 months? This includes any type of sexual intercourse you willingly engaged in, including getting or giving oral sex, vaginal sex, and/or anal sex with people of any gender” with response options: yes, no, and prefer not to answer. In the Wave 1 questionnaire only, participants were also prompted with “[t]hese next questions are in regards to your sexual activity in the past month. Have you been sexually active during the past month?” and the same response options. Participants deemed sexually active using the first item answered the SRPS at Wave 2, and participants deemed sexually active using the second item answered the SRPS at Wave 1, yielding the smaller sample and leading us to focus on the Wave 2 data. Thus, those who answered no to item 1 in Wave 2 were coded as “sexually inactive”, with this binary item being used to enhance the interpretability of the SRPS at Wave 2. The amount of missing information for this variable in the analytic sample was 6.2%.

**HIV Stigma**

HIV stigma was measured using Wright’s ten-item version of the forty-item HIV Stigma Scale, which was originally developed by Berger and colleagues. Both the original and abbreviated scales contain four subscales: personalized stigma (fear of rejection), disclosure concerns, negative self-image, and concerns related to public attitudes about people with HIV. Similarly, both versions of the scale demonstrated good internal consistency and construct validity for subscales as well as the total scale. Response options are on a five-point Likert scale (strongly agree, agree, neither, disagree, strongly disagree) with stronger agreement corresponding to higher scores. Items were summed
resulting in final scores ranging from 0 to 40. As with other continuous variables, participants missing ≥ 20% of items (i.e. ≥2/10 items) were excluded from analysis of this outcome (missingness rate of 1.15%).

**Perceived Social Support**

Perceived social support was captured using a shortened four-item version of the Medical Outcomes Study Social Support Scale (MOS-SSS).\textsuperscript{119,230} The abbreviated MOS-SSS has been shown to have comparable internal consistency, test-retest reliability, and construct validity to the original 18-item scale.\textsuperscript{230} Each of the four items taps a different domain of social support as identified in factor analysis of the original scale: emotional-informational support, tangible support, affectionate support, and positive social interaction.\textsuperscript{119,230} In CHIWOS, the abbreviated MOS-SSS items are preceded by the statement: “People sometimes look to others for companionship, assistance, or other types of support. How often is each of the following kinds of support available to you if you need it?” and followed by a five-point Likert scale: all, most, some, a little, or none of the time. Greater frequency of support corresponds to higher scores, which are summed to create the final score which ranges from 4 to 20. As with other continuous measures, participants missing ≥ 20% of items (i.e. ≥1/4 items) were excluded from analysis of this outcome (missingness rate of 3.29%). The abbreviated MOS-SSS was only asked in the Wave 1 questionnaire.

### 3.4.2 Explanatory Variables

**Childhood Physical or Sexual Abuse**

Experience of physical or sexual abuse during childhood was assessed using two yes/no items broadly defining each type of abuse: “During your childhood, did an adult ever physically hurt you?” and “During your childhood, did someone ever sexually force themselves on you, or forced you to have sex?” Prior to asking these questions PRAs informed participants that here “child” is defined as less than 16 years old. PRAs could also further define physical abuse, stating “in some cultures, physical discipline of children is common; for our purposes, we are including such physical discipline” as well
as sexual abuse, stating “this can include the fondling of your private parts, oral sex, vaginal sex, and anal intercourse. It can be either forced or with your consent because you feared the consequences of resisting the person”. These items were only asked in Wave 1. A single variable was created using these two items: participants who answered yes to either question were coded as having experienced CPSA, while participants who no to both were coded as not experiencing CPSA. Those missing one item (n=19) were coded according to their response on the other, while those missing both items were excluded from the analysis, yielding an analytic sample size of n=1307/1422.

Childhood Physical Abuse (for sensitivity analysis)

CPA was coded as a binary variable using only the physical abuse item from the CPSA measure. The analytic sample for the CPA sensitivity analysis included the n=1311/1422 participants for which the CPA variable was non-missing.

Childhood Sexual Abuse (for sensitivity analysis)

CSA was coded as a binary variable using only the sexual abuse item from the CPSA measure. The analytic sample for the CSA sensitivity analysis included the n=1291/1422 participants for which the CSA variable was non-missing.

Ethnoracial Group

Participants self-identified their racial and/or ethnic background on an extensive “check all that apply” list including: Aboriginal person living in Canada, Indigenous Person from a country outside of Canada, Black African, Black Caribbean, Black Other, Caucasian/White, Chinese of Taiwanese, Filipino, Japanese, Korean, Latin American, South Asian, Southeast Asian, Arab, West Asian, Central Asian, Multiple races / Multiracial / “Mixed”, or “Other, specify”. Any participant who reported identifying as an Indigenous person in Canada was classified as Indigenous (n=289), while participants who identified as Black African only or Black African and Black Other were classified as Black African (n=302). Similarly, those who identified as Black Caribbean only or Black Caribbean and Black Other were classified as Black Caribbean (n=64). Participants who selected only Caucasian/white were classified as white (n=549). The “other” category
(n=103) included participants who did not fall into the parameters above, including multiracial individuals, those who selected single ethnoracial groups for which the sub-sample was not large enough to constitute its own strata, and 6 participants who selected “Black, Other” with no additional Black ethnoracial group. This coding resulted in no participants missing data on race.

Because of within-group heterogeneity, results pertaining to the “other” group were not interpretable in any way other than to serve as a call for further research. To emphasize this necessity and remain accountable to the women from under-represented ethnoracial groups who participated in CHIWOS, results for the “other” group were reported alongside those for the four main ethnoracial groups, and of course included in effect estimates for the total sample. In order to provide some insight into the individuals who constituted the “other” group, Table 8 provides a breakdown of their ethnoracial identities.
Table 8: Ethnoracial groups identified by the women classified in the "other' ethnoracial group (n=103)

<table>
<thead>
<tr>
<th>Ethnoracial group</th>
<th>Percentage (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Indigenous person living in Canada (e.g., First Nations, Métis, Inuit)</td>
<td>0</td>
</tr>
<tr>
<td>Indigenous person from a country outside of Canada</td>
<td>0</td>
</tr>
<tr>
<td>Black African (e.g., Nigerian, Somali)</td>
<td>5.83% (6)</td>
</tr>
<tr>
<td>Black Caribbean (e.g., Haitian)</td>
<td>1.94% (2)</td>
</tr>
<tr>
<td>Black Other (e.g., Black Canadian)</td>
<td>5.83% (6)</td>
</tr>
<tr>
<td>Caucasian/White</td>
<td>13.6% (14)</td>
</tr>
<tr>
<td>Chinese or Taiwanese</td>
<td>3.88% (4)</td>
</tr>
<tr>
<td>Filipino</td>
<td>2.91% (3)</td>
</tr>
<tr>
<td>Japanese</td>
<td>5.83% (6)</td>
</tr>
<tr>
<td>Korean</td>
<td>3.88% (4)</td>
</tr>
<tr>
<td>Latin American (e.g., Chilean, Costa Rican, Mexican)</td>
<td>20.4% (21)</td>
</tr>
<tr>
<td>South Asian (e.g., Indian, Bangladeshi, Pakistani, Punjabi, and Sri Lankan)</td>
<td>13.6% (14)</td>
</tr>
<tr>
<td>Southeast Asian (e.g., Cambodian, Laotian, Malaysian, Vietnamese)</td>
<td>5.83% (6)</td>
</tr>
<tr>
<td>Arab (e.g., Egyptian, Kuwaiti, and Libyan)</td>
<td>5.83% (6)</td>
</tr>
<tr>
<td>West Asian (e.g. Iraqi, Isreali, Lebanese, Afghani, Iranian)</td>
<td>2.91% (3)</td>
</tr>
<tr>
<td>Central Asian (e.g., Kazakhstan, Kyrgyzstan, Tajikistan, Turkmenistan)</td>
<td>0</td>
</tr>
<tr>
<td>Multiple races / Multiracial / “Mixed”</td>
<td>34.0% (35)</td>
</tr>
</tbody>
</table>

Participants could select all ethnoracial groups that applied and thus percentages do not sum to 100%.

3.4.3 Propensity Score Covariates

Age

Age was measured continuously in years, subtracting the participant’s date of birth from the baseline interview date. No participants in the analytic sample were missing data on age.
Sexual Minority

Sexual orientation was measured by self-identification, using the item “With respect to your sexual orientation, how do you currently identify?” and a check-all-that-apply list including Heterosexual/Straight, Lesbian, Gay, Queer, Bisexual, Two-Spirited, Questioning, and Other, specify. Participants who only selected heterosexual/straight were coded as the reference category, while participants who selected any other identity alone or in combination with each other or heterosexual were coded as sexual minorities. Write-in responses without accompanying checkbox responses were coded into these two categories, with two participants identifying other sexual orientations coded as sexual minorities. Five participants endorsed “don’t know” or “prefer not to answer” for this item, and were marked missing. The amount of missing information for this variable in the analytic sample was 0.38%.

Gender Minority

Gender identity was measured by self-identification using the items related to current gender identity: “With respect to your gender, how do you currently identify?” and sex assigned at birth: “What was your biological sex at birth?”. Response options for gender identity included Woman, Trans Man (Female to Male), Trans Woman (Male to Female), Two-spirited, Intersex, Gender Queer, and Other, specify. Response options for sex at birth included “Male, Female, Intersex, Undetermined, and Other, specify. Respondents who identified as trans men in the gender identity item were not included in the study as per the inclusion criteria. Participants who were assigned female at birth and identified as women were coded as cisgender (reference group), while participants who selected any other combination of biological sex and gender identity were classified as gender minorities. There was no missing data for this variable in the analytic sample.

Immigrating to Canada at or before age 16

Immigration to Canada at or before age 16 was measured using items on year and country of birth, and year of immigration to Canada. Among participants born outside Canada, age at immigration was calculated by subtracting year of birth from year of immigration,
with those who immigrated after age 16 being classified as the reference group. Non-immigrants were also placed in this reference group, creating a binary indicator variable for those who came to Canada at or before age 16. Participants who immigrated but were missing information on year of immigration were marked as missing. The amount of missing information for this variable in the analytic sample was 0.69%.

**Stress-related immigration circumstances among those who immigrated to Canada at or before age 16**

Among those who immigrated to Canada at or before age 16, stress-related immigration circumstances were coded using the item “What were your reason(s)/your family’s reasons for immigrating to Canada?”. If participants selected 1 or more stress-related reason (Living conditions, Escape socio-political conditions in home country: political persecution, Persecution as a member of a sexual minority group or because of sexual orientation, Religious persecution, Conditions of war, slavery, or forced labour, or Domestic violence/intimate partner violence) and immigrated at or before 16 they were coded as 1, while others, non-immigrants, and those who immigrated after made up the reference group. The amount of missing information for this variable in the analytic sample was 4.44%.

**Survivor of the residential school system**

Participants who indicated that they were Indigenous and who agreed to answer questions about residential school were asked whether they ever attended one of these institutions. Participants who answered yes were coded as one, while those who did not attend or were not Indigenous constituted the reference group. Indigenous participants who declined to answer this section (n=123) or item (n=42) were marked missing. The total amount of missing information for this variable in the analytic sample was 12.6%.

**Family member(s) who attended residential school**

Similarly, Indigenous participants living in Canada who agreed to answer questions about residential school were asked whether their mother, father, maternal grandmother, maternal grandfather, paternal grandmother, paternal grandfather, or any siblings had
ever attended residential school. Those who had at one or more family member attend were classified accordingly, while those with no family members who attended or were not Indigenous were the reference group. Participants who declined to answer this section (n=123) or item (n=17) were marked missing. The total amount of missing information for this variable in the analytic sample was 10.7%.

3.5 Missing Data

3.5.1 Outcomes

In the analytic sample, outcome missingness ranged from 0.08% to 3.29%, with the exception of the SRPS which was considerably higher (62.8% at Wave 2). Because missing data for outcomes other than the SRPS was low and missing data for the SRPS was derived almost entirely from a known survey design feature for which further interpretation is provided (i.e. effect of CPSA on sexual inactivity), analyses were conducted on the available data for each outcome.

3.5.2 Explanatory Variables

The analysis was restricted to the 1307/1422 cases where the exposure variable (CPSA) was non-missing, as calculation of the propensity score depends on valid exposure data.

3.5.3 Propensity Score Covariates

Missingness for propensity score covariates ranged from 0% to 12.6% in the analytic sample. As addressed in 3.2.4, the generalized boosted modelling algorithm creates missingness indicators for each propensity score covariate, and balances missing data across exposure groups as it does for non-missing data.
Chapter 4

4 Results

This chapter will provide the results of IPTW using GBM in the form of propensity score overlap and balance diagnostics. It will then describe the unadjusted differences in analytic variables observed across ethnoracial strata. With respect to the analytic results, the propensity score analysis will provide AEE estimates (prevalence ratios, regression coefficients, and robust 95% confidence intervals) in tables and figures for the effect of CPSA on barriers to HIV disclosure among women who experienced CPSA. AEE estimates will be compared across ethnoracial groups. Finally, the results of the sensitivity analysis testing the effect of CPA and CSA on disclosure variables will be provided.

4.1 IPTW using GBM

The GBM algorithm with a KS mean stopping point successfully balanced all propensity score covariates and their missing values at 784 iterations for the main CPSA analysis (see Appendix 3 for the optimization plot). The propensity scores generated by the algorithm, while higher among women who experienced CPSA (as expected), did overlap (see Appendix 4 for side-by-side box plots of propensity scores for CPSA+ and CPSA- groups). This indicated that the weights derived from these scores would produce minimal off-support inference, and could be applied in analysis. The same was true for both the CPA and CSA sensitivity analyses, which achieved covariate balance at 1767 and 1889 iterations, respectively.

4.2 Balance Diagnostics

Covariate balance in the total analytic sample:

As displayed in Table 9, there were several significant differences between women with and without a history of CPSA prior to weighting. These differences rendered the unweighted groups incomparable with respect to measured confounders. As expected, women who experienced CPSA were more likely to report a minority sexual orientation
or a minority gender identity \((P=0.003)\). While the proportion of women who were survivors of residential school did not reach statistical significance, the proportion of women who declined to answer items on self-attendance at residential school was greater among women who experienced CPSA \((P=0.001)\). Women who experienced CPSA also were significantly more likely to have one or more family members who attended residential school \((P<.0001)\) and to decline to answer items on family attendance at residential school \((P=0.005)\). While no statistically significant difference was observed in the mean age of women in both groups, a Kolmogorov-Smirnov test (for continuous variables) did detect a marginally significant difference in the distribution of age across the two groups \((P=0.062)\). Other imbalances approached statistical significance, such as the difference across exposure groups in proportions of women reporting that they immigrated at or before age 16 \((P=0.101)\), or experienced stress-related circumstances surrounding that immigration \((P=0.106)\). In both cases, the outcome was more common among women who experienced CPSA.

As displayed in Table 10, no statistically significant differences remained across the weighted exposure groups. Thus, weighting rendered the groups comparable with respect to measured confounders. In addition to all variables and their missingness indicators rising above the significance cut-off of \(\alpha = 0.05\), the majority of these variables (even those that were not significant before weighting) had \(P\)-values much higher than those prior to weighting. For example, experiencing stress related to immigration was approaching significance at \(P=0.106\) prior to weighting, but any differences could be definitively ruled out after weighting \((P=0.993)\).
Table 9: Covariate balance, unweighted

<table>
<thead>
<tr>
<th>Variable</th>
<th>Measure</th>
<th>CPSA- N=488</th>
<th>CPSA+ N=819</th>
<th>Standardized effect size difference</th>
<th>P-value *</th>
<th>P-value (KS-test)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>x (SE)</td>
<td>42.9 (0.53)</td>
<td>42.7 (0.35)</td>
<td>-0.025</td>
<td>0.689</td>
<td>0.062</td>
</tr>
<tr>
<td>Sexual minority</td>
<td>% (SE)</td>
<td>6.78 (1.14)</td>
<td>16.8 (1.31)</td>
<td>0.267</td>
<td>&lt;.0001</td>
<td>N/A</td>
</tr>
<tr>
<td>Missing</td>
<td>% (SE)</td>
<td>0.20 (0.20)</td>
<td>0.49 (0.24)</td>
<td>0.041</td>
<td>0.373</td>
<td>N/A</td>
</tr>
<tr>
<td>Gender minority</td>
<td>% (SE)</td>
<td>2.46 (0.70)</td>
<td>5.62 (0.81)</td>
<td>0.137</td>
<td>0.003</td>
<td>N/A</td>
</tr>
<tr>
<td>Immigrated at or before age 16</td>
<td>% (SE)</td>
<td>3.92 (0.88)</td>
<td>5.90 (0.83)</td>
<td>0.084</td>
<td>0.101</td>
<td>N/A</td>
</tr>
<tr>
<td>Missing</td>
<td>% (SE)</td>
<td>0.61 (0.35)</td>
<td>0.73 (0.30)</td>
<td>0.014</td>
<td>0.799</td>
<td>N/A</td>
</tr>
<tr>
<td>Immigration related-stress at or before age 16</td>
<td>% (SE)</td>
<td>0.86 (0.43)</td>
<td>1.91 (0.49)</td>
<td>0.075</td>
<td>0.106</td>
<td>N/A</td>
</tr>
<tr>
<td>Missing</td>
<td>% (SE)</td>
<td>4.71 (0.96)</td>
<td>4.27 (0.71)</td>
<td>-0.022</td>
<td>0.712</td>
<td>N/A</td>
</tr>
<tr>
<td>Survivor of residential school</td>
<td>% (SE)</td>
<td>0.45 (0.32)</td>
<td>1.15 (0.40)</td>
<td>0.058</td>
<td>0.176</td>
<td>N/A</td>
</tr>
<tr>
<td>Missing</td>
<td>% (SE)</td>
<td>9.02 (1.30)</td>
<td>14.8 (1.24)</td>
<td>0.162</td>
<td>0.001</td>
<td>N/A</td>
</tr>
<tr>
<td>Family member(s) attended residential school</td>
<td>% (SE)</td>
<td>3.33 (0.85)</td>
<td>11.3 (1.18)</td>
<td>0.228</td>
<td>&lt;.0001</td>
<td>N/A</td>
</tr>
<tr>
<td>Missing</td>
<td>% (SE)</td>
<td>7.79 (1.21)</td>
<td>12.5 (1.15)</td>
<td>0.141</td>
<td>0.005</td>
<td>N/A</td>
</tr>
</tbody>
</table>

SE denotes standard error.

