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Addressing Cigarette-Related Health Inequities Through a Trauma-Informed Lens

A dissertation submitted in partial satisfaction of the requirement for the degree Doctor in Philosophy

in

Interdisciplinary Research on Substance Use

by

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The dissertation of Charles Hayden Marks is approved, and it is acceptable in quality and form for publication on microfilm and electronically.

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DEDICATION

I would like to dedicate this dissertation to two people that would not have come into my life if it were not for this program that brought me to San Diego.

To Jenna, for your constant support, your love, and your light. It has meant everything to me to dream together and to take steps to make those dreams into reality. Thank you for always supporting me and uplifting me over the course of these past 4 years, for growing with me, for taking such huge chances to make our dreams come true. I love you, b.

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EPIGRAPH

"Be ruthless with systems, be kind with people" – Michael Brooks

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SELECTED PUBLICATIONS

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ABSTRACT OF THE DISSERTATION

Addressing Cigarette-Related Health Disparities Through a Trauma-Informed Lens

By

Charles Hayden Marks

Doctor in Philosophy in Interdisciplinary Research on Substance Use

University of California San Diego, 2021

San Diego State University, 2021

Professor Dan Werb, Chair

Background: Research has indicated that exposure to traumatic events is associated with cigarette use and with related outcomes such as pulmonary disease. Better characterizing the connection between trauma exposure and cigarette use and related outcomes remains a critical gap.

Theoretical Framework: We developed the Trauma-Informed Theory of Individual Health Behavior (TTB) to guide the aims of this dissertation. TTB articulates three types of trauma: historical trauma, acute experiences of trauma, and trauma-replicating environments.

Aims: Aim 1 (Historical Trauma): Identify how historical trauma has been conceptualized within the academic literature studying substance use and produce recommendations for incorporating the historical trauma concept into tobacco use research. Aim 2 (Trauma-Replicating Environments [i.e., poverty]): Examine the impact of income level on cigarette use transitions (initiation, cessation, reinstatement) and on cigarette use prevalence. Aim 3 (Acute Experiences of Trauma): Evaluate the mediating role of cigarette smoking on the relationship between trauma exposure during childhood and chronic obstructive pulmonary disorder (COPD) and chronic heart disease (CHD).

Methods: Aim 1 employed a scoping review strategy to identify relevant literature. Aim 2 was undertaken through the development of statistical models and a novel mathematical model of income and cigarette use. Aim 3 employed a causal mediation approach, based on inverse probability weighting, to assess the mediating role of lifetime cigarette smoking on the relationship between childhood trauma exposure and COPD and CHD incidence.

Results: Aim 1: We identified literature examining the relationship between historical trauma and substance use. We highlight that quantitative methods may be poorly suited to the study of this topic. Aim 2: We identified that lower income level is associated with increased risk

of adopting cigarette use and a diminished likelihood of cessating use. Further, that income is attributable to a substantial amount of cigarette use prevalence, particularly among those who have never used cigarettes before. Aim 3: We identified that cigarette use mediates the relationship between childhood trauma exposure and COPD and CHD.

Conclusions: We have explored how different trauma constructs may be applied to better understanding tobacco use disparities. The findings hold important implications for policy endeavors to reduce the harms of tobacco use and for future research directions.

Chapter 1 Introduction

Background

Tobacco control initiatives over the past half century have resulted in a substantial reduction in the use of cigarettes in the United States (US). The cigarette smoking prevalence has fallen from over 40% in the 1960s to 14% in 2019 (Cornelius et al.; Cummings and Proctor). Despite these reductions, it is estimated that cigarette use is attributable to 500,000 deaths annually, along with costing approximately $300-billion in medical costs and lost productivity each year (National Center for Chronic Disease Prevention and Health Promotion (US) Office on Smoking and Health). Further, concerningly, the cigarette use prevalence remains disparately within many demographics, including: historically marginalized communities such as Indigenous peoples and lesbian, gay, and bisexual individuals; people affected by disability and/or psychological distress; as well as individuals with access to fewer resources such as income, education, and health insurance (Cornelius et al.). Addressing these disparities requires both critical engagement with the inequitable impact of past tobacco control initiatives and new strategies to reduce these noted disparities.

Frohlich and Potvin describe "The Inequality Paradox", a phenomenon in which population-level public health approaches improve health metrics overall, but reinforce pre-existing health disparities (Frohlich and Potvin). They point out that while many public health initiatives, such as US tobacco control, effectively improve population-level health indicators, they do so by effectively concentrating risk within vulnerable communities (Frohlich and Potvin). In this case, we can understand that while tobacco control initiatives, such as excise taxes and smoke-free legislation, may incentivize people to stop using cigarettes, these initiatives do not address the underlying factors which place certain populations at greater risk of adopting and maintaining

cigarette use behaviors. Further, these approaches may reinforce factors which place individuals at higher risk of smoking. For example, given evidence of the association between poverty and cigarette use (Leventhal et al.), we may understand that excise taxes reinforce the situation of poverty and, thus, reinforce the context which places individuals at higher risk of cigarette use. Leventhal et al. specifically note how individuals exposed to multiple forms of "disadvantage", such as poverty, unemployment, and experiencing serious psychological distress, constitute a disproportionate percent of smokers as compared to individuals experience no or fewer forms of "disadvantage" (Leventhal et al.). As such, better understanding factors which increase vulnerability to cigarette use and, thus, drive cigarette use disparities, may improve our overall ability to address the noted disparities.

Over the past several decades, the role which trauma plays as a risk factor for cigarette use and cigarette-related health outcomes (e.g., pulmonary disease, heart disease, cancer), has been brought into light. The Adverse Childhood Experiences (ACE) Study, conducted in San Diego in the mid-1990s, sought to examine the relationship between exposure to neglect and abuse as a child and health behaviors and outcomes (Felitti et al.). The ACE Study developed the ACE score, an index from 0 to 8, measuring the number of distinct types of trauma (ranging from exposure to drug abuse and criminality in the household to emotional, physical, and sexual abuse) respondents experienced prior to the age of 18 (Felitti et al.). They found that reporting high levels of exposure to childhood trauma (i.e. an ACE score of 4 or more) corresponded to double the odds of reporting being a current smoker compared to those exposed to no ACE types (Felitti et al.). Further, they found that high levels of exposure to trauma doubled the odds that participants reported being diagnosed with heart disease and cancer and quadrupled the odds that participants reported chronic pulmonary disease (Felitti et al.). Results of a 2017 meta-analysis by Hughes et al. reinforce these

findings (Hughes et al.). Further, a recent study by Bellis et al. estimated that, in North America, exposure to trauma during childhood (as measured by the ACE score) could be attributed to 24% of smoking incidence, 10% of cancer incidence, 20% of cardiovascular disease incidence, and 28% of respiratory disease incidence, costing $750-billion annually (Bellis et al.).

The Health Inequality Paradox indicates that population-level public health interventions can increase health disparities by failing to account for factors that specific vulnerable communities face in reaping the benefits (such as cigarette cessation) of the intervention (Frohlich and Potvin). Many of the demographics which are subject to higher rates of cigarette use also report higher exposure to ACEs. For example, in data collected from 23 states between 2011 and 2014 found that specific demographic groups had significantly higher average ACE scores, including: people making less than $35,000 annually (compared to those making more than $50,000); those with a high school education or less (compared to those with a college degree); non-white individuals (compared white individuals); and, those who identify as gay, lesbian, or bisexual (compared to those who identify as heterosexual) (Merrick et al.). This indicates that many of the same demographic groups that are subject to cigarette use disparities also are subject to trauma exposure disparities. This provides an indication that better understanding the relationship between trauma exposure and cigarette use can be an important component of addressing tobacco use health disparities.

The Need for a Trauma-Informed Framework for Studying Cigarette Use

The declines in cigarette use prevalence over the past half century have been dispersed inequitably. Many vulnerable demographics remain subject to substantially higher rates of cigarettes use, many of which are also subject to higher levels of exposure to trauma. While research has made clear associations between trauma, cigarette use, and cigarette-related health

outcomes, tobacco control policies have largely not been influenced by such research. For example, the 2021 proposal to ban menthol cigarettes has been touted as a strategy to "reduce health disparities" by the US Food and Drug Administration (US Food and Drug Administration) – however, such an approach does not address underlying sources of vulnerability (as highlighted by the Health Inequality Paradox). As such, it is necessary to develop a trauma-informed framework for cigarette use research and policy development and to model how such a framework may be applied to undertaking research focused on addressing cigarette use disparities. Thus, the primary objective of this dissertation is to develop the **Trauma-Informed Theory of Individual Health Behavior** and then to conduct a series of studies applying constructs within this novel theory to display how it may be applied to undertake research into cigarette use health disparities.

Structure of this Dissertation & Dissertation Aims

In Chapter 2 of this dissertation, we introduce and define the Trauma-Informed Theory of Individual Health Behavior (TTB).[1] TTB is adapted from the Trauma-Informed Care (TIC) framework, synthesized by the US Substance Abuse and Mental Health Administration from the works of Harris and Fallot (SAMHSA; Harris and Fallot). TIC was developed to improve health care organizations ability to provide substance use and mental health care to individuals impacted by exposure to trauma (SAMHSA). However, TIC does not posit a model for how exposure to trauma impacts risk of engaging in harmful health behaviors, such as cigarette use. As such, TTB is developed to: 1) identifies the primary forms that trauma can take; 2) models the pathways by which exposure to trauma inhibits individual capacity to undertake positive health behavior change (via the trauma response); and 3) identifies key resilience factors that can help individuals mitigate the trauma response. Specifically, TTB identifies three primary forms of trauma exposure:

[1] The manuscript introducing the Trauma-Informed Theory of Individual Health Behavior has been accepted for publication in the journal Stress & Health

historical trauma, acute experiences of trauma, and trauma-replicating environments. Each aim of this dissertation corresponds to each of these three trauma constructs and their relation to cigarette use. Through the completion of such aims, this dissertation shall display the potential efficacy of applying TTB and, further, will build the foundations of a program of research aimed at better understanding the relationship between exposure to trauma and cigarette use. Of note, we have developed TTB as a general theory of health behavior (i.e., not specific to cigarette use) so that it may be applied more broadly to motivate research into other topics of health behavior, including substance use more broadly. The three aims of this dissertation are as follows:

Aim #1 (Historical Trauma): **To undertake a scoping review of the academic literature on the relationship between historical trauma and substance use.[2] The review shall aim to characterize the findings of the qualitative and quantitative literature on the topic, identify incongruities between the findings of each, and provide recommendations for how the historical trauma concept can be effectively incorporated into future substance use research, including cigarette use research.**

Aim #2 (Trauma-Replicating Environments): **To apply statistical and mathematical modeling techniques in order to: 1) assess the role of income (where we understand poverty as a trauma-replicating environment) as a risk factor for cigarette use transitions (initiation, cessation, reinstatement); and. 2) assess the attributable impact of income on cigarette use prevalence among the US adult population.**

Aim #3 (Acute Experiences of Trauma): **To assess the role of lifetime cigarette use as a mediator in the relationship between exposure to adverse childhood experiences and both**

[2] Due to a paucity of research on the association of historical trauma and cigarette use, it was deemed appropriate to focus on the literature studying the relation between historical trauma and substance use more broadly.

chronic obstructive pulmonary disease and coronary heart disease (two cigarette use-related health outcomes).

The aims are not dependent on one another, but in their entirety, display how applying TTB can provide new insights into reducing cigarette use-related health disparities. As well, each chapter of this dissertation (except for this chapter and the concluding chapter), have been completed as distinct, stand-alone manuscripts intended for individual publication. This format was chosen to increase the potential impact of this overall work and to display how TTB may be applied in the production of academic manuscripts.

Aim #1 shall be completed in Chapter 3. A scoping review of the literature will be undertaken to capture peer-reviewed, original research focusing on the relationship between historical trauma and substance use. A 2019 systematic review of quantitative literature focusing on the health harms of historical trauma found only two articles examining the relationship between historical trauma and cigarette use (Gone et al.) – given the paucity of such literature, it was determined that focusing on substance use research broadly (i.e., not specifically cigarette use) would provide a more impactful foundation for future research examining the relationship between historical trauma and cigarette use. In this review, we shall examine the findings of the qualitative and quantitative research identified, reflect on similarities and incongruities between how historical trauma has been conceptualized within the qualitative and quantitative research, and provide considerations for incorporating historical trauma into future substance use research.

Aim #2 shall be completed as two distinct manuscripts, presented in Chapters 4 and 5. The goal of these studies is to explore the impact of income level on cigarette use. As shall be discussed further in Chapter 2, we may understand living in poverty or with a low-income as a type of trauma-replicating environment. First, using data from the Population Assessment of Tobacco and

Health (PATH) Study (Hyland et al.), we shall develop statistical models to examine the association between income level and three cigarette transitions: cigarette-naïve individuals initiating cigarette use; people who current smoke cigarettes cessating cigarette use; and, people who formerly smoked cigarettes reinstating cigarette use. Second, using data from the PATH Study and from other publicly available sources, we shall develop a novel mathematical compartment model of cigarette use which factors in income level. We shall use this model to estimate the population attributable effect of income level on cigarette use prevalence over a 1-year, 5-year, and 10-year time horizon.

Aim #3 shall be completed in Chapter 6 using data collected by 7 US states as a part of the CDC Behavioral Risk Factor Surveillance System (BRFSS) survey (Centers for Disease Control and Prevention). The BRFSS survey collects data related to health risk behaviors, such as cigarette smoking, and health outcomes, such as pulmonary disease and heart disease. Optionally, states are permitted to ask participants to respond to the ACEs survey. We shall apply the potential outcomes framework to assess the mediating role of lifetime cigarette use on the relationship between elevated ACE type exposure and incidence of chronic obstructive pulmonary disorder (COPD) and coronary heart disease (CHD). Given the research which has indicated that increased ACE score is associated with increased risk of cigarette use and COPD and CHD (Felitti et al.; Hughes et al.; Bellis et al.), we hypothesize that cigarette use partially mediates the relationship between increased ACE score and COPD and CHD incidence.

Conclusion

Within this dissertation, we posit a novel trauma-informed theory of health behavior and display how it may be applied to furthering research aimed at addressing cigarette use disparities. As noted, each chapter (with the exception of the first and last) are intended to stand alone as

distinct academic manuscripts. These works, taken together, lay the foundation for a program of research which seeks to better capture the impact of exposure to trauma on cigarette use. Given the noted cigarette use disparities within the US, this program of research may provide new insights and policy directions which can be applied for the purpose of addressing these disparities.

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Chapter 2 Articulating the Trauma-Informed Theory of Behavior

Disclaimer of Publication

The following chapter has been published in the journal Stress & Health. This work was completed as part of this dissertation and approval was granted to include in the final dissertation. This article is subject to the journal Stress & Health's copyright. This chapter is the final submitted article, accepted by Stress & Health. The citation for the published article is:

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Abstract

Exposure to trauma increases the risk of engaging in detrimental health behaviors such as tobacco and substance use. In response, the United States Substance Abuse and Mental Health Services Administration developed Trauma-Informed Care (TIC), an organizational framework for improving the provision of behavioral health care to account for the role exposure to trauma plays in patients' lives. We adapt TIC to introduce a novel theory of behavior change, the Trauma-Informed Theory of Individual Health Behavior (TTB). TTB posits that individual capacity to undertake intentional health-promoting behavior change is dependent on three factors: 1) the forms and severity of trauma they have been and are exposed to; 2) how this trauma physiologically manifests (i.e., the trauma response); and 3) resilience to undertake behavior change despite this trauma response. We define each of these factors and their relationships to one another. We anticipate that the introduction of TTB will provide a foundation for developing theory-driven research, interventions, and policies that improve behavioral health outcomes in trauma-affected populations.

Introduction

Between 82% and 90% of people in the United States (US) are exposed to trauma in their lifetimes, including experiencing or witnessing violence and surviving war or a disaster (Koenen et al.; Kilpatrick et al.). This statistic, however, does not include exposure to traumatic environments, such as living in poverty (Gelkopf), nor the harms of historical trauma, such as forced enrollment in boarding schools for Indigenous children (Heart; Mohatt et al.). Indeed, a growing body of evidence indicates that exposure to trauma is a critical risk factor for development of harmful health behaviors and poor health outcomes (Sowder et al.). Exposure to trauma during childhood is associated with increased likelihood of cigarette smoking, alcohol and substance use disorders, sexual risk-taking, poor mental health, obesity, and greater incidence of heart disease, respiratory disease, and cancer in adulthood (Hughes et al.). Notably, not all communities experience trauma equally. Racial, ethnic, sexual, and gender minority communities disproportionately experience acute forms of trauma (Merrick et al.). Additionally, many groups are subject to additional identity-specific forms of historical trauma such as the history of genocide and forced assimilation faced by Indigenous people in the US (Mohatt et al.; Heart). In 2019, exposure to trauma during childhood cost North America approximately $748 billion annually in direct medical costs and lost labor productivity (Bellis et al.).

In response to the growing understanding of the role of trauma in influencing negative health behaviors, the US Substance Abuse and Mental Health Services Administration (SAMHSA) synthesized the Trauma-Informed Care (TIC) approach from the work of Harris and Fallot (2001) to achieve three primary goals within the behavioral health care context: "1) realiz[e] the prevalence of trauma; 2) recogniz[e] how trauma affects all individuals involved with the program, organization, or system, including its own workforce; and 3) respond by putting this knowledge

into practice" (SAMHSA xix). TIC informs the design and implementation of trauma-informed behavioral healthcare systems. It highlights the importance of patient recovery from experiences of trauma and protection from re-traumatization during treatment (SAMHSA).

TIC, however, does not provide an explicit theoretical framework explaining the mechanisms driving the relationship between trauma exposure and individual health behavior change. Generally, theories of individual health behavior are often critiqued for failing to effectively account for how individuals prioritize and enact behavior change (Kelly and Barker) and the effectiveness of interventions driven by such theories remains debated (Hagger and Weed). Of importance, most health behavior theories treat the individual as a "rational actor" without providing sufficient attention to the contextual factors that limit the range of choices available to a person (Kelly and Barker). As a result, they fail to capture the physiological, social, and structural factors which influence behavior. We posit that such health behavior theories fail to account for the ways in which individuals prioritize the need for potential behavior changes and the limited resources (e.g., time, energy) individuals have to address competing stressors. TIC addresses these shortcomings by acknowledging that 1) individuals tend to focus their efforts on the most immediate and severe threats in their lives (which often demand their attention) and that 2) individuals generally undertake behaviors they believe will best alleviate the most immediate and severe of these threats (SAMHSA). Thus, integration of TIC principles into an individual-level theory of health behavior holds promise to improve the effectiveness of health behavior interventions.

The purpose of this paper is to introduce the Trauma-Informed Theory of Individual Health Behavior (TTB) and explain how its application can guide research on the mechanisms linking trauma and poor health outcomes. In particular, this theory is rooted in the understanding that

exposure to trauma can lead to elevated stress responses (e.g., PTSD, anxiety) (van der Kolk) and that individuals make their best effort to address this response with limited resources. TTB acknowledges that the physiological response to trauma exposure often compels individuals to focus on alleviating the immediate harms and threats associated with this trauma. It is then hypothesized that, absent this physiological trauma response, individuals will be empowered to focus on the threat of the long-term health behaviors (e.g., diet, substance use) that are generally the focus of health behavior change interventions.

The Trauma-Informed Theory of Individual Health Behavior

The Trauma-Informed Theory of Individual Health Behavior (TTB, see **Figure 2.1**) is an extension of SAMHSA's TIC. The TTB theory: 1) identifies the primary forms that trauma can take; 2) models the pathways by which exposure to trauma inhibits individual capacity to undertake positive health behavior change (via the trauma response); and 3) identifies key resilience factors that can help individuals mitigate the trauma response.

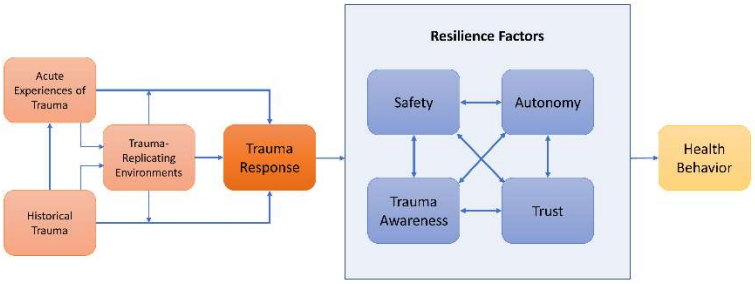

Figure 2.1 The Trauma-Informed Theory of Individual Health Behavior

Extending the TIC Definition of Trauma

TIC is an adapted version of the social-ecological model (SAMHSA). TIC identifies five social-ecological levels through which trauma occurs and exerts its effects, defined as individual, interpersonal, community/organizational, societal, and period of time in history (see Exhibit 1.1-2 in SAMHSA, 2014). The individual level of the TIC acknowledges that each person has a unique history of trauma exposure and a different capacity to mitigate the consequences of that trauma. At the interpersonal level, one person can directly inflict trauma upon another individual. At the community/organizational level, social networks and local organizations shape the environments within which individuals are exposed to trauma. A lack of social support may expose individuals to potentially traumatic environments. For example, an individual's risk of becoming homeless (a traumatic event) is shaped by the presence and ability of a family network to provide resources and protection from homelessness (Bramley and Fitzpatrick). Additionally, public safety and social service organizations may directly inflict harm on individuals. For example, the US Public Health Service conducted the 4-decades long Tuskegee Syphilis Study by recruiting Black men with syphilis, withholding their disease status, and pretending to provide them treatment despite the availability of effective treatment (Washington). At the societal level, state and federal laws and policies also shape the environments within which individuals are exposed to trauma. US federal policies passed in the 1990s, such as the 1996 Personal Responsibility and Work Opportunity Reconciliation Act and the 1998 Quality Housing and Work Responsibility Act, prohibited individuals charged with substance use convictions from accessing federal housing and income assistance (Alexander), reinforcing their circumstances of poverty and housing insecurity. TIC also integrates historical context into the social-ecological model, given its importance in shaping individuals' experiences and environments over the long term. For example, Indigenous populations in the United States and Canada have been subjected to the trauma of centuries of

genocide and assimilation policies (Heart). According to TIC, health care providers who fail to account for this history risk replicating these harms with their Indigenous patients (SAMHSA). These social-ecological levels are intertwined but differentiating them provides insight into pathways by which trauma is enacted and replicated on individuals.

Defining TTB Trauma Constructs

To facilitate replicability in measurement and targeted intervention, the TTB maps the TIC's five social-ecological levels onto three primary forms of trauma exposure. The individual and interpersonal levels map to **acute experiences of trauma**; the community/organizational and societal levels map to **trauma-replicating environments**; and period of time in history maps to **historical trauma** (see **Figure 2.2**). Below, we define each of these three forms of trauma.

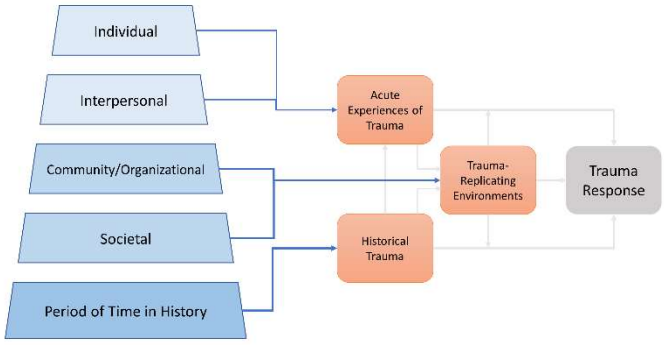

Figure 2.2 TIC social–ecological levels mapped onto TTB trauma constructs.

Acute Experiences of Trauma. SAMHSA defines acute trauma as "an event, series of events, or set of circumstances that is experienced by an individual as physically or emotionally harmful or threatening and that has lasting adverse effects on the individual's functioning and physical, social, emotional, or spiritual well-being" (SAMHSA xix). Acute traumatic events can take a variety of forms, such as experiencing assault or losing one's home or employment. They

are frequently defined by an accompanying loss of sense of safety, autonomy, and trust. In these moments, an individual is subject to direct harm at the hands of another individual or entity (such as a landlord or employer) in an interpersonal context. As shown in **Figure 2.2**, in the TTB, acute experiences of trauma encompass the "individual" and "interpersonal" social-ecological levels of the TIC. We also note that much of the research on the harms of trauma exposure focus on the impact of such exposure during childhood on health behaviors and outcomes during adulthood (Hughes et al.). This definition of acute trauma is intended to encompass both traumas experienced during childhood and adulthood and can be understood to measure the lifetime accumulation of traumatic exposure.

Trauma-Replicating Environments. Trauma-replicating environments impact individuals in two ways: 1) they prime individuals to anticipate a traumatic event (i.e., they are "triggering"), regardless of whether such trauma will occur; and, 2) they may expose individuals to acute experiences of trauma (SAMHSA). With the former, this "priming" represents a distinct harm that such environments enact on individuals and is partially dependent upon previous exposure to trauma. For example, individuals who have experienced or witnessed abusive behavior from law enforcement may find the presence of law enforcement to be "triggering", as they anticipate potential abuse. The latter emphasizes the inextricable link between individuals' environments and the acute forms of trauma they experience within those environments. This construct captures how social circumstances and environments, such as poverty or homelessness, similarly replicate experiences of trauma. Coates and Mckenzie-Mohr (2010) describe how, for homeless youth, becoming homeless represents an event of acute trauma and that the circumstances of being homeless continuously replicate that loss of safety and autonomy (Coates and McKenzie-Mohr). Poverty creates a similar cycle, where the experience of living in poverty continually replicates the

dynamics of acutely experiencing trauma. Poverty is not simply defined by a lack of resources, but by the threat of trauma in the forms of housing, food, and financial insecurity and loss. As put by Gelkopf (2) , "trauma begets trauma, trauma begets poverty, poverty begets poverty, poverty begets trauma, and the cycle goes on". Trauma-replicating environments overlay the "societal" and "community/organizational" social-ecological levels of the TIC (**Figure 2.2**).

Historical Trauma. Mohatt et al. summarize historical trauma as "a complex and collective trauma experienced over time and across generations by a group of people who share an identity, affiliation, or circumstance" (128). They go on to describe three components of historical trauma: "a 'trauma' or wounding; the trauma is shared by a group of people, rather than individually experienced; [and] the trauma spans multiple generations, such that contemporary members of the affected group may experience trauma-related symptoms without having been present for the past traumatizing events" (Mohatt et al. 128). Historical trauma overlays the "period of time in history" social-ecological level of the TIC. This outermost layer represents the historical context of the current moment, which "influences each other level" (SAMHSA 16). Historical harms are concentrated within specific communities that share a collective history and the burden of these harms is passed from generation to generation (Mohatt et al.; Heart). For example, in the US, the enslavement and disenfranchisement of Black people and, in the US and Canada, the genocide of Indigenous people are not historical relics. The health of Black communities today cannot be separated from enslavement, from the failures of Reconstruction, from Jim Crow, from redlining and racist housing policies, and from the War on Drugs policies that have led to the US mass incarceration crisis (Alexander). Similarly, the health of North American Indigenous communities today cannot be separated from the massacre of people, from the seizure of homelands, and from

the stealing and forced assimilation of children during the boarding/residential school era (Heart; Elias et al.).

The Trauma Response

In the face of imminent danger, it is natural for the body to invoke its protective stress responses. However, as van der Kolk (66) writes, "as long as trauma is not resolved, the stress hormones that the body secretes to protect itself keep circulating and the defensive movements and emotional responses keep getting replayed". Trauma responses are unique to the individual, their environments, and the circumstances of their trauma. Trauma responses manifest in myriad variations that cannot easily be boiled down to standardized classifications (van der Kolk). Until the trauma response is properly addressed, individuals are subject to reliving their experiences of trauma and this reliving "engrave[s] those memories [of trauma] ever more deeply in the mind" (van der Kolk 67). This experience then acts to disconnect individuals from the present and their immediate surroundings (i.e., dissociation) and their "physical reactions are dictated by the imprint of the past" (van der Kolk 67). TIC notes that, for individuals experiencing such a trauma response, their actions must be understood as a best effort to escape the cause of the trauma (as the stress system in their body is dictating) (SAMHSA). The trauma response activates the body's survival mechanisms. In this state of stress, survival becomes the primary concern and represents a primary barrier to engaging with behavior changes to achieve long-term health outcomes. Additionally, there are many instances where behaviors known to be detrimental in the long-term are used to cope with the trauma response. In such cases, survival takes priority, and the trauma response provides a physiological imperative for the individual to prioritize their immediate safety concerns over potential long-term health consequences. This is consistent with findings that indicate that higher and prolonged stress levels are associated with negative health behaviors and outcomes,

such as increased cigarette smoking, poor diet, lack of physical activity, and diminished physical, mental, and spiritual well-being, generally (Ng and Jeffery; Clark et al.; Park and Iacocca; Umberson et al.). The TTB includes a trauma response construct to ensure it is appropriately accounted for in trauma-related research endeavors.

Resilience Factors

From TIC, four key resilience factors which mediate the relationship between trauma and health behaviors are included in the TTB: *safety*, *autonomy*, *trauma awareness,* and *trust* (SAMHSA). The TTB defines resilience as an individual's capacity to cope with and mitigate the effects of the trauma response over time. Resilience should not be thought of as an individual attribute, but as a process in which an individual adapts to deleterious exposure to maintain a basal level of well-being (Southwick et al.; Lerner et al.). While an individual's trauma response is shaped by their exposure to trauma, an individual's capacity to undertake health promoting behaviors is shaped, in part, by their resilience. This understanding of health behavior highlights an important concept in trauma-informed approaches: an individual's risk of adopting poor health behaviors, such as cigarette smoking, and their ability to change such behaviors are related to their capacity to avoid exposure to trauma and to manage the sequelae of traumatic experiences and environments. Resilience factors are specific, measurable constructs which influence individual capacity to cope with and mitigate the trauma response. TIC notes that for health care providers to deliver more effective care, the impact of trauma must first be addressed (SAMHSA). Based on the forms of trauma defined above, this means that providing effective care is dependent first on addressing the present harms that acute experiences of trauma, trauma-replicating environments, and historical trauma are enacting on individuals (see **Figure 2.1**). As such, interventions aiming

to utilize TTB should understand resilience building as secondary to addressing exposure to trauma.

Safety. TIC identifies the creation of a "safe environment" as necessary to providing adequate behavioral health care (SAMHSA). "Safety" refers to an individual's perception that they are not currently at risk of nor actively being subjected to traumatizing events and that they feel protected from the sequelae of having experienced trauma (SAMHSA). Such an understanding of safety also requires provider and individual awareness of "triggers" which elicit a trauma response and decrease an individual's perception of safety (SAMHSA). If an individual feels unsafe, their capacity to undertake behavioral change is reduced as a consequence of their trauma response. As van der Kolk (2014) states, "traumatized people chronically feel unsafe inside their bodies: the past is alive in the form of gnawing interior discomfort" (van der Kolk 96).

Autonomy. TIC identifies fostering individual autonomy as a necessary step to providing adequate behavioral health care (SAMHSA). "Autonomy" refers to an individual's perception that they have control over themself and the environment around them – that they have agency over their life (van der Kolk). A lack of control over one's surroundings or of one's own body is a defining characteristic of traumatic experiences (SAMHSA; van der Kolk); a loss of autonomy can trigger and reinforce the trauma response.

Trauma Awareness. Body awareness and autonomy are inextricably linked. Van der Kolk notes that "agency starts with…our awareness of our subtle sensory, body based feeling: the greater that awareness, the greater our potential to control our lives" (van der Kolk 95). Being aware of internal feelings allows an individual to "feel in charge of [their] body, [their] feelings, and [their] self" (van der Kolk 96). Often, individuals experiencing a trauma response are not conscious of the connection between their current state of elevated stress and past experiences of

trauma (Payne et al.). Becoming conscious of this connection – such as through elevating interoceptive and proprioceptive awareness (Payne et al.) – can provide individuals a sense of agency over their own body, which represents a necessary step to overcoming the trauma response (van der Kolk). Mindfulness exercises, such as body scan and breathing exercises, represent a potential set of interventions which can improve awareness of the body and its internal sensations (Creswell).

Trust. TIC identifies that health care providers must be aware of the trauma their patient population has faced and must understand that, for trauma survivors, their behaviors are often a response to mitigating the harms of experienced trauma (SAMHSA). The inverse of this principle is that patients must trust their health care providers (or whoever is asking them to enact behavior change). The trauma response is often defined by not being able to trust one's self or others (van der Kolk). This is in line with research finding that decreased trust in health care systems and providers is associated with diminished communication, care retention and poor health care outcomes (Cuevas et al.).

The primary goal of improving safety, autonomy, trauma awareness, and trust is to help individuals overcome the trauma response and avoid future exposure to trauma. These resilience factors are inextricable and are related to a person's ability to overcome the impact of trauma on their life. Importantly, increased resilience positively affects patients' readiness to make positive behavior changes (Cook et al.). Further, we recognize that there are additional factors which are understood to influence resilience, such as social support, and encourage future research into TTB to expand upon these initial resilience factors adapted from TIC principles. TTB emphasizes, though, that enhancing individual resilience is understood as a secondary intervention to eliminating trauma exposure.

TTB Pathways Defined

TTB applies these three forms of trauma and four resiliency factors to understand how they relate to health behaviors (see **Figure 2.1**). TTB is comprised of three components: trauma and its response; resilience to mitigate the trauma response; and individual capacity to undertake positive health behaviors. First, we can understand the trauma response an individual may face in making behavior change as dependent on their past exposure to trauma. Historical trauma can influence how trauma is acutely experienced, can shape trauma-replicating environments, and can have direct impact on the trauma response. Acute experiences of trauma shape the individual's reactions to different types of environments that an individual is exposed to and define the environments that are trauma-replicating for an individual. The acute experiences of trauma also directly impact the trauma response. Exposure to trauma-replicating environments then has a direct impact on the trauma response but can also be understood to modify the relationship between historical trauma and acute experiences of trauma with the trauma response. For example, lifting an individual out of trauma-replicating environments may attenuate the harmful impacts that historical trauma and acute experiences of trauma have. As such, TTB-informed interventions can aim to improve health behavior outcomes through two pathways: first, by decreasing exposure to trauma and, thus, attenuating the trauma response; and second, by improving individual resilience to overcome the trauma response. It is important to understand, however, that individual resilience is defined by access to limited personal resources and that interventions which focus solely on resilience building will likely fail individuals who face the greatest burden of exposure to trauma. TTB is based on the TIC principle that individuals will always make their best effort to alleviate the harms they are currently facing (SAMHSA). TTB views trauma reduction as the most effective strategy for motivating health behavior change, with resilience building as an important (though secondary) mitigation strategy.

Discussion

Here we have presented a novel trauma-informed theory of health behavior, the Trauma-Informed Theory of Individual Health Behavior. Exposure to trauma is responsible for disparate health harms and billions of dollars in medical costs each year (Bellis et al.). As highlighted by SAMHSA's development of Trauma-Informed Care, stakeholders have mobilized initiatives to better understand and intervene on the damaging impact of trauma on behavioral health. The development of TTB builds upon this important work, providing an explicit model for how trauma impacts an individual's ability to undertake beneficial behavioral change.

TTB and theories of behavior change

Whereas health behavior theories traditionally aim to promote specific health behavior changes, TTB provides a framework for understanding how individuals prioritize responding to harms they have been, are, and will be exposed to. The TTB does not model the relationship between the individual and any specific behavior – this should not be viewed, necessarily, as a short-coming, but instead as a key feature. This key feature arises from a core tenet of TIC, that individuals will make their best effort to overcome their most immediate stressors. Individuals may not be able to take advantage of interventions aimed at specific long-term health behaviors when exposed to trauma. Not only is this conceptualization useful in contrasting the perceived immediacy the threat of trauma holds in comparison to, for example, the long-term harms of cigarette smoking or poor diet, but it also implies that if those long-term harms are the most immediate threat to an individual's well-being, then they will do their best to address it.

We may look to smoking cessation to highlight this key difference. An intervention based on theories such as the Theory of Planned Behavior (TPB) seeks to improve knowledge and change attitudes abouts the harms of cigarette smoking to incentivize cessation (Glanz et al.). While this

approach can help an individual make an informed decision about the relative threat of cigarette smoking, it does not consider whether an individual will perceive the long-term harms of cigarette smoking as a more immediate concern than other sources of harm. TTB assumes that the individual is best equipped to prioritize addressing sources of harm they are exposed to, which may explain why cigarette smoking prevalence remains disparately high among populations subject to more immediate threats to well-being, such as those subject to financial, food, and housing insecurity or those living with elevated levels of psychological distress (i.e., a potential proxy for an elevated trauma response) (Cornelius et al.). Interventions based on Social Cognitive Theory (SCT) focus on how a behavior change is learned and on promoting self-efficacy for behavior change (e.g., teaching youth how to say no to cigarettes and promoting their confidence to do so) (Glanz et al.). TTB is not concerned with how a behavior is learned or an individual's belief in their ability to undertake it but is instead intended to reflect on how trauma physiologically influences the behavioral choices an individual will prioritize. SCT's understanding that there is a dynamic interaction between person, environment, and their behavior (i.e., reciprocal determinism) is consistent with TTB. This similar understanding of the relation between the individual, their environment, and their behavior suggests that TTB and SCT may be used effectively in conjunction with one another. However, SCT rejects the idea that an individual's behavior is determined solely by their environment. TTB, on the other hand, suggests that environmental exposure to trauma can dictate individual behavior via an elevated trauma response. As such, TTB differs from many health behavior theories because it is focused on how people prioritize behaviors, whereas theories such as TPB and SCT focus on how behaviors are chosen, learned, and executed.

TTB also differs from the Transactional Model of Stress and Coping (TMSC), which explains how individuals cope in response to stressors, including trauma (Glanz et al.). Similar to TTB, TMSC models how external/environmental stressors and individual capacity to cope with these stressors impact individual capacity to undertake behavior change (Glanz et al.). Both theories suggest that the individual's ability to overcome stressors is subject to the individual's limited set of resources to do so. A primary difference is that TMSC is concerned with the *cognitive* pathway by which stressors are appraised and a coping strategy is developed to adapt to said stressor (Glanz et al.). In contrast, TTB models the relationship between exposure to stressors and the *physiological* response they invoke in the individual. TMSC presumes the rational actor conception of human behavior, in that it posits that when exposed to a given situation, an individual will undertake a cognitive appraisal and evaluate the harms and benefits, both in the short- and long-term (Glanz et al.). TTB suggests that, when exposed to trauma, an individual's response may be largely *physiological* and that the cognitive pathways described by TMSC may be more applicable in circumstances where the trauma response is minimal.

TTB may best be understood as an individual-level counterpart to participatory community-level frameworks such as the Empowerment Education model developed from the ideas of Paolo Freire (Wallerstein). Empowerment Education is an action-oriented model in which community members critically engage with their shared social conditions through group dialogue, which then motivates actions to alter their social conditions for the benefit of the community (Wallerstein). TTB models how individuals experience social conditions and how these conditions impact individuals' capacity to undertake behavior changes. Further, TTB, like Empowerment Education, emphasizes that no one can speak better to the health needs of an individual than the individual.

We additionally note limitations to the scope of TTB. The theory is not concerned with individual knowledge, beliefs, or attitudes toward given health behaviors, nor does it address how behaviors are learned and executed. As discussed in relation to TPB and SCT, TTB implies that an elevated trauma response may inhibit an individual from prioritizing adopting a health behavior related to a long-term harm, rendering knowledge, beliefs, and attitudes relatively moot. However, it will be important to better understand the role that knowledge and attitudes play in influencing health behavior when considered through the lens of TTB – for example, it is worth considering if increased knowledge or improved self-efficacy about health behaviors supports individual resilience. Additionally, as we have noted, the TTB resilience factors are limited to individual characteristics, whereas community and cultural factors such as social support (Ozbay et al.) and cultural resilience (Spence et al.) have been identified as important protective factors. Finally, while TTB is concerned with the role of trauma exposure in eliciting a physiological trauma response, TTB does not reflect on the biological mechanisms that may underly the trauma response such as the epigenetic component of the human stress response (Stankiewicz et al.). TTB-driven research which utilizes biometrics to operationalize the trauma response could provide a better understanding the pathways by which exposure to trauma impact health behavior.

Future directions

TTB could motivate research addressing health behavior disparities. Frohlich and Potvin describe "The Inequality Paradox", a phenomenon in which population-level public health approaches improve health metrics overall, but reinforce pre-existing health disparities (2008). They point out that while many public health initiatives, such as North American tobacco control, improve population-level behavioral health indicators, they also further concentrate risk within vulnerable communities (Frohlich and Potvin). To date, trauma-informed research and

interventions have largely focused on exposure to trauma during childhood development on a wide range of health behaviors during adulthood (such as cigarette and other drug use) and outcomes (such as heart disease and cancer) (Hughes et al.). By applying TTB, we can understand how trauma experienced by marginalized communities subjects them to concentrated health risks and health outcome disparities. Examining historical trauma provides a lens through which to understand how specific communities, such as Black and Indigenous people in North America, face increased barriers to making specific behavioral health choices. Examining trauma-replicating environments provides a lens through which to understand how specific demographics – such as those living in poverty, those without health insurance, and those who are houseless – face increased barriers to undertaking specific behavioral health promoting choices.

TTB may be very well suited to understand behavioral health disparities in the time of COVID-19. We may understand the COVID-19 pandemic as a traumatic event and, further, as reinforcing existing historical harms and trauma-replicating environments. The impacts of the epidemic have not been felt equally in the US, with lower-income, communities of color already facing the syndemic harms of historical trauma and chronic health inequities (Gravlee). The epidemic has not been solely defined by risk of contracting the disease, but by increased risk of housing, economic, and health care insecurity. For many, the COVID-19 era has been defined by both the acute trauma of more severe illness and greater cumulative loss and the reinforcement of trauma-replicating environments. Further, these harms have thus far been concentrated in many US populations, such as Indigenous and Black communities, that have been subject to well-documented historical harms. As such, TTB is well-suited to examine the impact of the COVID-19 epidemic on health behaviors and related disparities.

Conclusion

In line with the broader efforts to better understand the harmful health impacts of trauma exposure, we present the novel Trauma-Informed Theory of Individual Health Behavior. TTB holds the potential to help researchers and policymakers better understand and intervene on the harms of trauma, and to ultimately support the development of interventions to reduce health behavior disparities. A primary implication of TTB is that health practitioners and agencies promoting behavior changes to address specific health harms (especially long-term health consequences, such as cigarette-related outcomes) must account for the role of competing stressors and sources of more immediate harm individuals are also facing – specifically indicating that alleviating the conditions of trauma-replicating environments (such as poverty, homelessness, and lack of access to healthcare) may be necessary steps to addressing health disparities faced by individuals living in these environments. Next steps should aim to apply and evaluate the validity and utility of TTB as a research framework.

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Chapter 3 Aim #1 – The Association Between Historical Trauma and Substance Use: A Scoping Review

Abstract

Background. Historical trauma, defined as a collective wound, experienced by a community, with lasting detrimental impact across future generations, has been indicated as a source of many modern harms, including substance use.

Objectives. In this scoping review, we aimed to capture and reflect on original research examining the relationship between historical trauma and substance use. The goal of this review was to characterize how historical trauma has been conceptualized and associated with substance use in both the qualitative and quantitative literature, to discuss incongruities between the two, and to set forth considerations for future endeavors seeking to study the relationship between historical trauma and substance use.

Eligibility Criteria. Original, peer-reviewed research in English published after 1980 was considered. To be included, qualitative studies needed to connect historical trauma with modern substance use and quantitative studies needed to measure both historical trauma and substance use and measure their association.

Sources of Evidence. PubMed, Scopus, PsycInfo, and the Cumulative Index to Nursing and Allied Health Literature

Results. Overall, our review included 26 papers, 19 qualitative and 7 quantitative. A majority focused on Indigenous peoples living in the United States, Canada, and Australia. Throughout the qualitative literature, participants reflected on the inextricable nature of historical harms their people have faced and modern substance use harms they now experience – naming

both centuries of colonization broadly and specific historic policies, such as residential schools, as sources of modern substance use harms. The quantitative literature generally focused specifically on how thoughts and feelings towards historical harms were associated with substance use behaviors. Qualitative studies indicated that historical harms impact people regardless of their awareness of them, indicating that self-report quantitative data may be inadequate for fully capturing the relation between historical harms and modern substance use.

Conclusion. As has been discussed by Mohatt and colleagues, historical trauma may best be conceptualized as a narrative tool that communities can employ to understand how historical traumas have resulted in modern harms. Further, the connections consistently drawn in the literature between historical trauma and modern systems of oppression, in which substance use is understood as a symptom, raise ethical questions of the appropriateness of research endeavors narrowly focused on substance use. The use of action-oriented, participatory, decolonizing research frameworks which center research participant voices and their right to liberation from oppressive systems is recommended for future research efforts. While most of the literature focused on Indigenous communities impacted by European settler-colonialism, the historical trauma narrative can be considered when conducting research with other communities subject to modern systems of oppression.

Introduction

Over the past two decades, the deleterious impact of historical trauma on present-day health has been identified in the literature (Brave Heart and DeBruyn; Mohatt et al.; Gone et al.). Mohatt et al. summarize historical trauma as "a complex and collective trauma experienced over time and across generations by a group of people who share an identity, affiliation, or circumstance" (Mohatt et al. 128). The historical trauma concept, developed to better describe the experiences of survivors of the Holocaust (Kellerman), has been applied to studying oppressed and persecuted peoples globally such as Palestinians displaced during the Nakba (Daoud et al.), Black descendants of slaves in the United States (Degruy-Leary), and descendants of Armenian refugees (Karenian et al.). In particular, a growing body of quantitative research has highlighted the connection between historical trauma and poor health outcomes among Indigenous peoples of the United States and Canada (Gone et al.).

Of particular interest is the relationship between historical trauma and substance use behaviors and outcomes. Writing about research among Indigenous peoples of North America in 2003, Maria Brave Heart Yellow Horse stated that, "continued research must include … [the] study and assessment of the [Historical Trauma Response] and its relationship with substance use" (Brave Heart, "The Historical Trauma Response among Natives and Its Relationship with Substance Abuse: A Lakota Illustration" 12). The importance of this is highlighted by the fact that Indigenous people in the United States report higher rates of recent tobacco and illicit drug use than any other single racial/ethnic group (SAMHSA). However, a 2019 systematic review by Gone et al. examining the impact of historical trauma on health outcomes among North American Indigenous populations identified only 5 manuscripts which quantitatively assessed the relationship between historical trauma and substance use outcomes (Gone et al.). The paucity of

available quantitative research indicates that the study of the impact of historical trauma on substance use remains a developing line of inquiry. This lack of research is even more stark for other populations subject to historical harms.

The purpose of this scoping review is to present the current state of research focusing on the impact of historical trauma on substance use. The objectives of this study are: 1) to summarize how historical trauma's impact on substance use has been characterized in the qualitative literature; 2) to identify how historical trauma has been operationalized in the quantitative literature; 3) to summarize the findings of the quantitative literature; and 4) to describe incongruities between the qualitative and quantitative literature. From these findings, we will discuss considerations for incorporating historical trauma into future substance use research.

Methods

We conducted a scoping review of peer-reviewed, original research reflecting on the impact of historical trauma on substance use behaviors and outcomes. Given that 1) the breadth and comparability of the literature on historical trauma and substance use are not readily clear and that 2) the objectives of this review focus on identifying types of available evidence, examining how research is conducted, and on identifying and reflecting on gaps (and potential inconsistencies) in the literature, a scoping review was deemed preferable to a systematic review (Munn et al.). The review was conducted following the PRISMA Extension for Scoping Reviews (see **Appendix A** for completed PRISMA-ScR checklist) (Tricco et al.). Given challenges in anticipating the structure and scope of the results of this review, no protocol for this review was not registered.

Research Question

The research question driving this scoping review is, "What is the state of research exploring the association between historical trauma and substance use?" The four objectives highlighted will shape the presentation of results: first, reflecting on the findings of qualitative studies; second, on how historical trauma has been operationalized in the quantitative literature; third, on the findings of quantitative studies; and fourth, on similarities and incongruities between the qualitative and quantitative findings. In regard to the fourth objective, we highlight that examining incongruities between qualitative and quantitative findings can help illustrate key differences between how respondents conceptualize the question at hand (via qualitative methods) versus how researchers measure it (via quantitative methods) (Wagner et al.).

Search Protocol

Database searches were conducted in PubMed, Scopus, PsycInfo, and the Cumulative Index to Nursing and Allied Health Literature (CINAHL) on January 1st, 2021. Two search terms were developed: the first, capturing historical trauma and, the second, capturing substance use. The search strategy, adapted for each database, returned all papers which were captured by both the historical trauma and the substance use terms. The search queries for each database are available in **Appendix A**.

Formalizing the Definition of Historical Trauma for the Review

To drive this review, we apply Mohatt et al.'s definition of historical trauma. They highlight that historical traumas are defined by three components: "[the first,] a 'trauma' or wounding; [the second,] the trauma is shared by a group of people, rather than an individually experienced [sic]; [and, the third,] the trauma spans multiple generations such that contemporary members of the affected group may experience trauma-related symptoms without having been present for the past traumatizing events(s)" (Mohatt et al. 128). As such, to be included in this

review, manuscripts must have reflected on traumas which match this definition. Of note, we highlight that manuscripts that solely focused on the transmission of trauma from parent to child (such as measured by the Adverse Childhood Experiences Scale) without connecting these harms to an historical event (component number one) that is shared by a group of people (component number two) were excluded.

Inclusion Criteria

We included English, peer-reviewed, original research articles published after 1980 which applied qualitative, quantitative, or mixed methods approaches to study the association between historical trauma and substance use. To be included, qualitative studies had to identify the connection between historical trauma and substance use as a primary theme or had to present participant reflections on the connection between historical harms and substance use. To be included, quantitative studies had to operationalize both historical trauma and substance use and measure their association. We chose 1980 as a cut-off because the historical trauma construct was not broadly adopted within the health behavior literature until the late 1990s (Brave Heart and DeBruyn; Mohatt et al.; Gone et al.) and we wished to capture important papers that may have proceeded this adoption.

Screening and Selection

Search results were uploaded to Rayyan for title and abstract screenings (Ouzzani et al.). After removing duplicate articles, titles and abstracts were screened by 2 reviewers (CM and BD). After initial screening, authors met to resolve discrepancies in the screening process. Manuscripts chosen for full-text screening were then exported to an Excel document. Full texts were then screened by the same two reviewers with discrepancies adjudicated by the senior author.

Data Extraction

Data were extracted into a standardized form by CM and then reviewed by BD for consensus. Reviewers then met to resolve any discrepancies. First, study details were extracted including the geographic location, date, target population, and sample size. For qualitative studies, primary findings relating historical trauma and substance use were extracted. For quantitative studies, the tools used to operationalize historical trauma and substance use and the results were extracted.

Results

Study Selection and Included Articles

The initial search, run on January 1st, 2021, returned 742 citations (see **Figure 3.1**). After removing duplicates, 405 title/abstracts were screened by reviewers. At this stage, 340 citations were excluded. The remaining 65 citations were then subject to full-text screening and 26 citations were excluded. In total, 19 qualitative and 7 quantitative studies were judged to have met study inclusion criteria.

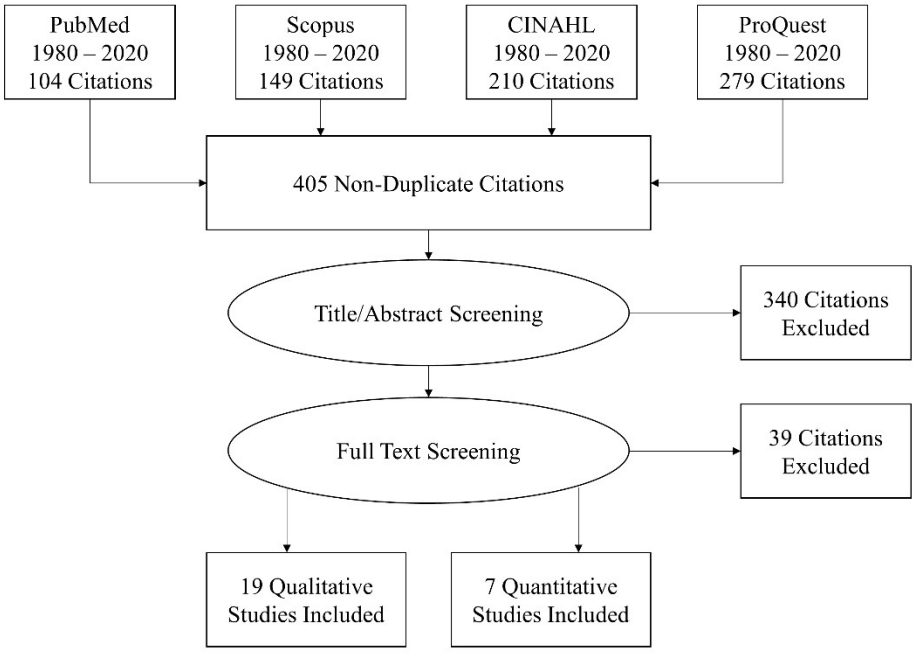

Figure 3.1 PRISMA Flowchart

Findings from Qualitative Literature

Of the 19 qualitative papers included (see **Table 3.1**), 14 focused on Indigenous peoples in the United States and Canada (Christiansen et al.; Skewes and Blume; Hartmann and Gone; Walls et al.; Goodkind et al.; Myhra; Bird et al.; Grayshield et al.; Reinschmidt et al.; Jervis and American Indian Service Utilization; Gonzales et al.; Shahram et al.; Brave Heart, "*Oyate Ptayela*: Rebuilding the Lakota Nation through Addressing Historical Trauma among Lakota Parents"; Gone), 3 focused on Indigenous peoples in Australia (MacLean et al.; Reid et al.; Kendall, Lighton, Sherwood, Baldry, Sullivan, et al.), and 2 focused on Ukrainian survivors of the Holodomor (a genocide by hunger enacted against Ukrainians by the Soviet Union in the 1930s) and their descendants (Bezo and Maggi, "Intergenerational Perceptions of Mass Trauma's Impact on Physical Health and Well-Being"; Bezo and Maggi, "Living in 'Survival Mode:' Intergenerational Transmission of Trauma from the Holodomor Genocide of 1932–1933 in Ukraine").

Table 3.1 Qualitative Studies Included in the Review.

Article	Geographic Location	Dates	Target Population	Sample Size	Primary Findings
(Reid et al.)	Remote, Northwest Queensland, Australia	2018-2020	Care Providers Who Work with Aboriginal Patients	87	History of colonization integral to understanding current alcohol use among Aboriginal Australians. Summarized by one participant, "if they want to stop all this [fetal alcohol spectrum disorder] and the chronic diseases that are affecting our people you have to address all these underlying issues that history has created that has put us in this situation."
(Christianse n et al.)	Upper Midwest and Southwest	July 2010 - August 2011	North American Indigenous Women	89	It is broadly stated that "Participants connected the prevalence of drug and alcohol misuse to…the disruption in family life across generations brough by boarding schools, and the loss of cultural knowledge and traditions under colonization."
(Skewes and Blume)	Montana	Fall 2015 - 2016	Tribal Members "Knoweldgeabl e about substance use, tribal culture, and reservation life"	25	Many participants initiated the interview by stating something akin to, "Before I answer your questions, you need to understand what has happened to our people in the past." Participants referred to concerns with meth use (as a result of oil company camps), alcohol, and prescription drug use -- they highlight that "community concerns are symptoms of larger problems -- namely a history of colonialism and racism -- that have affected the community for generations." Stated by a participant "Oppression is the overarching umbrella for all of sickness with drugs and alcohol".
(Gonzales et al.)	Portland, Oregon	Summer 2014	Indigenous People	74	Many participants "viewed alcohol as a result of colonization and oppression, and some believed that alcohol was 'intentionally introduced into Native communities by the colonizer to harm them' as a weapon of genocide and instability. An adult participant states, 'I think what's going on here is a 100-year old war that is meant to destroy us. That's why the colonizers brought [alcohol] here and it's hitting us from all directions.'" Participants described alcohol use as a consequence of historical trauma and source of contemporary trauma. In this way alcohol use perpetuates the cycle of historical harms.

Table 3.1 Qualitative Studies Included in the Review, continued.

Article	Geographic Location	Dates	Target Population	Sample Size	Primary Findings
(Bezo and Maggi, "Intergenerational Perceptions of Mass Trauma's Impact on Physical Health and Well-Being")	Ukraine	July - November 2010	Ukrainians Impacted by the Holodomar and their descendents	45	One participant reflected on how "the Holodomor destroyed the sense of community-belonging, which then created an intergenerational cycle of social isolation and low collective trust. In turn, these community-related consequences continue to foster an environment conducive for alcohol and drug abuse. Alcohol abuse was noted as a male risky health behavior that adversely affects men's health."
(MacLean et al.)	Mildura, Australia	Late 2014 - Early 2015	Aboriginal people who use methamphetamine, their family, and community care providers	26	While the study largely does not reflect on how historical events have lead to meth use in the present, one participant reflected that: "If you lose something towards your culture and identity, well then of course you're going to use more drugs."
(Shahram et al.)	Three Canadian Cities	2012-2016*	Aboriginal Women with history of pregnancy in Canada	23	Participants reflected that the residential schools have resulted in drug use in future generations. One participant highlighted how being 3rd generation impacted by residential schools fated her into her current situation involving drug use: "My mom, my grandma was down here [dealing with drug use], my mom is down here and I'm down here…Well I kind of figured it out now because of the residential school right. I don't know, it just seems like it was...there was no way out of being...for me to be down here." Another participant articulated that the cycle of trauma is passed on from the event (residential schools) through each generation.
(Bezo and Maggi, "Living in 'Survival Mode:' Intergenerational Transmission of Trauma from the Holodomor Genocide of 1932–1933 in Ukraine")	Ukraine	July - November 2010	Ukrainians Impacted by the Holodomar and their descendents	45	The Holodomor was a Soviet-led genocide of Ukrainians in the 1930s. Many reported that Ukrainian men used alcohol to lessen the appearance of being independent and anti-Soviet. Being weak and ill meant men were not a threat and this embodied weakness via alcohol was adopted in response to genocide and continues to impact Ukrainians today.

Table 3.1 Qualitative Studies Included in the Review, continued.

Article	Geographic Location	Dates	Target Population	Sample Size	Primary Findings
(Hartmann and Gone)	US Great Plains	August, 2012	Indigenous Medicine Men	2	Interviews with two medicine men, George and Henry. George discussed how substance use is a result of historical trauma. George shares how traditional dancing can help individuals heal from the harms of historical trauma and that this helps them avoid substance use. Further, that this healing, once done, is passed down to the next generation. Henry spoke of how modern systems of oppression, such as the reservation system, drive substance use problems. Henry viewed historical trauma as an important tool for discussing the past but felt that talking about modern systems of oppression were more relevant when discussing substance use outcomes.
(Walls et al.)	Central Canada Indigenous Reserves	Summer 2005	First Nations in Canada	~45; 3 focus groups	Participants point to the detrimental impact of colonization and policies such as the residential schools. One participant notes that "These problems we have are just symptoms of our main problems, I think… like we try to solve alcohol problems…those are just symptoms to me… Like residential schools, for example, that's where we lost pretty well everything" Another then notes that prior to European contact there was "no reason for committing suicide" but that with contact came many ills including how they turned to alcohol to cope.
(Goodkind et al.)	Diné Land/Navajo Nation	2006-2012*	Diné	37	Most children did not connect HT with current situations, many were unaware of history. Similar with parent-aged adults (~30 years old). One person shared an anecdote that their brother said "If it weren't for the White people, we wouldn't have been alcoholics", directly tying substance use to changes brough by European contact. When talking about the Long Walk, one participant responded by discussing modern day alcohol use in the community, indicating an understood connection. Elders appeared more conscious of the role history has played and the impact of colonization, as one states "All those substances, they are from the White people, they have plagued us with it. This is what my maternal grandma used to tell me, that all those substances have afflicted us as a people.... I think all of the things that affect us today, it is a disease, passed down to us from the [White people]. The alcohol is a disease that was put upon us by the [White people]."

Table 3.1 Qualitative Studies Included in the Review, continued.

Article	Geographic Location	Dates	Target Population	Sample Size	Primary Findings
(Myhra)	Minneapolis, Minnesota	*	Indigenous people recruited from sobriety maintenance programs	13	One participant reflects that they were "born too late" and that "my alcoholism can be linked to the notion that this society that we live in here is not my society; it's not my culture." This combination, of reflecting on being born at the wrong time indicates an understanding that their society/culture was taken, that this historical taking represents an historical harm which is embodied today in their alcohol use. Authors further reflect that "all but one participant connected their drinking or other substance abuse to their desire to numb themselves from cumulative stress related to historical trauma".
(Bird et al.)	Crow Reservation (SE Montana)	*	Crow	20	Quite generally, the results (theme 1) indicates that negative mental health is a result of historical harms and that these harms can lead to substance use and abuse.
(Grayshield et al.)	Western, Southwestern, and upper Midwest USA	*	Indigenous People in US	11	First, a participant reflected on how historical trauma of colonization manifests in drug use, stating: Describing how "thousands of alien people" came in an destroyed the earth by "digging everything up, tearing the tress down, muddying the waters..and shooting everything..putting fences up." Another participant then elaborated that just learning about this history "Your mind, your body, and your spirit don't know what to do with it. So you dwell on it. And to relieve that, you go to alcohol or some kind of addiction. As you do it, you destroy yourself and your whole being." Then, the paper directly identifies substance abuse as a direct harm of historical trauma
(Reinschmidt et al.)	Tucson Arizona	*	Indigenous in Tucson	13	Elders "understood alcohol and drugs as rooted in history with health and social consequences. They described the introduction of alcohol as part of historical trauma and described alcohol use as a coping strategy."
(Jervis and American Indian Service Utilization)	Northern Plains (US)	*	Indigenous	44	One participant reflected on how things were different "50 or 60 years ago or before the reservation days". She talks about many of the harms they are now subject to including "the alcohol factor too." This paper also highlights the "circular relationship" of community and individual trauma, where historical trauma and community trauma fuel "dysfunctional behavior (e.g., widespread alcohol abuse)" and then the alcohol use drives further traumatization.

Table 3.1 Qualitative Studies Included in the Review, continued.

Article	Geographic Location	Dates	Target Population	Sample Size	Primary Findings
(Brave Heart, "*Oyate Ptayela*: Rebuilding the Lakota Nation through Addressing Historical Trauma among Lakota Parents")	Lakota Nation	1990s*	Lakota Parents	10	This study provided participants with an educational curriculum aimed at learning about historical trauma and developing resilience through re-connection with traditional knowledge to improve parenting. After receiving the curriculum, multiple participants reflected on how alcohol use was a natural response to being forced into the boarding schools and how the internalization of this trauma is passed down to future generations.
(Gone)	Northern Algonquian Reserve (Canada)	2003-2004	Staff at a First Nation Healing Lodge	19	Reflecting on the impact of colonization, participants reflected on the connection between these historical harms and multiple issues within the community, including "drugs". When asked about the source of all these problems, including drugs, one participant said, "From the Western society. Colonizationists. Europeans." They also reflected on the impact of specific policies, such as one in which traditional hunting grounds were flooded. Participants reflected on the connection between these policies and people drinking alcohol more.
(Kendall, Lighton, Sherwood, Baldry, and Sullivan)	New South Wales (Australia)	2013 & 2016	Incarcerated Aboriginal Women	43	Many participants reflected on their experiences having their children taken from them. They reflected on how this was not simply an acute instance of trauma, but that for generations children had been taken from their parents. They reflect that the loss of a child in this way was connected with substance use, but also that these harms have manifested across generations.

*Precise dates of data collection are unclear based on text, general ranges are provided if text allows

Studies among Indigenous peoples in North America often reflected on the inextricable nature of the history of colonialism and modern harms, including those related to substance use. As noted by Skewes and Blume in their study aiming to understand the connection between racial trauma and substance use among Indigenous peoples in Montana, many participants began their interviews by saying a variation of: "Before I answer your questions, you need to understand what has happened to our people in the past" (Skewes and Blume 9). They note that several such

interviews began "with lengthy discussion about the history of the region and its peoples" (Skewes and Blume 9). Similarly, in Walls et al., an Indigenous man from central Canada spoke that, "These problems we have are just symptoms of our main problems … like we try to solve alcohol problems … [but] those are just symptoms to me" (Walls et al. 12). In Myhra et al., an Indigenous person in Minneapolis reflected on how they were "born too late" and that their "alcoholism can be linked to the notion that this society that we live in here is not my society; it's not my culture" (Myhra 23). Such accounts indicate that, for many Indigenous peoples in North America, that substance use must be understood within the context of historical harms enacted against their people, broadly.