* P-values for continuous variables were derived from weighted t-tests (PROC SURVEYMEANS) while P-values for binary variables were derived from weighted Wald Chi Square tests (PROC SURVEYFREQ). Kolmogorov-Smirnov tests are only intended for continuous variables and thus were only reported for age.
Table 10: Covariate balance, AEE weighted

<table>
<thead>
<tr>
<th>Variable</th>
<th>Measure</th>
<th>CPSA-N=744</th>
<th>CPSA+ N=819</th>
<th>Standardized effect size difference</th>
<th>P-value*</th>
<th>P-value (KS-test)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>x (SE)</td>
<td>42.8 (0.47)</td>
<td>42.7 (0.35)</td>
<td>-0.01</td>
<td>0.873</td>
<td>0.77</td>
</tr>
<tr>
<td>Sexual minority</td>
<td>% (SE)</td>
<td>14.2 (2.55)</td>
<td>16.8 (1.31)</td>
<td>0.068</td>
<td>0.362</td>
<td>N/A</td>
</tr>
<tr>
<td>Missing</td>
<td>% (SE)</td>
<td>0.17 (0.17)</td>
<td>0.49 (0.24)</td>
<td>0.046</td>
<td>0.281</td>
<td>N/A</td>
</tr>
<tr>
<td>Gender minority</td>
<td>% (SE)</td>
<td>5.49 (1.92)</td>
<td>5.62 (0.81)</td>
<td>0.006</td>
<td>0.949</td>
<td>N/A</td>
</tr>
<tr>
<td>Immigrated at or before age 16</td>
<td>% (SE)</td>
<td>5.82 (1.41)</td>
<td>5.90 (0.83)</td>
<td>0.004</td>
<td>0.959</td>
<td>N/A</td>
</tr>
<tr>
<td>Missing</td>
<td>% (SE)</td>
<td>0.95 (0.61)</td>
<td>0.73 (0.30)</td>
<td>-0.026</td>
<td>0.747</td>
<td>N/A</td>
</tr>
<tr>
<td>Immigration related-stress at or before age 16</td>
<td>% (SE)</td>
<td>1.92 (1.02)</td>
<td>1.91 (0.49)</td>
<td>0.000</td>
<td>0.993</td>
<td>N/A</td>
</tr>
<tr>
<td>Missing</td>
<td>% (SE)</td>
<td>4.74 (1.08)</td>
<td>4.27 (0.71)</td>
<td>-0.023</td>
<td>0.716</td>
<td>N/A</td>
</tr>
<tr>
<td>Survivor of residential school</td>
<td>% (SE)</td>
<td>0.97 (0.70)</td>
<td>1.15 (0.40)</td>
<td>0.013</td>
<td>0.831</td>
<td>N/A</td>
</tr>
<tr>
<td>Missing</td>
<td>% (SE)</td>
<td>12.4 (1.97)</td>
<td>14.8 (1.24)</td>
<td>0.068</td>
<td>0.296</td>
<td>N/A</td>
</tr>
<tr>
<td>Family member attended residential school</td>
<td>% (SE)</td>
<td>9.15 (2.33)</td>
<td>11.3 (1.18)</td>
<td>0.059</td>
<td>0.409</td>
<td>N/A</td>
</tr>
<tr>
<td>Missing</td>
<td>% (SE)</td>
<td>11.2 (1.92)</td>
<td>12.5 (1.15)</td>
<td>0.038</td>
<td>0.573</td>
<td>N/A</td>
</tr>
</tbody>
</table>

SE denotes standard error.

* P-values for continuous variables were derived from weighted t-tests (PROC SURVEYMEANS) while P-values for binary variables were derived from weighted Wald Chi Square tests (PROC SURVEYFREQ). Kolmogorov-Smirnov tests are only intended for continuous variables and thus were only reported for age. CPSA- sample size differs post-weighting because weights are calculated based on similarity to the CPSA+ group; CPSA- women with higher odds of exposure are weighted heavier, resulting in a greater post-weighting sample size.

**Covariate balance within ethnoracial strata:**

In order to ensure that the use of cohort PSs as opposed to stratum-specific PSs balanced measured confounders across exposure groups within each ethnoracial group, weighted and unweighted balance diagnostics were calculated for each stratum (see Appendix 6). As for the total sample, the unweighted prevalence of sexual minorities was higher...
among the CPSA+ groups for Black African and white women \((P=0.018, \ P<0.0001)\). Similarly, the prevalence of gender minorities was significantly higher among “other” women who experienced CPSA compared to those who did not \((P=0.025)\). Other imbalances in the unweighted data were related to CPSA+ women being younger among Black African women \((P=0.003)\) and CPSA+ women being more likely to immigrate in childhood among Black Caribbean women \((P=0.019)\). Gender identity, however, did not allow for calculation of balance diagnostics for any Black women, as no CPSA+ Black African women and no CPSA- Black Caribbean women were gender minorities. Similarly, no CPSA- Black Caribbean women had immigration-related stress in childhood. Because there was no heterogeneity across CPSA groups here prior to weighting, GBM could not balance said heterogeneity with weighting, and thus these stratum-specific instances of data insufficiency remained. Apart from these stratum-specific instances of homogeneity in the data, weighting successfully remedied all of the measured imbalances in the unweighted data apart from that with respect to the age distribution among Black African women. Specifically, CPSA+ Black African women had a mean age of 39.8 while CPSA- Black African women had a mean age of 43.4.

**Concerns related to absolute standard effect size difference**

While the AEE weights successfully balanced all measured exposure group differences in the total sample, and the majority of stratum-specific differences, one concern with the given weights was the limited effect sizes of the propensity score covariates on CPSA in the unweighted sample, as represented by the absolute standardized effect size difference (Table 9). McCaffrey and colleagues have loosely defined any standardized effect size difference greater than 0.20 to be a potentially problematic imbalance. The standardized effect size differences observed in the unweighted sample were low relative to this metric, with only the effects of sexual minority identity and family member attendance at residential school exceeding 0.20. The limited observed differences in the unweighted sample lead to limited potential for adjustment and thus affected the interpretation of AEE estimates as detailed in section 4.4.1.
4.3 Descriptive Statistics

Exposure variables:

The burden of CPSA (and CPA/CSA respectively) in the sample was very high, with the majority of women (62.7%) having some experience of physical or sexual abuse in childhood (Table 11). Physical abuse was more prevalent than sexual abuse in the total sample and within each ethnoracial group. Proportionally, CPSA experiences were highest among Indigenous women (76.8%) and lowest among Black Caribbean women (46.9%). This trend held for experiences of physical abuse and sexual abuse, respectively. More than half (56.7%) of the sample experienced CPA, while 39.6% of the sample experienced CSA. Notably, for all ethnoracial groups, CPA and CSA co-occurred more frequently than they occurred independently, with overall an prevalence of co-occurrence equal to 34.1%. When measuring mutually exclusive CPA and CSA, experiencing only physical abuse was consistently more common than experiencing only sexual abuse. The prevalence of co-occurrence and independent occurrence is important when considering the validity of the sensitivity analyses, which attempt to disentangle the effects of CPA and CSA (see 3.2.6 Sensitivity Analysis).

Outcome variables:

Similar to childhood experiences of trauma, the burden of physical and sexual abuse in adulthood in the sample was very high. More than half of women (63.6%) had ever experienced physical abuse in adulthood (Table 12). This proportion was higher among women who experienced CPSA compared to those who did not (79.0% and 37.7%, respectively). Just under half of women (45.8%) had ever experienced sexual abuse in adulthood. Similarly, adult sexual abuse was more prevalent in the CPSA+ group than the CPSA- group (56.7% and 28.6%, respectively). The level of sexual inactivity in the sample was also high. In the total sample, and across exposure groups, approximately half of women had not engaged in consensual sex in the past six months. Furthermore, among women who had engaged in consensual sex, levels of perceived sexual relationship power were relatively high overall ($\bar{x} = 3.30$ on a 1-4 scale) and high across the CPSA- and CPSA+ groups. These results cannot be taken as representative of the
level of sexual relationship power among the sexually inactive women in the sample. The sample mean score on the HIV stigma scale (which ranges from 0-40) was 22.8, with higher scores indicating higher HIV stigma. This measure indicated mid-level HIV stigma for the group as a whole. Exposure group means did not appear to differ meaningfully from the overall mean. The same was true for perceived social support. The sample mean score on the social support scale was 14.2 on a scale from 4-20, which may denote mid-to-high levels of perceived social support.

Table 11: Unweighted prevalence of childhood physical or sexual abuse and associated variables by ethnoracial group

<table>
<thead>
<tr>
<th>Exposure % (SE)</th>
<th>Total N=1307</th>
<th>Indigenous N=289</th>
<th>Black African N=302</th>
<th>Black Caribbean N=64</th>
<th>White N=549</th>
<th>Other N=103</th>
</tr>
</thead>
<tbody>
<tr>
<td>CPSA</td>
<td>62.7 (1.34)</td>
<td>76.8 (2.49)</td>
<td>50.0 (2.88)</td>
<td>46.9 (6.29)</td>
<td>62.3 (2.07)</td>
<td>71.8 (4.45)</td>
</tr>
<tr>
<td>CPA</td>
<td>56.7 (1.37)</td>
<td>70.8 (2.68)</td>
<td>47.0 (2.88)</td>
<td>45.3 (6.27)</td>
<td>54.8 (2.13)</td>
<td>63.1 (4.78)</td>
</tr>
<tr>
<td>CSA</td>
<td>40.1 (1.37)</td>
<td>60.9 (2.92)</td>
<td>17.8 (2.22)</td>
<td>14.1 (4.38)</td>
<td>43.9 (2.13)</td>
<td>44.6 (4.97)</td>
</tr>
<tr>
<td>CPA &amp; CSA</td>
<td>34.1 (1.32)</td>
<td>54.6 (2.98)</td>
<td>14.8 (2.06)</td>
<td>12.5 (4.17)</td>
<td>36.3 (2.06)</td>
<td>35.6 (4.79)</td>
</tr>
<tr>
<td>CPA only</td>
<td>22.0 (1.16)</td>
<td>15.4 (2.16)</td>
<td>31.5 (2.70)</td>
<td>32.8 (5.92)</td>
<td>18.2 (1.65)</td>
<td>26.7 (4.43)</td>
</tr>
<tr>
<td>CSA only</td>
<td>5.98 (0.66)</td>
<td>6.07 (1.43)</td>
<td>3.02 (0.99)</td>
<td>1.56 (1.56)</td>
<td>7.52 (1.13)</td>
<td>8.91 (2.85)</td>
</tr>
</tbody>
</table>

SE denotes standard error. CPSA= childhood physical or sexual abuse, CPA= childhood physical abuse, CSA= childhood sexual abuse, CPA & CSA = co-occurrence of childhood physical and sexual abuse, CPA only= occurrence of childhood physical abuse without sexual abuse, CSA only= occurrence of childhood sexual abuse without physical abuse.

Table 12: Unweighted distribution of outcomes by exposure status

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Total N=1307</th>
<th>CPSA- N=488</th>
<th>CPSA+ N=819</th>
</tr>
</thead>
<tbody>
<tr>
<td>Physical abuse in adulthood % (SE)</td>
<td>63.6 (1.33)</td>
<td>37.7 (2.20)</td>
<td>79.0 (1.43)</td>
</tr>
<tr>
<td>Sexual abuse in adulthood % (SE)</td>
<td>46.2 (1.39)</td>
<td>28.6 (2.05)</td>
<td>56.7 (1.74)</td>
</tr>
<tr>
<td>Sexual inactivity % (SE)</td>
<td>54.4 (1.51)</td>
<td>54.7 (2.43)</td>
<td>54.1 (1.93)</td>
</tr>
<tr>
<td>Sexual relationship power (scale 1-4) × (SE)</td>
<td>3.30 (0.03)</td>
<td>3.39 (0.04)</td>
<td>3.25 (0.03)</td>
</tr>
<tr>
<td>HIV Stigma (scale 0-40) × (SE)</td>
<td>22.9 (0.23)</td>
<td>22.3 (0.36)</td>
<td>23.2 (0.29)</td>
</tr>
<tr>
<td>Social support (scale 4-20) × (SE)</td>
<td>14.2 (0.13)</td>
<td>14.5 (0.20)</td>
<td>14.0 (0.16)</td>
</tr>
</tbody>
</table>

SE denotes standard error. Sample sizes for Sexual Relationship Power are reduced due to survey design.
4.4 Effect of CPSA on Disclosure Barriers

4.4.1 Interpretation of AEE

The AEE weighting scheme of the present analysis was meant to allow measures of effect to be interpreted from a counterfactual perspective. For binary outcomes, prevalence ratios and their robust 95% confidence intervals were calculated. An AEE weighted prevalence ratio (PR) can be interpreted as the effect of an exposure on a given outcome among those who actually experienced the exposure. For instance, “women who experienced CPSA had 1.96 (95% CI 1.72, 2.25) times greater risk of physical abuse in adulthood than they would have, had CPSA never occurred”. Similarly, for continuous outcomes, effects were estimated using regression coefficients representing the adjusted difference in mean outcome level for the CPSA+ group as compared to the CPSA- group. For instance, “women who experienced CPSA had scores on the 40-point HIV stigma scale that were 1.34 (95% CI 0.35, 2.40) points higher than they would have been, had CPSA never occurred”. For the current analyses, AEE estimates were intended to represent causal processes. However, the limited effects of propensity score covariates and resulting limited potential for adjustment resulted in fewer than expected differences between unweighted and weighted effect estimates. For this reason, among other limitations such as the potential for unmeasured confounding, it is cautioned that AEE estimates should be interpreted as adjusted risk estimates with CPSA+ women as the referent group, rather than causal effects. This limitation is discussed further in Chapter 5.2.

4.4.2 Analytic Results

Objectives 1 through 3 are addressed by outcome within these analytic results. Objective 1 is addressed by presenting the effect of CPSA on outcomes for the group as a whole. Objective 2 to compare the above effect across ethnoracial groups is then addressed by presenting effect estimates stratified by ethnoracial group, then further effect estimates comparing the groups with white women as the referent group. Finally, a sensitivity
analysis is undertaken by repeating steps taken in Objectives 1 and 2 with different exposure variables: CPA and CSA.

Physical abuse in adulthood

For the total sample as per Objective 1, CPSA had a positive effect on physical abuse in adulthood, with women who experienced CPSA having 1.97 (95% CI 1.72, 2.25) times greater risk of physical abuse attributable to their CPSA history after weighting for measured confounders. Per Objective 2, the effect of CPSA on physical abuse persisted within each ethnoracial group, both before and after weighting (Table 13). As reflected in Figure 4 (a), the robust 95% confidence intervals for Black Caribbean women, as well as for “other” women were relatively imprecise compared to other groups. Black Caribbean women made up the smallest ethnoracial group under consideration (n=64), and thus estimates for this group were relatively imprecise for all outcomes. Similarly, the mixed ethnoracial group was the second smallest group under consideration (n=104) and thus some imprecision was expected, however, the wide confidence intervals for this group were likely also impacted by within-group ethnoracial heterogeneity. Interestingly, despite the relative imprecision for the effect of CPSA on physical abuse among Black Caribbean women, comparisons of prevalence ratios across ethnoracial groups revealed that the effect was stronger among Black Caribbean women as compared to white women (PR=1.12 95% CI 1.12, 4.60).

The sensitivity analyses for this outcome revealed some heterogeneity of effect; it appeared that the effects of CPSA on physical abuse were more broadly attributable to CPA than CSA in the weighted analysis. Both CPA and CSA had overall and stratum-specific effects on adult physical abuse in the unweighted sample, however, after weighting, the effect of CSA on physical abuse was restricted to white women (Appendix 7). Conversely, the weighted effect of CPA on physical abuse persisted within each ethnoracial group, and even appeared to differ significantly across ethnoracial groups. As for CPSA, the effect of CPA on physical abuse was considerably stronger for Black Caribbean women when compared to white women (PR=3.61 95% CI 1.58, 8.24). However, in this case, the same was true for Black African women (PR=1.65 95% CI
Thus, for physical abuse in adulthood, CPA appeared to drive the observed effect of CPSA, and when examined independently the CPA analysis revealed additional nuance across ethnoracial groups.

**Sexual abuse in adulthood**

Similar to physical abuse in adulthood, CPSA increased risk for sexual abuse in adulthood among CPSA+ women (weighted PR=1.80 95% CI 1.51, 2.13). Furthermore, CPSA was found to increase risk of sexual abuse among all CPSA+ women outside of Black Caribbean women (Table 13). Tests for effect modification across ethnoracial groups did not find differences in the effect of CPSA on sexual abuse for any group when compared to white women. It is possible that no effect was detected among Black Caribbean women based on the small sample size for this group, allowing less power to detect an effect if one was truly present. As expected, the confidence interval for this estimate was wider relative to other ethnoracial groups (Figure 4 b).

As with the sensitivity analyses for adult physical abuse, it was found that the effect of CPSA on adult sexual abuse may have been driven more by CSA than CPA. Both CSA and CPA had an effect on adult sexual abuse prior to weighting, however, after weighting, the effect of CPA on adult physical abuse was much lower in magnitude than the weighted CPSA effect. The CPA confidence interval, while small, approached the null (PR=1.20, 95% CI 1.02, 1.43). Conversely, the effect of CSA on adult sexual abuse was more similar to the CPSA effect both overall (CSA PR=1.63, 95% CI 1.38, 1.93) and within ethnoracial groups (Appendix 7). Furthermore, like CPSA, neither CPA or CSA analyses revealed any effect modification across ethnoracial groups for adult sexual abuse.

**Sexual inactivity and sexual relationship power**

In order to understand the effect of CPSA on sexual relationship power (SRP), it was first necessary to examine its effect on sexual inactivity given that SRP was only asked of sexually active women. CPSA did not appear to have an effect on sexual inactivity for the overall sample (PR=1.02, 95% CI 0.90, 1.16) or within ethnoracial groups (Table 13).
Knowing that the sexually active women who answered the SRP items had comparable histories of CPSA to those not asked the SRP items, it was possible to proceed in interpreting the effect of CPSA on SRP. Overall, women who experienced CPSA had lower scores on the SRP scale than they would have had CPSA never occurred, after adjusting for confounding. The estimated difference in scores attributable to CPSA was -0.13 (95% CI -0.23, -0.02). On the SRP scale, which ranged from 1-4, this meant that women who experienced CPSA had an estimated score of 3.25 (95% CI 3.19, 3.31); according to the scale’s original unvalidated cut-points, this would still be considered as having high sexual relationship power. It is important to note that, because of the survey design which restricted the SRPS to sexually active women, sample sizes for this outcome were reduced overall, but especially within ethnoracial groups. When differences in SRPS scores attributable to CPSA were estimated within ethnoracial groups, confidence intervals were fairly wide and only indicated a significant CPSA effect among white women (Figure 5, Table 13). However, when compared to other ethnoracial groups no differences were observed.

The sensitivity analyses for this outcome revealed some further nuance, particularly with respect to the effect of CSA on SRP among white women. Neither CPA nor CSA had an overall effect on SRP after weighting (CPA β=-0.03 95% CI -0.14, 0.09; CSA β =-0.07 95% CI -0.26, 0.11). However, the weighted effect of CSA among CSA+ white women was relatively large (β=-0.41 95% CI -0.57, -0.24), such that white women who experienced CSA had lower SRP, attributable to CSA. Furthermore, the magnitude of effect was greater among white women compared to both Indigenous and Black African women (Appendix 7). Thus, the effect of CPSA on SRP was more broadly similar to the effect of CSA, and the CSA analysis revealed some further variability across ethnoracial groups.

**HIV stigma**

For the total sample, women who experienced CPSA had HIV stigma scores 1.37 (95% CI 0.35, 2.40) points higher than they would have had, had CPSA not occurred. On the HIV stigma scale, which ranged from 0-40, this meant that CPSA+ women had an
estimated score of 23.5 (95% CI 23.0, 24.1). While no reference scores for this scale were identified, this appears to indicate mid-range HIV stigma. Interestingly, among Black African women CPSA had a substantial effect on HIV stigma; Black African women who experienced CPSA had HIV stigma scores 3.93 (95% CI 2.16, 5.70) points higher than would be expected had CPSA not occurred (Table 13, Figure 6). This effect was 2.52 (95% CI 0.19, 4.86) points greater than that observed among white women.

Both CPA and CSA independently had overall effects on HIV stigma in the weighted sample (CPA $\beta$=1.83 95% CI 0.58, 3.08; CSA $\beta$=1.33 95% CI 0.19, 2.48). These estimated differences were broadly consistent with the result for CPSA which indicated that CPSA+ women experienced slightly elevated HIV stigma because of their CPSA experiences. However, in the CPA analysis, the effect among Black African women which was observed for CPSA appeared to be attenuated; HIV stigma was only 1.40 (95% CI 0.44, 5.92) points greater because of CPA (Appendix 7). Furthermore, while CSA had an effect among Black African women prior to weighting ($\beta$=2.83 95% CI 0.56, 5.09), the effect could not be attributed to CSA after weighting ($\beta$=2.34 95% CI -0.21, 4.90). It is possible that the construct driving the relationship between CPSA and HIV stigma was not the presence of either CPA or CSA alone, but their co-occurrence, which would be controlled for in the sensitivity analyses but not in the overall analysis where the exposure was child physical or sexual abuse. It is also possible that because CPA and CSA independently elevated HIV stigma among white women (CPA $\beta$=2.09 95% CI 0.24, 3.94; CSA $\beta$=1.75 95% CI 0.08, 3.41), their effects among Black African women were more comparable.

**Social support**

CPSA had no overall effect on perceived social support among women who experienced CPSA. In particular, a difference of -0.33 (95% CI -0.92, 0.24) points on the MOS-SSS scale was attributed to CPSA, though the confidence interval crossed the null value of 1. In the overall sample the estimated mean level of social support for women who experienced CPSA+ was 14.0 (95% CI 13.7, 14.4) on a scale from 4-20, a value not substantially different from mean level observed in the original 4-item MOS-SSS
validation study (\(\bar{x} = 14.88\), standard deviation=3.97). The mean observed from the validation study came from a sample of 330 low-income American mothers receiving services for children with emotional or behavioural problems.\(^{230}\) When considering stratum-specific estimates, among white women only a difference of -1.04 (95% CI -2.06, -0.02) points was attributable to CPSA (Table 13, Figure 7). This effect was not significantly different when compared to the effects among other ethnoracial groups.

The results of both sensitivity analyses were broadly similar to those from the main analysis. Neither CPA nor CSA had an independent effect on perceived social support among their exposed populations (CPA \(\beta = -0.43\) 95% CI -1.09, 0.21; CSA \(\beta = -0.31\) 95% CI -1.01, 0.40). Within ethnoracial strata, CSA had no effect on perceived social support, while CPA was linked to reduced social support among Black African women (\(\beta = -1.35\) 95% CI -2.58, -0.11) and women from the other ethnoracial group (\(\beta = -2.72\) 95% CI -4.88, -0.56) (Appendix 7). When the CPA effects were compared across strata, “other” women experienced -2.81 (95% CI -5.18, -0.44) fewer points attributable to CPA relative to white women. However, given the within-group heterogeneity of this stratum, the interpretability of this potential difference was compromised. Thus, overall the observed CPA and CSA effects did not diverge excessively from those observed in the main analysis.
Table 13: Effect of CPSA on barriers to HIV disclosure among women who experienced CPSA

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Ethnoracial group</th>
<th>Unweighted</th>
<th>Weighted</th>
<th>Effect modification</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adult physical abuse ¶</td>
<td>White</td>
<td>1.72 (1.47, 2.03)</td>
<td>1.56 (1.31, 1.85)</td>
<td>REF</td>
</tr>
<tr>
<td></td>
<td>Indigenous</td>
<td>2.34 (1.73, 3.18)</td>
<td>2.18 (1.56, 3.05)</td>
<td>1.40 (0.96, 2.04)</td>
</tr>
<tr>
<td></td>
<td>Black African</td>
<td>2.10 (1.62, 2.18)</td>
<td>2.11 (1.62, 2.74)</td>
<td>1.35 (0.99, 1.85)</td>
</tr>
<tr>
<td></td>
<td>Black Caribbean</td>
<td>3.12 (1.64, 5.93)</td>
<td>3.53 (1.78, 7.01)</td>
<td>2.27 (1.12, 4.60)</td>
</tr>
<tr>
<td></td>
<td>Other</td>
<td>2.69 (1.47, 4.93)</td>
<td>2.49 (1.27, 4.89)</td>
<td>1.60 (0.80, 3.21)</td>
</tr>
<tr>
<td>Adult sexual abuse ¶</td>
<td>White</td>
<td>1.83 (1.46, 2.28)</td>
<td>1.56 (1.23, 1.99)</td>
<td>REF</td>
</tr>
<tr>
<td></td>
<td>Indigenous</td>
<td>2.52 (1.62, 3.91)</td>
<td>2.02 (1.27, 3.20)</td>
<td>1.29 (0.77, 2.18)</td>
</tr>
<tr>
<td></td>
<td>Black African</td>
<td>1.77 (1.32, 2.37)</td>
<td>1.75 (1.30, 2.36)</td>
<td>1.12 (0.76, 1.65)</td>
</tr>
<tr>
<td></td>
<td>Black Caribbean</td>
<td>1.98 (0.97, 4.06)</td>
<td>1.69 (0.79, 3.61)</td>
<td>1.08 (0.49, 2.40)</td>
</tr>
<tr>
<td></td>
<td>Other</td>
<td>2.74 (1.31, 5.75)</td>
<td>2.90 (1.32, 6.38)</td>
<td>1.86 (0.81, 4.23)</td>
</tr>
<tr>
<td>Sexual inactivity ¶</td>
<td>White</td>
<td>1.00 (0.85, 1.19)</td>
<td>1.05 (0.87, 1.28)</td>
<td>REF</td>
</tr>
<tr>
<td></td>
<td>Indigenous</td>
<td>0.99 (0.75, 1.30)</td>
<td>1.05 (0.76, 1.46)</td>
<td>1.00 (0.68, 1.46)</td>
</tr>
<tr>
<td></td>
<td>Black African</td>
<td>1.10 (0.86, 1.41)</td>
<td>1.10 (0.86, 1.42)</td>
<td>1.05 (0.76, 1.44)</td>
</tr>
<tr>
<td></td>
<td>Black Caribbean</td>
<td>0.93 (0.61, 1.44)</td>
<td>0.91 (0.59, 1.40)</td>
<td>0.86 (0.54, 1.38)</td>
</tr>
<tr>
<td></td>
<td>Other</td>
<td>0.68 (0.48, 0.95)</td>
<td>0.69 (0.47, 1.02)</td>
<td>0.66 (0.43, 1.02)</td>
</tr>
<tr>
<td>Sexual relationship power ±</td>
<td>White</td>
<td>-0.19 (-0.35, -0.04)</td>
<td>-0.17 (-0.33, -0.01)</td>
<td>REF</td>
</tr>
<tr>
<td></td>
<td>Indigenous</td>
<td>-0.13 (-0.39, 0.13)</td>
<td>-0.06 (-0.32, 0.21)</td>
<td>0.12 (-0.19, 0.42)</td>
</tr>
<tr>
<td></td>
<td>Black African</td>
<td>-0.12 (-0.30, 0.06)</td>
<td>-0.15 (-0.33, 0.04)</td>
<td>0.02 (-0.22, 0.27)</td>
</tr>
<tr>
<td></td>
<td>Black Caribbean</td>
<td>0.04 (-0.34, 0.42)</td>
<td>-0.02 (-0.41, 0.36)</td>
<td>0.15 (-0.27, 0.56)</td>
</tr>
<tr>
<td></td>
<td>Other</td>
<td>-0.33 (-0.70, 0.05)</td>
<td>-0.32 (-0.66, 0.02)</td>
<td>-0.15 (-0.53, 0.23)</td>
</tr>
<tr>
<td>HIV stigma ±</td>
<td>White</td>
<td>1.90 (0.50, 3.30)</td>
<td>1.41 (-0.12, 2.93)</td>
<td>REF</td>
</tr>
<tr>
<td></td>
<td>Indigenous</td>
<td>0.07 (-2.25, 2.39)</td>
<td>-0.80 (-3.39, 1.78)</td>
<td>-2.21 (-5.21, 0.79)</td>
</tr>
<tr>
<td></td>
<td>Black African</td>
<td>3.76 (2.05, 5.48)</td>
<td>3.93 (2.16, 5.70)</td>
<td>2.52 (0.19, 4.86)</td>
</tr>
<tr>
<td></td>
<td>Black Caribbean</td>
<td>-0.54 (-3.93, 2.86)</td>
<td>-0.27 (-3.56, 3.02)</td>
<td>-1.68 (-5.31, 1.95)</td>
</tr>
<tr>
<td></td>
<td>Other</td>
<td>-0.25 (-3.31, 2.81)</td>
<td>0.53 (-2.39, 3.45)</td>
<td>-0.88 (-4.17, 2.42)</td>
</tr>
<tr>
<td>Social support ±</td>
<td>White</td>
<td>-1.36 (-2.11, -0.61)</td>
<td>-1.04 (-2.06, -0.02)</td>
<td>REF</td>
</tr>
<tr>
<td></td>
<td>Indigenous</td>
<td>-0.36 (-1.43, 0.70)</td>
<td>0.39 (-0.91, 1.69)</td>
<td>1.43 (-0.22, 3.08)</td>
</tr>
<tr>
<td></td>
<td>Black African</td>
<td>-0.36 (-1.35, 0.62)</td>
<td>-0.31 (-1.32, 0.70)</td>
<td>0.72 (-0.71, 2.16)</td>
</tr>
<tr>
<td></td>
<td>Black Caribbean</td>
<td>0.90 (-1.21, 3.01)</td>
<td>0.82 (-1.19, 2.83)</td>
<td>1.86 (-0.39, 4.11)</td>
</tr>
<tr>
<td></td>
<td>Other</td>
<td>-2.33 (-4.32, -0.35)</td>
<td>-2.83 (-4.70, -0.96)</td>
<td>-1.79 (-3.92, 0.34)</td>
</tr>
</tbody>
</table>

¶ Indicates binary outcome variables. Effect estimates are prevalence ratios and their robust 95% confidence intervals. Shaded cells represent those where the confidence interval does not cross the null value of 1.

± Indicates continuous outcome variables. Effect estimates are coefficients from linear regression (i.e. estimated difference in mean outcome for CPSA+ compared to CPSA-) and their 95% confidence intervals. Shaded cells represent those where the confidence interval does not cross the null value of 0.
Figure 4: (a) Effect of CPSA on physical abuse and (b) sexual abuse by ethnoracial group

Points represent estimated prevalence ratios for the effect of CPSA, the vertical axis at x=1 represents the null value, and stems represent robust 95% confidence intervals.

Figure 5: Effect of CPSA on sexual relationship power by ethnoracial group

Bars represent estimated mean outcome level by exposure status for the AEE weighted sample, while stems represent robust 95% confidence intervals.
Figure 6: Effect of CPSA on HIV stigma by ethnoracial group
Bars represent estimated mean outcome level by exposure status for the AEE weighted sample, while stems represent robust 95% confidence intervals.

Figure 7: Effect of CPSA on social support by ethnoracial group
Bars represent estimated mean outcome level by exposure status for the AEE weighted sample, while stems represent robust 95% confidence intervals.
Chapter 5

5 Discussion and Implications

This chapter will reiterate the main descriptive and analytic findings, discussing them in the context of the literature and evaluating them in light of the study’s strengths and limitations. Extensions for future research will be identified, and implications for policy and practice will be described.

5.1 Main Findings

5.1.1 Summary

This thesis adapted a novel theoretical model based on eco-social theory and intersectionality which contextualizes the causes and consequences of CPSA among women with HIV in Canada. The framework relayed the importance of unpacking culture as related to child abuse, described how intersecting systems of oppression operate at the four eco-social levels to produce societies, communities, families, and individuals vulnerable to child abuse, and introduced colonialism as an axis of oppression affecting Indigenous women with HIV in Canada. In accordance with this model, it was of interest to determine whether child maltreatment affected barriers to HIV status disclosure among women with HIV after controlling for eco-social risk, and to determine whether these relationships differed across intersectional ethnoracial groups. Results showed that CPSA appeared to generate barriers to HIV disclosure, specifically increasing prevalence of both physical and sexual abuse, and increasing levels of HIV stigma. Furthermore, subgroup analysis showed that the effect of CPSA on some outcomes did vary across the intersectional ethnoracial groups; CPSA significantly decreased sexual relationship power among white women only, while increasing HIV stigma among only Black African women. A sensitivity analysis found that CPA may account for effects of CPSA on physical abuse in adulthood, while CSA may account for effects on sexual abuse in adulthood, and sexual relationship power. In summary, women with HIV who experienced CPSA did appear to face greater barriers to partner disclosure than those without such experiences, and this relationship may be of a causal nature. The relationship between CPSA and some barriers may not be homogeneous across
ethnoracial groups, and thus intersectional perspectives should be of great importance for research, intervention planning, and policy making in the future.

5.1.2 Descriptive Findings

Burden of Risk for CPSA

CPSA has been theorized to have causes at the level of the individual child, their family, their community, and their society. As research has further disentangled the causes of CPSA, Belsky’s original eco-social model has been adapted and refined, resulting in several theoretical principles and broad risk factors for CPSA at each level.136,142 These principles were applied in the current analysis when selecting propensity score covariates. As expected, the prevalence of all CPSA risk factors was elevated among those who reported experiencing CPSA, though not all differences reached statistical significance. For example, the prevalence of sexual minority and gender minority women among those who experienced CPSA was significantly greater than that among women who did not experience CPSA (16.8% vs. 6.78% sexual minorities, 5.62% vs. 2.46% gender minorities). These, and other observed disparities across exposure groups (e.g. with respect to stress-related immigration in childhood and family residential school attendance) illustrate the life circumstances of these women that may have preceded both child maltreatment and HIV diagnosis. While the CHIWOS sample is not population-based and exposure-stratified prevalence estimates for CPSA risk factors cannot be interpreted as causal, the fact that the retrospective burden of risk for CPSA was consistently greater among women who experienced CPSA lends support to the proposed theoretical model and to the analysis as a whole.

5.1.3 Analytic Findings

Revictimization

Arguably the most important finding from this analysis was the rate of revictimization from childhood to adulthood, in terms of both adult physical abuse and sexual abuse. Women who experienced CPSA were consistently at higher risk for experiencing further violence in adulthood. This relationship was expected, and contributes to the body of
evidence reporting not only the descriptive rate of revictimization, but controlled risk estimates. The relationship between CPSA (and CPA/CSA independently) and further abuse in adulthood among the general population has been documented extensively in longitudinal\textsuperscript{6,231} and meta-analytic studies.\textsuperscript{7} However, revictimization has more frequently been studied as a risk factor for HIV (eg. \textsuperscript{232,233}) than a construct generating further risk among people with HIV. Thus, the present study contributes new evidence suggesting that the relationship between CPSA and violence in adulthood persists among women with HIV in ways that may further complicate their lives and health.

Comparing the magnitude of the relationship between childhood violence and later revictimization for women with HIV and the general population presents methodological difficulties. Population-based studies of women with HIV are scant and likely under-represent marginalized women, and thus convenience and quota samples become the most optimal method for studying risk within the population of women with HIV. However, the lack of a population-based sampling frame complicates comparisons to the larger population of women in Canada. On the basis of measurement and study design (though not geography or ethnoracial composition), the sample most comparable to CHIWOS may be that of Simoni and Ng who produced descriptive estimates of revictimization among a community-recruited sample of 230 women with HIV in New York City.\textsuperscript{128} As with CHIWOS, prevalence estimates for CPSA (50%), adult physical abuse (63%), and adult sexual abuse (46%) were alarmingly high. Furthermore, the study found a zero-order correlation of 0.50 (p<0.01) between CPSA and physical or sexual abuse in adulthood, demonstrating a substantial amount of recurring abuse. Community samples like CHIWOS and Simoni and Ng demonstrate the high burden of abuse and revictimization among women with HIV. When considered alongside studies implicating revictimization as a risk factor for HIV in the first place,\textsuperscript{232,233} one can conclude that revictimization, regardless of the magnitude of risk compared to the general population, is an issue among women with HIV. While risk comparisons to the general population or men with HIV may be interesting, they are not necessary in order to justify interventions to reduce risk for violence against women with HIV, based on the high levels of violence alone and the unique socio-legal implications that violence generates for this population.
The present analysis also demonstrated the effect of CPSA on adult physical and sexual abuse largely persisting within each ethnoracial group. The single exception to this statement was the effect of CPSA on adult sexual abuse among Black Caribbean women, where the effect was in the positive direction but the robust 95% confidence interval crossed 1. Black Caribbean women constituted the smallest ethnoracial group under consideration (n=64), and consequently estimates for this group were relatively imprecise across all outcomes. The lack of observed relationship between CPSA and adult sexual abuse for this group should not be interpreted as a lack of grounds for intervention among this group, but rather as a potential type II error which warrants further investigation. This point is bolstered by the observed relationships between CPSA and abuse in adulthood across all other ethnoracial groups.

Another important result reflected in the main CPSA analysis as well as the CPA sensitivity analysis was that the magnitude of effect for childhood maltreatment on adult physical abuse was greater for Black women when compared to white women. This phenomenon was especially prominent among Black Caribbean women who experienced CPA; for this group, CPA multiplied risk of adult physical abuse by 3.61 (95% CI 1.58, 8.24) relative to white women. Published rates of physical and/or sexual revictimization disaggregated by ethnoracial groups for comparison were difficult to locate. Two recent studies have found that children and adolescents who identified as members of ethnoracial minority groups were more likely to report sexual revictimization than their white peers, however, further research is needed to examine revictimization at the intersection of race, gender, and class. There is some evidence to suggest that that women of colour are disproportionately affected by violence.236 However, when interpreting ethnoracial inequities and especially when planning interventions, it is important to be cognisant of the social and structural factors that may impact vulnerability to and reporting of violence against women of colour. The results of the present study provide further justification for developing anti-violence interventions that are appropriate and accessible for women of colour with HIV.

Sexual Relationship Power and Sexual Inactivity
This analysis found that, overall, women who experienced CPSA had lower sexual relationship power than they would have had CPSA not occurred. When broken down by ethnoracial group, it was revealed that this overall effect was primarily attributable to the strong effect among white women. Furthermore, the adjusted mean level of sexual relationship power possessed by women who experienced CPSA was still within the range of high sexual relationship power, as dictated by the scale’s unvalidated tertile cut-points. It was expected that this effect would be larger in magnitude and would persist across ethnoracial groups, however, given the inherent limitations due to survey design and measurement, this result was not entirely surprising. Sample sizes for the SRPS items within ethnoracial groups were reduced compared those of other outcomes, lowering the probability of observing the effect of CPSA if it was truly present.

During Wave 2 of the survey, the SRPS was only asked of women who reported engaging in consensual sexual activity within the past 6 months. While the scale was developed and validated among women with male sexual partners, it is possible that sexual relationship power as a construct (or a related construct) may exist outside the confines of a recent sexual encounter. For instance, the knowledge that one has the tendency to become involved in controlling relationships, sexual or otherwise, may prompt the conscious decision to avoid sexual relationships. This may be especially true for women with HIV; a manipulative sexual partner can use one’s HIV status as a point of power. Furthermore, CPSA may affect this potential sexual relationship power and decision making just as it may affect enacted sexual relationship power. However, because it is unknown whether the SRPS could measure potential sexual relationship power, and thus sexual relationship power was only measured among sexually active women, the relationship between CPSA and sexual agency cannot fully be described in the present analysis.