Further, that these historical harms continue to manifest in myriad ways, including via drug use. As noted in Reinschmidt et al., Indigenous elders in Tucson, Arizona "understood [the use of] alcohol and drugs as rooted in history with health and social consequences" (Reinschmidt et al. 7). In such accountings, substance use is understood as a natural response to centuries of enacted genocide.

While many Indigenous study participants understood current drug use as being a result of centuries of harm enacted by European colonizers, many spoke of specific traumatic historical events and how they relate to current circumstances. The residential/boarding school era, in which Indigenous children in Canada and the United States were stolen from their homes and forced to assimilate at the threat and enactment of abuse, was frequently cited as a specific event driving many harms today, including substance use. As one participant in Canada noted, "the residential schools, for example, that's where we lost pretty well everything" (Walls et al. 12). For adults and elders, they generally understood the residential schools as a wounding and described how this had impact across multiple generations. Christiansen et al. write that "some women described their own boarding school experience, or that of their parents, and how it disrupted family ties and the

transmission of cultural knowledge" (Christiansen et al.). An Indigenous woman in Canada whose grandmother was forced into the residential schools spoke of how she understood her fate to be inextricably tied to her grandmothers, stating "my grandma was down here [dealing with drug use], my mom is down here and I'm down here…because of the boarding school right" (Shahram et al. 253). The residential schools represent a tangible example of the broader historical harms enacted against Indigenous people and, for many participants, discussing them provided the context to describe the passage of trauma from one generation to the next. While residential schools were the most prominent specific harm identified in the literature, other policies with detrimental harm were noted, such as environmental policies which destroyed traditional hunting grounds (Gone).

To understand the impact of historical trauma on current circumstances, a knowledge of historical harms appears to be necessary. The goal of the residential schools was to destroy Indigenous culture and force assimilation into the dominant Euro-Christian society (Truth and Reconciliation Commission of Canada) – the recent discovery of a mass unmarked grave outside the Kamloops residential school in British Columbia, Canada displays both the severity of harms enacted and that these harms remain a living trauma which have not been resolved or rectified (Paperny). Those alive during that time period or with parents impacted by it displayed awareness for the detrimental impact of these institutions. However, Goodkind et al. noted that most of the Diné (Navajo) youth they interviewed did not connect the current circumstances of their people to historical events – when asked about hardships their ancestors faced, many youth reflected on the lack of electricity and running water as opposed to the harms of colonization highlighted by elders (Goodkind et al.). The importance of learning about historical harms was further exemplified in the educational curriculum taught to Lakota parents by Brave Heart. Lakota parents were provided

a curriculum which went over the impact of residential schools on their people and respondents, after receiving the curriculum, displayed an awareness between the harms of the residential schools and current substance use among Lakota parents (Brave Heart, "*Oyate Ptayela*: Rebuilding the Lakota Nation through Addressing Historical Trauma among Lakota Parents"). This indicates the role that learning about history plays in understanding the impact of historical harms – individuals cannot draw connections between historical events and present circumstances without awareness of those events.

Additionally, many participants did not only situate substance use as a result of historical trauma, but, that substances (alcohol in particular) were used as a form of warfare to enact said traumas. The arrival of Europeans to the continent was understood as a harbinger of violence, as one participant described how the colonizers arrived and destroyed the earth by "digging everything up, tearing trees down, muddying the waters…and shooting everything…putting fences up" (Grayshield et al. 301). Gonzales et al. discussed how many participants "believed that alcohol was 'intentionally introduced into Native communities by the colonizers to harm them' as a weapon of genocide and instability" (Gonzales et al. 288). This indicates that substance use should not be thought of as solely a result of historical harms, but that substance use can be a vector by which harms are enacted ("wounding") and by which these harms are propagated from generation to generation. Participants in a study by Jervis discussed a repeating cycle by which historical harms drive increases in alcohol use and how alcohol use likewise then drives additional harms, and so on (Jervis and American Indian Service Utilization).

Finally, participants across studies generally articulated a wide range of conceptions of historical trauma, which included not using the language of historical trauma. For example, in discussions with two medicine men from Great Plains Indigenous communities: the first described

historical trauma as a direct cause of current harms facing their community; the second discussed how historical trauma was an important construct for discussing the past, but that current focus should be placed on modern systems of oppression facing their community (Hartmann and Gone). As discussed in Reinschmidt et al., many participants did not use the phrase "historical trauma" when discussing the impact of historical events on modern circumstances, but that the many different ways people conceptualized the link between the past and present were consistent with academic definitions of historical trauma (Reinschmidt et al.).

The three studies of Aboriginal peoples in Australia identified similar themes of the role of historical harms related to colonization and modern substance use harms. As stated by a participant in Reid et al., "Realistically you know if they want to stop all this [Fetal Alcohol Spectrum Disorder] and the chronic diseases that are affecting our people you have to address all these underlying issues that history has created that has put us in this situation" (Reid et al. 6). Another participant highlighted how the weight of historical harms continues to impact them, stating that, "I am only young, but I still feel the stress of 50 years ago" (Reid et al. 6). All three studies highlighted the broad understanding that community substance use harms were tied directly to historical harms. This point was directly addressed by a participant in MacLean et al., who stated, "If you lose something towards your culture and identity, well then of course you're going to use more drugs" (MacLean et al. 506). In the work by Kendall et al., participants reflected specifically on how the state-sanctioned stealing of children across generations was directly tied to substance use (Kendall, Lighton, Sherwood, Baldry, Sullivan, et al.).

While most of the studies focused on the impact of colonization on Indigenous communities impacted by European colonization, two manuscripts by Bezo et al. from the same underlying study focused on the lasting harms faced by survivors and their descendants of the

Holodomor, a genocide via starvation enacted in the 1930s by the Soviet Union against Ukrainians (Bezo and Maggi, "Living in 'Survival Mode:' Intergenerational Transmission of Trauma from the Holodomor Genocide of 1932–1933 in Ukraine"; Bezo and Maggi, "Intergenerational Perceptions of Mass Trauma's Impact on Physical Health and Well-Being"). They discuss how "the Holodomor destroyed the sense of community-belonging, which then created an intergenerational cycle of social isolation and low collective trust. In turn, these community-related consequences continue to foster an environment conducive for alcohol and drug abuse" (Bezo and Maggi, "Intergenerational Perceptions of Mass Trauma's Impact on Physical Health and Well-Being" 90). Men were noted to be at higher risk of adopting alcohol use as a response to historical harms (Bezo and Maggi, "Intergenerational Perceptions of Mass Trauma's Impact on Physical Health and Well-Being"), with several participants discussing how many Ukrainian men adopted alcohol use in the wake of the Holodomor in order to adopt the appearance of weakness as a form of protection against future persecution (Bezo and Maggi, "Living in 'Survival Mode:' Intergenerational Transmission of Trauma from the Holodomor Genocide of 1932–1933 in Ukraine"). Such reports indicate that drug use may emerge as a survival response to cope with past harms or to mitigate the replication of such harms again. Overall, this further highlights the importance of contextualizing substance use in historical harms to better understand their origin and trajectory.

Operationalization of Historical Trauma in the Quantitative Literature

Six of the seven quantitative studies included focused on Indigenous peoples in the US and Canada (see **Table 3.2**). All 6 of these studies used the Historical Loss Scale (HLS) (Les B. Whitbeck et al.). The HLS was developed to capture the frequency with which participants thought about a series of historical harms specific to Indigenous people across North America, including

how often they think about loss of land, loss of language, the harm of the boarding schools, among other harms (Les B. Whitbeck et al.). Of note, one of the harms in the scale is how often an individual thinks about "the losses from the effects of alcoholism on our people" (Les B. Whitbeck et al.). Responses to each of the 12 items are summed such that lower scores correspond to higher levels of thinking about historical losses.

Table 3.2 Quantitative Studies Included in the Review

Title	Geographic Location	Dates	Target Population	Sample Size	HT Measurement	SU Measurement	Primary Findings
(Aviad-Wilchek et al.)	Israel	*	Ethiopian Adolescents in Israel	510	Participants were asked if their parents experienced immigration-related trauma and then whether the impact of this trauma was "positive" or "negative" (Likert scale)	Readiness to use Alcohol and Readiness to use Illegal Drugs, adopted from Aziza and Abu-Asaba	Study found that adolescents whose parents faced traumas or deaths of family members during migration to Israel reported higher readiness to use alcohol and to use illegal drugs.
(Soto et al.)	California	2008	Indigenous Youth	969	Historical Loss Scale	Asked if participant had ever tried cigarettes and then how many days in prior month they had smoked cigarettes?	Found that historical trauma was predictive of experimental smoking and past month smoking. They also reflected on mediating pathways.

Table 3.2 Quantitative Studies Included in the Review, continued.

Title	Geographic Location	Dates	Target Population	Sample Size	HT Measurement	SU Measurement	Primary Findings
(Pokhrel and Herzog)	Hawaii	*	Native Hawaiian Community College Students	128	Historical Loss Scale and Historical Traumatic Events Scale	Past 30 days use and frequency of use of cigarettes, alcohol, and marijuana use. Final variable was any use of either cigarettes, alcohol, or marijuana in past 30 days	Found that historical trauma increases substance use via indirect path through perceived discrimination, but that it directly diminished risk of historical trauma. In the discussion, they do argue that this may be because people who report higher levels of historical trauma might just be expressing greater awareness and greater familiarity with HT -- this in turn is likely associated with recognizing and identifying discrimination.
(Ehlers et al.)	US	*	Indigenous People	306	Historical Loss Scale and Historical Loss Associated Symptoms Scale	Semi-Structured Assessment for the Genetics of Alcoholism survey was used to make substance use disorder diagnoses using DSM 3 criteria	Found that historical loss scale scores and historical loss associated symptoms were associated with any substance dependence.
(Wiechelt et al.)	Baltimore, MD	*	Urban Indigenous People in US	120	Historical Loss Scale and Historical Loss Associated Symptoms Scale	Lifetime and Past 30 Day Substance Use	Found that elevated historical loss associated symptoms (i.e., negative feelings when thinking about historical loss) were associated with increased odds of reporting past 30 days alcohol use and of reporting lifetime illicit drug use. No significant findings were found for thoughts about historical loss and recent or lifetime drug use.

Table 3.2 Quantitative Studies Included in the Review, continued.

Title	Geographic Location	Dates	Target Population	Sample Size	HT Measurement	SU Measurement	Primary Findings
(Les B Whitbeck et al.)	Upper Midwest, US and Central Canada	*	Indigenous People in US and Canada Who are the Parent/Guardians of Children Aged 10 - 12	452	Historical Loss Scale and Historical Loss Associated Symptoms Scale	UofM Composite International Diagnostic Interview to assess 12-month diagnosis of alcohol abuse (DSM-3)	Found that elevated levels of historical loss are associated with an increased risk of past year alcohol abuse (DSM-3). Further, found that feelings of historical loss mediate the relationship between perceived discrimination and alcohol abuse.
(Spence et al.)	Ontario, Canada	2012-3	Indigenous Adults -- Kettle and Stony Point Communities	340	Historical Loss Scale	"Ever Use Cannabis more than once a week?"	Found a null relationship between historical loss measurement and cannabis use.

*Precise dates of data collection are unclear based on text, general ranges are provided if text allows

Three of these studies also applied the Historical Loss Associated Symptoms Scale (HLASS), developed alongside the HLS (Les B. Whitbeck et al.). The HLASS is intended to accompany the HLS and asks the participant to reflect on how they feel when they think about the historical losses asked about in the HLS. Respondents are given a series of emotional (i.e., "Often feel sadness or depression", "Often feel anger", etc.) and cognitive (i.e., "Feel like it is happening again", "Feel like avoiding places or people that remind you of these losses") prompts and respond via Likert scale. The responses are then summed up such that a higher score indicates more frequent negative feelings associated with thinking about historical loss.

An important note about both the Historical Loss Scale (HLS) and the Historical Loss Associated Symptoms Scale (HLASS) is that both measurement tools focus on how often the individual thinks about historical loss and how they feel when thinking about historical loss. Quantitative studies which use these scales are, thus, able to reflect on how thinking about historical loss and the feeling associated with such thoughts impact substance use. We may understand that perceptions of and feeling towards historical harms represent one pathway by which historical harms manifest in the current day. For example, people who are unaware of or

don't think about certain historical harms, such as the Diné youth interviewed by Goodkind et al., are still subject to current circumstances have manifested as a result of historical harms.

The final quantitative study focused on the drug use outcomes of the children of Ethiopian Jews who had to emigrate to Israel as a result of religious persecution in the 1980s (Aviad-Wilchek et al.). Instead of using a validated scale, participants were asked if their parents were exposed to trauma during their migration and if a family member died during the migration process. The need to immigrate was framed as a collective wound and these questions were used to try to capture the severity of these wounds. Like with the scales described above, these questions capture the individual's perceptions of harms enacted on their ancestors. The framing of this study also highlights potential types of study our review may have missed – in which a specific historical traumatic event impacts a marginalized community. Such studies may not use the language of historical trauma but do reflect on an event or series of events that fit within the definition of historical trauma used for this study.

Findings from Quantitative Literature

As noted, 6 of the 7 quantitative studies examined the relationship between historical trauma and substance use among Indigenous populations in the US and Canada. Soto et al. found that higher HLS scores were associated with elevated likelihood of ever having tried smoking commercial cigarettes and of reporting commercial cigarette use in the prior month (Soto et al.). Wiechelt et al. found that higher HLS/HLASS scores were associated with elevated risk of past 30 days alcohol use and lifetime illicit drug use (Wiechelt et al.). Whitbeck et al. also found that elevated HLS/HLASS scores were associated with an elevated risk of having a diagnosable alcohol use disorder (DSM-III) in the prior year (Les Whitbeck et al.). Ehlers et al. found that elevated HLS and HLASS scores were associated with an elevated likelihood of reporting substance

dependence (Ehlers et al.). Interestingly, Pokhrel et al. found that elevated HLS scores were indirectly associated with elevated risk of substance use via perceived discrimination, but that elevated HLS scores directly diminished risk of substance use (Pokhrel and Herzog). Finally, Spence et al. investigated the relationship between HLS scores and cannabis use and their findings were inconclusive (Spence et al.).

The final study, from Aviad-Wilchek et al., explored readiness to use psychoactive substances among second-generation adolescent Ethiopian immigrants living in Israel (Aviad-Wilchek et al.). Ethiopian Jews migrated to Israel in two waves (Operation Moses [1985-1986] and Operation Solomon [1990-1991]) – both waves were precipitated by violent persecution and poor living conditions, representing a collective wounding (Aviad-Wilchek et al.). Participants were asked if their family members who immigrated experienced specific traumas or deaths of family members to attempt to capture the severity of the harms experienced during the migration. Their study found that adolescents whose parents faced increased traumas and deaths of family members during the migration were more likely to report higher levels of readiness to use alcohol and illegal drugs (Aviad-Wilchek et al.).

Incongruities Between the Qualitative and Quantitative Literature

An immediate distinction between the qualitative and quantitative literature is in how historical trauma is conceptualized and understood. Across qualitative studies included in this review, participants indicated that it is necessary to understand the role historical traumas have played in shaping modern day circumstances, including substance use. The quantitative literature, on the other hand, largely focuses on a single mechanism (i.e., thinking about historical events and the emotions that accompany those thoughts) by which historical trauma may influence modern substance use harms. The qualitative literature does reflect on this mechanism, as one participant

in Grayshield et al. reflects that, “Your mind, your body, and your spirit don’t know what to do with [the historical harms of colonization]. So, you dwell on it. And to relieve that, you go to alcohol or some kind of addiction” (Grayshield et al. 301). However, the qualitative literature indicates that there are other mechanisms by which historical harms drive current substance use and that awareness of historical harms is not necessary to be impacted by those historical harms.

Of note, several quantitative studies contextualized historical trauma within mediating pathways (via structural equation modeling). While participants in qualitative studies often discussed how all modern circumstances must be understood in relation to historical harms enacted, several quantitative studies framed perceptions of the harms of historical trauma in a mediating role. This is possible, in part, because the quantitative studies in question measure individuals’ perceptions of historical harms, not the actual historical harms themselves (as historical harms themselves temporally precede the individual and cannot be a mediator). This is important when considering the qualitative study among Diné people in which many youths, less familiar with historical events, did not connect historical harms with modern circumstances. The narratives of historical harms depicted in the qualitative literature were generally focused on events and their impact on people and communities. Individual-level, quantitative studies were limited in their ability to reflect on historical trauma which is defined by community-level harms and outcomes. In essence, a person need not be aware of a historical event to be impacted by it and, as a result, quantitative research relying on self-report data may not be able to effectively contextualize current circumstances within the breadth of relevant historical harms. This concern is highlighted in Pokhrel et al., which found that greater perceived historical trauma was associated with a decreased direct effect on substance use and an increased indirect effect via discrimination (Pokhrel and Herzog). Pokhrel et al. argue in the discussion of their work that their measure of

historical trauma likely is capturing knowledge of historical harms and that individuals with greater awareness of historical harms may also be more resilient to said harms (Pokhrel and Herzog). Such a finding further highlights concerns about whether or not self-report measures of historical trauma can adequately capture the health impacts of historical harms.

Additionally, the qualitative literature provides clear examples of how historical trauma results in modern substance use harms today. In this literature, participants articulate an understanding that historical harms lead to modern day substance use outcomes and, further, many point to specific examples of historical harms (i.e., residential schools) and how those historical harms have been passed down between generations and manifest today. The quantitative literature, on the other hand, focuses on individuals' perceptions of historical trauma and their impact on substance use. As a result, such studies are not directly addressing the impact of historical harms on current substance use circumstances, but instead (the related process) of the impact of awareness of historical harms. This indicates that individual-level, quantitative research may not be adequately capable of studying the impact of historical trauma, but instead should be considered for the more limited application of studying perceptions of historical trauma on modern circumstances.

Discussion

In this scoping review, we identified peer-reviewed, original research which reflects on the relationship between historical trauma and substance use. This review reflects on how historical trauma has been conceptualized and operationalized within both the qualitative and quantitative literature and reflects on incongruities between the findings of the two. A majority of the research identified focused on Indigenous communities in North America and Australia impacted by European colonization. Indigenous participants in qualitative studies frequently indicated that one

cannot fully understand substance use within their communities without understanding the context of historical harms that their people have faced and continue to face. These harms were described both in the abstract (i.e., genocide and colonization broadly) and by referencing a specific historical "wounding" (i.e., residential schools). Whereas quantitative studies (which applied the HLS and HLAS scales) attempted to understand how perceptions and feelings towards historical harms, broadly, influence substance use outcomes. The limitation of measuring historical trauma via self-report is highlighted by Pokhrel et al. who argue that their findings indicate that people who are more aware of historical harms are more likely to be resilient to it (Pokhrel and Herzog).

Thus, it is important to reflect on future steps to study the relationship between historical trauma and substance use. The qualitative research among Indigenous people in the US indicates that modern substance use must be contextualized within historical harms. The quantitative research has reflected on one mechanism by which historical trauma may manifest and impact substance use but does not directly connect historical harms with substance use outcomes. While individual perceptions and feelings towards historical harms represent one mechanism by which historical trauma can manifest in the current moment, the qualitative research provides indication that historical harms can impact people regardless of awareness of said harms. This indicates, broadly, that quantitative, self-report data is likely not capable of capturing the full pathway by which historical harms manifest in modern day substance use outcomes. Further, given that the qualitative literature indicates that the modern circumstances of Indigenous peoples must be understood within the context of historical harms, it is unclear if research modalities which attempt to study narrow and pre-defined (i.e., researcher defined) questions related to substance use behaviors are capable of addressing root causes of substance use disparities. As such, we reflect

here on considerations for conducting future research into the relationship between historical trauma and substance use.

Consideration: What Does It Mean to Study Historical Trauma?

We must first ask what it means to evaluate the relationship between historical trauma and substance use. Epidemiological research is frequently concerned with how given factors may drive individual behavior, but historical trauma, by definition, is not concerned with an individual-level process. Historical trauma is generally intended to capture how historical harms impact a group of people with a shared identity and how these harms manifest in future generations. The qualitative research identified in this review makes clear that, for communities such as Indigenous peoples of North America, that substance use outcomes today must be understood within the historical context of the harms faced by their people. The quantitative research identified attempts to identify how thoughts and feelings towards historical harms impact individuals, but such approaches do not capture information about historical harms, nor the pathway by which said historical harms manifest in modern day substance use. This issue is highlighted by Pokhrel et al.'s finding that awareness of historical harms may confer certain forms of protectivity – indicating that awareness of historical harms isn't a proxy for being impacted by them. In either case, it appears self-report studies via quantitative or qualitative methods cannot, alone, articulate the pathway by which historical harms impact modern circumstances.

As such, historical trauma, as a concept, does not appear to be defined in a manner that can easily be captured or studied via quantitative and statistical methods. Mohatt et al. argue that historical trauma research should not exclusively focus on identifying historical causal variables that influence health (as is typically done in quantitative research), but that instead historical trauma should be understood as a public narrative which a group of people share (Mohatt et al.).

Through this shared narrative, communities are able to contextualize their current circumstances in the historical harms that their people have faced. In this review, the qualitative research provided the space for individuals to reflect on their understanding of the historical trauma narrative their people have been exposed and how they understand said narrative to have led to modern day harms. The quantitative literature, on the other hand, provides neither participants nor researchers the medium through which they can reflect upon or generate such a narrative. As Mohatt and colleagues note, for Indigenous communities globally, the public narrative of colonialism and colonization provides a unifying history which can be applied to make sense of current circumstances (including substance use) (Mohatt et al.).

Consideration: Merging the Historical and the Current

Another important question is how we delineate the past from the present. In Hartmann and Gone's discussions with two Great Plains Indigenous medicine men, both elders conceptualized historical trauma differently: one viewed historical trauma as a direct cause of current harms; the other viewed historical trauma as an important lens for discussing the past, but that greater focus should be placed on modern systems of oppression facing their community (Hartmann and Gone). These two conceptions articulate two important aspects of historical trauma and modern circumstances: the first is that historical harms directly impact the present; and the second is that present day oppression may be understood as an extension of these historical harms. To address the inextricable nature of historical harms from modern circumstances, Burnette and Figley synthesized the Historical Oppression framework (Burnette and Figley). The historical oppression construct is inclusive of historical trauma and contemporary forms of oppression and seeks to capture how historically oppressive power dynamics perpetuate inequality and oppression into the present day (Burnette and Figley). While Mohatt and colleagues discuss historical trauma

as a public narrative, the Historical Oppression framework makes explicit that this public narrative is one of both historical harms and modern oppression.

The connection between historical harms and modern oppression demands that we, as researchers, reflect on the intent of research endeavors – whether it is in the pursuit of knowledge or that of liberation. The qualitative literature identified in this review makes clear that historical harms result in modern substance use harms, as well as shape modern systems of oppression more broadly. While substance use epidemiology tends to situate substance use risk as an individual phenomenon (and that intervention generally targets the individual), systems of oppression act at the societal level. The Belmont Report's second principle is that of beneficence, which indicates that researchers must respect the decisions of study participants, must protect study participants from harm, and must also work to secure their well-being and ensure they are not exploited (Department of Health, Education and National Commission for the Protection of Human Subjects of Biomedical and Behavioral Research). If the evidence indicates the modern oppression of research participants (in the case of this review, Indigenous peoples impacted by ongoing colonization), we must ask if there is an "ethical" way to study historical harms among oppressed communities without also advocating and fighting for their liberation.

Consideration: Emancipatory Praxis and De-Colonizing Methodologies

The Historical Oppression framework grounds its understanding of liberation from oppression in Freirean terms, in that historical systems of oppression are self-perpetuating and evolve into present day systems of oppression (Burnette and Figley; Freire). Paolo Freire posits that humanization (i.e., the dismantling of systems of oppression and resultant freedom of individuals and communities) can be brought about via *praxis* – which is the coupling of critical dialogue to name and characterize systems of oppression with social action to dismantle these

systems (Burnette and Figley; Freire). The growing awareness of the ethical responsibility of researchers to aid in the emancipation of the researched (a relationship frequently defined by an oppressor-oppressed duality) has led to the development of alternate participatory, action-oriented, decolonial research modalities (Zavala; Simonds and Christopher; Smith; Evans et al.). These approaches, in line with Freirean ideals, advocate for the prioritization of Indigenous knowledge, both in terms of understanding how systems of oppression manifest and in prescribing steps to dismantle said systems. Within these frameworks, in line with Mohatt et al.'s conception of historical trauma as a public narrative, we may understand historical trauma as an important narrative tool which provides communities a framework through which to conceptualize how historical systems of oppression manifest in the present.

Consideration: Which History Are We Studying?

Inherent to this idea of historical trauma as a public narrative which can inform praxis, is allowing communities the autonomy to tell their own history. We may understand that an "objective" analysis of history may provide a pathway through which we may disentangle and explain the relation between historical harms and modern-day substance use, however the determination of history is far from an "objective" exercise and may result in a different narrative depending on who is telling it and what sources of knowledge they have access to. It is important to reflect that, as Michel Foucault has said, "each society has its regime of truth" ("TRUTH AND POWER : An Interview with Michel Foucault"). In Foucault's rendering, the ruling class is incentivized to develop a historical account which justifies their position of power. Zembylas and Bekerman argue that the narratives of historical trauma represent "dangerous memories" in that they disrupt the historical narratives that the ruling class evangelize (Zembylas and Bekerman). Examples of such "dangerous memories" abound: to tell the history of the Armenian genocide is

a direct threat to Turkish identity (Akçam); to challenge the state of Israel's accounting of the Nakba (Palestinian exodus, directly translated from Arabic as "catastrophe") is to challenge the legitimacy of the state of Israel itself (Anziska); and, to reflect on US interventionism in Central and Latin America is to contradict the nation's imagine as a global protector of democracy (Chomsky). It is important that we acknowledge and reckon with the reality that modern systems of oppression frequently rely upon the suppression of "dangerous memories" and that approaching historical trauma research from a decolonial and action-oriented perspective may be viewed as undesirable by people and institutions in positions of power.

Limitations

We highlight here that our review strategy may not have captured all relevant literature. Research which focuses on specific historical harms may not have employed the language of "historical trauma" (or any of the variations we used within our search strategy). It appears that there has been a stronger emphasis of using the historical trauma narrative in research focusing on Indigenous communities, perhaps explaining why a majority of the literature identified was amongst such communities. For example, our review captured studies of specific traumatic historical periods such as the Holodomor and the persecution of Ethiopian Jews. While these studies were captured in this review, it is likely that there exist studies which reflect on a specific historical harm which would meet our studies definition of historical trauma but did not employ the language of historical trauma. As well, it appears that an awareness of history may also drive awareness of historical harms – for example, our research team is made up of researchers from the United States and Canada, and as such, we may more immediately recognize policies such as residential schools as representing a historical trauma, whereas, we might be more limited in our

capacity to recognize historical harms in other geographical contexts if it is not explicitly identified as a historical trauma.

Conclusion

In this scoping review, we identified literature which reflects on the relationship between historical trauma and substance use outcomes. The findings from the qualitative literature in our review indicate that, for Indigenous peoples subject to colonization, that understanding the perpetration of historical harms against them is necessary to understanding the modern context, including that of substance use. It appears that self-report quantitative methodologies are insufficient for making connections between the historical events and modern substance use outcomes, though quantitative tools such as the Historical Loss Scale may be applied to assess specific, measurable mechanisms by which historical harms influence present day substance use. Historical trauma, as a narrative, provides a framework which communities can understand how historical harms have resulted in modern day harms and systems of oppression. Through the lens of Freire's work, historical trauma as a public narrative represents a tool through which communities can dialog, build critical consciousness and consensus, and develop strategies to address modern systems of oppression. Researchers must engage with the ethical implications of the communities they research explicitly naming both historical and modern systems of oppression they face – while the focus of substance use research is typically on specific behaviors, the findings of the qualitative literature in this review indicate that substance use is inextricable from both historical and modern harms. A narrow focus on substance use behaviors, while ignoring how historical harms have reinforced modern systems of oppression, may be inadequate for characterizing substance use behaviors amongst historically oppressed populations – this is supported by the reflections within the qualitative literature identified in this review. Further, if

substance use is a symptom of historical and modern oppression, then efforts to address substance use disparities without addressing the underlying systems of oppression will likely fail to improve health equity. Additionally, decolonial, participatory research methodologies imply the need to let communities frame their historical and modern circumstances themselves, and communities may prioritize addressing historical and modern systemic harms they are subject to over efforts to understand the precise mechanisms that link historical harms with modern substance use outcomes. As well, researchers should be conscious that elevating knowledge and histories from the perspective of marginalized and oppressed peoples will likely involve elevating "dangerous memories" – this is particularly important when considering that academic research is frequently funded through government institutions that might view the promotion of "alternate" histories as subversive to current systems of power. Finally, we note that while much of the research identified in this review focuses on Indigenous peoples of North America and Australia impacted by colonization, the historical trauma narrative can be applied to action-oriented research with any community subject to historical harms and modern systems of oppression.

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Chapter 4 Aim #2, Part 1 – The Association Between Income Level and Income Change and Future Cigarette Use Trajectories

Abstract

Background. Cigarette use patterns in the United States are closely linked to income disparities. However, the temporal relationship between income level and future cigarette transitions (i.e., initiation, cessation) has not been well studied. The primary objective of this study was to explore the association between income level and future cigarette initiation and cessation. We also sought to evaluate the relationship between income level and future e-cigarette initiation and cessation.

Methods. We used nationally representative, weighted data from Waves 1 – 3 of the Population Assessment of Tobacco and Health (PATH) Study, administered annually between 2012 and 2015. Using modified Poisson regressions, we first evaluated the impact of income level and e-cigarette use at Wave 2 on 1) cigarette initiation at Wave 3 among the cigarette-naïve at Wave 2 and 2) cigarette cessation at Wave 3 among those reporting current cigarette use at Wave 2. We then evaluated the impact of change in income between Waves 1 and 2 on both outcomes at Wave 3. Finally, we evaluated the impact of income level and cigarette use at Wave 2 on 1) e-cigarette initiation at Wave 3 and 2) e-cigarette cessation at Wave 3.

Results. There was dose-response relationship between lower Wave 2 annual income level and: 1) elevated risk of cigarette initiation at Wave 3; and 2) diminished likelihood of cigarette cessation at Wave 3. For people who increased income level from Waves 1 to 2, the protectivity of higher income level was attenuated. We did not find similar patterns between income level and e-cigarette use transitions.

Conclusion. We established a temporal association between lower income level and: 1) elevated risk of future cigarette initiation and 2) diminished likelihood of future cigarette cessation. Additionally, we found that short-term changes in income likely do not confer the full protective benefit associated with higher income level. Policies which increase financial resources (such as minimum wage increases and universal basic income) should be evaluated to determine their impact on cigarette trajectories. Our findings indicate that temporary financial relief (e.g., COVID stimulus checks) will not confer the same health benefits as long-term financial assistance.

Introduction

While cigarette use has fallen consistently for decades in the United States (US), cigarette use among people living with a low income remains disparately high. In 2019, over 21% of those making less than $35,000 annually report smoking cigarettes, compared to just 7% of those making more than $100,000 (Cornelius et al.). Among those living at or below the poverty line, 40% have smoked cigarettes in the past month (SAMHSA). Additionally, people living at or below the poverty line who smoke cigarettes generally smoke for a greater number of years than those living with a higher income (Siahpush et al.). While these findings are consistent with global research indicating that lower income is associated with higher prevalence of cigarette use (Casetta et al.), there is a paucity of literature focusing on the impact of income on future cigarette use transitions.

The association between lower income and increased risk of negative health behaviors and outcomes is well-documented (Chetty et al.), but the mechanisms underlying these relationships remain unclear (Pampel et al.). Link and Phelan posited that social deprivation (including lack of financial resources) is a fundamental cause of disease on its own (B G Link and Phelan). Frohlich and Potvin, in particular, highlight how vulnerable populations (such as those living in poverty) are subject to a greater risk of health risk exposure, and that population-level interventions—including tobacco control initiatives—often fail to address health disparities by not accounting for this (Frohlich and Potvin). Based on these arguments, we hypothesize that living in poverty or with a low income is associated with an increased likelihood of cigarette initiation among the cigarette-naïve and with a decreased likelihood of cigarette cessation among those who currently smoke cigarettes.

The introduction of e-cigarettes has complicated the tobacco use landscape. E-cigarette use is associated with both smoking cessation among those who current smoke cigarettes and future

cigarette use among the cigarette-naïve (Soneji et al.; Pierce, Chen, et al.; Abrams et al.; Hartmann-Boyce et al.; Khouja et al.). While lower income is associated with a higher prevalence of cigarette use, e-cigarette use does not appear to be defined by the same income dynamics (Friedman and Horn). In fact, people with higher educational levels (which is highly correlated with income) are more likely to utilize e-cigarettes as a smoking cessation aid, indicating that the potential benefits of e-cigarette use may be concentrated within higher income populations of people who smoke cigarettes (Friedman and Horn). This is supported by recent work which found that high-income white people who smoke are more likely to utilize e-cigarettes as a cigarette cessation aid than people of color and/or people living with a lower income (Harlow et al.). This indicates that the use of e-cigarettes as cessation aids may further drive income-based and race-based cigarette use disparities.

Therefore, the primary aim of this study was to leverage longitudinal data from the Population Assessment of Tobacco and Health (PATH) study to assess the relationship between income level and both future cigarette initiation and future cigarette cessation. Additionally, we sought to assess the relationship between income level and future e-cigarette initiation and future e-cigarette cessation.

Methods

Study Sample

The PATH Study is a US nationally representative longitudinal study focused on tobacco product use and related health outcomes among individuals 12 years and older (Hyland et al.). The PATH Study employs a four-stage stratified area probability approach to draw its sample and over sampled people who use tobacco products, young adults, and Black people. The PATH Study then generated weights to correct for over-sampling and ensure the population was representative of the

US population. Wave 1 of the PATH Study was administered between September 2013 and December 2014. Waves 2 (October 2014 – 2015) and 3 (October 2015 – 2016) were administered with the same sample in subsequent years. Our analysis included all participants who were at least 18 years old at Wave 1 who responded to each of Waves 1 – 3 of the PATH Study.

Analytic Design

The goal of this study was to determine the association between Wave 2 income and Wave 3 tobacco product transitions: cigarette initiation (among those reporting less than 100 cigarettes smoked lifetime at Wave 2); cigarette cessation (among those reporting current cigarette use and reporting at least 100 cigarettes smoked lifetime at Wave 2); e-cigarette initiation (among those reporting never having used e-cigarettes at Wave 2); and e-cigarette cessation (among those reporting current e-cigarette use at Wave 2). We fit four Zou's modified Poisson regression models (one for each outcome) (Zou). The modified regression was chosen in order to extract relative risks (as opposed to odds ratios from a logistic regression), given the utility of relative risks and their application to future mathematical modeling endeavors.

The primary predictor of interest was annual household income level at Wave 2 (>$100k [referent], $50-100k, $25-50k, $10-25k, <$10k). We hypothesized a dose-response relationship between decreased income and 1) increased likelihood of initiation of cigarettes; and 2) decreased likelihood of cessation of cigarettes. Based on available evidence, it was less clear if we should expect these relationships to exist for e-cigarettes as well. Additionally, we included a three-category variable to capture use of the alternate tobacco product: for models whose outcome was an cigarette transition, a measure capturing e-cigarette use status at Wave 2 (never use [ref], current use, former use) was included; and, for models whose outcome was an e-cigarette transition, a measure capturing cigarette use status at Wave 2 (never use [ref], current use, former use) was

included. For cigarette use, never use was defined as not having smoked 100 cigarettes lifetime and former use was defined as having smoked 100 cigarettes lifetime but reporting not currently smoking cigarettes. For each model, we additionally included the following covariates as controls: age (in years) at Wave 2 (18 – 24 [referent], 25-34, 35-44, 45-54, 55-64, >65); sex (male [referent], female); race (white [referent], Black, other); and Hispanic ethnicity (no [referent], yes).

Finally, we hypothesized that, if a dose-response relationship exists between income level and tobacco product transitions, the protective benefits of increased income may be attenuated for individuals whose income has only recently increased. This is consistent with research that indicates that long-term income transitions confer the greatest health benefits, as opposed to short-term or temporary fluctuations in income (Schöllgen et al.; Benzeval and Judge; Blakely). As such, we ran a secondary analysis for all outcomes for which we identified a dose-response relationship with income. In this secondary analysis, we repeated the primary analysis with the inclusion of an additional covariate capturing income change between Waves 1 and 2 (no change in income stratum [referent], increased 1 or more income strata, decreased 1 or more income strata). We hypothesized that long-term income stability confers the greatest health benefit and, therefore, that having recently increased income strata (from Waves 1 to 2) would attenuate the positive impact of income at Wave 2 on Wave 3 outcomes.

Weighting and Imputation

We applied the Wave 3 all-waves longitudinal weights to ensure that our sample was representative of the adult US population. Missing data was imputed heuristically and then using Gibbs sampling via the *mice()* function in the ***mice*** library in R (see **Appendix B** for descriptions of missing data patterns and for imputation description and diagnostics) (Buuren and Groothuis-Oudshoorn). In total, 15 imputed datasets were generated. The analytic design described above

was implemented on each of the 15 imputed datasets and regression coefficients and standard errors were calculated by pooling results of each iteration via Rubin's rules (Buuren and Groothuis-Oudshoorn). For descriptive statistics, we present the variable breakdowns for both the unimputed and unweighted data and the average imputed, weighted dataset. Additionally, as a sensitivity analysis, we ran the analyses on the weighted, unimputed dataset and present the results in the **Appendix B** – this was done to determine if the imputation procedure introduced bias into the final results.

Results Evaluation Framework

Consistent with recommendations (Amrhein et al.), we evaluate and present the results of this study by applying the Post-significance Communications Structure (POCS) (Cummins and Marks). Instead of evaluating results by establishing significance via null hypothesis testing based on a *p*-value threshold (generally 0.05), we present point estimates, confidence intervals, and *p*-values together and then comprehensively interpret them in relation to our primary scientific research questions.

Results

Description of the sample.

In total, 23,670 individuals responded to the adult questionnaire for Waves 1 – 3 and were included in this study. The PATH Study Waves 1 – 3 weights were applied to ensure the study sample was representative of the US population (estimated to be 234 million people by 2010 US census) (Howden and Meyer) (see **Table 4.1** for unweighted and weighted variable distribution). Of the 23,760 individuals in the study, 24.1% reported being 18-24 years old at Wave 2, 16.4% reported being Black, and 36.8% percent reported currently smoking cigarettes at Wave 2. The weighting procedure brought the average sample distribution of the factors closer to distribution of the US population, with 12.9% being aged 18-24, 12.3% reporting being Black, and 18.8%

reporting current smoking status. For reference, at the 2010 census, of those aged 18 and over, 13.1% were estimated to be between ages of 18-24 (Howden and Meyer) and 12.6% were estimated to be Black (Humes et al.). Further, in 2013-2014, it was estimated that 17.0% of adults in the US currently used cigarettes (Hu et al.). As well, the weighting procedure brought the sample income distribution in greater alignment with the US population income distribution. While 42.7% of the sample reported household annual income less than $25,000, the weighted proportion dropped to 32.9%. This is closer to the 2014 census estimate that 23.6% of households had an annual income less than $25,000 (DeNavas-Walt and Proctor). Additionally, while only 13.2% of the sample reported an income greater than $100,000 annually, the weighted proportion increased to 18.4%. This is closer to the 2014 census estimate that 24.7% of households had an annual income of at least $100,000 (DeNavas-Walt and Proctor).

Table 4.1 Descriptive statistics and average weighted distribution of variables

Variable		N (%)	Average Imputed Weighted %*
Wave 2 Age			
	18-24	5,538 (24.1)	12.9%
	25-34	5,078 (22.1)	19.5%
	35-44	3,753 (16.4)	17.9%
	45-54	3,679 (16.0)	18.7%
	55-64	3,188 (13.9)	18.4%
	>65	1,711 (7.5)	12.7%
Sex			
	Male	11,514 (48.7)	47.9%
	Female	12,132 (51.3)	52.1%
Race			
	White	16,987 (73.6)	77.9%
	Black	3,779 (16.4)	12.3%
	Other	2,317 (10.0)	9.8%
Hispanic			
	Not Hispanic	19,256 (84.9)	84.8%
	Hispanic	4,068 (15.1)	15.2%
Wave 2 Annual Income			
	<$10k	4,210 (19.5)	12.7%
	$10-25k	5,032 (23.2)	20.2%
	$25-50k	4,937 (22.8)	23.0%
	$50-100k	4,590 (21.2)	25.7%
	>$100k	2,850 (13.2)	18.4%
Income Change from Wave 1 to Wave 2			
	Stayed the Same	12,926 (62.5)	71.0%
	Increased 1+ Strata	4,441 (21.5)	20.5%
	Decreased 1+ Strata	3,326 (16.1)	8.6%
Cigarette Use Status Wave 2			
	Never Cigarette Use	9,938 (43.4)	57.9%
	Current Cigarette Use	8,427 (36.8)	18.8%
	Former Cigarette Use	4,540 (19.8)	23.3%
E-Cigarette Use Status Wave 2			
	Never E-Cigarette Use	19,120 (88.2)	94.0%
	Current E-Cigarette Use	1,478 (6.8)	3.3%
	Former E-Cigarette Use	1,086 (5.0)	2.7%

***** Fifteen imputed datasets were calculated. The percentages presented reflect the average distribution of each variable across the fifteen imputed datasets.

Association Between Income and Cigarette & E-Cigarette Transitions

There was a dose-response relationship between Wave 2 income level and both Wave 3 cigarette initiation and Wave 3 cigarette cessation (see **Table 4.2**). Among individuals reporting never cigarette use, lower income level was associated with an increased risk of initiating cigarette use at Wave 3. Compared to those making more than $100k annually, those making $50-100k were 2.33 times as likely (95% Confidence Interval [CI]: 1.54-3.52), those making $25-50k were

3.51 times as likely (95% CI: 2.15-5.74), those making $10-25k were 5.12 times as likely (3.34-7.84), and those making <$10k were 9.46 times as likely (95% CI: 5.62-15.93) to initiate cigarette use at Wave 3. Notably, cigarette-naïve individuals reporting current e-cigarette use at Wave 2 (adjusted Relative Risk [aRR]: 6.87 95% CI: 5.20-9.80) and reporting former e-cigarette use at Wave 2 (aRR: 5.59 95% CI: 4.56-6.84) were substantially more likely to initiate cigarette use at Wave 3 than those reporting never using e-cigarettes.

Table 4.2 Results of modified Poisson regression examining the relationship between Wave 2 income and Wave 2 alternate tobacco product use on 1) cigarette initiation at Wave 3 among the cigarette-naïve at Wave 2; 2) cigarette cessation at Wave 3 among those who currently used cigarettes at Wave 2; 3) e-cigarette initiation at Wave 3 among the e-cigarette-naïve at Wave 2; and, 4) e-cigarette cessation at Wave 3 among those who currently used e-cigarettes at Wave 2.

Variable		Cigarette Initiation	Cigarette Cessation	E-Cigarette Initiation	E-Cigarette Cessation
		aRR* (95% CI)	aRR (95% CI)	aRR (95% CI)	aRR (95% CI)
Wave 2 Annual Income					
	<$10k	9.46 (5.62-15.93)	0.52 (0.37-0.71)	1.07 (0.83-1.38)	1.23 (0.39-3.89)
	$10-25k	5.12 (3.34-7.84)	0.55 (0.44-0.68)	0.87 (0.71-1.07)	1.25 (0.48-3.28)
	$25-50k	3.51 (2.15-5.74)	0.75 (0.58-0.96)	0.92 (0.75-1.12)	1.23 (0.44-3.43)
	$50-100k	2.33 (1.54-3.52)	0.97 (0.75-1.26)	0.73 (0.55-0.97)	1.18 (0.39-3.62)
	>$100k	Ref	Ref	Ref	Ref
Wave 2 Cigarette Use Status					
	Never Cigarette Use	--	--	Ref	Ref
	Current Cigarette Use	--	--	9.68 (7.99-11.74)	1.05 (0.54-2.04)
	Former Cigarette Use	--	--	2.17 (1.81-2.61)	0.50 (0.12-2.16)
Wave 2 E-Cigarette Use Status					
	Never E-Cigarette Use	Ref	Ref	--	--
	Current E-Cigarette Use	6.87 (5.20-9.80)	1.30 (1.06-1.59)	--	--
	Former E-Cigarette Use	5.59 (4.56-6.84)	1.21 (0.98-1.48)	--	--

*All regression control for Wave 2 Age, Gender, Race, and Hispanic Ethnicity. Further, coefficients and confidence intervals were computed by pooling the results of the regressions run on each of the 15 imputed datasets via Rubin's rules.

aRR: adjusted Relative Risk; 95% CI: 95% Confidence Interval

Among individuals reporting current cigarette use at Wave 2, lower income level was associated with a decreased likelihood of cigarette use cessation at Wave 3. Compared to those making more than $100k annually, those making $50-100k were 0.97 times as likely (95% CI: 0.75-1.26), those making $25-50k were 0.75 times as likely (95% CI: 0.58-0.96), those making

$10-25k were 0.55 times as likely (95% CI: 0.44-0.68), and those making <$10k were 0.52 times as likely (95% CI: 0.37-0.71) to report cigarette use cessation at Wave 3. Current e-cigarette use among those reporting current cigarette use at Wave 2 was associated with being 1.30 times as likely (95% CI: 1.06 – 1.59) of reporting smoking cessation at Wave 3 compared to never having used e-cigarettes.

We did not observe a similar dose-response relationship between Wave 2 income level and e-cigarette transitions at Wave 3. Among those reporting never e-cigarette use at Wave 2, those making $50 – 100k annually were less likely to initiate e-cigarette use (aRR: 0.73 95% CI: 0.55 – 0.97) than those making >$100k annually. Among participants who reported never using e-cigarettes at Wave 2, those who currently smoked cigarettes at Wave 2 were 9.68 times as likely of initiating e-cigarette use at Wave 3 compared to those reporting never cigarette use. Former cigarette use at Wave 2 was associated with being 2.17% times as likely (95% CI: 1.81-2.61) of initiating e-cigarette use.

Impact of Income Change on Future Cigarette Transitions

Since we identified a dose-response relationship between Wave 2 income and both Wave 3 smoking initiation and cessation, we fit two additional models to assess our hypothesis that the protective effect of higher income level would be attenuated for those who have only recently entered that higher income strata. Our hypothesis was confirmed for both cigarette initiation and cigarette cessation (see **Table 4.3**). Among those reporting never cigarette use at Wave 2, those who had increased income strata from Waves 1 to 2 were at an increased risk (aRR: 2.34 95% CI: 1.66-3.30) of initiating cigarette use at Wave 3 compared to those who stayed at the same income strata. This means that someone who had recently increased to income level X is more likely to initiate cigarette use than someone who had been at income level X for a longer period of time.

Similarly, among those reporting current cigarette use at Wave 2, those who had increased income strata from Waves 1 to 2 had a diminished likelihood (aRR: 0.74 95% CI: 0.56-0.98) of reporting cigarette cessation at Wave 3 than those whose income stayed the same.

Table 4.3 Results of modified Poisson regression looking at the impact of change in income from Wave 1 to Wave 2, income level at Wave 2, and e-cigarette use at Wave 2 on: 1) cigarette initiation at Wave 3 among the cigarette-naïve at Wave 2; and, 2) cigarette cessation at Wave 3 among those who currently used cigarettes at Wave 2.

Variable		Cigarette Initiation	Cigarette Cessation
		aRR (95% CI)	aRR (95% CI)
Wave 2 Annual Income			
	<$10k	14.32 (7.54-27.17)	0.47 (0.31-0.72)
	$10-25k	5.49 (3.13-9.63)	0.53 (0.39-0.72)
	$25-50k	2.96 (1.56-5.60)	0.72 (0.50-1.05)
	$50-100k	2.20 (1.35-3.58)	0.99 (0.72-1.36)
	>$100k	Ref	Ref
Income Change Wave 1 to Wave 2			
	Stayed the Same	Ref	Ref
	Increased 1+ Income Strata	2.34 (1.66-3.30)	0.74 (0.56-0.98)
	Decreased 1+ Income Strata	0.82 (0.59-1.15)	1.01 (0.75-1.37)
Wave 2 E-Cigarette Use Status			
	Never E-Cigarette Use	Ref	Ref
	Current E-Cigarette Use	5.16 (3.87-6.89)	1.31 (1.04-1.66)
	Former E-Cigarette Use	6.45 (4.97-8.38)	1.23 (0.96-1.57)

*All regression control for Wave 2 Age, Gender, Race, Hispanic Ethnicity

aRR: adjusted Relative Risk; 95% CI: 95% Confidence Interval

Discussion

In a sample representative of the adult US population, we identified a dose-response relationship between lower income level and 1) an elevated likelihood of initiating cigarette use in the following year among the cigarette-naïve and 2) a decreased likelihood of smoking cessation in the following year among people who currently smoke cigarettes. Further, our findings indicate that the protective impact of elevated income level is attenuated for individuals whose income had only recently increased (in the year prior) to their current income level. We additionally did not find the same dose-response relationship between income level and e-cigarette use.

While our findings indicate that there is a temporal connection between income level and future cigarette use transitions, our findings do not identify the mechanisms which drive these associations. First, cigarettes play a specific functional role for people living with lower incomes in terms of alleviating negative affect and improving positive affect. For people living with a low-income, cigarette smoking is one of the few, affordable ways to access the positive feelings associated with cigarette use such as stress relief and leisure (Peretti-Watel and Constance). Second, parental smoking is predictive of child initiation of smoking (Mays et al.). Given established income disparities in cigarette use, social exposure to cigarette use during development likely reinforces income-based cigarette use disparities. Third, tobacco marketing and stores are disproportionately concentrated within low income communities (Lee et al.), which may also be a driver of income-based cigarette use disparities. As well, access to health insurance has been found to be associated with greater likelihood of cessating cigarette use (Bailey et al.). Lower prevalence of health insurance coverage among low-income individuals may contribute to the impact of income on cigarette cessation. Finally, it has been argued that the decline in smoking in the US has largely been driven by the stigmatization of cigarette use and its concentration within communities with diminished social power (Stuber et al.). Link and Phelan highlight how the ability to generate stigma is dependent on having the social power to do so (Bruce G. Link and Phelan). If we understand higher income level as a proxy measure for membership in social classes with greater social power, then as people's income increase they may be exposed to more environments in which cigarette use is more stigmatized. This may explain why, in our model, increases in income level attenuated the protective impact of higher income level: short-term increases in income likely do not lead to immediate changes in social class membership or social environment. Future research should aim to better characterize the mechanisms underlying the

relationship between income and cigarette use transitions so that we may effectively identify fundamental causes of cigarette disparities and motivate interventions and policy strategies to directly address them.

Our study identifies a temporal connection between income level and future cigarette use trajectories, and these findings hold important policy implications. Of note, excise taxes have been proposed as the most effective tobacco control policy tool available (National Center for Chronic Disease Prevention and Health Promotion (US) Office on Smoking and Health). The proposed mechanism of action is that excise taxes place a higher financial burden on smoking cigarettes, which then dissuades people from initiating use or continuing their use. The underlying logic of regressive taxation assumes that people with the fewest financial resources will be most responsive to such tax increases. However, by contrast, cigarette use has most substantially fallen amongst high-income populations who are unlikely to be dissuaded from cigarette use solely for financial reasons. This indicates that a non-financial mechanism has driven cigarette use reductions, at least among those with higher incomes. As has been noted, the stigmatization of cigarette use among people at higher income levels has played an important role in driving reductions in cigarette use among people with higher incomes. Given challenges in disentangling the impact of growing social stigmatization and the use of excise taxes on cigarette use, it is likely that evaluations of the efficacy of excise taxes over the last several decades have inadvertently also captured the impact of elevated stigmatization of cigarette use. If this is the case, the efficacy of excise taxes, broadly, may be overstated. Even so, excise taxes remain an important tobacco control tool. They operate by imposing financial barriers onto people who smoke. Our findings provide indication that tobacco control approaches which eliminate financial and income-based barriers to cigarette initiation and cessation are necessary to alleviate income-based cigarette disparities. Studies

focusing on the broader impact of income-based social interventions, such as an increased minimum wage or a universal basic income, should seek to evaluate the impact of such policies on cigarette use trajectories.

Finally, our findings indicate that e-cigarette use is associated with cigarette initiation and, to a lesser extent, promotes cigarette cessation, consistent with prior literature (Pearson et al.; Levy et al.; Stanton et al.; Pierce, Chen, et al.). These dual findings imply, that the population-level benefit of introducing e-cigarettes as a cigarette cessation aid is dependent on ensuring that the use of e-cigarettes is not adopted by cigarette-naïve individuals. Further, given evidence that higher-income people are more likely to use e-cigarettes as a cigarette cessation aid (Harlow et al.), e-cigarettes may exacerbate smoking cessation disparities based on income. Such a result would be consistent with Frohlich and Potvin's health inequality paradox in which policies which improve population-level metrics may also expand the disparity between those at greatest risk and the general population (Frohlich and Potvin).

Limitations

While our study leveraged nationally representative longitudinal data, limitations remain. First, the weighting procedure was limited to race, age, and smoking status and was not designed to ensure that the income distribution of the sample was reflective of that of the US population. As noted in the results, the sample over-represented people with lower incomes (which is potentially explained by the over-sampling of young adults), and, consequently, may have resulted in biased point estimates on the impact of income. As well, our measure of former cigarette and e-cigarette use are unable to capture the duration of cessation. As such, our findings around cessation reflect on short-term quitting, as opposed to the long-term cessation which may confer the most meaningful long-term health benefits Additionally, to conduct the present study, we had to impute

missing datapoints. Consistent with prior research using the PATH Study data (Pierce, Benmarhnia, et al.), we treated data as missing at random given that the primary covariate of interest (income level) was asked prior to the outcomes of interest. We then ran the analysis on 15 distinct imputed datasets and reported to pooled results to control for potential bias introduced by each imputation. Additional information on the imputation is included in the **Appendix B**.

Conclusion

Patterns of cigarette use in the United States can be explained in part by disparities in income. While these disparities have long been recognized, this is the first study to assess the temporal relationship between income level and future cigarette use transitions. We found a dose-response relationship between lower income and 1) increased likelihood of initiating cigarette use over the subsequent year and 2) decreased likelihood of quitting cigarette use over the subsequent year. Further, our findings indicated that e-cigarette use was associated with 1) an elevated risk of adopting cigarette use among the cigarette-naïve and, to a lesser degree, 2) an elevated likelihood of quitting cigarette use. While this study establishes the temporal connection between income and cigarette transitions, future research must aim to better characterize the mechanisms underlying this association. Such research represents an important step in effectively addressing income-based cigarette use disparities, which current tobacco control approaches have thus far failed to alleviate. Of particular note, it will be important to evaluate the impact of policies which aim to increase financial resources (such as raising the minimum wage or a universal basic income) on cigarette use trajectories.

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Chapter 5 Aim #2, Part 2 – Evaluating the Contribution of Income Level to Cigarette Use Prevalence: A Mathematical Modelling Study

Abstract

Background. Despite the long-term declines in cigarette use prevalence in the United States (US), there remain stark income-based disparities. While higher cigarette use initiation rates and lower cessation rates among lower income populations are well-documented, efforts to evaluate to contribution of income to cigarette use have not been undertaken.

Objective. Determine the contribution of income (population attributable fraction) to cigarette use among adults in the US.

Methods. We developed a novel mathematical model of cigarette and e-cigarette use among adults, incorporating stratification by age, sex, and annual household income level (allowing for transitions by age). Data from the Population Assessment of Tobacco and Health Study, Monitoring the Future Study and Centers for Disease Control and Prevention were used to parametrize the model. Data from the National Health Interview Survey and World Bank were used to calibrate the model to historical trends (2014 – 2019) in population growth, cigarette use, and e-cigarette use. The model projected 10 years of cigarette use trajectories (from 2015 to 2024). We then modeled the counterfactual scenario in which all participants had the same protectivity associated with being in the highest income group (>$100k annually) and compared the results. PAF was calculated by determining the change in cumulative person-years of smoking between the status quo and counterfactual scenarios over 1, 5, and 10-year periods, both overall and among different subgroups in 2014 (never smokers, current smokers, former smokers).

Results. Overall, our baseline model accurately projected the cigarette use prevalence among US adults would fall from 17% in 2014 to just below 14% in 2019, projecting that it would

fall to 13.6% in 2024. In comparison, the high-income scenario projected that the cigarette use prevalence would fall to 7.7% in 2024, resulting in 113.9-million fewer cigarette use-years over ten years – approximately half of which were among those reporting current cigarette use at model start in 2014. Overall, we found that income contributed to 12.3%, 25.2%, and 32.8% of cigarette use-years after 1, 5, and 10 years, respectively. Among individuals who reported never smoking cigarettes in 2014, income contributed to 75.6%, 79.2%, and 81.2% of cigarette use-years after 1, 5, and 10 years, respectively.

Discussion. Our findings indicate that income contributes substantially to cigarette use-years among adults in the US, especially among individuals who have never smoked cigarettes before. This implies that policies which directly improve individual finances, such as a universal basic income, are likely to result in reductions in cigarette use among the US adult population.

Introduction

Despite the population-level decline in cigarette use prevalence in the United States (US), cigarette use remains disparately high among those living with a low income. Although the national prevalence of cigarette use has steadily declined to 14% as of 2019, the smoking prevalence is 3 times higher for people with an annual household income less than $35,000 (21%) compared to those making more than $100,000 (7%) (Cornelius et al.). The disparity is even more stark for those living at or below the poverty line, as it estimated that 40% of such individuals have smoked cigarettes in the prior month (SAMHSA). Further, research has indicated that individuals living at or below the poverty line who smoke tend to have substantially longer median smoking duration (40 years) compared to those living at 3 times the poverty level or greater (22 years) (Siahpush et al.). Despite awareness of the association between lower income and higher prevalence of cigarette use, there is a paucity of research aimed at understanding and measuring the impact of income on cigarette use.

Of particular note, mathematical modeling endeavors aimed at evaluating the impact of policy interventions on cigarette use prevalence have largely failed to incorporate income as a determinant factor (National Center for Chronic Disease Prevention and Health Promotion (US) Office on Smoking and Health; Shari P. Feirman et al.; Shari P Feirman et al.). Such models represent a powerful set of tools for evaluating the potential impact of tobacco control policy approaches. Three of the most prominent tobacco simulation models – SimSmoke (D T Levy et al.; David T. Levy et al.), BENESCO (Howard et al.), and the Tobacco Policy Model (Tengs et al.) – do not stratify the population by income level. Given that US population-level cigarette use prevalence trajectories have been distinct across socio-economic class, there is a need for modeling approaches which do factor in income level.

As such, the purpose of this study is to present a novel, compartment model of cigarette use – the Socio-Economic Tobacco (SET) Model – which accounts for the impact of income and income changes over time in the adult U.S. population. We primarily parameterized the model using US weighted, nationally-representative data from Waves 1 – 3 of the Population Assessment of Tobacco and Health (PATH) Study (Hyland et al.). The primary objectives of this study were to: 1) present and evaluate the performance of the SET model; and, 2) use the SET model to calculate the population attributable impact of income level on cigarette use over ten years among the US adult population.

Methods

Defining the SET Model

The Socio-Economic Tobacco (SET) compartmental model was designed to simulate cigarette and e-cigarette use trajectories among the US adult (18+) population, with a focus on income level and transitions. The model stratifies the population by five factors: sex, age, cigarette use status, e-cigarette use status, and income level (see **Figure 5.1** for schematic of each strata). The operationalization of each strata was determined by how each variable was measured in the PATH Study. Sex was measured dichotomously (male, female) and was considered immutable for this iteration of the SET model. Age was measured categorically (18 – 24, 25 – 34, 35 – 44, 45 – 54, 55 – 64, 65+). Both cigarette and e-cigarette use were captured using three categories (never use, current use, former use). For cigarette use, current use was defined as having smoked at least 100 cigarettes lifetime and reporting current use of cigarettes (i.e., some days or every day use). Former use was defined as having smoked at least 100 cigarettes lifetime and reporting not currently using cigarettes. Never use was defined as not having smoked 100 cigarettes lifetime. For e-cigarette use, current use was defined as reporting currently using e-cigarettes “some days” or “every day”. Former use was defined as having ever previously used e-cigarettes “some days”

or “every day”. Never use was defined as never having previously used e-cigarettes “some days or “every day”. Finally, income level was captured using a 13-category construct. First, PATH captured five distinct annual household income levels: <$10,000, $10-25,000, $25-50,000, $50-100,000, and >$100,000. Next, as was displayed in **Chapter 4,** PATH data indicates those who have recently (i.e., in the past year) changed income level are at differential risk of cigarette use transitions compared to those whose income has remained stable in the long-term. Notably, those who have recently increased income level are at higher risk of adopting or maintaining cigarette use compared to individuals who have remained stable at the same income level. As such, 5 compartments were included to capture stable income (one for each income level) and 8 compartments were included to capture recent (past-year) changes in income and the direction of that income change (increased or decreased): increased to $10-25,000, to $25-50,000, to $50-100,000, and to >$100,000; and, decreased to <$10,000, to $10-25,000, to $25-50,000, and to $50-100,000. We refer to these 8 compartments as “transitionary” income compartments. The full model was generated as the Cartesian product of each of these five strata (Worden and Porco) resulting in a total of 1,404 compartments. Each compartment represents the set of people of a given sex, at a given age, with a given cigarette use status, with a given e-cigarette use status, and a given income level status.

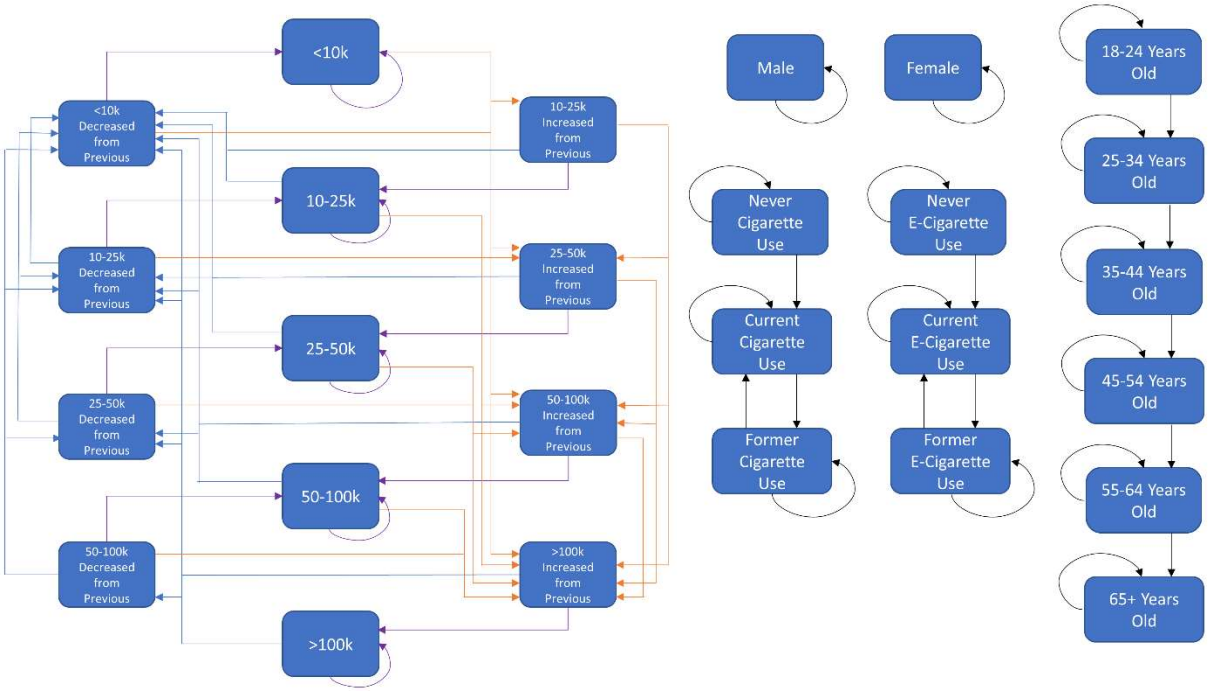

Figure 5.1 Model Schematics. *For the 5 model strata (sex, age, cigarette use, e-cigarette use, and income level), we present the model schematics. First, the sex stratum is made of two compartments (male and female). No transitions between the two compartments are permitted. Second, the age stratum is made of 6 compartments (18-24, 25-34, 35-44, 45-54, 55-64, and 65+). Transitions between age compartments occur sequentially, based on participant age. Both the cigarette and e-cigarette strata use the same structure. Those who have never used cigarettes/e-cigarettes can transition to current use, those currently using can transition to former use, and those who have formerly used the product can return to current use. Finally, the income strata has 13 compartments: 5 income-stable compartments (represented by the middle column) and 8 income-transitionary compartments (represented by the outer columns). If an individual changes income level, they must transition to a "transitionary" state. For example, if someone is in the "$10-25k" compartment and their income increases to "$50-100k" then they would transition to the "$50-100k, increased from previous" transitionary compartment. If someone remains in the same transitionary compartment for a year, then they transition to the income stable compartment of the same income level. The income transitions are color-coded such that: orange represents increases to income level, blue indicates decreases in income level, and purple represents income staying the same.*

Given the unlikelihood of transitions across strata occurring simultaneously, our model only permits transitions between one stratum at a time. In terms of sex, it was our preference to use a measure capturing gender-identity, but no such measure was available in the PATH Study. Individuals were able to transition to the next age group, up until the 65+ group. For both cigarette and e-cigarette use, those reporting never use status were able to transition to current use, those reporting current use status were able to transition to former use, and those reporting former use status were able to transition back to current use. For income, individuals in one of the five income-

stable compartments could either remain in the same income group or transition to one of the transitionary income compartments. For example, an individual in the $25-50,000 income-stable compartment whose income increased to $50-100,000 would then move to the "Increased to $50-100,000" transitionary compartment. Individuals in the transitionary compartment either can transition to the stable income compartment (if in the same transitionary compartment for 1 year) or can move to a different transitionary income state if their income level changes or decreases prior to stabilization. Additionally, background death can occur from every compartment. Entries into the model are permitted only for youngest age group to simulate children entering adulthood, and distributed by sex, income group, and cigarette use status.