In an effort to rule out or rule in the posited relationship between CPSA and consensual sex, we measured the effect of CPSA on sexual inactivity over the past 6 months, and found no statistically significant effects. This provided some indication that CPSA may affect women who are sexually inactive in the same way as it does women who are sexually active, though we cannot know this for sure. Furthermore, because the sub-
sample who were sexually active and completed the SRPS consisted of only 486 of n=1307 women, effect estimates for the SRPS (especially within under-represented ethnoracial groups) were relatively imprecise. The fact that the only confidence interval that does not cross zero was among white women, the largest ethnoracial group in the sample, lends further support to the theory that CPSA driven differences in SRP (or lack thereof) could be attributable to a lack of precision. Some imprecision relative to other outcomes was expected, however, the SRPS outcome remained a part of the analysis plan on the basis of its importance as a risk factor for HIV non-disclosure and as an effort to encourage other researchers to incorporate SRP as not just a risk factor for HIV, but as a potential cause of further vulnerability among people with HIV. To our knowledge, no other studies have used specific measures of sexual agency or sexual relationship power in relation to CPSA or HIV disclosure, rather opting to measure characteristics of relationships with power imbalances. Because sexual relationship power is a construct that aggregates across multiple controlling characteristics and is already in use within the HIV literature, it is our hope that this concept can be further refined and applied more widely in the future.

**HIV Stigma**

An unexpected finding from this analysis was the effect of CPSA on HIV stigma being limited to Black African women. The relationship between CPSA and HIV stigma was examined on the basis of HIV stigma’s ability to impact HIV disclosure, and the ability of CPSA to engender perceived and internalized stigma related to the abuse. Furthermore, in the context of HIV, stigmas are often co-occurring and not necessarily independent (for example, stigmatizing attitudes about AIDS and people who inject drugs tend to co-occur,\(^237\) and internalized HIV stigma can be related to internalized blame about experiencing abuse).\(^238\) Furthermore, dimensions of multiple stigmas can vary across ethnoracial groups, for example, in one study negative attitudes towards people who inject drugs predicted AIDS stigma among Black participants while negative attitudes towards gay people predicted AIDS stigma among white participants.\(^237\) On the basis of the present analysis, future research should explore what drives the relationship between
CPSA and HIV stigma, whether it be cultural differences in measurement, attitudes, or unmeasured confounders.

Wright’s abbreviated HIV stigma scale measured stigma across four dimensions: personalized stigma, disclosure, negative self-image, and public attitudes – all of which have been associated with HIV disclosure in previous research using various measures. In investigating the unique relationship between CPSA and HIV stigma among Black African women, we should consider whether the effect is driven by HIV stigma in the broadest sense as captured in the given measure, or by certain dimensions within the given measure. The full HIV stigma scale was chosen based on existing evidence linking multiple dimensions of HIV stigma to partner disclosure, and the desire to examine multiple barriers to disclosure rather than the dimensionality of a single barrier. The latter option would have necessitated three additional study outcomes, which could potentially overwhelm results pertinent to other important barriers to HIV disclosure. However, in validation studies, both the full and abbreviated HIV stigma scales have demonstrated differential relationships between the subscales and construct validity outcomes such as perceived social support. Because HIV stigma as measured is truly a multidimensional construct, further research should explore the relationship between CPSA and multidimensional HIV stigma across ethnoracial groups.

Intersectionality theory helps us to understand that interlocking matrices of domination affect the experiences of people as they sit at the intersections of multiple social positions. A qualitative study of women with HIV in Ontario has explored the unique experiences of Black women with HIV produced by the intersection of sexism, racism, and HIV stigma. One Black African woman highlighted the enacted stigma she had experienced while receiving HIV services, stating “I need you to listen me and to help me. But you’re thinking ‘you come from Africa you don’t understand’”. This intersection of HIV stigma, sexism, and racial discrimination against Black African (and Caribbean) women and its effect on lived experiences has been explored in qualitative and quantitative research in North America. However, it is less clear how experiences of CPSA may fit into this picture.
While no studies specific to experiences of CPSA and HIV stigma among Black African women in North America were identified, the narratives of Black African women who experienced CPSA may provide a starting point for further inquiry among women with HIV. One qualitative study of African American women who were survivors of CSA highlighted how children often blame themselves for their own abuse. Through “truth-telling”, or reaching out to others who believed and validated their experiences, some women were able to find greater self-acceptance. This pattern is common among survivors of child abuse more generally. Among women with HIV, it is possible that internalized blame about CPSA could be a component of internalized HIV stigma, especially if the women perceive the two to be related. An aspect of this cycle of internalization and truth-telling that may be unique to the intersection of anti-Black racism and sexism is the pressure on Black CSA survivors to avoid disclosing their abuse. In the same qualitative CSA study, one participant described hearing other women express the idea that Black women who experienced abuse at the hands of Black men should remain silent so as not to contribute to a legal system which is already pitted against Black men. In addition to suppressing CSA disclosure, this pressure may close women off from community support and self-acceptance. Thus, it is possible that barriers to CPSA disclosure may mediate the relationship between CPSA and internalized stigma related to abuse and HIV. However, this hypothesis results from piecing together CPSA research on the intersections of gender and ethnoracial group with HIV research that is not specific to CPSA. Further research should explore lived experiences at this particular intersection.

Social Support

The effect of CPSA on perceived social support appeared to be negligible in the present study. The weighted mean level of social support reported by women who experienced CPSA was comparable to both the estimated mean level had those same women not experienced CPSA, as well as to the average social support observed in the abbreviated MOS-SSS validation study. Similar to the measure of HIV stigma, the abbreviated MOS-SSS captures four dimensions of perceived social support: emotional-informational support, tangible support, affectionate support, and positive social interaction.
literature review provided some indication that perceived or anticipated social support was most salient to partner disclosure, such that women who felt that their partners would support them regardless of their HIV status were empowered to disclose, while women who were doubtful or unsure of how their partner would react were less likely to disclose. While this was not the exact construct measured by the MOS-SSS, the two share some overlapping dimensions such as love and acceptance. The most significant difference between the two was that social support from sexual partners was most relevant to partner disclosure, while the MOS-SSS measured global social support from no target in particular. This limitation should be noted when considering the implications of these results.

The lack of observed relationship between CPSA and perceived social support may have been attributable to construct multidimensionality, or the two may simply not be related. While the link between anticipated social support and HIV non-disclosure has been well developed in the literature, that between CPSA and later perceived social support has been under-researched among the general population and, to our knowledge, not explicitly studied among people with HIV. The outcome was included based on its relationship with HIV non-disclosure along with the rationalization that reduced social support may be one of the many downstream consequences of CPSA. Additional qualitative research should investigate whether and how CPSA may play into anticipated social support just as such research has shed light on the relationship between social support and disclosure. Furthermore, as qualitative research has identified anticipated HIV-related social support as a construct of interest in the study of HIV disclosure, a corresponding quantitative measure of the construct should be developed in order to further investigate its relationships with both CPSA and partner disclosure.

5.1.4 Sensitivity Analysis

The planned sensitivity analyses revealed some heterogeneity in the observed effects of CPA as compared to CSA. In particular, the relationships between CPSA and adult physical abuse bore more similarity to the effects of CPA than CSA, while those between CPSA and adult sexual abuse, as well as sexual relationship power appeared to be driven by CSA. Furthermore, comparing the main analysis to the sensitivity analyses for the
HIV stigma outcome raised the question of whether the co-occurrence of CPA and CSA may have driven the observed effects. The observed heterogeneity according to the type of abuse suggested that the sensitivity analyses were valuable additions to the study, and that they may offer direction for future research. The segregation of effect such that physical abuse more consistently predicted physical revictimization and sexual abuse more consistently predicted adverse sexual experiences in adulthood was unexpected, but not unreasonable. Furthermore, the propensity score model which allowed for the collapsing of multiple confounders may have been an optimal way to control for the effect of a covariate that is highly collinear to the exposure (i.e. controlling CSA when studying CPA). However, it is acknowledged that the outcome analysis was unable to definitively account for variability in the original propensity score model, and thus the potential for misestimation exists as with traditional regression models. Future studies should further explore the ability of propensity score models to collapse multiple covariates in order to avoid multicollinearity issues; given the high frequency of co-occurring abuse, accurately estimated propensity score models may be ideally suited to this problem.

Also related to abuse co-occurrence, the findings related to HIV stigma and the potential interactivity of multiple types of abuse presented thought provoking ideas novel to the current study. A growing body of literature has addressed the consequences of co-occurring types of abuse, termed “poly-victimization”. When studied as an antecedent to mental health conditions and trauma symptomology in childhood, poly-victimization has been found to almost completely eclipse single-type victimizations as a predictor. Recent findings from poly-victimization research along with the results of the sensitivity analyses suggest that future research should pay attention to the potential for divergent effects by type of child abuse, as well as the potential synergistic effects of multiple types of adverse childhood experiences.

5.1.5 Eco-Social Intersectionality

In fulfillment of Objective 1, this thesis produced a novel theoretical model to contextualize the relationship between CPSA and HIV disclosure among women with HIV in Canada. This model paired intersectionality with eco-social theory to understand
vulnerability to CPSA at multiple levels, and at the intersection of gender, HIV status, and ethnoracial group. The model illustrated how systemic oppression drives inequalities, acknowledged culture as context rather than a cause of CPSA, described the effects of colonialism and intergenerational trauma on Indigenous women, and integrated all of these factors in the design of a quantitative analysis. While eco-social theory and intersectionality have been considered jointly in the past,168 this model extended the theories to incorporate novel concepts vital to the population at hand. Specifically, the determinants of CPSA for Indigenous women are known to be qualitatively different from populations that have not experienced colonial disruption of culture and family. For this reason, it was important to not only be conscious of potential effect modification across ethnoracial groups, but to include culturally specific determinants of CPSA in the causal model. Unfortunately, this practice is less common in the wider literature, with some publications reporting Indigenous data without giving consideration to Indigenous determinants of health and wellbeing. Notably, in the present analysis, when survival and family attendance at residential school were controlled for, there was no significant difference in the effect of CPSA on disclosure barriers among Indigenous women compared to white women. The value of approaches which model or control for the effects of inequitable processes such as residential school attendance is their ability to demonstrate that population inequities do not represent intractable differences. Furthermore, some inequitable processes are amenable to intervention and thus reduction of population inequities. Future research should take this eco-social and intersectional approach into consideration, adapting the relevant determinants and axes of oppression to the context at hand.

In addition to marking novel theoretical territory, this model appeared to demonstrate some empirical validity in its use of eco-social risk factors for CPSA. Of the risk factors considered, all those which different significantly across the CPSA+ and CPSA-exposure groups did so in the expected direction. Specifically, the CPSA+ group was composed of significantly more sexual minority women, trans or intersex women, women with a family member who attended residential school, women and who declined to answer items on self-attendance and family attendance at residential school. One area where the theoretical underpinnings of this model could have been better incorporated
into analysis is the inclusion of protective factors for CPSA, as well as additional higher-
level factors such as quality of the child protection system and societal attitudes towards
corporal punishment. However, these represented limitations to the data rather than the
theoretical model itself.

5.2 Limitations

Quota sampling

The use of quota sampling as opposed to population based sampling in CHIWOS
presented both a limitation and an advantage. Prevalence estimates reported in the
descriptive statistics section of this analysis are not necessarily generalizable to the larger
population of women with HIV in Canada. However, it is arguable that a population
based study of women with HIV (i.e. using household or clinical sampling frames) would
be inefficient and/or only representative of women with HIV accessing clinical care. It
is unlikely that a clinical sample would represent marginalized women with HIV, making
this strategy incongruent with the CHIWOS objective to apply an intersectional approach
in understanding the positionality and determinants of health for women with HIV. Thus,
the quota sampling method which allowed for relatively precise effect estimates within
key populations of women with HIV was likely an optimal approach. Furthermore,
CHIWOS coverage of the base population is numerically strong; the study is estimated to
capture 10% of women with HIV in Canada. Sociodemographic coverage of CHIWOS
is more difficult to evaluate, given the known under-coverage of women with HIV in
both surveillance and clinical data. Manipulation of the data to represent either of these
populations would therefore be inappropriate, as it would not meet the study objectives or
justifiably improve representativeness. Thus, the ability to draw causal inference from the
CHIWOS dataset fell on the assumption that exposure groups were comparable with
respect to potential confounders; the accuracy of this assumption is evaluated below.

Limited capability for adjustment

One potential limitation of the given study was the small effect size for propensity score
covariates on the exposure variable, leading to fewer than expected observable
differences between the weighted and unweighted results. While all propensity score covariates were selected in an *a priori* protocol developed using epidemiological theory and were within the confines of an established theoretical model, the data presented limitations that could not have been foreseen. The small covariate effect sizes and limited effect of weighting may be indicative of issues with covariate selection, measurement, temporality, or unmeasured confounding, as discussed below. However, given that CPSA is an unfortunately common experience characterized more so by a network of risk factors than strong causal factors, it is possible that the effect sizes for predictors on CPSA are truly as small as estimated here. The consequence of these secondary data limitations was that AEE estimates, which were intended to be causal, should be interpreted with caution. As the first known study to suggest a link between CPSA and barriers to HIV disclosure to sexual partners among women, the resulting exploratory effect estimates are still valuable. Furthermore, where significant effects were observed they were consistently in the hypothesized direction, providing support for the underlying theory, and justification for further inquiry into the magnitude of any potentially causal effects.

**Temporality**

The inability to definitively establish temporality presents an issue with most cross-sectional research, including the present study. This issue is particularly common in child maltreatment research, where most measures of CPSA are retrospective regardless of study design. The cut-off age (<16) for the exposure variable in conjunction with the CHIWOS inclusion criteria (age ≥16) allowed us to establish a temporal sequence such that CPSA and propensity score covariates preceded measurement of outcomes. Unfortunately, it was not possible to establish whether propensity score covariates preceded the exposure. In an effort to mitigate this, propensity score covariates where chosen from a pool of variables which were identifiable as having the potential to take effect prior to age 16. Within this pool of variables, those which theoretically were more likely to mediate relationships between CPSA and outcomes (i.e. experience with foster care or adoption) than to cause CPSA were eliminated. However, it is possible that some time-dependent propensity score covariates occurred after CPSA, meaning that
propensity score models may control the effect of mediators along the causal pathway. The consequence of this limitation may be that some of the true exposure effect was adjusted away. However, this limitation was considered to be less significant in light of the more considerable limitation presented by the small effect sizes for propensity score covariates on CPSA.

Unmeasured confounders

Because CHIWOS is a study of adults focusing primarily on variables measured at present, the ability to include variables from the eco-social intersectional model of child maltreatment was limited. Some notable variables that were unmeasured included: childhood health issues, caregiver (un)employment, family socioeconomic status, family isolation and household composition, and instability outside of the context of immigration. It is expected that the inclusion of a wider range of childhood variables would block additional non-causal pathways and result in a more accurately specified propensity score model. The implications of being unable to balance a more robust set of variables are that the weighting did not render the CPSA+ and CPSA- groups sufficiently balanced, and thus that the observed effects may have been attributable to unmeasured factors. This possibility was mitigated to the best of our abilities by controlling for factors not only identifiable as occurring during childhood (such as immigration prior to age 16), but for factors which are measured in adulthood but retrospectively known to affect childhood maltreatment (such as sexual and gender minority identities). Some results from the present analysis such as the impact of CPSA on further abuse in adulthood have been heavily documented in the literature (though not necessarily among women with HIV) and thus are not expected to change dramatically (i.e. in significance or direction) with additional adjustment. However, other results such as the effect of CPSA on HIV stigma represent novel additions to the quantitative literature, and should be both interpreted with caution and subjected to further scrutiny.

Collider-stratification bias

The CHIWOS sample, like most samples of people with HIV is a “selected population”, defined by the condition that every woman in the study has been diagnosed with HIV.
From a causal modelling perspective, a selected population may be problematic when the exposure of interest (CPSA) precedes the selection/stratification factor (here, HIV) along a causal pathway. In the present example, CPSA is a known risk factor for HIV, while the causal pathway of interest is that between CPSA and barriers to HIV disclosure. The issue of collider-stratification bias presents itself here when a common cause exists for both HIV and an outcome, making HIV a collider along the causal CPSA to outcome pathway. In this situation, effects observed between CPSA and the outcome among the selected population would be biased unless the common causes could be controlled. However, if no common causes of HIV and an outcome could be identified, then the selected population variable would not be a collider, and collider-stratification bias would not be an issue. Thus, for the present study, it is important to consider potential common causes of HIV and outcomes in order to evaluate the risk and potential consequences of collider-stratification bias. Because HIV risk has been studied extensively while some outcomes such as sexual relationship power represent emergent concepts, a logical strategy is to evaluate whether any of the causes of HIV may also cause the given outcomes. A review of broad risk factors for HIV revealed three categories of potential confounders as they relate to the present analysis: (1) those that were controlled by propensity score weighting, (2) those what were not controlled, and (3) those that represent mediators along the CPSA to outcome pathway.

Within the first category, sexual orientation and gender identity have been associated with differential HIV risk profiles, especially among MSM. Risk differences also exist among women, though these have been addressed less frequently in the literature. In particular, self-identified lesbian and bisexual women may differ from heterosexual women in terms of HIV testing and HIV risk behaviors. While differences between sexual minority and heterosexual women were addressed by propensity score weighting, differences between lesbian and bisexual women may have been obscured by collapsing these groups into a single category. Transgender women also likely differ from cisgender women in terms of HIV risk behaviours and testing, though formal comparisons between these populations are complicated by the frequent lack of explicit inclusion of gender diverse people in population-based health studies. These differences were addressed in the current study by propensity score weighting. Thus, it is unlikely
that the effects of sexual orientation and gender identity on HIV risk and study outcomes contributed to collider-stratification bias. However, knowledge about HIV is one factor which affects HIV risk\textsuperscript{253} as well as HIV stigma,\textsuperscript{254} and thus may have biased measures of effect for this outcome. Thus, it is possible that the observed positive relationship between CPSA and HIV stigma may have been partially attributable to HIV related knowledge. No explicit evaluation of HIV-related knowledge was available in the CHIWOS dataset, but future researchers should be cognizant of this potential bias.

The third category of confounders which could potentially induce collider-stratification bias are those which both affect HIV and outcomes, but may also be mediators along the causal pathway from CPSA to outcomes. These include factors influenced by CPSA, such as: alcohol use,\textsuperscript{255} injection drug use,\textsuperscript{256,257} and sex work.\textsuperscript{258} The existence of such factors introduces a methodological limitation: controlling for these variables would block causal pathways between the exposure and the outcome, while failing to control for them leaves the analysis vulnerable to collider-stratification bias and spurious associations. The consequence of this limitation in the present analysis is that the observed effects may have been attributable to the effect of CPSA on stratifier-outcome confounders. A review of methods to address such exposure-induced collider-stratification bias revealed no clear solution to this issue, as well as a lack of attention to collider-stratification bias more generally. Given the high prevalence of “selected populations” in clinical research, and the threat to validity posed by collider-stratification bias, this is an issue that should be addressed with further methodological work.

**Measures**

All measures in CHIWOS were reliant on self-report, which meant that results were vulnerable to potential response biases such as social desirability bias and recall bias. Given the emotionally burdensome content of the survey, social desirability bias may present the largest threat to measurement validity. For many reasons including embarrassment or mistrust of researchers, participants may have felt uncomfortable disclosing information on items related to experiences of abuse throughout the life course, as well as sexual relationship power and residential school experiences. Notably,
Indigenous women declined to answer the block of questions on residential school; given the Canada’s history of exploitative research against Indigenous peoples, this level of refusal was not unsurprising. While this was the largest identifiable example of item non-response in the survey and other item refusals were much smaller in magnitude, there was also the possibility for unidentifiable response bias. Most concerning given the subject matter was the possibility for under-reporting of adverse experiences such as physical or sexual abuse. Self-reporting of abuse is recognized as able to capture more cases of violence as compared to abuse reported to police or child protective services, however self-report measures are still vulnerable to response bias as well as recall bias. Multiple strategies were used by the CHIWOS team to mitigate such response bias, including: having interviews conducted by community-involved women with HIV in order to establish rapport, assuring participants of data confidentiality, allowing for self-completion of sensitive survey items, and using behavioural rather than definitional measures of self-reported violence.

No disclosure measure

Readers may wonder why no effort was made in the present thesis to establish links between not only CPSA and barriers to disclosure, but between barriers to disclosure and a measure of HIV disclosure to sexual partners. Given that much of the existing research on disclosure to sexual partners is qualitative in nature, such an extension would bolster the study’s relevance and make a valuable addition to the literature. However, it is worth noting that the Canadian legal context which justified the current study on barriers to HIV disclosure also made it unethical to measure HIV disclosure to sexual partners. The Canadian Criminal Code allows courts to subpoena information from those who may have information related to criminal cases. This information can include communications between people with HIV and clinicians, counsellors and researchers, depending on whether or not it was gained under certain circumstances which qualify the information as “privileged” (i.e. inadmissible in court). Even in situations where privacy and/or anonymity has been guaranteed by the researcher or service provider, information can sometimes be subpoenaed. Not only do these conditions this make research on non-disclosure ethically unacceptable, but they bar people with HIV from seeking
professional counsel related to HIV disclosure. Thus, the CHIWOS team made a conscious decision not to ask directly about HIV disclosure to sexual partners in order to protect their participants and PRAs from legal vulnerability. Instead, the team surveyed general knowledge and opinions on Canadian criminalization of non-disclosure, as well as barriers to HIV disclosure as operationalized in the current study.

5.3 Strengths

One of the main strengths of the given analysis was its innovative approach to the study of HIV disclosure via barriers and facilitators. Members of the CHIWOS team, legal practitioners specializing in HIV non-disclosure, and women with HIV in Canada have made it clear that the present study is well-justified and innovative, and that the results will be valuable in informing policy and practice. Actionability was a main goal of this thesis from the identification of research questions through design of the statistical methods and interpretation of the results. In line with this goal, the present study (1) adapted a theoretical framework which renders results (and future research on this topic) interpretable in ways that are non-stigmatizing and true to community experience; (2) centred effect estimates on CPSA+ women rather than the population as a whole in order to estimate the impact of targeted intervention; and (3) leveraged data that was rendered limited by the surrounding policy environment (i.e. no disclosure measure) in a way that sheds light on the inadequacy of that same policy. While this thesis endeavoured to produce causal effect estimates in order to accurately evaluate the level of socio-legal vulnerability directly attributable to adverse childhood experiences, the resulting adjusted risk estimates are still valuable. Both the descriptive and analytic results, while not necessarily representative of all women with HIV, indicated an incredibly high burden of recurring violence among marginalized women with HIV. Regardless of the causal root of these experiences, the majority of women in CHIWOS had experienced physical or sexual assault in adulthood, which translates easily into socio-legal vulnerability. While estimates of the level of disclosure barriers attributable to HIV may have been vulnerable to misspecification, this thesis succeeded in showing the heavy burden of violence in adulthood and consequent need for interventions to temper the impact of such violence, especially in the context of HIV criminalization.
This project also successfully responded to calls for research that conceptualizes violence as not only a cause of HIV, but as a cause of further vulnerability among women with HIV. Research on the causes and consequences of violence against women is plentiful, and the concepts relayed in this body of work are easily extendable to women with HIV. Furthermore, studying established concepts such as stigma and revictimization among women with HIV provides the opportunity to investigate not only whether such concepts hold across populations, but also how HIV may nuance these concepts. In 2018, when medical advances have significantly bridged the life expectancy gap between people with HIV and people without HIV, we must turn our focus on how people with HIV move through the world, which is often skewed against them. The extension of existing research paradigms to this population is one way to accomplish this. Here, the application of a suite of barriers to HIV disclosure and their attribution to distal factors outside of one’s own control represented a novel approach in the quantitative HIV literature. It is our hope that, on the basis of these promising initial results, further research will build on this conceptualization in an effort to necessarily complicate the legal system’s understanding of HIV disclosure and its antecedents.

5.4 Directions for Future Research

Directions for future research have been identified intermittently throughout this discussion, but will be briefly summarized in this section. In direct response to the potential shortcomings of the present study, it is of interest to estimate the effects of CPSA on barriers to disclosure while controlling for a more robust suite of potential confounders in order to block all identifiable non-causal pathways. This could be accomplished with access to a dataset that has a heavier focus on childhood experiences. Alternatively, shifting the approach to confounding control from blocking non-causal pathways proximal to the exposure to an approach controlling true confounders or parents of the outcome may present viable methods for estimating causal effects with datasets not focused on childhood variables. Furthermore, studies of HIV disclosure barriers should pay attention to measurement limitations in the present study, with an eye towards potentially developing new measures. The literature review identified several relevant constructs for which no measure existed (e.g. potential sexual relationship power), as
well as constructs for which the CHIWOS measure did not map neatly onto the construct of interest (e.g. perceived social support versus anticipated partner support), and multidimensional constructs that warranted further exploration (e.g. HIV stigma). If ethically permissible, it would also be ideal to formally study the relationship between the barriers to HIV disclosure and partner disclosure. Hopefully this will be possible in the future given changes to the legal circumstances surrounding HIV disclosure or research confidentiality.

The sensitivity analyses of the independent effects of CPA and CSA on barriers to HIV disclosure revealed the potential for interactive effects (i.e. poly-victimization) when compared to the main analysis of the effects of childhood physical or sexual abuse. Moving forward, researchers should think critically about whether a unitary approach to victimization is sufficient to address a given research question, and whether data will allow for the estimation of interaction effects between multiple types of adverse childhood experiences. These are not limited to childhood physical and sexual abuse, but may also include emotional abuse (when appropriately measured), exposure to intimate partner violence in childhood, and neglect. The relatively recent body of work on poly-victimization is compelling, and should be taken into consideration by researchers across disciplines where childhood adversities are relevant predictors.246

The present analysis also provided a hypothesized causal framework well-suited to mediation analysis. A wide range of factors may mediate the observed effects, and even the relationships where no significant effect was detected.264 For some outcomes where causal mechanisms have been well-established among the general population (e.g. known mediators between CSA and revictimization include dissociation, targetability, and risk-recognition),265 concept validation and extension among women with HIV should prove fairly straightforward. For others, such as the relationship between CPSA and HIV stigma among Black African women, mediation analysis represents uncharted territory. While navigating this territory, researchers should consider potential mediators that have proven to be salient determinants of other CPSA sequelae in the literature. These include coping strategies,266 exposure to interventions,267 self-blame,268 and family support.266 Mediation
analysis will prove extremely valuable in identifying modifiable factors to target for intervention.

5.5 Implications for Practice and Policy

Practice

The results of the present study in conjunction with prior research present several potential points for intervention, including enhancing protection for children, intervening on mediating pathways between CPSA and revictimization and/or HIV, and interventions for adults who have experienced CPSA with the goal of maintaining personal safety and understanding disclosure. In line with this study’s theoretical framework, any intervention should be accessible and acceptable to women and girls across all intersections of gender and ethnoracial group. In some cases, this will necessitate the development of intersection specific resources. Authors Etherington and Baker provide a useful guide for applying intersectionality theory in practice related to children exposed to intimate partner violence, which could be easily adapted to interventions related to CPSA.269 Institutions and individuals that are positioned to provide interventions and resources along this pathway include: child protection services, schools, women’s shelters, AIDS service organizations, physicians, psychologists, and other health/wellbeing practitioners. Some potential interventions focused on recognizing and reducing CPSA may include: educating children on personal autonomy and consent, providing access to parenting classes, educating parents and teachers on the signs of child abuse, recalibrating the child protection system to allow for lower caseloads, and providing intersectional anti-oppression training to those who work closely with children. Other interventions may focus on providing remedial support to children who have been victims of CPSA, including services such as professional counselling for children and their caregivers.

The results of this analysis also have implications specific to individuals and institutions that work with HIV positive women. However, service providers must be aware of how they as counsellors may open their HIV positive clients to legal vulnerability, and should receive education on the intricacies of disclosure related counselling before engaging in
any discussions on disclosure. The Canadian HIV/AIDS Legal Network in partnership with other organizations has developed an online resource intended to educate those who regularly work with people with HIV regarding client confidentiality, recommended record keeping practices, and counselling people with HIV on matters related to disclosure.\textsuperscript{263} This information is well disseminated among AIDS service organizations, but should be increasingly targeted towards general practitioners and other health care providers, who tend to engage less frequently in discussions of HIV criminalization with their HIV positive patients even though they may also be vulnerable to disclosure related legal action.\textsuperscript{270} On top of existing educational resources about HIV disclosure, the current project presents new information that may be informative for individuals who engage in disclosure counselling. Providers should understand that women with a history of CPSA may be at greater risk for further abuse at present, and thus may be legally vulnerable. At the individual level, this information may provide HIV positive women who have experienced CPSA with some peace of mind in knowing that their experiences negotiating partner disclosure are valid and at least partially rooted in factors outside of their own control. At present, steps are already being taken to incorporate this information into the harmonized volunteer training materials for AIDS service organizations across Toronto, and consultations are underway with legal practitioners at HIV & AIDS Legal Clinic Ontario (HALCO) to develop additional knowledge translation strategies.

The information collated in this thesis on the burden of violence and abuse among women with HIV merits further attention beyond knowledge translation strategies. Where they are not already doing to, AIDS service organizations and health care providers should offer programming teaching women with HIV who are at risk for violence about how the law has typically handled cases of HIV non-disclosure, and how they can best negotiate their bodily safety and legal vulnerability. A search for potential resources yielded several components of what would make for a larger and more comprehensive strategy. In 2015, HALCO developed a text-based resource for women with HIV who are experiencing or at risk of experiencing violence.\textsuperscript{271} This resource was intended to provide education on navigating the legal system as related to violence and HIV, including non-disclosure. The legal information contained in this resource is highly valuable and could
provide the basis for a more accessible (i.e. face-to-face) intervention related to violence against women with HIV. Additionally, a 2015 systematic review of interventions to facilitate HIV disclosure in the context of violence identified two randomized trials from Africa which both appeared to increase rates of partner disclosure, though the effect could not conclusively be attributed to the disclosure counselling components as they were both part of larger multifaceted interventions.\textsuperscript{272} The interventions included screening all new HIV diagnoses among women for histories of violence, followed by an evaluation of safety and best options for disclosure or non-disclosure if that was deemed the best option.\textsuperscript{273,274} Concurrent screening for HIV and intimate partner violence is already in practice in some Canadian clinics,\textsuperscript{275} though it is unclear the extent to which this approach has been adopted nationally. Furthermore, screening for violence represents only the first step in a comprehensive strategy to protect women with HIV from additional vulnerability. An ideal intervention would incorporate the already established strategies above, along with acknowledgement of intersectionality and social power in the educational material and in the delivery of the intervention to women from multiply marginalized populations.

\textbf{Policy}

The findings of this analysis indicated that women with HIV, like other women, are vulnerable to physical and sexual violence in adulthood, and that experiences of CPSA increase risk for such violence. This evidence, coupled with research establishing a relationship between CPSA and HIV risk among women reiterates that recurring violence is an issue within this community. Furthermore, in the context of criminalization of HIV non-disclosure, violence against women with HIV has socio-legal implications beyond the already devastating implications of violence against women in general. Experiences of physical, sexual, and emotional violence undermine the power of women and girls, including women’s power to disclose their HIV status to their sexual partners, whether or not the current relationship is abusive. Within an abusive relationship, the decision to disclose one’s HIV status is often a negotiation where acceptance, reputation, shelter, financial security, bodily safety, and even one’s own life are on the line. In most of Canada where HIV non-disclosure is a crime, women with HIV must also consider the
potential for criminal charges and incarceration when negotiating disclosure. Sadly, even if women do disclose their HIV status to their sexual partners, they are not protected from false legal accusations by abusive partners attempting to exact control or revenge.

What the current analysis provides is an additional piece within the larger body of evidence which tells us that children cannot cause their own abuse, girls who experience abuse are at risk for HIV as well as revictimization, and the co-occurrence of HIV and abuse makes HIV disclosure dangerous. When legal bodies ignore scientific evidence on HIV transmission and fail to recognize disclosure-related risks for people with HIV they are not only complacent to this cycle of abuse and vulnerability, but they enable it. As a consequence, these systems may be disproportionately punitive to those who are especially vulnerable based on early-life violence.

Steps are being take to remedy this as newer scientific evidence becomes incorporated into the legal system. Most recently, in December 2017 the Ontario Attorney General and Ontario Minister of Health and Long Term Care announced that in light of scientific developments, Ontario’s crown prosecutors would no longer pursue HIV non-disclosure cases where an individual has had a suppressed viral load for six months. This decision represents an important step towards achieving justice for people with HIV in Canada, however, the policy’s reach is limited to Ontario and does not yet address the legal precedent for other situations where HIV transmission risk is known to be negligible, including: oral sex, anal or vaginal sex with a condom, and anal or vaginal sex without a condom while having low viral load. A community consensus statement signed by over 150 Canadian organizations in 2017 further advocated for the application of non-disclosure law only as a last resort in the case of intentional HIV transmission, which is very rare. Their call to action included the following demands:

1. Federal and provincial Attorneys-General should develop sound prosecutorial guidelines to preclude unjust HIV prosecutions.
2. The federal government should reform the Criminal Code to limit the unjust use of the criminal law against people living with HIV.
3. All three levels of government should support the development of resources and training to address misinformation, fear and stigma related to HIV.
This position was echoed by the Department of Justice’s 2017 commissioned report on the criminal justice system’s response to non-disclosure of HIV. These advances are highly promising, and represent exciting potential for advancement of human rights in Canada.

5.6 Conclusion

In summary, the findings of this thesis confirmed that women with HIV in Canada experience a high burden of violence which may be at least partially attributable to earlier experiences of violence in childhood. Such experiences may also be associated with adverse outcomes in adulthood, such as low sexual relationship power or high HIV stigma, that disenable women from disclosing their HIV status to their sexual partners. These barriers and facilitators to HIV disclosure are especially relevant in Canada where, as of August 2018, people with HIV continue to be charged and convicted for HIV non-disclosure despite heavily documented scientific and ethical objections to this practice. While the current analysis was not able to ascertain causality, adjusted risk estimates provided some indication that women with HIV who experienced child abuse may be more vulnerable than women without such experiences to non-disclosure, and thus legal actions or threats. These results introduce the possibility that HIV non-disclosure law over-criminalizes women with HIV who experienced child abuse, punishing them for making what is deemed to be the wrong decision when their personal safety and autonomy is at risk. On the basis of this reasoning, interventions which restore autonomy to women with HIV by way of education, empowerment, and national policy change are recommended. This includes explicit decriminalization of HIV non-disclosure at the national level.
References


9. Mullen PEE, Martin JLL, Anderson JCC, Romans SEE, Herbison GPP. The long-


DOI:10.1037/12289-000.


92 Riley NE. Gender, power, and population change. *Popul Bull* 1997; **52**: [2], 1-48.


Straus MA, Hamby SL, Finkelhor D, Moore DW, Runyan D. Identification of Child Maltreatment With the Parent-Child Conflict Tactics Scales: Development


161 Turner HA, Finkelhor D, Ormrod R, *et al.* Family context, victimization, and child


Blackstock C. After the apology: Why are so many First Nations children still in foster care? A summary of the research on ethnic over-representation and structural bias. 2017. DOI:10.1017/S103507720000050X.


Skype. 2018.


Hume D. An Enquiry Concerning Human Understanding. 1748 DOI:10.1353/hms.0.0023.


Lunceford JK. Correction to: Stratification and weighting via the propensity score in estimation of causal treatment effects: a comparative study (Statistics in


Austin PC. The relative ability of different propensity score methods to balance measured covariates between treated and untreated subjects in observational studies. *Med Decis Mak* 2009; 29: 661–77.


203 Rubin DB. Using propensity scores to help design observational studies: 
Application to the tobacco litigation. *Health Serv Outcomes Res Methodol* 2001; 2: 
169–88.

204 Friedman JH. Greedy function approximation: a gradient boosting machine. *Source 

78.


207 Lee BK, Lessler J, Stuart EA. Improving propensity score weighting using 

208 Harder VSS, Stuart EA, Hopkins J, Anthony JC. Propensity score techniques and 
the assessment of measured covariate balance to test causal associations in 
psychological research. DOI:10.1037/a0019623.

209 Ridgeway G, McCaffrey D, Morral A, Burgette L, Griffin BA. Toolkit for 
weighting and analysis of nonequivalent groups: A tutorial for the twang package. 


https://www.r-project.org/.


213 McCaffrey DF, Griffin BA, Almirall D, Slaughter ME, Ramchand R, Burgette LF. 
A tutorial on propensity score estimation for multiple treatments using generalized 


Marcus SM, Gibbons RD. Caution should be used in applying propensity scores estimated in a full cohort to adjust for confounding in subgroup analyses: Commentary on “Applying propensity scores estimated in a full cohort to adjust for confounding in subgroup analyses”. *Pharmacoepidemiol Drug Saf* 2012; 21: 710–2.


Matsuda Y, McGrath JM, Jallo N. Use of the Sexual Relationship Power Scale in Research: An Integrative Review. *Hisp Heal Care Int* 2012; **10**: 175–89.


Halpern CT, Spriggs AL, Martin SL, Kupper LL. Patterns of Intimate Partner Violence Victimization from Adolescence to Young Adulthood in a Nationally Representative Sample. *J Adolesc Heal* 2009; 45: 508–16.


Logie C, James LL, Tharao W, Loutfy M. Associations Between HIV-Related Stigma, Racial Discrimination, Gender Discrimination, and Depression Among


268 Whiffen VE, Macintosh HB. Mediators of the Link between Childhood Sexual


271 Women living with HIV and intimate partner violence. 2016.


Appendices

Appendix 1: Hierarchy of HIV exposure categories

Source: Public Health Agency of Canada

1. MSM: Men who have sex with men. This category includes men who report either homosexual or bisexual sexual contact.
2. MSM/IDU: Men who have sex with men and use injection drugs.
3. IDU: Injection drug use.
4. Blood/blood products
   a. Recipient of blood/clotting factor: Before 1998, it was not possible to separate this exposure category. However, where possible, it has been separated into subcategories b and c.
   b. Recipient of blood: Received transfusion of whole blood or blood components, such as packed red cells, plasma, platelets, or cryoprecipitate.
   c. Recipient of clotting factor: Received pooled concentrates of clotting factor VIII or IX for treatment of hemophilia/coagulation disorder.
5. Heterosexual contact
   a. Origin from an HIV-endemic country (Het-Endemic): People who were born in a country where HIV is endemic. An HIV-endemic country is defined as having an adult (ages 15–49) prevalence of HIV that is 1.0% or greater and one of the following: • 50% or more of HIV cases attributed to heterosexual transmission • a male to female ratio of 2:1 or less • HIV prevalence greater than or equal to 2% among women receiving prenatal care Before 1998, it was not always possible to separate Origin from an HIV-endemic country and sexual contact with a person at risk. However, where possible, it has been separated into subcategories a and b.
   b. Sexual contact with a person at risk (Het-Risk): People who report heterosexual contact with someone who is either HIV-infected or who is at
increased risk of HIV infection (e.g. a person who injects drugs, a bisexual male, or a person from an HIV-endemic country).

c. No Identified Risk-Heterosexual (NIR-Het): If heterosexual contact is the only risk factor reported and nothing is known about the HIV-related factors associated with the partner, the case is classified as NIR–Het.