Cigarette transition rates (initiation, cessation, reinstatement) were modified by sex, age, e-cigarette use status (never [referent], current, former), income level, and recent change in income level (no change [referent], increased 1+ income strata, decreased 1+ income strata). E-cigarette transitions (initiation, cessation, reinstatement) were modified by sex, age, cigarette use, income level, and recent change in income level. As described below, a series of six modified Poisson regressions (corresponding to each of the six cigarette and e-cigarette transitions) were fit to extract relative risks of each of the modifying factors.

Initializing Model Start State

We generated a starting state of the initial population that matched with available data on the distribution of age, sex, cigarette use status, e-cigarette use status, and income level for the U.S. population in year 2014. We initialized the model to represent the 234-million adults in the US in 2014. First, we used US Census data from 2014 to determine the population distribution across age and sex (US Census, *Age and Sex Composition in the United States: 2014*). We then used data on the household income and average number of people per household to determine the

population income distribution (US Census, *HINC-01. Selected Characteristics of Households by Total Money Income.*). We assumed that income distribution was the same for both men and women. At the start, individuals were placed in the five income-stable compartments. Next, we used National Health Interview Survey (NHIS) data to determine the distribution of cigarette and e-cigarette use. In 2014, NHIS estimates indicate that 17% of the adult population were currently using cigarettes (Hu et al.) and that 20.9% formerly used cigarettes (United States Public Health Service Office of the Surgeon General and National Center for Chronic Disease Prevention and Health Promotion (US) Office on Smoking and Health). The initial start state also captured the higher prevalence of current cigarette use reported by those with a lower income (Hu et al.). For former smoking prevalence, we assumed that older individuals have a higher former smoking prevalence, that men have a higher smoking prevalence than women (supported by (Kramarow)), and that those at higher incomes have a higher prevalence of former smoking (given that they generally report more successful cessation attempts (Vijayaraghavan et al.; Reid et al.)). Finally, we used NHIS data to determine the distribution of e-cigarette use. In 2014 it was estimated that 3.3% of the US adult population was currently using e-cigarettes (Hu et al.). Additionally, information on the distribution of e-cigarette use based on cigarette use status and age was available ("QuickStats: Cigarette Smoking Status* Among Current Adult E-Cigarette Users,† by Age Group — National Health Interview Survey,§ United States, 2015"). Given challenges in estimating former e-cigarette use, we assumed an equal prevalence of former e-cigarette prevalence as current use prevalence.

Parameterizing the Model

Data from Waves 1 – 3 of the Population Assessment of Tobacco and Health (PATH) Study, NHIS, Centers for Disease Control and Prevention (CDC), and Monitoring the Future

(MTF) were used to parameterize the model. The PATH study is a longitudinal study representative of the US population (Hyland et al.). We used data for all participants who responded to the adult survey at each of Waves 1 – 3, imputing missing data and applying weights such that our study sample is representative of the US adult population (see **Appendix C** for further details on imputation and weighting procedures).

First, transition rates were calculated. Transition rates between the five income levels were calculated by examining observed income transitions between Waves 2 and 3 of the weighted PATH Study data. The proportion of people that transitioned from income level X to income level Y were extracted and then converted into rates using the appropriate conversion equation (Fleurence and Hollenbeak). Next, to calculate cigarette use and e-cigarette use transition rates, we defined a referent group (men, aged 18-24, making <$10k annually). We then calculated the proportion of people in this referent group at cigarette or e-cigarette status X at Wave 2 who transitioned to status Y at Wave 3 and converted these values into rates (Fleurence and Hollenbeak).

We then fit a series of modified Poisson regression models to evaluate the relationship between sex, age, income level, change in income, and the alternate tobacco product (i.e., cigarette status was included as a predictor in the e-cigarette transition models and vice versa) and cigarette/e-cigarette transitions (i.e., initiation, cessation, reinstatement) from Wave 2 to Wave 3. The relative risk and standard errors were extracted from the models and applied to modify compartmental transition rates based on sex, age, income level, change in income status, and alternate tobacco product use. Consistent with the models fit in **Chapter 4**, the models found a dose-response relationship between higher income level and 1) a lower risk of initiating cigarette use among the cigarette naïve, 2) a higher likelihood of cigarette cessation among those who

currently smoke, and 3) a lower risk of reinstating cigarette use among those reporting formerly smoking (see **Appendix C** for parameter tables).

Age-specific death rates were extracted from the CDC life tables by taking the median death probability for the age grouping and converting it into a rate (Arias; Fleurence and Hollenbeak). Relative risks were then applied to capture elevated risk of mortality associated with both current and former use of cigarettes (Lariscy et al.).

Annual entries into the model simulate children entering adulthood (i.e., aging from 17 to 18 years old). First, we assumed that the people entering the 18-24 age group had the same sex and income distribution as 18-24 age group in 2014. In 2014, for the first time, it was estimated by MTF that youth reported higher prevalence of e-cigarette use than cigarette use – with 17.1% of 12^{th} graders reporting e-cigarette use in the prior month compared to just 12.7% reporting cigarette use (R. A. Miech et al.). Interestingly, MTF estimates for 12^{th} grade e-cigarette use prevalence have fluctuated dramatically, with estimates falling to 11.0% in 2017 before climbing to 25.4% in 2019 (R. Miech, Johnston, P. M. O'Malley, et al.). As this model was not designed to capture changes in the dynamics of youth product use, we chose to match the distribution of cigarette and e-cigarette use to MTF data for the year 2018, in which 7.6% currently used cigarettes (in the past month) and 20.9% currently used nicotine e-cigarettes (in the past month) (R. Miech, Johnston, P. O'Malley, et al.). The year 2018 was chosen as it captures the recent increase in e-cigarette use and decline in cigarette use among adolescents and appears less likely to be an outlying estimate.

Finally, to capture historical trends in declining population growth from 2014-2019 (World Bank), we used a logistic growth function to capture this declining acceleration in population size. Such a function requires both a population growth rate and a population carrying capacity (i.e., the

maximum number of people a society can theoretically hold). We applied this function as a multiplier to determine the size of entry population at a given time *t.* The population growth rate and population carrying capacity were determined via calibration, as discussed next.

Calibrating the Model

To ensure that the model output was consistent with historical data (from 2014 to 2019), we calibrated the model to annual population growth estimates from 2014-2015 to 2018-2019, current cigarette use prevalence from 2014 to 2019, former cigarette use prevalence from 2014-2019, and current e-cigarette use prevalence from 2014 to 2019.

First, we calculated the per capita growth rate and carrying capacity which would generate the observed U.S. population annual growth rate from 2014-2015 to 2018-2019 (i.e., we calibrated to 5 data points). In 2014, it was estimated that the US population increased by 0.7% and that over the subsequent years the US population has continued to grow by a declining acceleration (fell to 0.5% increase in 2019) (World Bank).

Then, we calibrated the model to time trends in prevalence of current and former cigarette use in the US in from 2014 to 2019 (i.e., six calibration points for both current and former use), through varying two multiplicative scalars: the first modifying the cigarette initiation rate; and the second modifying the cigarette cessation rate. Our model start state was initiated to the NHIS estimated cigarette use prevalence for 2013-2014 of 17% (the first calibration data point). By 2019, the estimated cigarette use prevalence in the US fell to 14.0% (Jamal et al.; Cornelius et al.). Further, estimates have indicated that the population prevalence of former cigarette use has remained relatively stable at 20.9% from 2014 to 2019 (United States Public Health Service Office of the Surgeon General and National Center for Chronic Disease Prevention and Health Promotion

(US) Office on Smoking and Health). As such, we calibrated the initiation and cessation rates such that model output matched the 2019 current and former cigarette use prevalence.

Finally, we calibrated the model to time trends in prevalence of current e-cigarette use in the US from 2014 to 2019, excluding the year 2016 for which the annual NHIS e-cigarette use prevalence was not reported (i.e., five calibration points total). The current e-cigarette use prevalence rose from 3.3% in 2013-2014 (Hu et al.) to 4.5% in 2019 (Cornelius et al.).

Calibration for each parameter set was undertaken using a the ***nls.lm*** function in the ***minpack.lm*** R package (Elzhov et al.). This function uses a modified Levenberg-Marquardt algorithm to minimize the sum-square of the difference between model output and target values.

Uncertainty Analyses

To account for uncertainty in underlying model parameters, we generated 1,000 parameter sets by randomly sampling from parameter distributions (see **Table 10.X** for full parameter list and their distributions). For transition rates for income, cigarette, and e-cigarette use, transition probabilities were sampled from Dirichlet distributions prior to being converted into rates. For relative risks modifying cigarette and e-cigarette transitions, log-relative risks extracted from regression models were sampled from a normal distribution prior to converting sampled values into relative risks (via exponentiation).

Model Scenarios and Outputs

For each of our 1,000 sampled and calibrated parameter sets, we simulated the future trajectory of cigarette smoking prevalence among the US adult population across 10 years. We simulated trajectories with the following two scenarios:

- **Baseline scenario:** Relative risks of cigarette and e-cigarette initiation and cessation remain stable through time from 2014 onwards.
- **All high-income scenario:** All risks of cigarette and e-cigarette initiation and cessation are set to those among the highest income group (>$100k annually) from 2014 onwards.

We calculate the cumulative number of cigarette use-years and the prevalence of current cigarette use for each scenario at 1, 5, and 10 years. One cigarette use-year is defined as an individual meeting the criteria for "Current Cigarette Use" status for one calendar year. An individual who smokes cigarettes for 5 years would be understood to contribute 5 cigarette use-years. The all high-income scenario represents the counterfactual in which all members of the population receive the same protective risk of cigarette use and e-cigarette use transitions as those in the highest income group.

Additionally, we defined the population attributable fraction (PAF) of income on cigarette use as the relative change in the cumulative number of cigarette use-years between the baseline model and the high-income scenario after 1, 5, and 10 years. We separately analyzed the PAF of income on cigarette use for people reporting never smoking status, current smoking status, and former smoking status in 2014 to determine differential impact of income on cigarette use trajectories.

Sensitivity Analyses

As noted, the distribution of cigarette and e-cigarette use among model entries was determined via MTF's 2018 estimates of 12th graders tobacco product use. In **Appendix C**, we include sensitivity analyses using the 2017 distributions (where e-cigarette use is estimated to be much lower [11.0%]) and the 2019 distributions (where e-cigarette use is estimated to be higher

[25.5%]) to determine if changes in the distribution of tobacco product use among model entries impacted this paper's primary findings.

Implementation

The model was implemented in R using the ***deSolve*** package (Soetaert et al.; R Core Team). To improve runtime, code was parallelized using the ***parallel, doParallel***, and ***foreach*** packages (Weston and Calaway) to run the model separately for the male and female populations (given that transitions were not permitted across sex, nor were any transitions dynamically dependent upon distribution of sex). Further, code was run separately for the populations reporting never, current, and former cigarette use at *t=0*. This was done so that we could effectively evaluate the attribution of income on each of these three groups cigarette use trajectories. Individuals who entered the model after *t=0* were included in the simulation corresponding to their cigarette use status at model entry.

Results

Calibration

The model was initiated to be representative of the 234-million adults in the US in 2014. The model calibrated well to historical population growth (**Figure 5.2a**), current cigarette use prevalence (**Figure 5.2b**), former cigarette use prevalence (**Figure 5.2c**), and current e-cigarette use prevalence (**Figure 5.2d**) trends from 2014 to 2019.

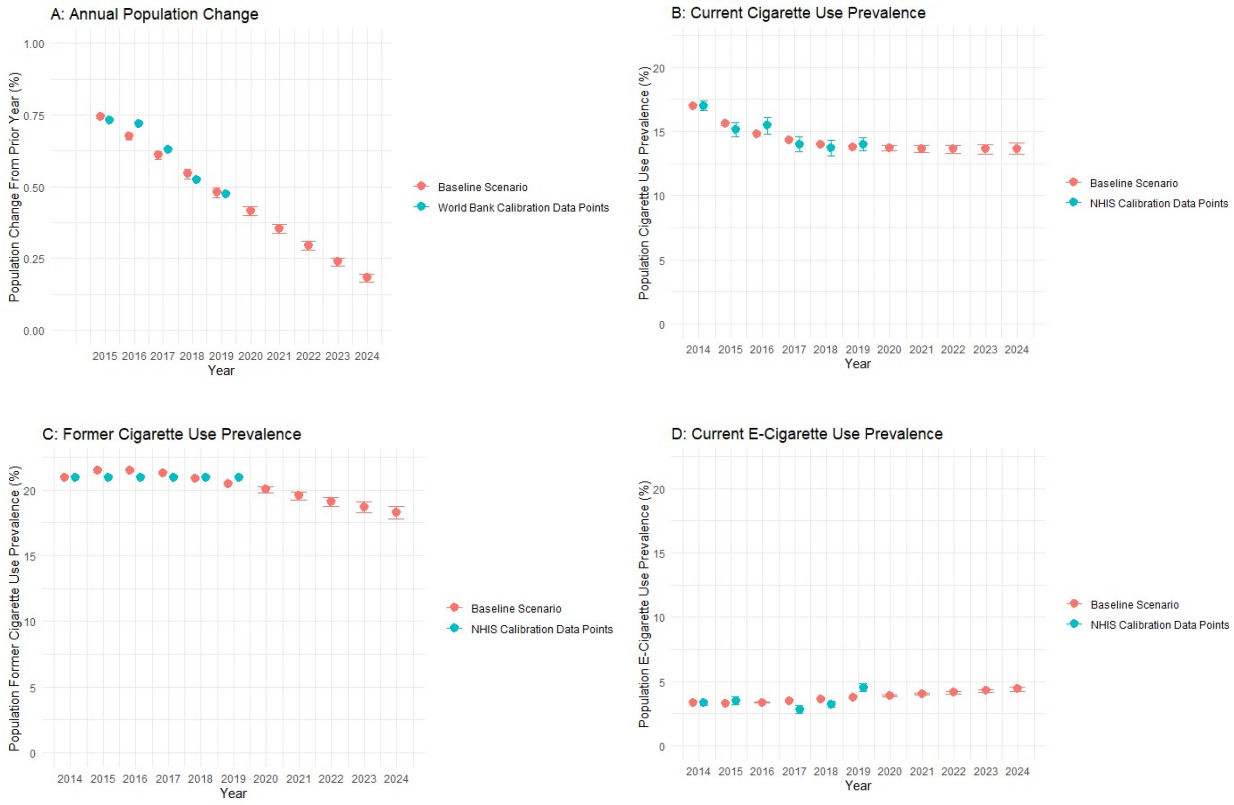

Figure 5.2. Model calibration to data*. Plots displaying mean(dots) and 95% confidence interval (whiskers) for baseline model projections (in red), along with calibration data points for (in green): (**a,** top left) annual population change; (**b,** top right) annual current cigarette use prevalence; (**c,** bottom left) annual former cigarette use prevalence; and (**d,** bottom right) annual current e-cigarette use prevalence.*

Baseline model projections

The baseline model projected that the cigarette prevalence would decline from 17% in 2014 to 13.6% (95% CI: 13.2% - 14.1%) in 2024. As well, the model projected that the former cigarette use prevalence would increase from 20.9% in 2014 to 21.5% (95% CI: 21.4 – 21.6) in 2016 before falling to 18.3% (95% CI: 17.8 - 18.7) in 2024. The baseline model projected that the e-cigarette prevalence would increase steadily from 3.3% in 2014 to 4.4% (95% CI: 4.2 - 4.5) in 2024.

All high-income scenario projections

In the high-income scenario, the average current cigarette use prevalence was projected to decline from 17% in 2014 to 7.7% (95% CI: 5.6% - 9.9%) in 2024 (**Figure 5.3**). As depicted in

Figure 5.3, the current cigarette use prevalence in the high-income scenario was significantly less than that of the baseline model for each of the ten years simulated.

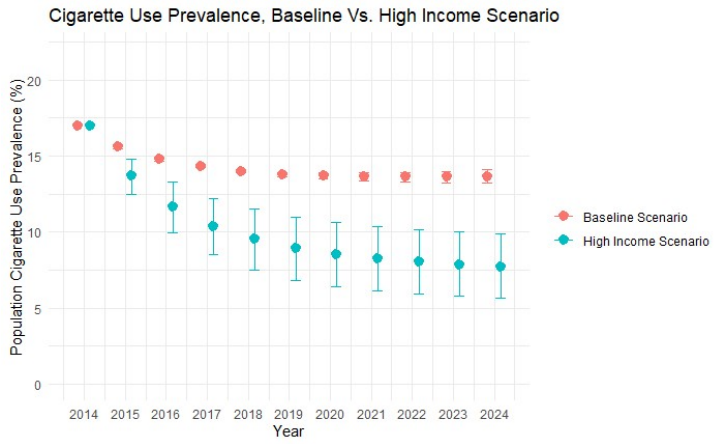

Figure 5.3 Model Projections for Annual Current Cigarette Use Prevalence for the Baseline Scenario Versus High-Income Scenario.

Comparing Cigarette Use Years: Baseline Scenario Vs. High-Income Scenario

As depicted in **Figure 5.4**, from 2014 to 2024, the baseline scenario projected a cumulative 347.4-million cigarette use years (95% CI: 343.9 – 351.2) among the U.S. adult population. Across the 10 years from 2014-2024, people reporting current cigarette smoking at simulation start in 2014 contributed a mean 216.6-million cigarette use-years (95% CI: 207.9 - 224.8), those reporting former cigarette smoking in 2014 contributed a mean 101.5-million cigarette use-years (95% CI: 89.9 – 113.7), and those reporting never smoking cigarettes in 2014 contributed a mean 29.4-million cigarette-years (95% CI: 27.6 - 31.0).

From 2014 to 2024, the high-income scenario projected a mean 233.5-million (95% CI: 185.7 - 279.9) cigarette years. On average, this represents a crude reduction in cigarette years of 113.9-million cigarette years over ten years, compared to the baseline scenario. People reporting current cigarette smoking in 2014 contributed 161.8-million cigarette years (95% CI: 131.4 - 190.4), those reporting former cigarette smoking contributed a mean 66.2-million cigarette-years

(95% CI: 45.0 – 90.9), and those reporting never smoking cigarettes contributed 5.5-million cigarette-years (95% CI: 3.3 – 8.4). On average, the crude reduction in cigarette-years between the baseline model and high-income scenario over the ten years projected was 54.8-million, 35.3-million, and 23.9-million cigarette-years for those reporting current, former, and never cigarette use, respectively.

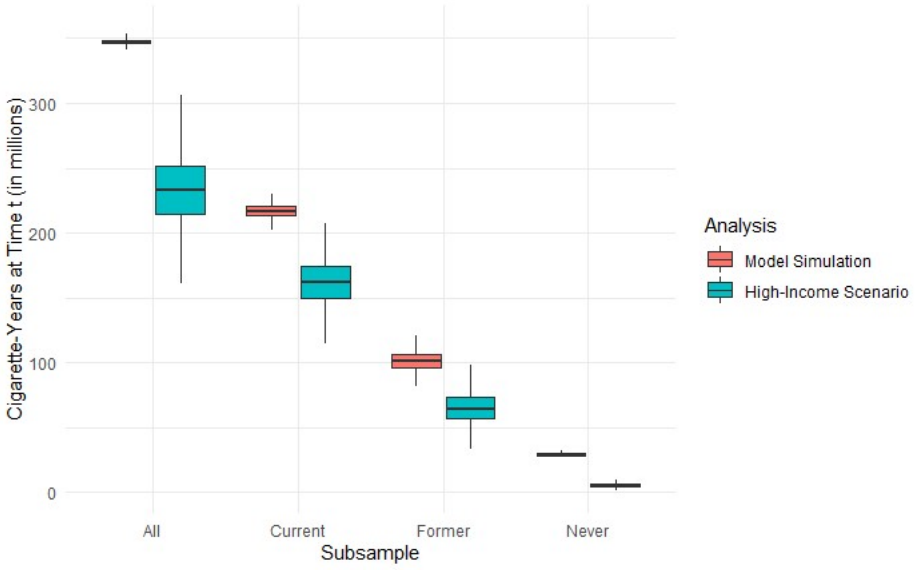

Figure 5.4 Cumulative cigarette use-years projected over 2014-2024 *for the baseline scenario (red) compared to the all high-income scenario (blue). In the high-income scenario, there was an average reduction of 111.4 cigarette use-years over the ten years projected. As well, results are displayed for the population of people who reported current, former, and never cigarette smoking status at model entry, respectively, as indicated on the x-axis. Approximately half of the overall reduction was accounted for by the reduction in cigarette use-years amongst those currently smoking at model entry (reduction of 52.9-million cigarette use-years). Boxes represent the 1st through 3rd quartile of values and whiskers extend to the furthest data point within 1.5-times the interquartile range of observed values.*

Population Attributable Fraction of Income on Cigarette Use

The PAF of income on cigarette-use increased over time. Overall, after 1, 5, and 10 years respectively, the PAF associated with income on cigarette-use years was 12.3% (95% CI: 5.7 - 19.7), 25.2% (95% CI: 13.5 - 37.1), and 32.8% (95% CI: 19.8% - 46.5%), respectively (**Figure 5.5**). However, income contributed to a greater proportion of cigarette use-years among never smokers at model start in 2014 compared to both current and former smoker in 2014. Among never

smokers in 2014, the income PAF was 75.6% (95% CI: 63/1 - 84.8%), 79.2% (95% CI: 68.3 – 87.3), and 81.2% (95% CI: 71.2 – 88.7) after 1, 5, and 10 years, respectively. Among current smokers in 2014, the income PAF was 8.5% (95% CI: 2.6 – 15.4), 19.2% (95% CI: 8.3 – 30.5), and 25.3% (95% CI: 12.5 – 38.1) after 1, 5, and 10 years, respectively. Among former smokers in 2014, the income PAF was 31.9% (95% CI: 8.6 - 52.8), 33.5% (95% CI: 12.9 - 52.9), and 34.9% (95% CI: 15.0 – 54.0) after 1, 5, and 10 years, respectively. Our sensitivity analyses (see **Appendix C**) indicate that varying the distribution of cigarette and e-cigarette use among model entries did not impact the interpretation of study findings.

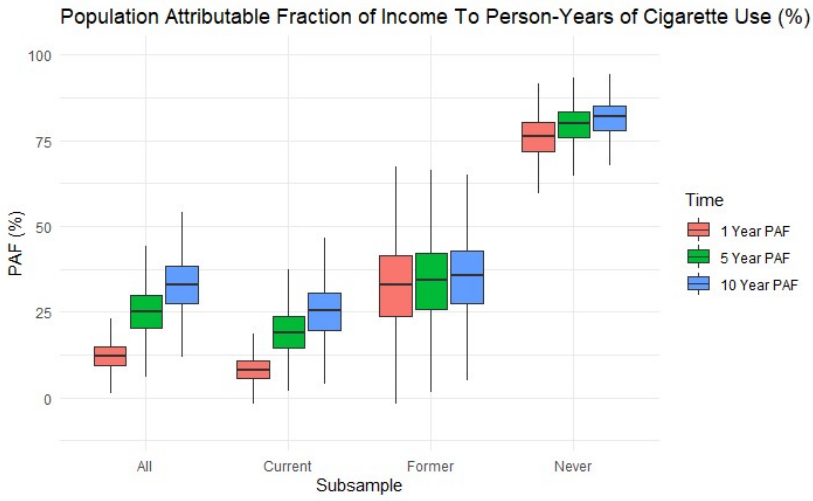

Figure 5.5 Population attributable fraction of the contribution of income to cigarette use across 1 year (red), 5 years (green) and 10 years (blue). *As well, results are displayed for the population of people who reported current, former, and never cigarette smoking status at model entry, respectively, as indicated on the x-axis. PAF: Population Attributable Fraction*

Discussion

In this study we sought to assess the contribution of income on cigarette use among adults in the US. We developed a novel model of cigarette and e-cigarette use which accounts for both individual annual income level and changes in income level. We found that, over ten years from 2014-2024, income contributes to 32.8% (95% CI: 19.8% - 46.5%) of cigarette use-years among adults in the United States. Further, we projected, over ten years, that income contributes to

approximately 81.2% (95% CI: 71.2 – 88.7) of cigarette use-years among those never using cigarettes in 2014, indicating that increasing household income can reduce the initiation of cigarette use at the population level. Our projections showed that, from 2014 to 2024, that 113.9-million cigarette use-years could be prevented if all US adults were exposed to the highest income level, with approximately half of these reductions occurring amongst people who currently smoke cigarettes at model start in 2014. Overall, are findings indicate that improvements to household financial resources could promote population-level reductions in cigarette use by reducing cigarette initiation, promoting cigarette cessation, and reducing cigarette reinstatement. Further, while the purpose of this study was not to reflect on e-cigarette use outcomes, this novel model may also be employed to explore the relationship between income and e-cigarette use as well.

These findings hold immediate policy implications and directions for future research. There have been growing calls to consider universal basic income (a standard wage provided to individuals regardless of employment status) as a remedy for many social health inequalities (Ruckert et al.). While few studies have been able to study the impact of universal basic income on health behaviors, it has been noted that dynamic modeling studies (such as this one) are an ideal way to project the potential health benefits of such policy (Gibson et al.). Our findings indicate that policies aimed at elevating income level among those making a lower income will lead to reductions in population-level cigarette use prevalence. However, it is not clear if there is a linear relationship between elevated income and decreased cigarette use (i.e., the absolute income hypothesis) or if there is an income threshold (i.e., poverty line) under which individuals are subject to outsized health harms (i.e., the poverty hypothesis), or a combination of the two (Mullahy et al.). Research should seek to better identify how income drives cigarette use behaviors – a universal basic income would be particularly well-equipped to address health inequalities

driven by the poverty hypothesis. This model may be modified and applied to project the potential impact of policies which directly increase household income, such as a universal basic income.

Additionally, these findings require that we reflect on how income has historically been factored into tobacco control policy initiatives. First, it is generally accepted that people with a lower income are more responsive to cigarette excise taxes (considered the most effective tobacco control strategy) (Townsend et al.; National Center for Chronic Disease Prevention and Health Promotion (US) Office on Smoking and Health). Excise taxes are intended to work by increasing the financial burden of using cigarettes, resulting in lower demand, particularly among individuals with fewer financial resources – however, after many decades of ever-present income-based cigarette use disparities (Hu et al.; Cornelius et al.), it is clear that excise taxes (as currently implemented in the US) are not sufficient for the purpose of reducing income-based cigarette use disparities. The logic of excise taxes, which relies on the "rational actor" model of human behavior (Kelly and Barker), would indicate, if extended to its logical ends, that if people were provided more income, their demand for cigarettes would be less responsive to increases in cigarette taxes. Our findings, however, indicate that increasing income level will result in population-level reductions in cigarette use. It is thus important to make explicit the assumptions of human behavior implicit within tobacco policies, such as excise taxes. As a potential alternative, the Trauma-Informed Theory of Individual Health Behavior (TTB) was developed to challenge the "rational actor" model of human behavior (Marks et al.). TTB posits that individuals living in trauma-replicating environments (such as poverty or resource insecurity) must prioritize addressing the harms of their environment (i.e., immediate threats to their well-being) before addressing long term threats to their health, such as those caused by cigarette use. While TTB represents one such

lens, it is important that future tobacco policy endeavors make explicit their (typically implicit) understanding of human behavior.

Limitations

First, it is important to note that this study does not reflect on the mechanisms by which increased income levels may result in lower levels of cigarette use at the population level. While relationships between lower income level and elevated risk of mortality have been established within the academic literature for at least half of a century (Preston), quantitative research approaches have largely failed to capture the mechanism linking income to poor health behaviors and outcomes (Mullahy et al.). One issue is that the quantitative measurement of income represents one operationalization of individual resource security – income is associated with other potential causal factors such as parental use of cigarettes, neighborhood cigarette advertising, and lack of access to health care which might also drive cigarette use. The use of qualitative methods may better illuminate the nature of the relationship between income level and cigarette use – such research has identified a wide range of factors which drive cigarette use among low-income populations, including lack of resources and no comparable, available alternative for stress management (Twyman et al.). Regardless of underlying mechanism, available data indicates that increasing household income levels will result in reductions in cigarette use.

Second, we highlight that our model does not capture realistic dynamics of youth tobacco product use. We have chosen static values for the distribution of cigarette and e-cigarette use among emerging adults to reflect the recent shift in youth product use to primarily e-cigarettes. In our sensitivity analyses (see **Appendix C**), we found that changing the distribution of e-cigarette and cigarette use among emerging adults did not meaningfully change our primary result (i.e., PAF of contribution of income on current cigarette use). However, we highlight that future adaptations

of this model should seek to explicitly model youth tobacco product use. Unfortunately, the PATH study, whose data was used to inform much of this model, did not ask youth participants about their household income. Surveys of youth tobacco use can alleviate such limitations by surveying youth about their household income.

Third, while the PATH Study is weighted to be representative of the US population (Hyland et al.), the weighted sample was not representative along two key variables: the former smoking prevalence and the income distribution of the population. While we used data from other sources to instantiate the starting state of our model to ensure the model was representative of adults in the US, we highlight that parameters may have been biased. The PATH Study over-estimated the prevalence of former smoking and the prevalence of people living with a low-income. While it is challenging to estimate the prevalence of former smoking amongst the population, it is important that we be able to differentiate between individuals who have consumed nicotine regularly in the past versus those who have not – this is because the relationship between an individual who has developed nicotine dependence and nicotine products (such as cigarettes and e-cigarettes) is different than that for someone who has never developed such a dependence (Koob et al.).

Finally, this compartmental model does not capture social dynamics related to cigarette use. Qualitative studies of cigarette use have indicated that the social acceptability of cigarette use and diminished stigma around its use are drivers of cigarette use among low-income populations (Twyman et al.). Agent-based modeling approaches which can account for both social interactions and environments (such as living in a low-income neighborhood with a greater prevalence of cigarette advertising) may be better suited to capturing some of these social dynamics. Future

efforts to develop agent-based models of cigarette use should include income level as a determinant factor.

Conclusion

We developed a novel, compartmental model of cigarette use, e-cigarette use, and income and parametrized it using publicly available data. Over the ten years projected, we found that income contributed to 32% of cigarette use. Among individuals who had never smoked cigarettes at model entry, income contributed to over 80% of cigarette smoking over ten years. In all, our findings indicate that increasing annual household income levels will result in decreased population-level cigarette use prevalence. Policies which directly increase household income, such as a universal basic income, should be considered as potential tobacco control policy approaches.

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Chapter 6 Aim #3 – The Mediating Role of Cigarette Smoking on the Relationship Between Exposure to Trauma During Childhood and Both Chronic Obstructive Pulmonary Disease and Coronary Heart Disease

Abstract

Background. Exposure to trauma during childhood is a risk factor for initiating cigarette smoking, which is the primary cause of preventable death in the USA. The objective of this study was to evaluate the mediating role of lifetime cigarette smoking on the relationship between childhood trauma and two smoking-related health outcomes: chronic obstructive pulmonary disease (COPD) and coronary heart disease (CHD).

Methods. We used cross-sectional data from the 2011-2017 Behavioral Risk Factor Surveillance System, including participants ages 40+ years living in one of 7 US states (n = 44,734). Inverse-probability-weighted marginal structural models were fit to estimate: 1) the total effect of exposure to Adverse Childhood Experiences (ACEs) on each outcome (ever diagnosed with COPD [yes/no], ever diagnosed with CHD [yes/no]; 2) the controlled direct effect of ACE exposure on each outcome, accounting for the mediating role of lifetime cigarette smoking; and 3) the proportion of the total effect of ACE exposure on each outcome that would be eliminated if everyone in the sample had never smoker 100+ cigarettes.

Results. Compared to experiencing 0 ACEs, experiencing a greater number of ACE types was associated with additional cases of both COPD and CHD per 1,000 participants. For COPD, 1 ACE was associated with an additional 22 cases per 1,000 [95% CI 16-29], 2 ACEs an additional 37 cases [95% CI 28-46], 3 ACEs an additional 58 cases [95% CI 46-70], and 4+ ACEs with an additional 112 cases [95% CI 100-124]. For CHD, 1 ACE was associated with an additional 19 cases per 1,000 [95% CI 11-26]), 2 ACEs an additional 33 cases [95% CI 23-44], 3 ACEs an

additional 34 cases [95% CI 21-47], and 4+ ACEs with an additional 48 cases [95% CI 36-59]. It was estimated that between 48.2%-65.5% of the additional cases of COPD and between -4.2%-38.2% of the additional cases of CHD associated with increased ACE exposure would be eliminated if everyone in the sample never smoked 100+ cigarettes.

Discussion. Our results suggest a dose-dependent relationship between ACE exposure and each of our smoking-related health outcomes (COPD and CHD), which are partially mediated by lifetime cigarette use. As reducing the prevalence of cigarette smoking remains a top public health priority, upstream prevention efforts that seek to alleviate the burden of childhood trauma exposure will likely result in reductions in both cigarette use and smoking-related health outcomes such as COPD and CHD.

Introduction

While cigarette smoking prevalence in the US has fallen from over 40% of the total population in the mid-20th century to 14% as of 2019 (Cornelius et al.; Cummings and Proctor), these reductions have not been equitably distributed. For instance, populations such as those living with a low income, those living with a disability, and those living anxiety still report a higher prevalence of cigarette smoking compared to the population average (Cornelius et al.). This indicates a need to identify factors which may drive these troubling disparities in the impact of smoking prevention approaches. Relatedly, exposure to trauma during childhood (ages 0-17) is a significant risk factor for adopting cigarette use and for developing smoking-related health outcomes such as chronic obstructive pulmonary disease (COPD) and coronary heart disease (CHD) (Hughes et al.). Bellis et al. found that in North America, exposure to trauma during childhood were attributed to 24% of smoking incidence, along with 28% of respiratory disease incidence, and 20% of cardiovascular disease incidence, costing approximately $750-billion annually (Bellis et al.). Despite the identification of these associations, research has yet to quantify the mediating role of cigarette use on the relationship between exposure to trauma during adolescence and smoking-related health outcomes such as COPD and CHD. Better understanding how upstream factors (i.e., childhood trauma) predict increased levels of cigarette smoking and related health outcomes can inform future tobacco control and public health initiatives, and can also support efforts to reduce the disease burdens associated with both COPD and CHD.

In this study, our primary aim was to assess whether (and to what degree) lifetime cigarette smoking mediates the relationship between exposure to trauma during childhood – measured using the Adverse Childhood Experiences (ACEs) Scale (Felitti et al.) – on COPD and CHD. To achieve this aim we: 1) estimated the total direct effect of experiencing cumulative ACE types on COPD

and CHD incidence, not accounting for cigarette use; 2) calculated the controlled direct effect of experiencing ACEs on COPD and CHD incidence accounting for the mediating effect of lifetime cigarette smoking; and 3) calculated the proportion of both COPD and CHD as a result of increased ACE exposure that would be eliminated if the study population had never smoked 100+ lifetime cigarettes (smoking 100+ cigarettes is a commonly used threshold for determining if an individual has ever regularly used cigarettes). These results may provide insight into the potential effectiveness of upstream prevention approaches for tobacco control among populations exposed to higher levels of trauma.

Methods

Data Sources

Data were collected via the Behavioral Risk Factor Surveillance System (BRFSS), an annual, cross-sectional health-related telephone survey of adults in the US established by the Centers for Disease Control and Prevention (CDC) in 1984 (Centers for Disease Control and Prevention, *Behavioral Risk Factor Surveillance Survey: History*). Participants were required to be reachable by telephone (BRFSS uses random digit dialing to identify participants) and to be at least 18 years of age. The BRFSS questionnaire is administered at the state-level by CDC-partnered state public health agencies and consists of three parts: 1) a mandatory core component, which includes fixed questions on demographic characteristics and current health behaviors (e.g., cigarette use); 2) optional modules which states are permitted to include; and 3) state-added questions. Response rates vary both by state and type of phone (landline versus cellular device), with the annual median response rates consistently between 40 and 50% (CDC, *2017 Summary Data Quality Report*; CDC, *2011 Summary Data Quality Report*). Cell phone response rates have improved from 2011 (27.9%) to 2017 (44.5%) (CDC, *2017 Summary Data Quality Report*; CDC, *2011 Summary Data Quality Report*).

Data applied in this study were collected between 2011 and 2017. Due to changes to the BRFSS methodology in 2011 to start including cellphone-based respondents (Centers for Disease Control and Prevention (CDC)), we did not request data corresponding to the years 2009 and 2010 in order to ensure that datasets were all comparable. States are not required to share the results of optional modules with the CDC and, as such, we contacted all states that reported including the ACE module (Centers for Disease Control and Prevention, *BRFSS ACE Data*). We received BRFSS data including the results of the ACEs module from: California (2015), New York (2016), North Carolina (2012, 2014), Oklahoma (2014, 2016), Texas (2015), Virginia (2017), and Wisconsin (2011-2015). Because data was collected in a sub-set of states across different years, we did not attempt to weight the sample to represent the US population.

Measures

Our exposure was defined as experiencing trauma during childhood, measured using ACEs. To define participants' exposure to ACEs, we used data collected on their responses to 11 questions on the BRFSS optional module about trauma experienced during childhood (i.e., prior to the age of 18). **Table 6.1** presents these items and how they correspond with 8 distinct types of trauma exposure: emotional abuse, physical abuse, sexual abuse, witnessing intimate partner violence, living with someone who displayed substance use dependence or used illicit drugs, living with someone with a mental health disorder, parental separation or divorce, and living with someone who has been incarcerated. We derived an indicator variable denoting whether a participant reported childhood exposure to each ACE type (1 if yes, 0 if no). For ACE types derived using multiple questions (e.g., sexual abuse), we considered participants who answered in the affirmative to at least one of the questions as exposed to that ACE type; whereas, for questions that asked participants "how often" an ACE type occurred, those responding ≥1 occurrence(s) were

considered exposed to that type of trauma. We then summed across all 8 indicator variables per participant to calculate their cumulative exposure to ACEs (ranging from 0 to 8). Lastly, we then re-coded the cumulative ACE value into a categorical variable (0 ACEs, 1 ACE, 2 ACEs, 3 ACEs, 4+ ACEs) (Hughes et al.). Consistent with prior literature, this categorical definition of exposure to ACEs was chosen to mitigate potential issues around sample size at the margins of exposure; specifically, regarding low anticipated numbers of participants reporting 5+ ACEs, both overall and by state (Hughes et al.).

Table 6.1 Types of trauma exposure and corresponding BRFSS survey items.

Type of Trauma Exposure	**BRFSS Survey Items: "Now looking back, before you were 18 years of age…":**
Emotional Abuse	*How often did a parent or adult in your home ever swear at you, insult you or put you down?*
Physical Abuse	*How often did a parent or other adult in your home ever hit, beat, kick or physically hurt you in any way? Do not include spanking.*
Sexual Abuse	*How often did anyone at least 5 years older than you, or an adult, touch you sexually?* *How often did anyone at least 5 years older than you, or an adult, try to make you touch them sexually?* *How often did anyone at least 5 years older than you, or an adult, force you to have sex?*
Witnessed Intimate Partner Violence	*How often did your parents or adults in your home ever slap, hit, kick, punch, or beat each other up?*
Substance Use in Household	*Did you live with anyone who was a problem drinker or alcoholic?* *Did you live with anyone who used illegal street drugs or who abused prescription medications?*
Mental Health Disorder in Household	*Did you live with anyone who was depressed, mentally ill, or suicidal?*
Parental Separation/Divorce	*Were your parents separated or divorced?*
Incarceration in Household	*Did you live with anyone who served time or was sentenced to serve time in a prison, jail or other correctional facility?*

The mediator was self-reporting having smoked 100+ cigarettes lifetime (yes/no). While cigarette use can be adopted prior to the age of 18 (and thus potentially preceding the time window in which ACEs can occur), maintenance of smoking into later adulthood (over the age of 40) is responsible for most of the health harms associated with cigarette use (Jha et al.).

For the outcomes, participants reported their history of COPD and CHD using the following yes or no questions in the BRFSS: 1) "*[Were you] Ever told you have chronic obstructive pulmonary disease, C.O.P.D., emphysema or chronic bronchitis?"* and *2) "[Were you] Ever told you had angina or coronary heart disease?"*. For simplicity, we refer to each of these dichotomous outcomes as COPD and CHD, respectively.

We captured several covariates that were either deemed to be (1) baseline confounders, which were assumed to precede and potentially confound the exposure-mediator, exposure-outcome and mediator-outcome relationships, or (2) intermediary confounders, which were assumed to follow the exposure and potentially confound the mediator-outcome relationship (**Figure 6.1**). Specifically, we measured the following (1) confounders – age (in years; treated as continuous), sex (male [referent], female), race (white [referent], American Indian/Alaskan Native, Asian, Black, Multiracial, Native Hawaiian/Pacific Islander, Other), and ethnicity (Not Hispanic [referent], Hispanic) – and (2) intermediate confounders – annual income (in USD; >$50k [referent], $35-$50k, $25-$35k, $15-$25k, <$15k), education level (attended or completed higher education [referent], completed high school only, did not complete high school), and marital status (Married/Coupled [Referent], Never Married, Separated/Widowed/Divorced).

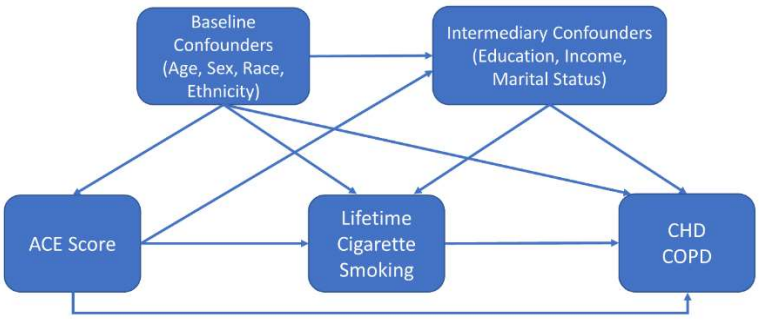

Figure 6.1 Hypothesized Relationship between Study Measures

Inclusion Criteria

BRFSS participants from the 7 states data was received from who responded to the ACEs module, to the lifetime cigarette use survey item, all the covariate survey items, and at least one of the two outcome survey items were included in this study. We restricted the analysis to participants who reported being at least 40 years old at the time of their survey, given that the onset of COPD and CHD generally occurs later in life (Jha et al.). To ensure that our choice to restrict participants to respondents aged 40 or older, we replicated the analysis described herein among the full sample with no age restrictions (see **Appendix D** for sensitivity analysis results).

Analytic Approach

We used a potential outcomes framework to assess the mediating role of 100+ lifetime cigarette smoking on the relationship between experiencing ACEs and COPD and CHD (Rubin; VanderWeele, "Mediation Analysis: A Practitioner's Guide"). Our analytic objectives were to estimate: 1) the total effect of ACE exposure on each outcome (expressed as risk differences); 2) the controlled direct effect of ACE exposure – at each level of the mediator (lifetime cigarette smoking) – on each outcome (expressed as risk differences); and 3) the proportion of the total effect of ACE exposure on each outcome that could be eliminated if tobacco control measures had successfully prevented the study sample from smoking 100+ lifetime cigarettes. To do so, we employ inverse probability weight (IPW)-weighted marginal structural models (MSM) as has been described by Robins and colleagues (Robins et al.), VanderWeele (VanderWeele, "Marginal Structural Models for the Estimation of Direct and Indirect Effects"), and Nandi and colleagues (Nandi et al.). The application of IPWs is an effective strategy for adjusting for different sets of confounding (Cole and Hernan) and has been previously applied by Nandi and colleagues to

explore the mediating role of income on the relationship between ACE exposure and heart disease (Nandi et al.). In **Figure 6.1**, we present a directed acyclic graph that illustrates the hypothesized relationships between ACE exposure, lifetime cigarette use, baseline confounders, intermediary confounders, and the outcomes of interest.

This approach involves two analytic steps, which we undertook separately for each of our outcomes. First, we calculated IPWs to account for exposure-outcome and mediator-outcome confounders. Second, we fit IPW-weighted MSM to calculate our effect measures of interest. The stabilized IPW were calculated for each participant as the product of two separate components: (1) an exposure weight, which accounts for measured confounding of the exposure-outcome relationships and (2) a mediator weight, which accounts for confounding of the mediator-outcome relationships. After multiplying the exposure and mediator weights together to derive a participant's final stabilized IPW, we truncated weights below the 1st or above the 99th percentile weight values to further stabilize their distribution. The ***ipwpoint*** function in the ***ipw*** package in R was used to estimate these weights (van der Wal and Geskus).

After calculating the weights, two IPW-weighted MSMs were fit for each outcome: the first, to assess the total effect of ACE exposure on the outcome of interest (i.e. not accounting for the mediating role of cigarette smoking); and the second, to assess the controlled direct effect of ACE exposure on the outcome while accounting for the mediating role of cigarette smoking. We fit binomial models with an identity link to estimate effects on the risk difference scale. To evaluate total effect, we fit an IPW-weighted model regressing the outcome on ACE exposure. Then to evaluate controlled direct effect and account for potential exposure-mediator confounding, we fit an IPTW-weighted model regressing the outcome on ACE exposure, cigarette smoking status, and an interaction term of the two. The ***contrast*** function in the ***rms*** package in R was used to calculate

controlled direct effect across both mediator levels (<100 lifetime cigarettes/100+ lifetime cigarettes) and corresponding confidence intervals (Harrell Jr). Finally, we calculated the proportion eliminated at the <100 lifetime cigarettes mediator level (VanderWeele, "Policy-Relevant Proportions for Direct Effects"). This metric informs us of the proportion of the effect of ACE exposure on each outcome that would be eliminated if the entire sample had never smoked 100+ lifetime cigarettes. While it is not possible for an individual to return to <100 lifetime cigarettes once they have smoked 100+ lifetime cigarettes, we may understand proportion eliminated as the reduction in the impact of ACE exposure on the outcome if cigarette use were not adopted among a given population – this can be understood to reflect on the protective impact that successful tobacco control policy can have on the relationship between ACE exposure and COPD/CHD. The proportion eliminated is calculated as the difference between the total effect and controlled direct effect divided by the total effect at each ACE exposure level.

Additionally, we fit an MSM regressing cigarette use on ACE exposure, controlling for confounding by applying the exposure-level IPWs and an MSM regressing COPD/CHD on cigarette use by applying the mediator-level IPWs. We provide the results of these models to further contextualize the results. As is discussed in greater detail in **Appendix D,** the mediation approach we have employed is not equipped to reflect on the indirect, mediating pathway given failure to meet necessary assumptions. All analyses were conducted using R (R Core Team) and the code for undertaking this analysis is available in the **Appendix D.** Additionally, in the **Appendix D**, we provide more thorough rationale for using the potential outcomes framework for mediation over traditional mediation approaches.

Evaluation Framework

In response to concerns raised by the replication crisis within the social sciences, leading statisticians have advocated for the evaluation of quantitative findings without the use bright-line significance testing (Amrhein et al.; Wasserstein et al.). As such, we shall evaluate results using the Post-Significance Communication Style (POCS) (Cummins and Marks) – instead of relying on determinations of statistical significance to drive results evaluation, POCS relies upon a thorough description of effect size, effect direction, and uncertainty in order to directly answer the research question at hand.

Results

Sample Characteristics

Overall, 44,734 participants were included in the final analysis (see **Table 6.2**). The median age was 61 years and 42.4% of the sample was male. Nearly 85% of the sample identified as white, 9.0% identified as Black, and 6.8% of the sample identified as Hispanic. Over 45% of the sample reported an annual income greater than $50,000, with only 10.5% reporting an annual income below $15,000. Within the sample, 36.1% reported high school or less as their highest level of formal educational attainment. A majority (57.5%) of the sample reporting being married or in a couple. The state contributing the fewest participants was California (7.8%) and the state contributing the most participants was Wisconsin (23.2%). In total, 44.7% of the sample reported experiencing 0 ACEs during childhood, 23.2% reported experiencing 1 ACE type, 12.3% reported experiencing 2 ACE types, 8.0% reported experiencing 3 ACE types, and 11.8% reported experiencing at least 4 ACE types. Nearly half (47.4%) of the sample reported smoking at least 100 cigarettes in their lifetime. Of those who reported, 9.4% reported ever having been diagnosed with COPD and 11.0% reported ever having been diagnosed with CHD.

Table 6.2 Sample Characteristics

		N = 44,734
Demographic Information		
Age (in years) median (IQR)		61 (52-70)
Sex		
	Male	18,936 (42.3%)
	Female	25,798 (57.7%)
Race		
	White	37,874 (84.7%)
	Black	4,009 (9.0%)
	Asian	502 (1.1%)
	American Indian/Alaskan Native	665 (1.5%)
	Native Hawaiian/Pacific Islander	385 (0.9%)
	Multi-Racial	717 (1.6%)
	Other	582 (1.3%)
Ethnicity		
	Hispanic	3,058 (6.8%)
	Not Hispanic	41,676 (93.2%)
Annual Income Level		
	>$50k	20,443 (45.7%)
	$35-$50k	6,572 (14.7%)
	$25-$35k	5,387 (12.0%)
	$15-$25k	7,636 (17.1%)
	<$15k	4,696 (10.5%)
Highest Educational Attainment		
	Did Not Complete High School	3,756 (8.4%)
	Completed High School	12,394 (27.7%)
	Completed Some College or More	28,584 (63.9%)
Marital Status		
	Married/Coupled	25,730 (57.5%)
	Never Married	3,802 (8.5%)
	Separated/Divorced/Widowed	15,202 (34.0%)
State		
	California	3,493 (7.8%)
	New York	6,260 (14.0%)
	North Carolina	8,333 (18.6%)
	Oklahoma	4,199 (9.4%)
	Texas	6,924 (15.5%)
	Virginia	5,133 (11.5%)
	Wisconsin	10,392 (23.2%)
Mediation Variables		
ACE Score		
	0 ACEs	19,996 (44.7%)
	1 ACE	10,370 (23.2%)
	2 ACEs	5,506 (12.3%)
	3 ACEs	3,593 (8.0%)
	4+ ACEs	5,269 (11.8%)
Lifetime Cigarette Smoking		
	Smoked Less Than 100 Cigarettes	23,479 (52.5%)
	Smoked 100 or More Cigarettes	21,255 (47.5%)
Lifetime, COPD		
	No	40,366 (90.6%)
	Yes	4,203 (9.4%)
	Did Not Respond	165
Lifetime, CHD		
	No	39,507 (88.9%)
	Yes	4,918 (11.1%)
	Did Not Respond	309

Impact of ACE Exposure on Cigarette Use

There was a dose-response relationship between number of ACE types and risk of smoking at least 100 lifetime cigarettes (see **Table 6.3**), such that exposure to 1 ACE type was yielded an additional 64 people per 1,000 (95% CI: 52 – 76), exposure to 2 ACE types with an additional 107 per 1,000 (95% CI: 91 – 122), exposure to 3 ACE types with an additional 157 per 1,000 (95% CI: 138 – 175), and exposure to 4+ ACE types with an additional 199 per 1,000 (95% CI: 182 – 215).

Table 6.3 Impact of elevated ACE exposure on absolute risk of having smoked at least 100 cigarettes in one's lifetime.

ACE Exposure		Risk Difference (95% CI)*
	0 ACEs	
	1 ACE	0.064 (0.052 – 0.076)
	2 ACEs	0.107 (0.091 – 0.122)
	3 ACEs	0.157 (0.138 – 0.175)
	4+ ACEs	0.199 (0.182 – 0.215)

*Risk differences calculated using IPW-weighted binomial model with an identity link. IPWs were calculated to control for the potential confounding effect of age, sex, race, and ethnicity.

Association between Cigarette Smoking, COPD, and CHD

Having smoked at least 100 cigarettes lifetime was associated with an additional 94 cases of COPD per 1,000 people (95% CI: 89 – 100) and with an additional 40 cases of CHD per 1,000 people (95% CI: 34 – 46) (see **Table 6.4**).

Table 6.4 Impact of smoking at least 100 cigarettes lifetime on absolute risk of COPD and CHD incidence.

		COPD	CHD
Lifetime Cigarette Use		RD (95% CI)*	RD (95% CI)*
	Smoked <100 Cigarettes	Ref	Ref
	Smoked >= 100 Cigarettes	0.094 (0.089 – 0.100)	0.040 (0.034 – 0.046)

*Risk differences calculated using IPW-weighted binomial model with an identity link. IPWs were calculated to control for the potential confounding effect of age, sex, race, ethnicity, income, education level, marital status, and ACE exposure.

The Mediating Role of Cigarette Smoking on the Impact of ACE Exposure on COPD and CHD

We found a dose-dependent relationship between elevated ACE exposure and COPD incidence (total effect) (see **Table 6.5**). Compared to individuals exposed to 0 ACEs during childhood, exposure to 1 ACE was associated with an additional 22 cases of COPD per 1,000 (95% CI: 0.016-0.029), exposure to 2 ACEs was associated with an additional 37 cases per 1,000 (95%

CI: 0.028-0.046), exposure to 3 ACEs was associated with an additional 58 cases per 1,000 (95% CI: 0.046-0.070), and exposure to 4 or more ACEs was associated with an additional 112 cases per 1,000 (95% CI: 0.100-0.124). Compared to those exposed to 0 ACEs, the controlled direct effect of ACE exposure for those reporting <100 lifetime cigarettes, increased from an additional 10 cases per 1,000 (95% CI: 0.004-0.017) for those exposed to 1 ACE up to an additional 58 cases per 1,000 (95% CI: 0.045-0.071) for those exposed to 4 or more ACEs. If all BRFSS participants had never smoked 100+ lifetime cigarettes, between 48.2% and 65.5% of the total effect of ACEs on COPD would be eliminated, depending on ACE exposure level.

Table 6.5 The Total and Controlled Direct Effects of Adverse Childhood Experiences (ACEs) on Ever Being Diagnosed with COPD or Coronary Heart Disease among participants in the *Behavioral Risk Factor Surveillance System*, 2011 to 2017.

		CDE (95% CI)			PE
Outcome	ACE Exposure	<100 Cigarettes	100+ Cigarettes	TE (95% CI)	<100 Cigarettes
COPD[a]	1 ACE	0.010 (0.004-0.017)	0.019 (0.008-0.031)	0.022 (0.016-0.029)	54.5%
	2 ACEs	0.015 (0.006-0.024)	0.039 (0.024-0.053)	0.037 (0.028-0.046)	59.5%
	3 ACEs	0.020 (0.008-0.031)	0.062 (0.045-0.080)	0.058 (0.046-0.070)	65.5%
	4+ ACEs	0.058 (0.045-0.071)	0.115 (0.099-0.132)	0.112 (0.100-0.124)	48.2%
CHD[b]	1 ACE	0.013 (0.004-0.022)	0.016 (0.004-0.027)	0.019 (0.011-0.026)	31.5%
	2 ACEs	0.031 (0.019-0.044)	0.024 (0.009-0.039)	0.033 (0.023-0.044)	6.1%
	3 ACEs	0.021 (0.006-0.037)	0.026 (0.009-0.043)	0.034 (0.021-0.047)	38.2%
	4+ ACEs	0.050 (0.034-0.065)	0.030 (0.015-0.044)	0.048 (0.036-0.059)	-4.2%

Notes: CDE = controlled direct effect, TE = total effect, PE = proportion eliminated, CI = Confidence Interval.
[a] 44,569 participants included in analysis
[b] 44,425 participants included in analysis

We also found a dose-dependent relationship between elevated ACE exposure and CHD incidence. Compared to individuals exposed to 0 ACEs during childhood, exposure to 1 ACE was associated with an additional 19 cases of CHD per 1,000 (95% CI: 0.011-0.026), exposure to 2 ACEs was associated with an additional 33 cases per 1,000 (95% CI: 0.023-0.044), exposure to 3 ACEs was associated with an additional 34 cases per 1,000 (95% CI: 0.021-0.047), and exposure to 4 or more ACEs was associated with an additional 48 cases per 1,000 (95% CI: 0.036-0.059). After accounting for the mediating role of cigarette use, compared to those exposed to 0 ACEs,

the controlled direct effect of ACE exposure on CHD increased from an additional 13 cases per 1,000 (95% CI: 0.004-0.022) for those exposed to 1 ACE up to an additional 50 cases per 1,000 (95% CI: 0.034-0.065) for those exposed to 4 or more ACEs. If participants exposed to 1-3 ACEs had never smoked 100+ lifetime cigarettes, between 6.1% and 38.2% of the total effect of ACEs on CHD would be eliminated, depending on ACE exposure level. Interestingly, we found that the controlled direct effect of experiencing 4 or more ACEs among people reporting <100 lifetime cigarettes was greater than the total effect. This indicates that while cigarette smoking is associated with an elevated risk of CHD incidence, that eliminating smoking would not attenuate the increased risk of CHD associated with exposure to 4 or more ACEs.

Discussion

Our results show that exposure to childhood trauma is related to COPD and CHD, and that cigarette smoking helps explain part of the relationship between these two variables. We found that the total effect of exposure to 4 or more ACEs, compared to exposure to 0 ACEs, was an additional 112 cases of COPD per 1,000 individuals and an additional 48 cases of CHD per 1,000 individuals. If tobacco control initiatives had successfully prevented people in the sample from smoking 100+ lifetime cigarettes, over half of COPD incidence and one-quarter of CHD incidence would be eliminated. We note that our findings suggest that preventing the sample from smoking 100+ cigarettes would not attenuate the elevated risk of CHD incidence associated with the exposure to 4 or more ACEs. This suggests that reductions in smoking prevalence will be unlikely to reduce heart disease outcomes among populations with the greatest trauma burden. Our findings suggesting that reductions in smoking would substantially reduce the impact of ACE exposure on COPD incidence are consistent with the fact that cigarette smoking is understood to be a primary risk factor for COPD (Lundbäck et al.). Overall, our findings also indicate that, even in the absence

of cigarette smoking, elevated ACE exposure substantially contributes to COPD and CHD incidence.

While both our ACE measurement and cigarette use measurement fail to capture frequency, duration, and intensity of both exposure types, findings also indicate that elevated ACE exposure and lifetime cigarette use are associated with similar additional absolute risk of developing COPD and CHD. Future research into the role of trauma during development and cigarette use on health outcomes should apply more robust operationalizations of both variables. These findings suggest that 1) efforts to reduce cigarette smoking will partially attenuate the elevated risk of COPD and CHD associated with higher ACE exposure, but that 2) even if no one smoked 100+ lifetime cigarettes, ACE exposure remains a substantial risk factor for the incidence of both COPD and CHD via other pathways. Our findings are consistent with previous research (Hughes et al.; Bellis et al.) and are the first to specifically quantify the mediating effect of cigarette smoking on the relationship between childhood trauma exposure and smoking-related health outcomes.

As well, to appropriately characterize the direct effect of ACE exposure on COPD and CHD, it is important to reflect on the additional mechanisms by which elevated exposure to ACE types is associated with increased risk of both COPD and CHD, which we do not account for in this study. From a practical standpoint, it is not readily clear the mechanisms which underly the direct effect of ACE exposure on COPD and CHD as defined in our study. Su et al. reflect on a series of factors that may help explain this link (with an emphasis on heart disease) and motivate future research extending our results: additional behavioral factors may also mediate the relationship between ACE exposure, such as alcohol consumption, physical inactivity, and sleep abnormalities; elevated ACE exposure is also associated with emotional dysregulation, leading to greater incidence of depression, anxiety, and post-traumatic stress disorder, which are also

understood to elevate risk for a range of disease outcomes; and, finally, elevated exposure to ACEs during development has also been shown to alter immune system and neurocognitive development and has been found to be associated with elevated basal cortisol levels (indicating chronic activation of the body's stress response) (Su et al.). This provides indication that a focus on reducing exposure to trauma during development can lead to improved long-term health outcomes through several pathways, ranging from behavioral to physiological.

This study holds important implications for public health efforts to reduce the mortality burden associated with cigarette use. Our findings indicate exposure to trauma during childhood increases the likelihood of developing COPD and CHD via cigarette use and via additional pathways. This indicates that reducing exposure to trauma and better treating the sequelae of trauma may substantially reduce the prevalence of cigarette smoking and the long-term disease burden associated with these outcomes. Given that between 80% and 90% of people in the US are exposed to trauma in their lifetimes (Koenen et al.; Kilpatrick et al.), it is fair to suggest that programs and interventions which help individuals manage the sequelae of exposure to trauma will meaningfully improve smoking-related outcomes. Further, given the known association between exposure to trauma and a wide range of additional health outcomes (Hughes et al.), tobacco control initiatives would likely be more effective if undertaking in tandem with other public health sub-disciplines seeking to improve the social determinants of health, particularly among children and adolescents.

Limitations

This study used self-reported, cross-sectional data to attempt to assess a causal mediating pathway. We argue that there is a natural temporality within our variables of interest – ACEs occur during childhood, the cigarette smoking that can lead to CHD and COPD generally must be

prolonged into later adulthood (Jha et al.), and the onset of CHD and COPD generally occur later in life. We applied a counterfactual mediation analytic approach which is better suited for assessing such causal pathways than traditional frequentists approaches (VanderWeele, "Mediation Analysis: A Practitioner's Guide"). Ideally, we would have a measure for the intensity and frequency of exposure to trauma during childhood. While the ACE score is commonly utilized, it has noted limitations as an ideal measure of childhood exposure to trauma (McLennan et al.). For example, an individual experiencing only 1 ACE type (e.g., emotional abuse) chronically, would rank lower on ACE exposure than someone who experienced multiple ACE types (e.g., emotional abuse and substance use in the home) only once. Additionally, we would also like to have a more precise measure of lifetime smoking intensity and duration (as opposed to a dichotomous measure) – we may hypothesize that increased exposure to ACEs may be associated with increased intensity of smoking, but, due to our measure for cigarette smoking, we were unable to capture this effect. It is important that we be able to capture the intensity (pack of cigarettes per day) and duration of high-intensity use (in months or years) prior to disease onset. By using the 100-cigarette (i.e., 5 pack) threshold to determine lifetime cigarette smoking, our study may also be subject to a floor effect in which people who smoked cigarettes infrequently may have been assigned to the smoking exposure. If our study included a large number of people who smoked infrequently (e.g., between 100 to 200 cigarettes lifetime), then we will likely have under-estimated the impact of cigarette use on the outcomes of interest. This is because these individuals are less likely to have developed cigarette-related outcomes compared to those who smoked frequently for a long duration (for context, someone who smokes a pack of cigarettes a day smokes over 100 cigarettes each week). Further, we did not attempt to account for the use of other tobacco products (both combustible and non-combustible). This is especially important with the increased adoption of e-cigarette products

such as JUUL, which will not confer the same harms as combustible smoking products, but may also potentiate cigarette use among adolescents (Pierce et al.). Additionally, precise measurement of other high risk health behaviors (such as alcohol use), which may be associated with cigarette smoking, are needed to ensure that all sources of potential confounding are better controlled for. As well, due to concerns of reverse causality (i.e., that a CHD or COPD diagnosis may increase likelihood of smoking cessation), we felt it was inappropriate to use a measurement of current cigarette use status. This limitation can be addressed in future research endeavors by asking survey questions related to lifetime duration of use in addition to intensity of use over this period. In addition, exposure to ACEs may be collinear with other factors related to initiating cigarette use (such as parental cigarette use, exposure to secondhand smoke) and, as such, future research endeavors should seek to disentangle the impact of such potential confounders. Finally, we note that due to limitations in the data used for this analysis, measuring the indirect effect of ACE exposure mediated through smoking was not possible.