6. Occupational exposure: Exposure to HIV-contaminated blood or body fluids, or concentrated virus in an occupational setting. This applies only to reported AIDS cases and not to HIV-cases where the occupational exposure category is captured under “other”. The Canada Communicable Disease Report (CCDR) contains more information about occupational exposure.

7. Perinatal transmission: The transmission of HIV from a woman infected with HIV to her infant, either in utero, during childbirth, or through breastfeeding.

8. Other: Used to classify cases where the mode of HIV transmission is known but cannot be classified into any of the major exposure categories listed here; for example, a recipient of semen from an HIV-positive donor.

9. No identified risk (NIR): Used when the history of exposure to HIV through any of the other modes listed is unknown, or there is no reported history (e.g. because of death, or loss to follow-up).

10. Not reported: In certain provinces and territories, exposure categories are not reported to the Public Health Agency of Canada and are classified as Not Reported.
Appendix 2: SAS Code

File 1: Data cleaning, generating propensity scores, evaluating balance diagnostics

*Import macros;
%include 'U:\SAS\Macros\twang_mac.sas';
/*-IMPORT AND SORT W1/W2 DATA BY PART_ID*/
LIBNAME USB 'E:\Data';
DATA CHIWOSW1;
   SET USB.CHIWOSW1;
PROC SORT DATA=CHIWOSW1;
   BY PART_ID;
DATA CHIWOSW2;
   SET USB.CHIWOSW2;
PROC SORT DATA=CHIWOSW2;
   BY PART_ID;
/*CREATE TEMP DATASET WITH COMBINED W1+W2 by variable PART_ID (match-merging)*/
DATA CHIWOSW1_2;
   MERGE CHIWOSW1 CHIWOSW2;
      BY PART_ID;
/*DEFINE ALL ARRAYS. AAA=SRPS W1&2; BBB=Y,N,DK,PA; CCC=MOS-SSS; DDD=HIV Stigma W1&2; EEE=HAT-QoL-Disclosure; FFF=HIV Stigma w1&w2; GGG=HAT QoL Disclosure W1&W2;*/
ARRAY AAA S8Q24j_1-S8Q24j_15 W2S9Q15_0-W2S9Q15_14;
ARRAY BBB S7Q2a W2S8Q02 S7Q5a W2S8Q14 S7Q6a S7Q8a S1Q5a S1Q24 S1Q26 S1Q27 S1Q80_1-S1Q80_7;
ARRAY CCC S9Q4_1-S9Q4_4;
ARRAY DDD S5Q1_1-S5Q1_10 W2S6Q01_0-W2S6Q01_9;
ARRAY EEE S5Q5_1-S5Q5_6 W2S6Q02_0-W2S6Q02_5;
ARRAY FFF S5Q1_1-S5Q1_10 W2S6Q01_0-W2S6Q01_9;
ARRAY GGG S5Q5_1-S5Q5_5 W2S6Q02_0-W2S6Q02_4;
/*------------------- REMOVE MISSINGS -------------------*/
DO OVER AAA;
   IF AAA=5 then AAA=.;
END;
DO OVER BBB;
   IF BBB= 3 then BBB=.;
   IF BBB= 4 then BBB=.;
END;
DO OVER CCC;
   IF CCC= 6 then CCC=.;
   IF CCC= 7 then CCC=.;
END;
DO OVER DDD;
   IF DDD= 6 then DDD=.;
END;
DO OVER EEE;
   IF EEE= 6 then EEE=.;
END;
/*---------------- OUTCOMES ----------------*/
*(a) Sexual Relationship Power Scale (SRPS) Relationship Control Subscale Waves 1 and 2; *
1. Summing total of 15 items [CHECKED];
sum_SRPSw1 = sum(of S8Q24j_1-S8Q24j_15);
sum_SRPSw2 = sum(of W2S9Q15_0-W2S9Q15_14);
2. Determining number of items answered;
total_SRPSw1 = n(of S8Q24j_1-S8Q24j_15);
total_SRPSw2 = n(of W2S9Q15_0-W2S9Q15_14);
3 Calculating scale value as SUM of items/#nonmissing items (average of answered items).
*When instructions they say missing items *automatically* assigned mean score for completed items, this is what they mean;
SRPSw1 = sum_SRPSw1/total_SRPSw1; *Score is average across completed items;
SRPSw2 = sum_SRPSw2/total_SRPSw2;
*Those missing 3 or more (15-3=12) items are dropped;
if total_SRPSw1 le 12 then SRPSw1=.; *Missing SRPSw1=;
if total_SRPSw2 le 12 then SRPSw2=.; *Missing SRPSw2=;

/*(b) Adult physical abuse, ever (W1 ever, W2 since last CHIWOS interview) W1: S7Q2a, W2: W2S8Q02
If w1 missing and w2 is no --> missing. If w1 is missing and w2 is yes --> yes. If w2 is missing always default to w1 answer*/
APABUSE=.;
if S7Q2a=2 and W2S8Q02=2 then APABUSE=0; *no adult physical abuse ever;
if S7Q2a=1 and W2S8Q02=2 then APABUSE=.; *n=65 if w1 missing and w2 (since) is no --> missing since this is not "ever";
if S7Q2a=2 and W2S8Q02=1 then APABUSE=0; *no adult physical abuse ever as reported to CHIWOS;
if S7Q2a=1 or W2S8Q02=1 then APABUSE=1; *yes adult physical abuse ever (including if w1 missing and w2(since) is yes --> if happened since, this is part of "ever";
if S7Q2a=1 and W2S8Q02=2 then APABUSE=.; *if both waves missing, var is missing;

*Checking source of missingness;
if s7q1=1 then w1section7=1;
if s7q1=2 then w1section7=2;
if s7q1=3 then w1section7=.;
if w2s8q01=1 then w2section8=1;
if w2s8q01=2 then w2section8=2;
if w2s8q01=3 then w2section8=.;

/*@c) Adult sexual abuse (W1 ever, W2 since last CHIWOS interview) W1: S7Q5a, W2: W2S8Q14 [CHECKED]
If w1 missing and w2 is no --> missing. If w1 is missing and w2 is yes --> yes. If w2 is missing always default to w1 answer*/
ASABUSE=.;
if S7Q5a=2 and W2S8Q14=2 then ASABUSE=0; *no adult sexual abuse ever;
if S7Q5a=1 and W2S8Q14=2 then ASABUSE=.; *if w1 missing and w2 (since) is no --> missing since this is not "ever";
if S7Q5a=2 and W2S8Q14=1 then ASABUSE=0; *no adult sexual abuse ever as reported to CHIWOS;
if S7Q5a=1 or W2S8Q14=1 then ASABUSE=1; *yes adult sexual abuse ever (including if w1 missing and w2(since) is yes --> if happened since, this is part of "ever";
if S7Q5a=1 and W2S8Q14=2 then ASABUSE=.; *if both waves missing, var is missing;
*(d) Social Support [Medical Outcomes Study – Social Support Scale 4 item]. W1: S9Q4_1 to S9Q4_4, DV: MOS_SSS_Score (W1).*

1. Reverse code all items (not officially RC, response options just asked in opposite order in CHIWOS) (see http://www.theanalysisfactor.com/easy-reverse-code/);
   - S9Q4_RC_1 = 6 - S9Q4_1;
   - S9Q4_RC_2 = 6 - S9Q4_2;
   - S9Q4_RC_3 = 6 - S9Q4_3;
   - S9Q4_RC_4 = 6 - S9Q4_4;

2. Summing all 4 items;
   - MOSSw1 = sum(of S9Q4_RC_1 - S9Q4_RC_4);

3. Determining number of items answered;
   - total_MOSSw1 = n(of S9Q4_RC_1 - S9Q4_RC_4);

4. If any item is missing, drop from analysis;
   - if total_MOSSw1 lt 4 then MOSSw1 = .

*(e) HIV Stigma Scale W1: S5Q1_1 to S5Q1_10; W2: W2S6Q01_0 to W2S6Q01_9; DV: None*

1. Recode response options [CHECKED];
   - DO OVER FFF;
     - FFF = 5 - FFF;
   - END;

2. Sum all 10 items for each wave [CHECKED: scales range from 0-40];
   - STIGMAw1 = sum(of S5Q1_1 - S5Q1_10);
   - STIGMAw2 = sum(of W2S6Q01_0 - W2S6Q01_9);

3. If missing 2 or more Items on scale of 10, drop from analysis;
   - total_STIGMAw1 = n(of S5Q1_1 - S5Q1_10);
   - total_STIGMAw2 = n(of W2S6Q01_0 - W2S6Q01_9);
   - if total_STIGMAw1 le 8 then STIGMAw1 = .;
   - if total_STIGMAw2 le 8 then STIGMAw2 = .;

*(g)Sexual Inactivity (6 months) W2: S902 (Have you had consensual sex in the past 6 months 1 yes 2 no 3 DKPN);
   - if W2S9Q02=1 then inactivityw2=0; *Yes to consensual sex --> not inactive;
   - if W2S9Q02=2 then inactivityw2=1; *No to consensual sex --> not active;
   - if W2S9Q02=. then inactivityw2=.;

/*------------------- EXPLANATORY VARIABLES ------------------*/

/*1a. CPSA  W1: S7Q6a [physical] W1: S7Q8a [sexual]*/

CPSA = .;
   - if S7Q6a=2 and S7Q8a=2 then CPSA=0; *no physical or sexual abuse;
   - if S7Q6a=1 or S7Q8a=1 then CPSA=1; *yes physical and/or sexual abuse;
   - if S7Q6a=. and S7Q8a=. then CPSA=.; *if both are missing, var is missing;

/*1b. Child physical abuse S7Q6a*/
   - if S7Q6a=2 then CPA=0; *no physical abuse;
   - if S7Q6a=1 then CPA=1; *yes physical abuse;
   - if S7Q6a=. then CPA=.;

/*1c. Child sexual abuse S7Q8a*/
   - if S7Q8a=2 then CSA=0; *no sexual abuse;
   - if S7Q8a=1 then CSA=1; *yes sexual abuse;
   - if S7Q8a=. then CSA=.;
/*2. Race/ethnicity W1: S1Q7_1 to S1Q7_18 and S1Q7_18_sp (other,spec). Black african=S1Q7_3, Black Caribbean=S1Q7_4, Black Other=S1Q7_5, DV: S1Q7_dv. */
RACE=.;
if S1Q7_dv=1 then RACE=1; *Aboriginal;
if S1Q7_dv=2 and S1Q7_3=3 then RACE=2; *Black African;
if S1Q7_dv=2 and S1Q7_4=4 then RACE=3; *Black Caribbean;
if S1Q7_dv=2 and S1Q7_5=5 and S1Q7_3=3 and S1Q7_4=. then RACE=5; *Black other with no other black ID=other [5/11 "Black other" fall into this category];
if S1Q7_dv=3 then RACE=4; *Caucasian;
if S1Q7_dv=4 then RACE=5; *Other;
if S1Q7_dv=. then RACE=.;

/*/---------- PS VARIABLES ----------*/
/*1. Gender Identity. W1/DV: S1Q2a_dv. Combine transwomen and other [checked]*/
GENDERID=.;
if S1Q2a_dv=1 then GENDERID=0; *cis women;
if S1Q2a_dv=2 or S1Q2a_dv=3 then GENDERID=1; *trans women and other;
if S1Q2a_dv=. then GENDERID=.;

/*2. Leaving home country at or before age 16. W1: S1Q5a (Were you born in Canada); W1: S1Q5c_1 (in what year did you come to Canada to live); W1: S0QDOB_Y (Year of birth) [CHECKED]*/
*A. Create var for age of immigration (year of imm to canada subtract birth year);
IMMAGE=sum(S1Q5c_1-S0QDOB_Y);
*B. Dichotomize IMMAGE into le 16, gt 16;
if IMMAGE le 16 then IMLE16=1; *immigrated at or before 16;
if IMMAGE gt 16 or S1Q5a=1 then IMLE16=0; *immigrated after 16 or not an immigrant;
if S1Q5a=. or S0QDOB_Y=. then IMLE16=.;
if PART_ID in(REDACTED FOR DATA SECURITY) then IMLE16=.; *immigrants without year of immigration;
if PART_ID in REDACTED FOR DATA SECURITY) then IMLE16=0; *Non-immigrants without year of birth;
if PART_ID in (REDACTED FOR DATA SECURITY) then IMLE16=.; *immigrants without year of birth;

/*3. Stress-related reason for immigration during childhood. W2:
W2S1Q14_1 to W2S1Q14_16 and W2S1Q14_14_SP (Reason for moving to Canada - check all)*/
*if IMLE16=1;
*A. Create non age-restricted stress related immigration var;
if S1Q5a=1 then IMSTRESS=0; *not an immigrant;
if S1Q5a ne 1 and W2S1Q14_3 ne 3 and W2S1Q14_7 ne 7 and W2S1Q14_8 ne 8 and W2S1Q14_9 ne 9 and W2S1Q14_10 ne 10 and W2S1Q14_11 ne 11 then IMSTRESS=0; *immigrant w/no stress related reason;
if W2S1Q14_3=3 or W2S1Q14_7=7 or W2S1Q14_8=8 or W2S1Q14_9=9 or W2S1Q14_10=10 or W2S1Q14_11=11 then IMSTRESS=1; *yes any stress related reason;
if S1Q5a=. then IMSTRESS=.; *if born in Canada is missing, item is missing;
if S1Q5a=2 and W2S1Q14_1= and W2S1Q14_2= and W2S1Q14_3= and W2S1Q14_4= and W2S1Q14_5= and W2S1Q14_6= and W2S1Q14_7= and W2S1Q14_8= and W2S1Q14_9= and W2S1Q14_10= and W2S1Q14_11= and W2S1Q14_12= and W2S1Q14_13= and W2S1Q14_14= and W2S1Q14_15= and W2S1Q14_16= then IMSTRESS=; *immigrant w/no answers for reason of immigration;
if PART_ID in(REDACTED FOR DATA SECURITY) then IMSTRESS=1; *other specify indicating stress where not captured in quant vars;
*B. Create IMSTRESS <=16;
if IMLE16=0 or IMSTRESS=0 then IMSTRESS16=0; *didn't immigrate at/before 16 or didn't experience immigration stress;
if IMLE16=1 and IMSTRESS=1 then IMSTRESS16=1; *immigrated at/before 16 AND experienced immigration stress;
if IMLE16=. OR IMSTRESS=. then IMSTRESS16=.;

/*4. Attendance at residential school W1: RACE (aboriginal=1), S1Q8k (skip section?), S1Q8L (did you attend)*/
if RACE ne 1 then SELFATTEND=0; *not aboriginal in Canada;
if RACE=1 and S1Q8k=2 then SELFATTEND=.; *Aboriginal but skip residential school section;
if RACE=1 and S1Q8k=1 and S1Q8L=2 then SELFATTEND=0; *aboriginal, answered, didn't attend;
if RACE=1 and S1Q8k=1 and S1Q8L=1 then SELFATTEND=1; *aboriginal, answered, attended;
if RACE=1 and S1Q8k=1 and (S1Q8L=4 or S1Q8L=.) then SELFATTEND=.; *aboriginal, answered, missing self attendance;

/*5. Any family attended residential school*/
if S1Q8O_dv=0 then FAMATTEND=0; *not aboriginal in Canada;
if S1Q8O_dv=1 then FAMATTEND=1; *at least 1 fam said yes;
if S1Q8O_dv=2 then FAMATTEND=0; *all family members said no;
if S1Q8O_dv=5 then FAMATTEND=0; *does not id as first nation;
if S1Q8O_dv=8 then FAMATTEND=.; *skipped rschool questions;
if S1Q8O_dv=9 then FAMATTEND=.; *DK/PA;

/*6. Sexual orientation - S1Q3_dv (prev sex minority = 12.66%)*/
if S1Q3_dv=1 then sexid=0; *heterosexual;
if S1Q3_dv=2 then sexid=1; *not heterosexual;
if S1Q3_dv=9 then sexid=.; *missing;

/*7. Age. var=age*/

/*Remove obs with no outcome data (n=115) and unused vars*/
DATA mainanalysis;
  SET CHIWOSW1_2(KEEP=CPSA CSA CPA GENDERID IMLE16 IMSTRESS16 SELFATTEND FAMATTEND PART_ID RACE SRPSw1 SRPSw2 APABUSE ASABUSE MOSSw1 STIGMAW1 STIGMAW2 inactivityw2 sexid age);
  IF CPSA ne .;

/---------------- END OF DATA STEP "----------------*/

/-------------------Analysis with TWANG Macros "-------------------*/
*ESTIMATE PSs USING GBM, EVALUATE QUALITY USING COVARIATE BALANCE;
%ps(treatvar=CPSA,
vars=GENDERID IMLE16 IMSTRESS16 SELFATTEND FAMATTEND sexid age,
class=GENDERID IMLE16 IMSTRESS16 SELFATTEND FAMATTEND sexid,
dataset=mainanalysis,
ntrees=5000,
intdepth=3,
shrinkage=0.01,
permtsetiters=500,
stopmethod=ks.mean es.mean,
sampw=,
estimand=ATT,
output_dataset=USB.mainanalysis,
Rcmd=C:\Program Files\R\R-3.4.3\bin\x64\R.exe,
plotname=Weight_QualityCPSA.pdf,
objpath=U:\SAS\TWANG);
RUN;
/*----------------- END OF PS CODE -----------------*/

File 2: Outcome analysis

*IMPORT AND SORT W1/W2 DATA BY PART_ID;
LIBNAME USB 'E:\Data';
DATA chiwosw1_2_attwgtsv7;
   SET USB.chiwosw1_2_attwgtsv7;

*Indicator variables for missingness of PS vars;
ARRAY AAA age sexid genderid imle16 imstress16 selfattend famattend;
ARRAY BBB ageNA sexidNA genderidNA imle16NA imstress16NA selfattendNA famattendNA;
do over AAA;
   if AAA=. then BBB=1;
   if AAA=1 then BBB=0;
   if AAA=0 then BBB=0;
end;

*recode race with white reference group;
if race=4 then raceref=0; *white;
if race=1 then raceref=4; *Indigenous;
if race=2 then raceref=3; *Black African;
if race=3 then raceref=2; *Black Caribbean;
if race=5 then raceref=1; *Other;
if race=. then raceref=.;
LABEL raceref="Race ((4=Indig, 3=BA, 2=BC, 1=Other, 0=White))";

/*/----------------- END OF DATA STEP ----------------*/

PROC SORT DATA=chiwosw1_2_attwgtsv7;
   BY descending raceref descending CPSA;

/*/----------------- APABUSE (BINARY) ----------------*/
PROC GENMOD DATA=chiwosw1_2_attwgtsv7 order=data
de;
**APABUSE: Unw, Slice**

```plaintext
PROC GENMOD DATA=chiwosw1_2_attwgtsv7 order=data desc;
CLASS PART_ID raceref CPSA;
MODEL APABUSE=CPSA|raceref /dist=poisson link=log;
WEIGHT ks_mean_att;
REPEATED sub=PART_ID / type=ind;
SLICE CPSA*raceref /sliceby=raceref exp diff cl plots=none; *stratum specific effects;
TITLE "APABUSE: Weighted, slice, relative";
```