Conclusion

Exposure to trauma during childhood was causally linked to increased risk of developing COPD and CHD among a large sample of people in the United States and this relationship held in the absence of a mediated pathway via cigarette smoking. We further identified that these were dose-dependent relationships, and that they were only partially mediated through lifetime cigarette use. Future efforts to reduce the disease mortality of smoking-related health harms should focus on up-stream strategies which aim to minimize exposure to trauma and alleviate the sequelae of experiencing trauma. By effectively accounting for the role that trauma plays in the adoption and maintenance of cigarette use and the development of smoking-related diseases, public health institutions will be better equipped to reduce smoking-related mortality overall. Further, a focus

on reducing the harms of trauma will likely confer additional behavioral health outcome benefits, given the established associations between elevated ACE exposure and increased risk of adopting a range of harmful health behaviors.

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Chapter 7 Discussion

In this dissertation, we have presented a series of works which display how trauma-informed concepts can drive cigarette use research. In Chapter 2, we have presented (and published) the Trauma-Informed Theory of Individual Health Behavior (TTB) (Marks, Pearson, et al.). The three trauma constructs of TTB – historical trauma, trauma-replicating environments, and acute experiences of trauma – drove the remaining structure of this dissertation. In Chapter 3, we completed a scoping review of literature examining the relationship between historical trauma and substance use.[3] In Chapters 4 & 5 we sought to incorporate our understanding of living with a low-income as representing a trauma-replicating environment. We used data from the Population Assessment of Tobacco and Health (PATH) Study to determine the relationship between income and future cigarette and e-cigarette transitions (Chapter 4) and developed a novel mathematical model of cigarette use, e-cigarette use, and income (Chapter 5). Finally, we sought to assess the mediating role of cigarette use in the relationship between exposure to trauma during childhood and the incidence of heart disease and pulmonary disease later in life (Chapter 6). In all, these research projects provide information that can assist in better remedying tobacco-related health disparities.

While each of these chapters represent important contributions to the health behavior, substance use, and cigarette use literature, this dissertation lays a foundation for new avenues of research which may be promising in addressing health inequities, broadly. Herein, I discuss the implications of this dissertation on the public health mission to address health inequities and then

[3] Due to a paucity of literature looking at the relationship of historical trauma and tobacco use, it was determined more impactful to look at substance use more broadly.

identify future avenues of research that can further this mission. While this dissertation focused on cigarette use, many of the implications of these works extend to the use of other substances and to other health behaviors of interest to public health practitioners. As such, the implications discussed are intended to be broad in scope. As well, throughout my doctoral training, I have been challenged to consider the appropriateness of many of the practices and logics that are applied ubiquitously (and often unchallenged) across the public health research and policy fields.

Implications

TTB & Addressing Behavioral Health Disparities

Upon quick inspection of TTB, one might note that the theory does not hypothesize the processes by which an individual considers and adopts a given health behavior, such as ending cigarette use. TTB, instead, focuses on how various traumatic factors impact an individual's ability to engage in behavioral change – the specifics of the health behavior are secondary to the traumatic exposures and circumstances the individual is facing. A core axiom of TTB (extended from the Trauma-Informed Care framework (SAMHSA)) is that individuals will make their best effort to address the most immediate harms they are currently facing. TTB indicates that individuals with greater exposure to traumatic histories, exposures, and environments will have a greater physiological imperative to escape and/or mitigate the immediate harms these traumas represent to them. Often, these self-protective efforts will result in behaviors that have short-term benefit but long-term negative consequences, such as the use of cigarettes. However, public health policy, such as tobacco control initiatives, too often focus on promoting and coercing specific health behaviors while failing to account for heterogeneity of underlying risk. As a result, while such policy endeavors are effective at reducing population-level metrics (e.g., the cigarette use prevalence in the US has fallen consistently for decades), they frequently drive health inequities further (e.g., cigarette use remains disparately prevalent among many vulnerable populations).

This phenomenon has been captured by Frohlich and Potvin's "Health Inequality Paradox", which argues that public health initiatives that ignore heterogeneity in risk will reinforce health disparities (Frohlich and Potvin). The TTB "Trauma Response" represents a construct through which we may understand this heterogeneity in risk. As such, TTB represents a novel and timely theory through which we may understand how upstream factors generate and reinforce population-level health disparities.

TTB, Individual Autonomy, & Public Health Paternalism

TTB also highlights the importance of autonomy in impacting an individual's behavioral choices. Not only does TTB position autonomy as a key resilience factor, but, further, TTB indicates that environments which reduce individual autonomy can replicate traumatic exposures and drive the trauma response. Many public health institutions, such as those of tobacco control, have singular missions to promote specific behavioral health choices – often such institutions promote and implement policies which directly diminish individual autonomy. For example, excise taxes (which are considered the most effective tobacco control policy tool (National Center for Chronic Disease Prevention and Health Promotion (US) Office on Smoking and Health)) are coercive in their intended mechanism – by raising prices, people who smoke are forced to either take on an additional financial burden to continue smoking or to diminish or end their use of cigarettes. While such policies have been instrumental in reducing population-level cigarette use prevalence, they operate by diminishing personal autonomy. This is especially pronounced for individuals living in poverty or with a low-income (which, through TTB, we can conceptualize as a trauma-replicating environment), where excise taxes represent a greater relative financial burden. By increasing the financial burden on individuals who may be experiencing financial, food, and/or housing insecurity, the perceived immediate threat of harms from these insecurities will only be

elevated. This understanding of autonomy can be important for understanding why cigarette use in the US remains disparately high among individuals living with a low income or in poverty (Cornelius et al.).

More broadly, TTB's promotion of individual autonomy stands in opposition to the (often implicit) paternalism of public health institutions. Buchanan provides an enlightening conversation of the ethical dilemma of public health paternalism (i.e., when are public health institutions justified in overriding individual autonomy?) (Buchanan). Buchanan draws a distinction between endeavors to stop the spread of infectious disease (which until the 20th century, were a leading cause of mortality) versus addressing behaviors with long-term health consequences (which represent leading causes of death in many countries today). The restriction of individual autonomy to mitigate the spread of deadly infectious diseases (whether smallpox or COVID-19) is justified by the logic that if such actions are not taken, large swaths of the population will die in the immediate short-term. Buchanan notes, however, that public health institutions which focus on chronic behaviors and diseases tacitly rely on the same justification (i.e., that limiting individual autonomy is justified by the resultant reduction in mortality in the long-term), whereas research which focuses on the "social determinants of health" have indicated that people with the greatest degree of individual autonomy generally enjoy the most healthful lives (Marmot; Marmot and Wilkinson). In this sense, we may understand autonomy itself as a determinant of long-term health. Through this understanding, reductions in individual autonomy via paternalistic policies and interventions must be understood to have long-term negative consequences which may potentially cause more harm than benefit. As Buchanan highlights, the justification for reducing individual autonomy in the name of preventing chronic diseases must be made explicit and must account for the long-term health costs of diminishing individuals' autonomy.

Upstream Policies Which Promote Autonomy and Better Health

While there is an ethical imperative to consider the potential harms of paternalistic public health policies and practices, this dissertation also displays that such policies which focus on promoting individual autonomy can be highly effective at addressing specific harmful health behaviors. In Chapter 5, we projected that increasing household income levels would lead to substantial decreases in the cigarette use prevalence in the US. Here, we understand that elevated income provides an individual increased levels of autonomy by reducing insecurities that may cause them harm and that control and/or limit their behavior. Given the understood connection between living with a low income and well-being/mortality (Chetty et al.), it is also clear that such promotions in household income would likely result in health benefits outside those we projected for cigarette use (e.g., improvements in diet, exercise, mental health). Thus, policies and interventions which promote individual autonomy may not only be ethically preferable to paternalistic approaches, but they also may be more effective at promoting long-term health.

Community Self-Determination, Historical Trauma, and "Dangerous Memories"

Inextricable from the notion of personal autonomy is that of community self-determination. As we reflected on in Chapter 3, studying historical trauma makes it clear that there are many competing narratives of history and that certain narratives are understood to be more "dangerous" to current systems of power than others (Zembylas and Bekerman). US policy, since the nation's conception, has focused on the control of Indigenous and Black bodies, however, modern efforts to name these histories and the modern systems of oppression they have generated continue to face mainstream backlash – this is exemplified by the current fears of the "critical race theory bogeyman" which Fox News commentator Tucker Carlson referred to as "civilization-ending

poison" (Mudde; Porter).[4] As such, it is currently culturally and politically contentious to suggest that communities that have long been subjected to the harms of colonization should be permitted to define their own histories. This reality holds important implications for what it means, as researchers, to study the impact of historical trauma on modern day circumstances, including cigarette use and substance use broadly.

Mohatt et al. suggest that historical trauma should not be applied as a construct to identify the causal mechanisms by which historical harms have manifested in modern day harms and systems of oppression, but instead should be considered as a public narrative tool that communities can use to understand their modern circumstances in relation to their own histories (Mohatt et al.). While substance use epidemiology is generally focused on identifying associations and causal relationships between various factors and outcomes, our scoping review in Chapter 3 indicates that our statistical tools for making causal assessments may be poorly suited to incorporating the effects of history. The quantitative studies we identified measured one mechanism by which historical trauma impacts individuals (i.e., thoughts and feelings towards specific historical harms) (Whitbeck et al.) – while an important mechanism that is worthy of continued study, it appears that individual-level, self-report data is inadequate for the purposes of identifying how population-level historical harms have manifested in modern circumstances. Given reliance on quantitative strategies to determine causation within modern social sciences, it is important to question the best ways to approach the study of historical trauma and its impact on modern day substance use.

A brief overview of the history of social statistics and the study of race[5] is further illuminating in understanding how modern quantitative practices were developed to ignore

[4] While Mr. Carlson is a leading expert on "civilization-ending poison", I have presented his quotation facetiously.
[5] Given the racialized nature of societies impacted by European colonization, the study of race and the study of historical trauma are, at minimum, inter-related and, at maximum, inextricable.

historical context. In their work *Thicker Than Blood: How Racial Statistics Lie*, Zuberi discusses two competing visions for the use of quantitative statistics in the social sciences at the start of the 20th century (Zuberi). First, were the approaches developed by the forefathers of modern statistics, Galton, Pearson, and Fischer. As proponents of eugenic thought, they developed statistical techniques that could be used to compare racial groups – often for the explicit purpose of justifying white Anglo-Europeans position atop the racial hierarchy.[6] This approach took an essentialist view towards race – while the logic under-pinning the eugenics movement has been discredited, modern quantitative social scientists continue to rely upon the same methods of categorizing people by essentialist racial categories and ascribing associations and causal effects to them. In contrast, scientists such as W.E.B. DuBois contextualized racial statistics within arguments that connected historical context to differential outcomes experienced across socially constructed racial populations. Where Galton, Pearson, and Fischer conceptualized race as an essential human quality which explained observed population differences, DuBois conceptualized race as socially constructed and that measurable differences across racial groups were evidence of the historical, structural, and culture differential treatment of people based on constructed race. Where Galton, Pearson, and Fischer saw racial differences as evidence supporting essential racial hierarchy, DuBois saw racial differences as the evidence of differential historical and cultural treatment on the basis of race. Notably, modern quantitative epidemiological practices are based on the works of Galton, Pearson, and Fischer, not DuBois, which provides historical context and imperative for the need to critically challenge how we consider statistics which focus on the impact of historical

[6] See Pearson's 1925 publication in the *Annals of Eugenics*, "The Problem of Alien Immigration into Great Britain, Illustrated by an Examination of Russian and Polish Jewish Children" as one such example.

harms (which, in colonial settings, are frequently defined along these socially constructed racial stratifications).

Mohatt's understanding of historical trauma as a public narrative is in alignment with DuBois analyses of racial differences as the result of historical and cultural factors. DuBois did not capture historical harms quantitatively, but instead applied historical accounts to contextualize and understand current and measurable racial disparities. This indicates that historical accounts represent key pieces of data which can inform the construction of research hypotheses and the interpretation in findings. Racial health disparities are a natural consequence of disparate historical treatment. Research which focuses on identity-based health disparities (i.e., by race, by gender, by sexual orientation) should seek to contextualize their hypotheses within disparate histories. Instead of essentializing such factors through the implicit eugenic lens[7] and treating disparities as inherent to human differences, disparities can be understood as driven by differential treatment based on these socially constructed categories. However, as noted, which histories can be told remains culturally contentious and, thus, researchers studying historical trauma or seeking to incorporate historical context in their work are likely to receive pushback for elevating "dangerous memories." It is important to highlight that DuBois work was not contentious on its methodological merits, but instead because his work elevated such "dangerous memories" and connected them to modern day circumstances and systems (Zuberi). As such, there is an ethical imperative for communities to have the right to name their own histories and to conceptualize how they shape the present. Researchers, especially those focused on historical trauma and population-disparities research,

[7] This is not to suggest that researchers who use this lens are eugenicists, but to suggest that the tools we possess were developed in the name of eugenics and that we, as academics, hold a responsibility to engage with the consequences – in the next section, I will reflect on how the essentialist lens was applied several times within this dissertation and how this can be avoided moving forward.

hold a social position that can allow them to facilitate the process of communities naming their own histories.

Reflecting on the Use of Race Essentialism and Erasure of Historical Context Within This Dissertation

While it is easy enough to levy broad criticisms of applying eugenic, essentialist methods against the majority of quantitative social science disciplines, I believe it is important to reflect on how such essentialist methodology appeared within this dissertation[8] – which it did. In both Chapters 4 and 6, race and ethnicity were treated in this essential fashion and applied as covariates within the modeling endeavors. This, of course, is common practice and it is quite likely that a reviewer of such work might balk if these variables were not included as model controls. As Martin and Yeung highlight, even in studies where researchers are not attempting to study race, they are forced to reify the essentialist definition of race and take a position "on the nature of race itself" (Martin and Yeung). In other words, even when researchers understand that race is socially constructed, we are often expected to apply these essentialized definitions of race within our work, regardless. An implication of this is, that as social scientists, it is crucial that we take explicit stances on "the nature of race" and other such socially constructed identity variables (e.g., ethnicity, gender) else we shall be forced to assume the implicit stance of essentialism. Ideally, this will involve avoiding such essentializing, but may also arise in discussions of study limitations which apply essential definitions.

Then, an important question is, "What does it mean to control for race?" While I direct the reader to other works which address this question in far greater depth (Zuberi; Zuberi and Bonilla-

[8] While the practice of self-reflexivity is commonplace within the qualitative sciences, it is clear that researcher position and subjectivity plays a large part in the design and interpretation of quantitative studies (the difference in Pearson/Fischer and DuBois approaches highlight this all too well).

Silva), it is relevant to the implications of studying historical trauma to discuss this here (i.e., because of the intertwined relation between historical harms and race in settler-colonial contexts). Generally, in non-randomized studies, we incorporate control variables into our model to control for potential confounding. In Chapter 4, we were interested in the relationship between income level and various outcomes. It is widely accepted that race should be included as a control if available (i.e., no justification is required to include an essentialist definition of race into a regression model). However, can we truly hope to eliminate the effect of race on wealth in the US context? Given the legacy of slavery, the failures of Reconstruction, the implementation of Jim Crow and redlining policies, and then the War on Drugs and mass incarceration, the history of disparate treatment by racial status is a key predictor of the distribution of wealth across these constructed racial groups. Wealth and race in the US have an inextricable relationship which historical accounts make clear. The goal in Chapter 4 was to isolate the relationship between income level (our primary predictor) and a series of tobacco use related outcomes – however, history makes clear that measures of wealth cannot be isolated from the effects of race (i.e., assumption of predictor independence is violated). Given that race also captures modern disparities in treatment (e.g., exposures such as discrimination and microaggressions), it is not clear precisely what we are controlling for when we control for race in regression models. A primary implication of this discussion is that researchers have an ethical and methodological obligation to justify every variable included in their modeling endeavors. While this would put additional onus onto researchers, the alternative is to continue implicitly reifying the essentialist conception of race.

The importance of explicitly discussing the use of race essentialism in substance use research is exemplified by a line of inquiry I have conducted outside of this dissertation. Under the supervision of Dr. Annick Borquez, I led the development of a predictive model to determine

which counties are at highest risk of having an opioid overdose outbreak in the subsequent year (Marks, Abramovitz, et al.). An influential CDC report, which was used to identify counties in need of resources to stop potential HIV outbreaks related to injection drug use, had attempted a similar project years earlier and one of their key predictors of risk was the percentage of the population that was white in the county (Van Handel et al.). In other words, their model identified that counties with a higher proportion of white people were at higher risk of having an injection drug use-related HIV outbreak. Available data supported this finding because the early stages of the opioid crisis affected regions with a higher proportion of white people. However, by not reflecting and justifying the use of race in their model, they not only failed to capture the likely underlying mechanisms driving this trend, but their results then indicated that counties with a higher prevalence of white people were in greater need of resources than other counties (i.e., their model gave preferential treatment to regions with a higher prevalence of white people). It appears now that the early concentration of the opioids harms amongst white populations was not driven by some essential quality of white individuals that made them more susceptible to opioid abuse, but instead that doctors preferentially prescribed opioids for pain management to white patients because they, generally, took non-white patients' claims of pain less seriously (Om).[9] Without this historical context, one might interpret the racially disparate impact of the opioid crisis to suggest white people were more vulnerable to the harms of opioid abuse, but with historical context, it appears that differential treatment by race (i.e., racism) drove these initial disparities. Now that the opioid crisis is driven primarily by the illicit drug market (Ciccarone), there does not appear to be a justification for presuming that race is inherently associated with differential risk of experiencing

[9] Earlier, I discussed the importance of incorporating historical context in the design of research hypotheses and interpreting findings. In this example, we see that historical context illuminates the observed racial disparities and highlights the actual mechanism (i.e., not race, but racism) driving the phenomenon under study.

harms from opioid use. Further, a predictive model that incorporates race naively will predict (based on observed trends) that regions with a higher prevalence of white people are at highest risk of experiencing future harms, which could redirect resources to these communities based on their racial demography. I do not highlight the CDC report to disparage their important work, but to highlight how applying race essentialism implicitly replicated dynamics of racism and prioritized the needs of white communities over others as a result. Galton, Pearson, and Fischer developed their tools explicitly for the purpose of reinforcing racial hierarchy – to apply these methods naively is to risk replicating their intended purpose. As part of my research with Dr. Borquez, we also developed a framework for conducting predictive modeling studies focused on the opioid crisis and recommended that research teams explicitly justify the use of essentialized race in their models (Marks, Carrasco-Escobar, et al.). Our predictive modeling study refrained from using race as a covariate and we provided explicit justification for this choice, grounding our argument in historical context (Marks, Abramovitz, et al.).

Implications Summarized

To this point, I have highlighted several implications that I believe this dissertation is particularly well-suited to reflect upon. I summarize these concerns, for ease of future reflection, here:

1. The Trauma-Informed Theory of Individual Health Behavior is well-suited to investigate health disparities, particularly in applying trauma constructs to understand how risk is heterogeneously distributed across the population (a la Frohlich and Potvin's "Health Inequality Paradox")

2. Individual autonomy must be understood as a primary determinant of long-term health, in alignment with TTB principles, and that paternalistic public health initiatives aimed at improving long-term health must be able to justify the resultant reduction in autonomy.
3. Upstream policies which focus broadly on alleviating traumatic exposures and environments (such as income assistance) will increase individual autonomy and have benefits across a wide range of health behaviors and outcomes.
4. Researchers have an ethical responsibility to aid communities in naming their own histories, especially in the face of cultural and political contention surrounding who gets to tell history.
5. Health disparities research must apply an understanding of historical disparities in the treatment of populations. Historical context should be treated as data and should inform the design of studies, the research questions asked, and the interpretation of findings.
6. Researchers must justify the use of essentialized definitions of race within research studies or refrain from using them. Modern statistical practices were developed to further the eugenics mission of racial hierarchy and, while this does not mean that these methods cannot be applied for other purposes, our case example of predictive modeling studies displays how a lack of justification can result in replicating the intended (eugenic) purposes of essentializing race.

Future Directions

It is also important to reflect on what future research can be conducted to build from the work presented in this dissertation. Here I focus on two important directions: the first, validating the use of TTB to study health disparities; and the second, expanding the application of the SET

model to understand how upstream policy interventions may impact population-level health behaviors.

Validating the Trauma-Informed Theory of Individual Health Behavior

An important next step will be to test and validate the use of TTB to study, understand, and address health behavior and related disparities. While individual theories of health behavior typically capture how individual-level characteristics (e.g., knowledge, attitudes) shape behavioral processes, the three core TTB constructs attempt to capture traumatic sources at different socio-ecological levels (i.e., interpersonal, environmental, and historical) (SAMHSA). This requires critical thought into how these constructs should be captured and assessed in relation to one another. While, typically, theory validation is driven by identifying appropriate measurement tools, assessing their validity, and then applying them to validate the theory itself – I argue that TTB-driven health disparities research must start with formative, participatory, qualitative research to understand how constructs should be captured with respect to the population under study.

First, as highlighted earlier, historical trauma (and historical context, more broadly) likely cannot be fully captured by quantitative measurement in self-report studies. While measurements such as the Historical Loss Scale represent an important tool for capturing one mechanism by which historical trauma may manifest within the individual (Les Whitbeck et al.), the historical trauma construct is intended to capture how historical events experienced by populations shape their modern circumstances. Further, our previous discussions of "dangerous memories" also make clear that history is not a static, objective accounting of the past, but is made up of myriad accounts (many of which are not reconcilable) from specific vantages and which often arise to fulfill specific

purposes.[10] As such, we must not only ask how we are to incorporate history into this research, but which histories we elevate in our work.

Mohatt's understanding of historical trauma as a public narrative and DuBois' application of history to contextualize research questions and findings provide valuable guidance in capturing and applying historical trauma in TTB-driven health disparities research. The first step in applying TTB to study health disparities within a given population should be to work with members of said population to develop a consensus of important historical events which they understand to drive these modern disparities (i.e., allow communities to develop their "public narrative"). Then, in the spirit of DuBois' work, this understanding of history should be employed to contextualize research questions and findings. For example, in Chapter 3 we identified many qualitative studies with Indigenous peoples of North America which indicated that substance use in their communities today is inextricable from the impact of the residential/boarding school systems. Such studies represent ideal first steps in applying TTB to understand health disparities. Additional research questions and findings should then, in this example, be grounded within the residential/boarding school narrative. Future studies which employ and attempt to validate TTB should consider this type of historical narrative building as a starting place and basing next research decisions on the findings.

While less discussion has been dedicated thus far to the challenges in quantitatively capturing trauma-replicating environments, Chapters 4 & 5 of this dissertation lay a groundwork

[10] This is, then, where epistemological conversations on the nature of "history" as a concept are important to engage in, at least briefly. Without diving deeply into this dense philosophical forest, I believe it is important to highlight the difference between the past and history. Here I shall refer to the past as all that has preceded the present moment, regardless of whether humans have observed and can recall it and history as the human endeavor of trying to name aspects of this past, generally with a focus on the happenings and doings of human beings. Just as a painter cannot capture every vantage point of a landscape, no historical account can hope to capture the totality of the past. This distinction makes clear, as researchers, that we cannot assume a static conception of the past, but instead must wade through a multitude of (often conflicting) histories. Further, (as Foucault has described), specific versions of history are often employed by regimes of power to justify their hegemonic position ("TRUTH AND POWER : An Interview with Michel Foucault"). While, as scientists, we attempt to stake a neutral position in our work, it is important to recognize that there is no neutral historical position (i.e., every historical account is subjective in its nature and most serve some additional purpose of justifying positions of power).

for how we may attempt to both conceptualize and capture such environments. In these chapters, the research questions were motivated by the understanding that living in poverty or with a low-income may trigger the trauma response in individuals by replicating dynamics of previously experienced traumas (e.g., losing one's sense of autonomy and safety) or by directly exposing them to traumatic events (e.g., losing one's home). The variable used to capture this environmental exposure, however, was the commonly used measure of household income. In this dissertation, justification was provided for how living in poverty or with a low-income represents living in a trauma-replicating environment and then we applied the best available metric to capture this environmental exposure.

While this approach may suffice in many instances, formative qualitative research should be undertaken to better understand trauma-replicating environments among communities under study. While certain trauma-replicating environments may be readily apparent (such as poverty or homelessness), others may be less visible (especially if researchers are not members of the study population). For example, I worked at a behavioral health clinic that served an Indigenous community. There were several instances where an adolescent was taken by ambulance to an emergency room and their parents arrived in a state of panic, unable to locate their child – while their concern for their child's health was understandable, an additional cause of distress was that their child was taken by an unknown authority to a location they could not identify. This sequence directly replicated the dynamics of the boarding school era in which children were taken, often never to be heard from again. Even though, in these instances, the children were being provided the medical care they required, it was clear that the situation replicated past traumas that were harmful to the parents in that moment. Researchers (especially those not in the study population) should not attempt to identify such environments based solely on available research but should

apply participatory and qualitative strategies to identify them. Further, formative research into the historical traumas a population has faced will likely create a bridge to understanding trauma-replicating environments that they currently are exposed to.

TTB also indicates that many acute experiences of trauma are specific to historical trauma and trauma-replicating environment exposure. While we may understand that there are many traumatic events that can impact any person (i.e., being subject to abuse, being assaulted), there are also forms of trauma that are specific to populations. Slurs provide a clear example of this. In the United States, there are many violent terms that can be used to characterize Black people, Indigenous people, queer people, transgender people, among many other historically marginalized groups; whereas slurs with the same historical and violent weight are not available to characterize, for example, white, cis-gendered, men.[11] While slurs provide a clear example of how different populations may be subject to unique experiences of trauma, there are many instances in which population-specific traumas may be more abstract. For example, the weathering hypothesis discusses how the health of Black women is negatively impacted by constant exposure to daily slights and indignities (i.e., microaggressions) (Geronimus et al.). Such microaggressions may be rendered invisible to those who are never subject to them (for example, for Black women, having expertise dismissed at work, having someone be surprised that they are so "articulate", being ignored or profiled as a customer), yet they represent significant harms with lasting health impacts (Sue et al.). As with both historical trauma and trauma-replicating environments, it is important that researchers employing TTB apply participatory and qualitative methods to identify population-specific sources of acute trauma that may typically be rendered invisible.

[11] Here I simply mean that a slur used to characterize a white cisgender male is not rendered violent by historical or systemic context. As additional evidence, I offer the reader the following Portlandia skit, entitled "What About Men?": https://www.youtube.com/watch?v=z6IhNcnLwiw

Finally, it will be important to identify appropriate ways of capturing the trauma response. TTB indicates that an elevated trauma response reduces individual capacity to make health behavior choices aimed at long-term health outcomes, as individuals instead are forced to address short-term, immediate sources of harm. There may be interest to attempt to capture the trauma response using biometrics – cortisol levels have long been understood to be elevated in individuals with history of exposure to trauma (Elzinga et al.). However, TTB, as a theory, does not address biological mechanisms and any attempt to reduce the experience of the trauma response to biological mechanisms may fall short of capturing the perceived reality of experiencing an elevated trauma response. As such, biometrics should be applied with caution. As the primary axiom of TTB is that individuals make their best effort to address their most immediate harms, it will be important for TTB-driven research to attempt to capture the most immediate harms an individual is facing. The more immediate and severe the perceived harms are, the less likely individuals are to undertake health behaviors aimed at long-term, positive health outcomes. Capturing both the perceived immediacy and severity of harms is an ideal starting place for capturing the trauma response. Again, consistent with recommendations for each trauma construct, it will be important to use participatory, qualitative methods with the study population to capture their understanding of the trauma response, as this may also manifest in population- and culturally specific ways.

Overall, each application of TTB should be specific to the population under study and should employ participatory, qualitative strategies to lay the foundation for the research project. As noted in Chapter 2, it can be helpful to consider TTB as an individual-level counterpart to Freire's Empowerment Education which focuses on using participatory interventions to raise community consciousness and autonomy (Wallerstein). It will be important to develop quantitative

tools to measure various constructs, but the tools should be developed and adapted based on the results of initial qualitative findings.

Further Developing the Socio-Economic Tobacco Model

A second important area of future research will be the further development of the Socio-Economic Tobacco (SET) Model presented in Chapter 5. While income-related cigarette disparities have been long acknowledged, prominent models of cigarette use do not include income level as a factor of interest (Levy et al.; Howard et al.; Tengs et al.). This is peculiar, as well, given the accepted notion that people living with a low income are more responsive to common tobacco control policies such as excise taxes. As a result of incorporating income level in our model, our findings indicated that addressing income disparities can lead to substantial reductions in cigarette use. In particular, elevated population income may drastically reduce the initiation of cigarette use by the cigarette-naïve.

A first important adaptation will be developing modules that simulate common tobacco control policies, such as excise taxes and educational campaigns. The SET Model has the capacity to project how such policies may differentially impact populations based on their income level. It will be of interest to compare the efficacy of traditional tobacco control initiatives with upstream interventions aimed at elevating income household income level. If such upstream interventions appear to be more effective at reducing cigarette use levels, this implies they may a better use of resources than typical tobacco control initiatives. Such upstream interventions are also likely to have direct benefits across a range of health behaviors including diet, exercise, and illicit drug use, as well.

The larger task will be to expand the SET Model to also include these other health behaviors and more precise measurements of household financial resources. To appropriately

assess the impact of upstream interventions, it will require developing the SET model to account for health behaviors that are likely to be influenced by changes in income level. While the SET Model used publicly available data, it will be most effective to create a survey for the purpose of developing the SET Model. The income stratifications for the SET Model were based on how the Population Assessment of Tobacco and Health (PATH) Study defined income (categorically) (Hyland et al.), however, it would be ideal to capture a precise (continuous) measure of income level. Further, it would be useful to capture other factors related to financial resources such as home ownership, debt owed, and familial financial support systems. Through such a survey, the SET Model could be re-designed to account for income level, cigarette use, in addition to a range of other health behaviors of interest such as diet, exercise, and the use of other illicit drugs. It may be appropriate, to adapt the SET model into an agent-based model, as these may have a greater ability to assess the impact of income-based upstream interventions.

As such, while the SET Model and results presented in this dissertation represent an important step forward, the task of understanding the broad impacts of upstream, financial interventions represents a powerful next step.

Conclusion

In this dissertation, we have presented the Trauma-Informed Theory of Individual Health Behavior and have displayed how its trauma constructs can be applied to cigarette-related research. As discussed, this dissertation holds important implications for the future research of cigarette use and health behavior broadly. The findings challenge the use of methods and interventions that are largely accepted within our scientific fields. These works, further, set the foundation for multiple lifelong lines of scientific inquiry.