**APABUSE: Weighted, OVERALL**

```plaintext
PROC GENMOD DATA=chiwosw1_2_attwgtsv7 order=data desc;
CLASS PART_ID raceref CPSA;
MODEL APABUSE=CPSA|raceref /dist=poisson link=log;
WEIGHT ks_mean_att;
REPEATED sub=PART_ID / type=ind;
SLICE CPSA*raceref /sliceby=raceref exp diff cl plots=none; *stratum specific effects;
TITLE "APABUSE: Weighted, OVERALL";
```

**ASABUSE (BINARY)**

```plaintext
PROC GENMOD DATA=chiwosw1_2_attwgtsv7 order=data desc;
CLASS PART_ID raceref CPSA;
MODEL ASABUSE=CPSA|raceref /dist=poisson link=log;
WEIGHT ks_mean_att;
REPEATED sub=PART_ID / type=ind;
SLICE CPSA*raceref /sliceby=raceref exp diff cl plots=none; *stratum specific effects;
TITLE "ASABUSE: Weighted, slice, relative";
```

**ASABUSE: Weighted, OVERALL**

```plaintext
PROC GENMOD DATA=chiwosw1_2_attwgtsv7 order=data desc;
CLASS PART_ID raceref CPSA;
MODEL ASABUSE=CPSA|raceref /dist=poisson link=log;
WEIGHT ks_mean_att;
REPEATED sub=PART_ID / type=ind;
ESTIMATE 'Beta' CPSA 1 / exp;
TITLE "ASABUSE: Weighted, OVERALL";
```

**INACTIVITYW2 (BINARY)**

```plaintext
PROC GENMOD DATA=chiwosw1_2_attwgtsv7 order=data desc;
CLASS PART_ID raceref CPSA;
MODEL ASABUSE=CPSA|raceref /dist=poisson link=log;
WEIGHT ks_mean_att;
REPEATED sub=PART_ID / type=ind;
SLICE CPSA*raceref /sliceby=raceref exp diff cl plots=none; *stratum specific effects;
TITLE "INACTIVITYW2: Weighted, slice, relative";
```

**INACTIVITYW2: Weighted, OVERALL**

```plaintext
PROC GENMOD DATA=chiwosw1_2_attwgtsv7 order=data desc;
CLASS PART_ID raceref CPSA;
MODEL ASABUSE=CPSA /dist=poisson link=log;
WEIGHT ks_mean_att;
REPEATED sub=PART_ID / type=ind;
ESTIMATE 'Beta' CPSA 1 / exp;
TITLE "INACTIVITYW2: Weighted, OVERALL";
```
MODEL INACTIVITYW2=CPSA|raceref /dist=poisson link=log;
REPEATED sub=PART_ID / type=ind;
SLICE CPSA*raceref /sliceby=raceref exp diff cl plots=none; *stratum specific effects;
TITLE "INACTIVITYW2: Unw, Slice";

PROC GENMOD DATA=chiwosw1_2_attwgtsv7 order=data desc;
CLASS PART_ID raceref CPSA;
MODEL INACTIVITYW2=CPSA|raceref /dist=poisson link=log;
WEIGHT ks_mean_att;
REPEATED sub=PART_ID / type=ind; *gives risk estimates relative to white women;
SLICE CPSA*raceref /sliceby=raceref exp diff cl plots=none; *stratum specific effects;
TITLE "INACTIVITYW2: Weighted, slice, relative";

PROC GENMOD DATA=chiwosw1_2_attwgtsv7 order=data desc;
CLASS PART_ID;
MODEL INACTIVITYW2=CPSA /dist=poisson link=log;
WEIGHT ks_mean_att;
REPEATED sub=PART_ID / type=ind;
ESTIMATE 'Beta' CPSA 1 / exp;
TITLE "INACTIVITYW2: Weighted, OVERALL";

PROC GENMOD DATA=chiwosw1_2_attwgtsv7 order=data;
CLASS PART_ID raceref CPSA;
MODEL SRPSw2=CPSA|raceref /dist=normal link=id;
WEIGHT ks_mean_att;
REPEATED sub=PART_ID / type=ind; *gives risk estimates relative to white women;
SLICE CPSA*raceref /sliceby=raceref diff cl plots=none; *stratum specific effects;
TITLE "SRPSw2: Unw, Slice";

PROC GENMOD DATA=chiwosw1_2_attwgtsv7 order=data;
CLASS PART_ID raceref CPSA;
MODEL SRPSw2=CPSA /dist=normal link=id;
WEIGHT ks_mean_att;
REPEATED sub=PART_ID / type=ind; *gives risk estimates relative to white women;
SLICE CPSA*raceref /sliceby=raceref diff cl plots=none; *stratum specific effects;
TITLE "SRPSw2: Weighted, slice, relative";

PROC GENMOD DATA=chiwosw1_2_attwgtsv7 order=data;
CLASS PART_ID;
MODEL SRPSw2=CPSA /dist=normal link=id;
WEIGHT ks_mean_att;
REPEATED sub=PART_ID / type=ind;
ESTIMATE 'lincomb' intercept 1 CPSA 1;
TITLE "SRPSw2: Weighted, OVERALL";

PROC GENMOD DATA=chiwosw1_2_attwgtsv7 order=data;
CLASS PART_ID raceref CPSA;
MODEL MOSSw1=CPSA|raceref /dist=normal link=id;
REPEATED sub=PART_ID / type=ind;
SLICE CPSA*raceref /sliceby=raceref diff cl plots=none; *stratum specific effects;
TITLE "MOSSwl: Unw, Slice";
PROC GENMOD DATA=chiwoswl_2_attwgtsv7 order=data;
CLASS PART_ID raceref CPSA;
MODEL MOSSwl=CPSA|raceref /dist=normal link=id;
WEIGHT ks_mean_att;
REPEATED sub=PART_ID / type=ind; *gives risk estimates relative to white women;
SLICE CPSA*raceref /sliceby=raceref diff cl plots=none; *stratum specific effects;
TITLE "MOSSwl: Weighted, slice, relative";
PROC GENMOD DATA=chiwoswl_2_attwgtsv7 order=data;
CLASS PART_ID;
MODEL MOSSwl=CPSA /dist=normal link=id;
WEIGHT ks_mean_att;
REPEATED sub=PART_ID / type=ind;
ESTIMATE 'lincomb' intercept 1 CPSA 1;
TITLE "MOSSwl: Weighted, OVERALL";

PROC GENMOD DATA=chiwoswl_2_attwgtsv7 order=data;
CLASS PART_ID raceref CPSA;
MODEL STIGMAwl=CPSA|raceref /dist=normal link=id;
REPEATED sub=PART_ID / type=ind;
SLICE CPSA*raceref /sliceby=raceref diff cl plots=none; *stratum specific effects;
TITLE "STIGMAwl: Unw, Slice";
PROC GENMOD DATA=chiwoswl_2_attwgtsv7 order=data;
CLASS PART_ID raceref CPSA;
MODEL STIGMAwl=CPSA|raceref /dist=normal link=id;
WEIGHT ks_mean_att;
REPEATED sub=PART_ID / type=ind; *gives risk estimates relative to white women;
SLICE CPSA*raceref /sliceby=raceref diff cl plots=none; *stratum specific effects;
*SLICE CPSA*raceref /sliceby=CPSA exp diff cl plots=none; *pairwise if necessary;
TITLE "STIGMAwl: Weighted, slice, relative";
PROC GENMOD DATA=chiwoswl_2_attwgtsv7 order=data;
CLASS PART_ID;
MODEL STIGMAwl=CPSA /dist=normal link=id;
WEIGHT ks_mean_att;
REPEATED sub=PART_ID / type=ind;
ESTIMATE 'lincomb' intercept 1 CPSA 1;
TITLE "STIGMAwl: Weighted, OVERALL";

*MISSINGNESS OF PS COVARIATES;
PROC SURVEYFREQ DATA=chiwoswl_2_attwgtsv7;
tables ageNA sexid sexidNA genderid genderidNA imle16 imle16NA imstress16 imstress16NA selfattend selfattendNA famattend famattendNA /row;
title "Missingness of PS Covariates";

*BALANCE DIAGNOSTICS;
PROC SORT DATA=chiwosw1_2_attwgtsv7;
   BY CPSA;

PROC SURVEYFREQ DATA=chiwosw1_2_attwgtsv7;
   tables CPSA*(sexid sexidNA genderid genderidNA imle16 imle16NA imstress16 imstress16NA selfattend selfattendNA famattend famattendNA)
      /row wchisq;
   weight ks_mean_att;
   title "Weighted balance diagnostics for binary PS vars";

PROC SURVEYFREQ DATA=chiwosw1_2_attwgtsv7;
   tables CPSA*(ageNA sexid sexidNA genderid genderidNA imle16 imle16NA imstress16 imstress16NA selfattend selfattendNA famattend famattendNA)
      /row wchisq;
   title "Unweighted balance diagnostics for binary PS vars";

PROC TTEST;
   class cpsa;
   var age;
   weight ks_mean_att;
   title "Weighted balance diagnostic for age";

PROC TTEST;
   class cpsa;
   var age;
   title "Unweighted balance diagnostic for age";

*BALANCE DIAGNOSTICS BY ETHNORACIAL (i.e. testing for differences across CPSA within ethnoracial groups);

PROC SORT DATA=chiwosw1_2_attwgtsv7;
   BY race;

PROC SURVEYFREQ DATA=chiwosw1_2_attwgtsv7;
   tables CPSA*(sexid sexidNA genderid genderidNA imle16 imle16NA imstress16 imstress16NA selfattend selfattendNA famattend famattendNA)
      /row wchisq;
   weight ks_mean_att;
   by race;
   title "Weighted balance diagnostics for binary PS vars";

PROC SURVEYFREQ DATA=chiwosw1_2_attwgtsv7;
   tables CPSA*(ageNA sexid sexidNA genderid genderidNA imle16 imle16NA imstress16 imstress16NA selfattend selfattendNA famattend famattendNA)
      /row wchisq;
   by race;
   title "Unweighted balance diagnostics for binary PS vars";

PROC TTEST;
   by race;
   class cpsa;
var age;
weight ks_mean_att;
title "Weighted balance diagnostic for age by ethnoracial";

PROC TTEST;
by race;
class cpsa;
var age;
title "Unweighted balance diagnostic for age by ethnoracial";
RUN;
Appendix 3: GBM optimization for main CPSA analysis, ES mean and KS mean stopping points

“Balance measure” represents the absolute standardized mean difference (ES mean) and the mean Kolmogorov Smirnov test statistic (KS mean), respectively. Figures created using RAND corporation’s TWANG macro.  

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Appendix 4: Boxplots of propensity scores by exposure group

“Treatment” indicates CPSA status with 2 representing the CPSA+ group and 1 representing the CPSA- group. Figures created using RAND corporation’s TWANG macro.209

Boxplot 1: Propensity score overlap for main CPSA analysis, ES mean and KS mean stopping points
Boxplot 2: Propensity score overlap for CPA sensitivity analysis, KS mean stopping point

Boxplot 3: Propensity score overlap for CSA sensitivity analysis, KS mean stopping point
Appendix 5: Absolute standardized effect size differences pre and post AEE weighting

<table>
<thead>
<tr>
<th>es.mean.ATT</th>
<th>ks.mean.ATT</th>
</tr>
</thead>
<tbody>
<tr>
<td><img src="image" alt="Graph" /></td>
<td></td>
</tr>
</tbody>
</table>

ES mean refers to the absolute standardized effect size difference, while KS mean refers to the mean Kolmogorov Smirnov test statistic. Blue lines represent decreases in absolute standardized difference for covariates across exposure groups post-weighting, while red lines represent increases post-weighting. An absolute standard difference ≤0.20 is considered balanced. Figures created using RAND corporation’s TWANG macro.209
## Appendix 6: Stratum-specific balance diagnostics

### Table 1: Unweighted stratum-specific balance diagnostics for effect of CPSA

<table>
<thead>
<tr>
<th>PS Covariate</th>
<th>Indigenous</th>
<th>Black African</th>
<th>Black Caribbean</th>
<th>White</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age - years (SE)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CPSA-</td>
<td>37.9 (1.46)</td>
<td>43.1 (0.77)</td>
<td>45.4 (2.13)</td>
<td>44.0 (0.89)</td>
<td>43.4 (1.87)</td>
</tr>
<tr>
<td>CPSA+</td>
<td>40.9 (0.62)</td>
<td>39.8 (0.79)</td>
<td>45.1 (1.79)</td>
<td>44.7 (0.55)</td>
<td>43.6 (1.16)</td>
</tr>
<tr>
<td>P-value</td>
<td>0.0598</td>
<td>0.0032</td>
<td>0.9196</td>
<td>0.4957</td>
<td>0.9492</td>
</tr>
<tr>
<td><strong>Sexual Minority - % (SE)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CPSA-</td>
<td>17.9 (4.69)</td>
<td>1.32 (0.93)</td>
<td>2.94 (2.92)</td>
<td>7.28 (1.81)</td>
<td>10.3 (5.68)</td>
</tr>
<tr>
<td>CPSA+</td>
<td>21.8 (2.79)</td>
<td>6.71 (2.05)</td>
<td>10.0 (5.52)</td>
<td>19.3 (2.14)</td>
<td>13.51 (3.99)</td>
</tr>
<tr>
<td>P-value</td>
<td>0.4753</td>
<td>0.0175</td>
<td>0.2627</td>
<td>&lt;.0001</td>
<td>0.6495</td>
</tr>
<tr>
<td><strong>Gender Minority - % (SE)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CPSA-</td>
<td>5.97 (2.90)</td>
<td>0.66 (0.66)</td>
<td>0</td>
<td>2.90 (1.17)</td>
<td>3.44 (3.40)</td>
</tr>
<tr>
<td>CPSA+</td>
<td>6.76 (1.69)</td>
<td>0</td>
<td>10.0 (5.52)</td>
<td>4.68 (1.14)</td>
<td>16.2 (4.31)</td>
</tr>
<tr>
<td>P-value</td>
<td>0.8148</td>
<td>N/A</td>
<td>N/A</td>
<td>0.2765</td>
<td>0.0253</td>
</tr>
<tr>
<td><strong>Immigrated at or before age 16 - % (SE)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CPSA-</td>
<td>-</td>
<td>2.67 (1.32)</td>
<td>9.09 (5.04)</td>
<td>4.37 (1.43)</td>
<td>10.3 (5.68)</td>
</tr>
<tr>
<td>CPSA+</td>
<td>-</td>
<td>4.76 (1.76)</td>
<td>33.3 (8.68)</td>
<td>3.81 (1.04)</td>
<td>24.7 (5.07)</td>
</tr>
<tr>
<td>P-value</td>
<td>-</td>
<td>0.3413</td>
<td>0.0187</td>
<td>0.7524</td>
<td>0.0672</td>
</tr>
<tr>
<td><strong>Immigration-related stress at or before age 16 - % (SE)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CPSA-</td>
<td>-</td>
<td>0.74 (0.74)</td>
<td>0</td>
<td>0.49 (0.49)</td>
<td>7.69 (5.25)</td>
</tr>
<tr>
<td>CPSA+</td>
<td>-</td>
<td>3.05 (1.51)</td>
<td>11.5 (6.32)</td>
<td>0.30 (0.30)</td>
<td>10.4 (3.76)</td>
</tr>
<tr>
<td>P-value</td>
<td>-</td>
<td>0.1692</td>
<td>N/A</td>
<td>0.7341</td>
<td>0.6710</td>
</tr>
<tr>
<td><strong>Survivor of residential school - % (SE)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CPSA-</td>
<td>8.70 (5.90)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>CPSA+</td>
<td>7.92 (2.70)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>P-value</td>
<td>0.9051</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td><strong>Family member(s) attended residential school - % (SE)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CPSA-</td>
<td>51.7 (9.31)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>CPSA+</td>
<td>67.5 (4.29)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>P-value</td>
<td>0.1331</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

SE denotes standard error.

P-values for continuous variables were derived from unweighted t-tests (PROC SURVEYMEANS) while P-values for binary variables were derived from unweighted Wald Chi Square tests (PROC SURVEYFREQ).
### Table 2: AEE Weighted stratum-specific balance diagnostics for effect of CPSA

<table>
<thead>
<tr>
<th>PS Covariate</th>
<th>Indigenous</th>
<th>Black African</th>
<th>Black Caribbean</th>
<th>White</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N=288</td>
<td>N=302</td>
<td>N=64</td>
<td>N=554</td>
<td>N=103</td>
</tr>
<tr>
<td><strong>Age - years (SE)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CPSA-</td>
<td>39.2 (1.20)</td>
<td>43.4 (0.71)</td>
<td>45.8 (1.99)</td>
<td>44.0  (0.79)</td>
<td>42.8 (1.67)</td>
</tr>
<tr>
<td>CPSA+</td>
<td>40.9 (0.62)</td>
<td>39.8 (0.79)</td>
<td>45.1 (1.79)</td>
<td>44.7  (0.55)</td>
<td>43.6 (1.16)</td>
</tr>
<tr>
<td>P-value</td>
<td>0.2156</td>
<td>0.0008</td>
<td>0.7883</td>
<td>0.4385</td>
<td>0.7020</td>
</tr>
<tr>
<td><strong>Sexual Minority - % (SE)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CPSA-</td>
<td>23.6 (6.62)</td>
<td>2.81 (2.26)</td>
<td>8.93 (8.33)</td>
<td>16.4  (4.12)</td>
<td>21.0 (10.7)</td>
</tr>
<tr>
<td>CPSA+</td>
<td>21.8 (2.79)</td>
<td>6.71 (2.05)</td>
<td>10.0 (5.52)</td>
<td>19.3  (2.14)</td>
<td>13.5 (3.99)</td>
</tr>
<tr>
<td>P-value</td>
<td>0.8006</td>
<td>0.1996</td>
<td>0.9151</td>
<td>0.5574</td>
<td>0.5222</td>
</tr>
<tr>
<td><strong>Gender Minority - % (SE)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CPSA-</td>
<td>9.78 (5.08)</td>
<td>1.28 (1.27)</td>
<td>0</td>
<td>7.00  (3.78)</td>
<td>4.44 (4.37)</td>
</tr>
<tr>
<td>CPSA+</td>
<td>6.76 (1.69)</td>
<td>0</td>
<td>10.0 (5.52)</td>
<td>4.68  (1.14)</td>
<td>16.2 (4.31)</td>
</tr>
<tr>
<td>P-value</td>
<td>0.5752</td>
<td>N/A</td>
<td>N/A</td>
<td>0.5597</td>
<td>0.0621</td>
</tr>
<tr>
<td><strong>Immigrated at or before age 16 - % (SE)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CPSA-</td>
<td>-</td>
<td>3.37 (1.77)</td>
<td>11.7 (6.50)</td>
<td>7.24  (2.41)</td>
<td>23.1 (11.5)</td>
</tr>
<tr>
<td>CPSA+</td>
<td>-</td>
<td>4.76 (1.76)</td>
<td>33.3 (8.68)</td>
<td>3.81  (1.04)</td>
<td>24.7 (5.07)</td>
</tr>
<tr>
<td>P-value</td>
<td>-</td>
<td>0.5772</td>
<td>0.0504</td>
<td>0.1947</td>
<td>0.9022</td>
</tr>
<tr>
<td><strong>Immigration-related stress at or before age 16 - % (SE)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CPSA-</td>
<td>-</td>
<td>0.65 (0.65)</td>
<td>0</td>
<td>1.25  (1.24)</td>
<td>21.3 (12.4)</td>
</tr>
<tr>
<td>CPSA+</td>
<td>-</td>
<td>3.05 (1.51)</td>
<td>11.5 (6.32)</td>
<td>0.30  (0.30)</td>
<td>10.4 (3.76)</td>
</tr>
<tr>
<td>P-value</td>
<td>-</td>
<td>0.1448</td>
<td>N/A</td>
<td>0.4552</td>
<td>0.4300</td>
</tr>
<tr>
<td><strong>Survivor of residential school - % (SE)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CPSA-</td>
<td>8.17 (5.74)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>CPSA+</td>
<td>7.92 (2.70)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>P-value</td>
<td>0.9687</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td><strong>Family member(s) attended residential school - % (SE)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CPSA-</td>
<td>70.1 (8.49)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>CPSA+</td>
<td>67.5 (4.29)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>P-value</td>
<td>0.7901</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

SE denotes standard error.

P-values for continuous variables were derived from weighted t-tests (PROC SURVEYMEANS) while P-values for binary variables were derived from weighted Wald Chi Square tests (PROC SURVEYFREQ).
Appendix 7: Sensitivity analyses

Table 1: Effect of CPA on barriers to HIV disclosure, weighted to control for the effect of child sexual abuse and other potential confounders

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Ethnoracial group</th>
<th>Unweighted</th>
<th>AEE Weighted</th>
<th>Effect modification</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>REF</td>
</tr>
<tr>
<td>Adult physical abuse ¶</td>
<td>White</td>
<td>1.58 (1.38, 1.81)</td>
<td>1.23 (1.04, 1.44)</td>
<td>0.98 (0.78, 1.23)</td>
</tr>
<tr>
<td></td>
<td>Indigenous</td>
<td>1.87 (1.49, 2.34)</td>
<td>1.20 (1.02, 1.42)</td>
<td>1.65 (1.11, 2.46)</td>
</tr>
<tr>
<td></td>
<td>Black African</td>
<td>2.10 (1.64, 2.68)</td>
<td>2.03 (1.41, 2.92)</td>
<td>3.61 (1.58, 8.24)</td>
</tr>
<tr>
<td></td>
<td>Black Caribbean</td>
<td>3.32 (1.75, 6.31)</td>
<td>4.43 (1.97, 9.96)</td>
<td>3.61 (1.58, 8.24)</td>
</tr>
<tr>
<td></td>
<td>Other</td>
<td>1.58 (1.07, 2.33)</td>
<td>0.92 (0.72, 1.17)</td>
<td>0.75 (0.56, 1.00)</td>
</tr>
<tr>
<td>Adult sexual abuse ¶</td>
<td>White</td>
<td>1.63 (1.34, 1.98)</td>
<td>1.27 (0.98, 1.63)</td>
<td>0.95 (0.51, 1.78)</td>
</tr>
<tr>
<td></td>
<td>Indigenous</td>
<td>1.78 (1.28, 2.45)</td>
<td>0.98 (0.72, 1.32)</td>
<td>0.77 (0.52, 1.14)</td>
</tr>
<tr>
<td></td>
<td>Black African</td>
<td>1.68 (1.27, 2.23)</td>
<td>1.40 (0.95, 2.05)</td>
<td>1.10 (0.70, 1.74)</td>
</tr>
<tr>
<td></td>
<td>Black Caribbean</td>
<td>2.11 (1.03, 4.32)</td>
<td>2.20 (0.92, 5.27)</td>
<td>1.73 (0.70, 4.31)</td>
</tr>
<tr>
<td></td>
<td>Other</td>
<td>1.97 (1.14, 3.38)</td>
<td>1.21 (0.68, 2.14)</td>
<td>0.95 (0.51, 1.78)</td>
</tr>
<tr>
<td>Sexual inactivity ¶</td>
<td>White</td>
<td>1.03 (0.88, 1.21)</td>
<td>1.17 (0.91, 1.51)</td>
<td>0.75 (0.43, 1.32)</td>
</tr>
<tr>
<td></td>
<td>Indigenous</td>
<td>1.02 (0.78, 1.32)</td>
<td>0.97 (0.65, 1.45)</td>
<td>0.83 (0.52, 1.33)</td>
</tr>
<tr>
<td></td>
<td>Black African</td>
<td>1.11 (0.87, 1.41)</td>
<td>1.10 (0.79, 1.53)</td>
<td>0.94 (0.62, 1.42)</td>
</tr>
<tr>
<td></td>
<td>Black Caribbean</td>
<td>1.00 (0.65, 1.54)</td>
<td>1.31 (0.67, 2.54)</td>
<td>1.12 (0.55, 2.28)</td>
</tr>
<tr>
<td></td>
<td>Other</td>
<td>0.78 (0.54, 1.12)</td>
<td>0.88 (0.54, 1.45)</td>
<td>0.75 (0.43, 1.32)</td>
</tr>
<tr>
<td>Sexual relationship power ±</td>
<td>White</td>
<td>-0.18 (-0.33, -0.04)</td>
<td>-0.13 (-0.30, 0.03)</td>
<td>-0.10 (-0.16, 0.37)</td>
</tr>
<tr>
<td></td>
<td>Indigenous</td>
<td>-0.11 (-0.35, 0.13)</td>
<td>-0.03 (-0.24, 0.18)</td>
<td>-0.18 (-0.13, 0.49)</td>
</tr>
<tr>
<td></td>
<td>Black African</td>
<td>-0.08 (-0.27, 0.10)</td>
<td>0.05 (-0.22, 0.31)</td>
<td>-0.10 (-0.42, 0.22)</td>
</tr>
<tr>
<td></td>
<td>Black Caribbean</td>
<td>0.01 (-0.37, 0.39)</td>
<td>-0.10 (-0.42, 0.22)</td>
<td>-0.15 (-0.32, 0.39)</td>
</tr>
<tr>
<td></td>
<td>Other</td>
<td>0.24 (-0.21, 0.70)</td>
<td>0.59 (0.17, 1.02)</td>
<td>0.73 (0.27, 1.19)</td>
</tr>
<tr>
<td>HIV Stigma ±</td>
<td>White</td>
<td>2.17 (0.84, 3.51)</td>
<td>2.09 (0.24, 3.94)</td>
<td>REF</td>
</tr>
<tr>
<td></td>
<td>Indigenous</td>
<td>0.89 (-1.26, 3.04)</td>
<td>1.73 (-0.91, 4.37)</td>
<td>-0.36 (-3.59, 2.86)</td>
</tr>
<tr>
<td></td>
<td>Black African</td>
<td>3.72 (2.01, 5.43)</td>
<td>1.40 (0.44, 5.92)</td>
<td>1.09 (-2.22, 4.40)</td>
</tr>
<tr>
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<td>Black Caribbean</td>
<td>-0.37 (-3.75, 3.01)</td>
<td>0.50 (-2.54, 3.54)</td>
<td>-1.59 (-3.15, 1.97)</td>
</tr>
<tr>
<td></td>
<td>Other</td>
<td>-1.22 (-4.12, 1.69)</td>
<td>-2.83 (-6.49, 0.83)</td>
<td>-4.92 (-9.02, -0.82)</td>
</tr>
<tr>
<td>Social support ±</td>
<td>White</td>
<td>-0.83 (-1.56, -0.10)</td>
<td>0.09 (-0.89, 1.07)</td>
<td>REF</td>
</tr>
<tr>
<td></td>
<td>Indigenous</td>
<td>-0.54 (-1.53, 0.45)</td>
<td>-0.25 (-1.78, 1.27)</td>
<td>-0.34 (-2.15, 1.47)</td>
</tr>
<tr>
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<td>Black African</td>
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<td>-1.35 (-2.58, -0.11)</td>
<td>-1.43 (-3.01, 0.14)</td>
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<td>Black Caribbean</td>
<td>0.98 (-1.13, 3.09)</td>
<td>1.09 (-0.81, 3.00)</td>
<td>1.01 (-1.14, 3.15)</td>
</tr>
<tr>
<td></td>
<td>Other</td>
<td>-2.45 (-4.31, -0.59)</td>
<td>-2.72 (-4.88, -0.56)</td>
<td>-2.81 (-5.18, -0.44)</td>
</tr>
</tbody>
</table>

¶ Indicates binary outcome variables. Effect estimates are prevalence ratios and their robust 95% confidence intervals. Shaded cells represent those where the confidence interval does not cross the null value of 1.

± Indicates continuous outcome variables. Effect estimates are coefficients from linear regression (i.e. estimated difference in mean outcome for CPSA+ compared to CPSA-) and their 95% confidence intervals. Shaded cells represent those where the confidence interval does not cross the null value of 0.
Table 2: Effect of CSA on barriers to HIV disclosure, weighted to control for the effect of child physical abuse and other potential confounders

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Ethnoracial group</th>
<th>Unweighted</th>
<th>Weighted</th>
<th>Effect modification</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Adult physical abuse ¶</td>
<td>White</td>
<td>1.56 (1.38, 1.76)</td>
<td>1.26 (1.09, 1.47)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Indigenous</td>
<td>1.64 (1.38, 1.94)</td>
<td>1.18 (0.99, 1.41)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Black African</td>
<td>1.48 (1.18, 1.86)</td>
<td>1.12 (0.88, 1.43)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Black Caribbean</td>
<td>1.56 (0.90, 2.70)</td>
<td>0.99 (0.59, 1.68)</td>
</tr>
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<td></td>
<td></td>
<td>Other</td>
<td>1.68 (1.21, 2.31)</td>
<td>1.19 (0.88, 1.62)</td>
</tr>
<tr>
<td></td>
<td>Adult sexual abuse ¶</td>
<td>White</td>
<td>1.91 (1.60, 2.29)</td>
<td>1.49 (1.18, 1.88)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Indigenous</td>
<td>2.45 (1.77, 3.41)</td>
<td>1.97 (1.21, 3.21)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Black African</td>
<td>1.61 (1.22, 2.13)</td>
<td>1.32 (0.96, 1.81)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Black Caribbean</td>
<td>1.31 (0.58, 2.96)</td>
<td>1.06 (0.45, 2.53)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Other</td>
<td>2.93 (1.80, 4.77)</td>
<td>2.92 (1.53, 5.55)</td>
</tr>
<tr>
<td></td>
<td>Sexual inactivity ¶</td>
<td>White</td>
<td>0.95 (0.80, 1.12)</td>
<td>0.96 (0.77, 1.20)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Indigenous</td>
<td>0.91 (0.72, 1.17)</td>
<td>1.01 (0.68, 1.51)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Black African</td>
<td>0.92 (0.65, 1.29)</td>
<td>0.81 (0.56, 1.16)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Black Caribbean</td>
<td>0.37 (0.11, 1.25)</td>
<td>0.35 (0.10, 1.21)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Other</td>
<td>0.89 (0.61, 1.31)</td>
<td>1.07 (0.64, 1.78)</td>
</tr>
<tr>
<td></td>
<td>Sexual relationship power ±</td>
<td>White</td>
<td>-0.35 (-0.49, -0.21)</td>
<td>-0.41 (-0.57, -0.24)</td>
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<tr>
<td></td>
<td></td>
<td>Indigenous</td>
<td>-0.13 (-0.37, 0.11)</td>
<td>0.32 (-0.12, 0.76)</td>
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<tr>
<td></td>
<td></td>
<td>Black African</td>
<td>-0.08 (-0.32, 0.16)</td>
<td>-0.05 (-0.31, 0.21)</td>
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<tr>
<td></td>
<td></td>
<td>Black Caribbean</td>
<td>-0.05 (-0.44, 0.33)</td>
<td>-0.16 (-0.65, 0.33)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Other</td>
<td>-0.23 (-0.62, 0.16)</td>
<td>-0.08 (-0.52, 0.36)</td>
</tr>
<tr>
<td></td>
<td>HIV Stigma ±</td>
<td>White</td>
<td>1.68 (0.33, 3.03)</td>
<td>1.75 (0.08, 3.41)</td>
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<tr>
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<td>Indigenous</td>
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<td>0.55 (-2.07, 3.17)</td>
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<td>2.34 (-0.21, 4.90)</td>
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<tr>
<td></td>
<td></td>
<td>Black Caribbean</td>
<td>2.31 (-2.19, 6.81)</td>
<td>2.65 (-1.83, 7.13)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Other</td>
<td>1.30 (-1.59, 4.18)</td>
<td>1.84 (-1.33, 5.02)</td>
</tr>
<tr>
<td></td>
<td>Social support ±</td>
<td>White</td>
<td>-1.35 (-2.09, -0.61)</td>
<td>-0.94 (-1.91, 0.01)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Indigenous</td>
<td>-1.00 (-1.96, -0.04)</td>
<td>-0.40 (-2.11, 1.31)</td>
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<td>Black African</td>
<td>0.03 (-1.23, 1.29)</td>
<td>0.19 (-1.27, 1.64)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Black Caribbean</td>
<td>0.87 (-2.13, 3.87)</td>
<td>0.57 (-2.63, 3.77)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Other</td>
<td>-2.21 (-4.16, -0.26)</td>
<td>-1.82 (-4.29, 0.65)</td>
</tr>
</tbody>
</table>

¶ Indicates binary outcome variables. Effect estimates are prevalence ratios and their robust 95% confidence intervals. Shaded cells represent those where the confidence interval does not cross the null value of 1.
± Indicates continuous outcome variables. Effect estimates are coefficients from linear regression (i.e. estimated difference in mean outcome for CPSA+ compared to CPSA-) and their 95% confidence intervals. Shaded cells represent those where the confidence interval does not cross the null value of 0.
Appendix 8: Curriculum Vitae

SIOBHAN CHURCHILL

EDUCATION

MSc  University of Western Ontario (UWO), Epidemiology and Biostatistics  Advisor: Dr. Greta Bauer, PhD MPH  Thesis: Barriers to HIV status disclosure among women with a history of childhood physical or sexual abuse  Study: Canadian HIV Women’s Sexual & Reproductive Health Cohort Study

BSc  University of Guelph, Biomedical Science  Advisor: Dr. Andrew Papadopoulos, PhD, CPHI(C)  Thesis: Neonatal risks associated with maternal group B streptococcal colonization and intrapartum antibiotic prophylaxis  Study: Better Outcomes Registry and Network

HONORS AND AWARDS

Trainee Award for Innovative Thinking to Support LGBTQI2S Health 2017  CIHR grant funding valued at $25,000 to support student LGBTQI2S researchers in creating collaborative knowledge-to-action solutions in their areas of expertise. The final product for this particular grant will be a mobile, interactive education module providing home-care workers with training/certification in culturally competent care for LGBTQI2S older adults.

Carol Buck Graduate Scholarship in Epidemiology 2017  Annual scholarship valued at $7000 presented by UWO’s Department of Epidemiology and Biostatistics to an outstanding graduate student.

Ontario Graduate Scholarship 2017  Merit-based scholarship presented by the Province of Ontario and UWO, valued at $15,000.

Western Graduate Research Scholarship 2016  Entrance scholarship presented by UWO’s Department of Epidemiology and Biostatistics, valued at $4,200 annually.

Biomedical Sciences Research Award of Merit 2016  Award presented by the University of Guelph’s Department of Biomedical Sciences to students achieving over 90% in their fourth year research project.

Dean’s Honours List 2016  Award presented by the University of Guelph to students achieving an average grade at or above 80%.

Entrance Scholarship 2012  Merit-based entrance scholarship valued at $3000, presented by the University of Guelph.
**Research Experience**

**MSc Thesis**, Department of Epidemiology and Biostatistics, UWO (2016-2018)
Advisor: Dr. Greta Bauer, PhD MPH

- Completed quantitative analysis of barriers to HIV status disclosure using advanced causal epidemiological methods (propensity score weighting, machine learning)
- Adapted theoretical framework using eco-social theory and intersectionality to contextualize experiences of women living with HIV in Canada
- Participated in community-based research framework through ongoing collaboration with peer research associates/women living with HIV

**Research Assistant**, Department of Epidemiology and Biostatistics, UWO (2016-2018)
Advisor: Dr. Greta Bauer, PhD MPH

- Supervised team of five in conducting a systematic review on quantitative methods for applying intersectionality, to be submitted for publication in September 2018. *Grant: Improving Quantitative Health Research Methods: Multidimensionality and Intersectionality, CIHR*
- Contributed to design of parent and youth surveys as well as clinical case report forms for a national study of transgender youth in clinical care. This included selecting, adapting, and developing survey measures valid for transgender youth and their families. *Grant: Trans Youth CAN!, CIHR*
- Contributed to grant writing and study design for proposed national epidemiological study of transgender people in Canada. *Grant: CIHR Spring 2018 Project Grant Competition, results TBA*
- Contributed to grant writing for proposed project to expand cross-disciplinary methodological capacity for analytic intersectionality research in Canada. *Grant: CIHR Fall Project Grant Competition*
- Co-authored and edited manuscripts, including a study comparing Statistics Canada’s visible minority measure to a measure of self-identified visible minority status, and an item about being perceived as a person of colour

**Student Researcher**, TRANSforming Justice (2017-2018)
Supervisor: Dr. Greta Bauer, PhD MPH

- Completed descriptive quantitative analysis of legal needs of transgender people in Ontario, focusing on experiences with criminal law, police, sex work, and prison
- Analysis will be integrated with qualitative data for publication of mixed-methods report in 2018

**Contract Researcher**, International Centre for Science in Drug Policy (2017-2018)
Supervisor: Dr. Ayden Scheim, PhD

- Completed title/abstract/full-text screening, and data extraction for a systematic review on effects of drug decriminalization/regulation, to be published in 2018

**Contract Researcher**, LGBT Purge Class Action Lawsuit (2017)
Supervisor: Dr. Greta Bauer, PhD MPH

- Collaborated to identify archived quantitative data on unlawful treatment of LGBTQ members of the Canadian Armed Forces and Department of National Defence

**Health Data Analyst Summer Student**, Lambton Public Health (2015, 2016)
Supervisors: Crystal Palleschi, MSc and Dr. Sudit Ranade, MD MPH
• Analyzed and reported surveillance data (Canadian Community Health Study, Rapid Risk Factor Surveillance System) as well as health services data (Better Outcomes Registry and Network, National Ambulatory Care Reporting System, Vital Statistics Data, Ontario Mental Health Reporting System, Discharge Abstract Database) to public health professionals
• Consulted on survey design for Lambton County’s first study of LGBTQI2S health care access. Grant: Rainbow Health Ontario Breakthrough Grant
• Assisted with indicator development for internal program evaluation
• Consulted on survey design for a client satisfaction/public awareness survey, implemented by IPSOS for external program evaluation

SELECTED PUBLICATIONS

Journal Articles


Public Health Reports


PRESENTATIONS AND WORKSHOPS


LANGUAGES

**English:** Native Language

**French:** Written Comprehension, Written Expression, and Oral Proficiency Level A (Government of Canada Qualification Standards in Relation to Official Languages)

GRADUATE COURSEWORK

Systematic Reviews • Measurement in Epidemiology • Clinical Epidemiology • Principles of Biostatistics • Foundations of Epidemiology • Analytic Epidemiology • Survey Design and Implementation • Sampling Methods • Multivariable Methods

SOFTWARE

**SAS** (advanced), **Stata** (advanced), **R** (novice)