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Appendix A: Aim #1 Supplemental Materials

Search Protocol

Two search terms were generated to conduct this scoping review. Since we sought to capture all peer-reviewed, original research studies which focus on the association between historical trauma and substance use, we generated a search term for historical trauma and for substance use. For the historical trauma search term, we included variations of the term "historical trauma" as well as variations of similar terms including: "historical loss", "intergenerational trauma", "transgenerational trauma", and "collective trauma". For our PubMed search, the historical trauma search term was:

> **(**"Historical Trauma"[Mesh] OR "historical trauma" OR "historical traumas" OR "historical loss" OR "intergenerational trauma" OR "intergenerational traumas" OR "transgenerational trauma" OR "transgenerational traumas" OR "collective trauma" OR "collective traumas" OR "collective traumatic")

For substance use, we generated a search term which sought to capture specific substance types (e.g., "cigarettes"), substance behaviors (e.g., "cigarette use"), and specific populations who use drugs (e.g., "people who use drugs", "drug users"). We also included terms related to substance use disorders. Finally, we also included brought health behavior terms such as "health behavior" and "health risk behaviors" to ensure that we captured all relevant studies. For our PubMed search, the substance use search term was:

> (Tobacco[MeSH] OR "tobacco" OR Tobacco Use[MeSH] OR "tobacco use" OR Cigarette Smoking[MeSH] OR "cigarette" OR "cigarette smoking" OR Vaping[MeSH] "vaping" OR drinking behavior[MeSH] OR "drinking" OR alcohol drinking[MeSH] OR marijuana use[MeSH] OR marijuana smoking[MeSH] OR drug misuse[MeSH] OR "drug misuse" OR drug users[MeSH] OR "drug users" OR "drug injection" OR "injection drug users" OR "people who inject drugs" OR "drugs" OR "illicit drugs" OR "illegal drugs" OR "heroin" OR "cocaine" OR "crack cocaine" OR "opioids" OR "amphetamine" OR "methamphetamine" OR "injection drug use" OR "cannabis" OR "marijuana" OR "alcohol" OR Substance-Related Disorders[MeSH] OR "Substance-Related Disorders" OR Health Behavior[MeSH] OR "health behavior" OR "behavioral health" OR health risk behaviors[MeSH] OR "health risk behaviors" OR "risk behaviors")

We undertook our search in the following databases: PubMed, SCOPUS, CINAHL, and PsycInfo. Below are the full search terms used for each database:

PubMed: ("Historical Trauma"[Mesh] OR "historical trauma" OR "historical traumas" OR "historical loss" OR "intergenerational trauma" OR "intergenerational traumas" OR "transgenerational trauma" OR "transgenerational traumas" OR "collective trauma" OR "collective traumas" OR "collective traumatic"**)** AND (Tobacco[MeSH] OR "tobacco" OR Tobacco Use[MeSH] OR "tobacco use" OR Cigarette Smoking[MeSH] OR "cigarette" OR "cigarette smoking" OR Vaping[MeSH] "vaping" OR drinking behavior[MeSH] OR "drinking" OR alcohol drinking[MeSH] OR marijuana use[MeSH] OR marijuana smoking[MeSH] OR drug misuse[MeSH] OR "drug misuse" OR drug users[MeSH] OR "drug users" OR "drug injection" OR "injection drug users" OR "people who inject drugs" OR "drugs" OR "illicit drugs" OR

“illegal drugs” OR “heroin” OR “cocaine” OR “crack cocaine” OR “opioids” OR “amphetamine” OR “methamphetamine” OR “injection drug use” OR “cannabis” OR “marijuana” OR “alcohol” OR Substance-Related Disorders[MeSH] OR “Substance-Related Disorders” OR Health Behavior[MeSH] OR “health behavior” OR “behavioral health” OR health risk behaviors[MeSH] OR “health risk behaviors” OR “risk behaviors”)

SCOPUS: (TITLE-ABS-KEY((“historical trauma" OR "historical traumas" OR “historical loss” OR "intergenerational trauma" OR "intergenerational traumas" OR "transgenerational trauma" OR "transgenerational traumas" OR "collective trauma" OR "collective traumas" OR "collective traumatic") AND (“tobacco” OR “tobacco use” OR “cigarette” OR “cigarette smoking” OR “vaping” OR “drinking” OR“drug misuse” OR “drug users” OR “drug injection” OR “injection drug users” OR “people who inject drugs” OR “drugs” OR “illicit drugs” OR “illegal drugs” OR “heroin” OR “cocaine” OR “crack cocaine” OR “opioids” OR “amphetamine” OR “methamphetamine” OR “injection drug use” OR “cannabis” OR “marijuana” OR “alcohol” OR “Substance-Related Disorders” OR “health behavior” OR “behavioral health” OR “health risk behaviors” OR “risk behaviors”)))

CINAHL: ((MM "Historical Trauma") OR "historical trauma" OR "historical traumas" OR “historical loss” OR "intergenerational trauma" OR "intergenerational traumas" OR "transgenerational trauma" OR "transgenerational traumas" OR "collective trauma" OR "collective traumas" OR "collective traumatic") AND ((MM “Smoking+”) OR “tobacco” OR “tobacco use” OR “cigarette smoking” OR “cigarette” OR “smoking” OR (MM “Substance Use Disorders”) OR (MM “Substance Dependence”) OR (MM “Substance Abusers+”)OR (MM “Drinking Behavior+”) OR (MM “Cannabis”) OR (MM “Medical Marijuana”) OR “cannabis” OR “marijuana” OR (MM “Street Drugs+”) OR “drug misuse” OR “drug users” OR “drug injection” OR “injection drug use” OR “injection drug users” OR “people who inject drugs” OR “drugs” OR “illicit drugs” OR “illegal drugs” OR “heroin” OR “cocaine” OR “crack cocaine” OR “opioids” OR “amphetamine” OR “methamphetamine” OR (MM “Health Behavior”) OR “health behavior” OR “behavioral health” OR “health risk behaviors” OR “risk behaviors”)

PsycInfo: NOFT((“historical trauma" OR "historical traumas" OR “historical loss” OR "intergenerational trauma" OR "intergenerational traumas" OR "transgenerational trauma" OR "transgenerational traumas" OR "collective trauma" OR "collective traumas" OR "collective traumatic") AND (“tobacco” OR “tobacco use” OR “cigarette” OR “cigarette smoking” OR “vaping” OR “drinking” OR“drug misuse” OR “drug users” OR “drug injection” OR “injection drug users” OR “people who inject drugs” OR “drugs” OR “illicit drugs” OR “illegal drugs” OR “heroin” OR “cocaine” OR “crack cocaine” OR “opioids” OR “amphetamine” OR “methamphetamine” OR “injection drug use” OR “cannabis” OR “marijuana” OR “alcohol” OR “Substance-Related Disorders” OR “health behavior” OR “behavioral health” OR “health risk behaviors” OR “risk behaviors”))

PRISMA-ScR Checklist for Scoping Reviews

Page numbers filled out correspond to the page number of Chapter 3 of the dissertation. This is to ensure that when the chapter is submitted for publication, the checklist will not need to be updated.

Table A.1 PRISMA-ScR Checklist

SECTION	ITEM	PRISMA-ScR CHECKLIST ITEM	REPORTED ON PAGE #
TITLE			
Title	1	Identify the report as a scoping review.	1
ABSTRACT			
Structured summary	2	Provide a structured summary that includes (as applicable): background, objectives, eligibility criteria, sources of evidence, charting methods, results, and conclusions that relate to the review questions and objectives.	1
INTRODUCTION			
Rationale	3	Describe the rationale for the review in the context of what is already known. Explain why the review questions/objectives lend themselves to a scoping review approach.	2-3
Objectives	4	Provide an explicit statement of the questions and objectives being addressed with reference to their key elements (e.g., population or participants, concepts, and context) or other relevant key elements used to conceptualize the review questions and/or objectives.	2
METHODS			
Protocol and registration	5	Indicate whether a review protocol exists; state if and where it can be accessed (e.g., a Web address); and if available, provide registration information, including the registration number.	3
Eligibility criteria	6	Specify characteristics of the sources of evidence used as eligibility criteria (e.g., years considered, language, and publication status), and provide a rationale.	3-4
Information sources*	7	Describe all information sources in the search (e.g., databases with dates of coverage and contact with authors to identify additional sources), as well as the date the most recent search was executed.	3
Search	8	Present the full electronic search strategy for at least 1 database, including any limits used, such that it could be repeated.	Appendix
Selection of sources of evidence†	9	State the process for selecting sources of evidence (i.e., screening and eligibility) included in the scoping review.	4
Data charting process‡	10	Describe the methods of charting data from the included sources of evidence (e.g., calibrated forms or forms that have been tested by the team before their use, and whether data charting was done independently or in duplicate) and any processes for obtaining and confirming data from investigators.	4
Data items	11	List and define all variables for which data were sought and any assumptions and simplifications made.	4
Critical appraisal of individual sources of evidence§	12	If done, provide a rationale for conducting a critical appraisal of included sources of evidence; describe the methods used and how this information was used in any data synthesis (if appropriate).	NA
Synthesis of results	13	Describe the methods of handling and summarizing the data that were charted.	4

Table A.1, PRISMA-ScR Checklist, continued

RESULTS			
Selection of sources of evidence	14	Give numbers of sources of evidence screened, assessed for eligibility, and included in the review, with reasons for exclusions at each stage, ideally using a flow diagram.	4-5
Characteristics of sources of evidence	15	For each source of evidence, present characteristics for which data were charted and provide the citations.	Tables 1 & 2
Critical appraisal within sources of evidence	16	If done, present data on critical appraisal of included sources of evidence (see item 12).	NA
Results of individual sources of evidence	17	For each included source of evidence, present the relevant data that were charted that relate to the review questions and objectives.	Results Section and Tables 1 & 2
Synthesis of results	18	Summarize and/or present the charting results as they relate to the review questions and objectives.	Results
DISCUSSION			
Summary of evidence	19	Summarize the main results (including an overview of concepts, themes, and types of evidence available), link to the review questions and objectives, and consider the relevance to key groups.	Discussion Section
Limitations	20	Discuss the limitations of the scoping review process.	19
Conclusions	21	Provide a general interpretation of the results with respect to the review questions and objectives, as well as potential implications and/or next steps.	19
FUNDING			
Funding	22	Describe sources of funding for the included sources of evidence, as well as sources of funding for the scoping review. Describe the role of the funders of the scoping review.	20

JBI = Joanna Briggs Institute; PRISMA-ScR = Preferred Reporting Items for Systematic reviews and Meta-Analyses extension for Scoping Reviews.

* Where *sources of evidence* (see second footnote) are compiled from, such as bibliographic databases, social media platforms, and Web sites.

† A more inclusive/heterogeneous term used to account for the different types of evidence or data sources (e.g., quantitative and/or qualitative research, expert opinion, and policy documents) that may be eligible in a scoping review as opposed to only studies. This is not to be confused with *information sources* (see first footnote).

‡ The frameworks by Arksey and O'Malley (6) and Levac and colleagues (7) and the JBI guidance (4, 5) refer to the process of data extraction in a scoping review as data charting.

§ The process of systematically examining research evidence to assess its validity, results, and relevance before using it to inform a decision. This term is used for items 12 and 19 instead of "risk of bias" (which is more applicable to systematic reviews of interventions) to include and acknowledge the various sources of evidence that may be used in a scoping review (e.g., quantitative and/or qualitative research, expert opinion, and policy document).

Appendix B: Aim #2, Part 1 Supplemental Materials

Sensitivity Analysis: Unimputed Data

As a sensitivity analysis, we ran the primary 4 models using the unimputed data. Individuals with missing data were removed from the analysis. Weights were applied as in the primary analysis. The results of this sensitivity analysis are presented in **Table B.1**. The impact of income on all 4 transitions of interest were largely the same, though the confidence intervals for the cigarette transitions are notably wider. These results provide indication that the imputation improved study precision without introducing detrimental bias to the study.

Table B.1 Sensitivity Analyses with Unimputed Data. *Results of modified Poisson regression, using unimputed data, examining the relationship between Wave 2 income and Wave 2 alternate tobacco product use on 1) cigarette initiation at Wave 3 among the cigarette-naïve at Wave 2; 2) cigarette cessation at Wave 3 among those who currently used cigarettes at Wave 2; 3) e-cigarette initiation at Wave 3 among the e-cigarette-naïve at Wave 2; and, 4) e-cigarette cessation at Wave 3 among those who currently used e-cigarettes at Wave 2.*

Variable		Cigarette Initiation	Cigarette Cessation	E-Cigarette Initiation	E-Cigarette Cessation
		aRR* (95% CI)	aRR (95% CI)	aRR (95% CI)	aRR (95% CI)
Wave 2 Annual Income					
	<$10k	9.28 (4.28-20.12)	0.52 (0.31-0.86)	1.12 (0.92-1.38)	1.18 (0.36-3.88)
	$10-25k	5.13 (2.75-9.75)	0.53 (0.40-0.71)	0.93 (0.83-1.04)	1.22 (0.45-3.36)
	$25-50k	3.45 (1.87-6.39)	0.78 (0.53-1.16)	0.92 (0.83-1.04)	1.22 (0.41-3.63)
	$50-100k	2.16 (1.29-3.61)	1.01 (0.73-1.39)	0.68 (0.62-0.75)	1.13 (0.42-3.06)
	>$100k	Ref	Ref	Ref	Ref
Wave 2 Cigarette Use Status					
	Never Regular Cigarette Use	--	--	Ref	Ref
	Current Cigarette Use	--	--	12.22 (11.20-13.34)	1.03 (0.49-2.15)
	Former Regular Cigarette Use	--	--	3.01 (2.62-3.45)	0.44 (0.08-2.48)
Wave 2 E-Cigarette Use Status					
	Never E-Cigarette Use	Ref	Ref	--	--
	Current E-Cigarette Use	7.25 (4.56-11.53)	1.27 (0.97-1.67)	--	--
	Former E-Cigarette Use	7.30 (5.79-9.20)	1.19 (0.90-1.57)	--	--

*All regression control for Wave 2 Age, Gender, Race, and Hispanic Ethnicity. Further, coefficients and confidence intervals were computed by pooling the results of the regressions run on each of the 15 imputed datasets via Rubin's rules.

aRR: adjusted Relative Risk; 95% CI: 95% Confidence Interval

Imputation

Data was imputed by a two-step process. First, missing former tobacco product use and income data were imputed heuristically, where possible. For income, that if we know a participant's income at Wave X, then this value is the best guess for their missing income value at Wave Y. For former cigarette/e-cigarette use, many values could be imputed by looking at current and former use status at prior waves. While our study uses a 3-category (never, current, former) use variable, the PATH study has separate variables for current (yes/no) and former (yes/no) use. These former use variables were subject to greater missingness. Given information

about cigarette use at prior waves, we determined a set of rules for imputing former use status at Wave X (either Wave 2 or 3):

1. If an individual reports former smoking at Wave X - 1 and no current smoking at Wave X, then they are labeled as former smoking at Wave X;
2. If an individual reports no former smoking at Wave X - 1 and no current smoking at Wave X, then they are labeled as no former smoking at Wave X;
3. If an individual reports current smoking at Wave X, then they are labeled as no former smoking at Wave X (as they are current).

After these imputations were completed, we then used the ***mice()*** function in the ***mice*** (Multivariate Imputation by Chained Equations) package in R. We assumed missing-at-random missingness and used the default function options to impute data. In total, we created 15 distinct imputed datasets. As discussed in the primary manuscript, the analyses were run separately on each of the 15 imputed datasets and, then, the results were pooled to get our final estimates.

Imputation Diagnostics

It is important that we reflect upon patterns in the missing data to determine if our imputation process has been conducted appropriately. First, we note that because the PATH Study asked questions about tobacco product use last (outcome and primary predictor data), that at worst the missing data is missing at random. In **Table B.2**, we display the proportion of missing data for each key variable for the unweighted sample. All missing data is due to not responding to questions (i.e., no loss to follow-up to worry about because these individuals have already been removed from the data).

Table B.2 Proportion of each variable missing from the unweighted dataset.

Variable	% Missing
Age	3.05%
Gender	0.10%
Race	2.48%
Hispanic Ethnicity	1.46%
Wave 1 Income	8.66%
Wave 2 Income	6.82%
Wave 2 Current Cigarette Use	0.13%
Wave 2 Former Cigarette Use	3.13%
Wave 3 Current Cigarette Use	0.09%
Wave 2 Current E-Cigarette Use	0.54%
Wave 2 Former E-Cigarette Use	8.38%
Wave 3 Current E-Cigarette Use	0.21%

Next, we want to ensure that our imputed data are plausible. Essentially, it is important to ensure that the imputed values are logically reasonable. This is particularly important when there are continuous variables. For example, it would be concerning if our algorithm imputed that someone was 323 years old. However, all the variables we are imputing are categorical.

Therefore, it is good to check if the imputed values are appropriate categories. In **Figure B.1**, we display the distribution of imputed values for Wave 2 Income (5 Categories) and both Wave 2 Current Smoker Status (2 Categories) and Wave 2 Former Cigarette Use Status (2 Categories). In this plot, imputation 1 represents the unimputed data (in blue) and each additional column represents each additional imputation. As we can see, the imputed values for each of these three variables falls on one of the appropriate categories. As such, it appears the imputation appropriately assigned values to plausible values.

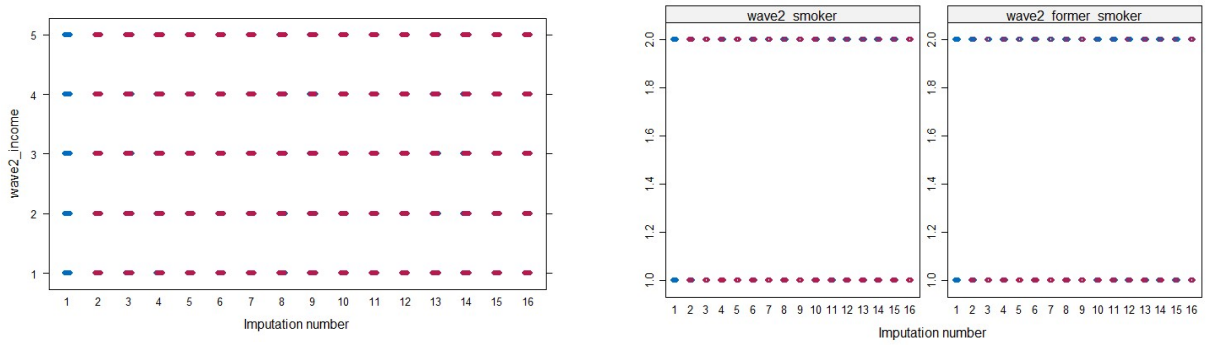

Figure B.1 Distributions of select imputed values *for Wave 2 Income, Current Smoking Status, and Former Smoking Status. Distributions indicate that imputed values represent each of the categorical levels of each variable displayed, indicating that the imputation appropriately imputed values across the domain of each variable.*

While the values of the imputation are plausible, we want to also examine if the distribution of the variables is impacted of the imputations. In **Figure B.2** we display the density plot for imputed current cigarette smoking values (2 categories) at Wave 2. In the first box (in blue), we see the distribution of observed current smoking at Wave 2. This distribution indicates that more individuals reported not being current smokers than being current smokers at Wave 2. Each of the other boxes shows the distribution of imputed current smoking at Wave 2. While distributions do vary, across all 15 imputations, the distribution of imputed current smoking at Wave 2 is similar to the initial distribution of the observed values.

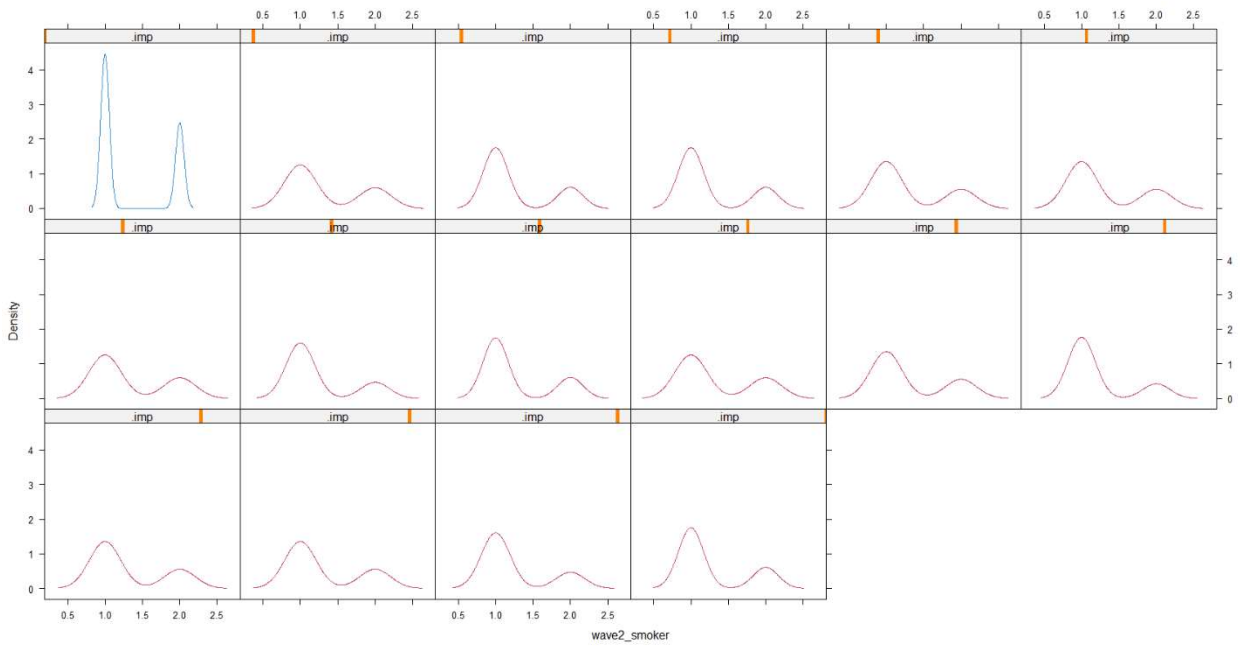

Figure B.2. Distributions of imputed values for Wave 2 Current Smoking. *Plot shows the distribution of the observed values (in the first square in blue) and the distribution of imputed values across each of the 15 imputations. We see that, like the observed data, a greater proportion of the imputed values are for the not current smoking value (= 1). This indicates appropriate behavior by our imputation.*

Finally, we want to reflect on if the imputations converged over the 5 iterations of each imputation. Ideally, the imputation will behave probabilistically (displaying no convergence trends) versus deterministically (i.e., approaching a steady state or displaying trends). In **Figure B.3**, we display convergence plots for the current smoker, former smoker, and income variables at Wave 2. The crossing of lines and lack of convergence indicates that the imputation behaved acceptably. We note a slight upward trajectory of the income values, however, the trajectories do not appear to be uniform across imputation and, as such, appear adequate for study purposes.

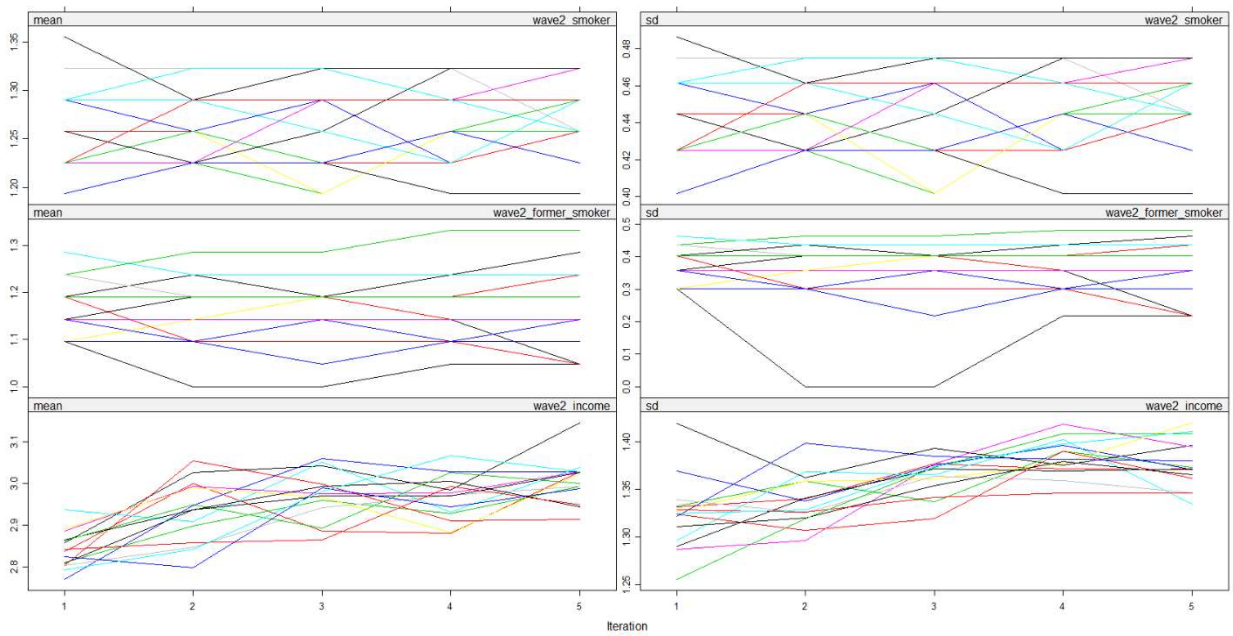

Figure B.3 Convergence plots for Wave 2 Income level, Current Smoking Status, and Former Smoking Status. *Each line represents the mean (left boxes) and standard deviations (right boxes) of each of the 15 imputations over the 5 iterations of the imputation process. For both the Current and Former Smoking variables, we see no trends of convergence, indicating that an appropriate imputation. Income, as well, does not display convergence, though we do note a slight upward trend, though we do not feel this indicates a poor imputational fit.*

Appendix C: Aim #2, Part 2 Supplemental Materials

Sensitivity Analyses

One limitation of the modeling approach we have employed is that we do not dynamically model cigarette and e-cigarette use among youth entering the model (i.e., 17-year-olds transitioning into adulthood). As such, we have included two sensitivity analyses to determine if changing the distribution of e-cigarette and cigarette use among model entries impacts our assessment of the contribution of income on cigarette use. First, our baseline model uses data from Monitoring the Future's (MTF) 2018 survey. They estimated that 7.6% of 12th graders used cigarettes in the past month and that 20.9% used e-cigarettes (Miech et al.). We used these values to generate our distributions for model entries. However, estimates from 2017 and 2019 MTF display drastically different distributions. In 2017, MTF estimated that 9.7% of 12th graders used cigarettes in the prior month and 11.0% used e-cigarettes (Miech et al.). In 2019, MTF estimated that 5.5% of 12th graders used cigarettes in the prior month and 25.5% used e-cigarettes (Johnston et al.). To determine if the distribution of cigarettes and e-cigarettes among model entries impacted our results, we re-ran our analyses using the 2017 and 2019 distributions and compared the results to the primary results. As seen in **Table C.1**, varying the distribution of cigarette use and e-cigarette on model entries had minimal impact on our primary finding.

Table C.1 Sensitivity Analyses*. Comparing the overall population attributable fraction of the contribution of income to current cigarette use at 1, 5, and 10 years. Results of the sensitivity analyses indicate that varying the model entries distribution of e-cigarette and cigarette use result in minimal variation in the primary findings of this study.*

	Baseline Model	2017 MTF Sensitivity Analysis	2019 MTF Sensitivity Analysis
1-Year PAF	12.3% (5.7 – 19.7)	12.1% (5.8 – 19.7)	12.2% (5.8 – 19.8)
5-Year PAF	25.2% (13.5 – 37.1)	25.2% (13.6 – 37.1)	25.4% (13.9 – 37.3)
10-Year PAF	32.8% (19.8 – 46.5)	32.7% (19.5 – 46.4)	33.5% (20.5 – 47.0)

Parameter Tables

Sampled model parameters of relative risks for modifying cigarette and e-cigarette transitions are in included in **Tables C.2 – C.7.** We note that while the same regression strategies were employed in both **Chapter 4 & 5**, the relative risks used in both chapters are different because the models in **Chapter 4** controlled for both race and Hispanic ethnicity, whereas the **Chapter 5** models did not (because race and ethnicity were not factored into the model design). Further, income change was not incorporated into **Chapter 4** models that focused on e-cigarette use transitions, for reasons described in **Chapter 4**. The five calibrated parameters are presented in **Table C.8**.

Table C.2 Cigarette Initiation Relative Risks

Cigarette Initiation				
Variable	**Level**	**Sampled Distribution Mean (95% I)**	**Distribution**	**Source**
Age	18-24	1 (1 – 1)	Log-normal	PATH Study
	24-35	1.60 (1.28 – 1.95)	Log-normal	PATH Study
	35-44	1.55 (0.90 – 2.44)	Log-normal	PATH Study
	45-54	1.56 (1.32 – 1.84)	Log-normal	PATH Study
	55-64	1.76 (1.51 – 2.03)	Log-normal	PATH Study
	65+	1.14 (0.75 – 1.62)	Log-normal	PATH Study
Sex	Male	1 (1 – 1)	Log-normal	PATH Study
	Female	0.55 (0.46 – 0.64)	Log-normal	PATH Study
E-Cigarette Use	Never	1 (1 – 1)	Log-normal	PATH Study
	Current	16.72 (11.67 – 23.48)	Log-normal	PATH Study
	Former	6.24 (5.18 – 7.45)	Log-normal	PATH Study
Annual Income	<$10k	1 (1 – 1)	Log-normal	PATH Study
	$10-25k	0.47 (0.37 – 0.59)	Log-normal	PATH Study
	$25-50k	0.27 (0.22 – 0.34)	Log-normal	PATH Study
	$50-100k	0.2 (0.12 – 0.31)	Log-normal	PATH Study
	>$100k	0.06 (0.04 – 0.09)	Log-normal	PATH Study
Income Change from Previous Year	Stayed the Same	1 (1 – 1)	Log-normal	PATH Study
	Decreased	0.84 (0.71 – 0.98)	Log-normal	PATH Study
	Increased	1.96 (1.63 – 2.33)	Log-normal	PATH Study

Table C.3 Cigarette Cessation Relative Risks

Cigarette Cessation				
Variable	**Level**	**Sampled Distribution Mean (95% I)**	**Distribution**	**Source**
Age	18-24	1 (1 – 1)	Log-normal	PATH Study
	24-35	0.73 (0.63 – 0.83)	Log-normal	PATH Study
	35-44	0.46 (0.39 – 0.53)	Log-normal	PATH Study
	45-54	0.42 (0.32 – 0.51)	Log-normal	PATH Study
	55-64	0.50 (0.37 – 0.66)	Log-normal	PATH Study
	65+	0.59 (0.42 – 0.77)	Log-normal	PATH Study
Sex	Male	1 (1 – 1)	Log-normal	PATH Study
	Female	0.91 (0.81 – 1.02)	Log-normal	PATH Study
E-Cigarette Use	Never	1 (1 – 1)	Log-normal	PATH Study
	Current	0.96 (0.80 – 1.14)	Log-normal	PATH Study
	Former	0.87 (0.74 – 1.03)	Log-normal	PATH Study
Annual Income	<$10k	1 (1 – 1)	Log-normal	PATH Study
	$10-25k	1.12 (0.92 – 1.35)	Log-normal	PATH Study
	$25-50k	1.57 (1.21 – 2.03)	Log-normal	PATH Study
	$50-100k	2.13 (1.78 – 2.49)	Log-normal	PATH Study
	>$100k	2.22 (1.71 – 2.80)	Log-normal	PATH Study
Income Change from Previous Year	Stayed the Same	1 (1 – 1)	Log-normal	PATH Study
	Decreased	1.11 (0.89 – 1.36)	Log-normal	PATH Study
	Increased	0.76 (0.64 – 0.89)	Log-normal	PATH Study

Table C.4 Cigarette Reinstatement Relative Risks

Cigarette Reinstatement				
Variable	**Level**	**Sampled Distribution Mean (95% I)**	**Distribution**	**Source**
Age	18-24	1 (1 – 1)	Log-normal	PATH Study
	24-35	0.79 (0.63 – 0.99)	Log-normal	PATH Study
	35-44	0.68 (0.47 – 0.94)	Log-normal	PATH Study
	45-54	0.63 (0.41 – 0.94)	Log-normal	PATH Study
	55-64	0.50 (0.36 – 0.28)	Log-normal	PATH Study
	65+	0.34 (0.16 – 0.57)	Log-normal	PATH Study
Sex	Male	1 (1 – 1)	Log-normal	PATH Study
	Female	1.14 (0.85 – 1.49)	Log-normal	PATH Study
E-Cigarette Use	Never	1 (1 – 1)	Log-normal	PATH Study
	Current	6.60 (3.55 – 10.76)	Log-normal	PATH Study
	Former	4.75 (2.83 – 7.50)	Log-normal	PATH Study
Annual Income	<$10k	1 (1 – 1)	Log-normal	PATH Study
	$10-25k	1.02 (0.77 – 1.32)	Log-normal	PATH Study
	$25-50k	0.67 (0.44 – 0.97)	Log-normal	PATH Study
	$50-100k	0.52 (0.37 – 0.70)	Log-normal	PATH Study
	>$100k	0.37 (0.23 – 0.56)	Log-normal	PATH Study
Income Change from Previous Year	Stayed the Same	1 (1 – 1)	Log-normal	PATH Study
	Decreased	1.07 (0.86 – 1.33)	Log-normal	PATH Study
	Increased	1.61 (0.91 – 2.50)	Log-normal	PATH Study

Table C.5 E-Cigarette Initiation Relative Risks

E-Cigarette Initiation				
Variable	**Level**	**Sampled Distribution Mean (95% I)**	**Distribution**	**Source**
Age	18-24	1 (1 – 1)	Log-normal	PATH Study
	24-35	0.44 (0.34 – 0.56)	Log-normal	PATH Study
	35-44	0.28 (0.24 – 0.31)	Log-normal	PATH Study
	45-54	0.19 (0.17 – 0.21)	Log-normal	PATH Study
	55-64	0.15 (0.13 – 0.17)	Log-normal	PATH Study
	65+	0.07 (0.06 – 0.09)	Log-normal	PATH Study
Sex	Male	1 (1 – 1)	Log-normal	PATH Study
	Female	0.68 (0.59 – 0.77)	Log-normal	PATH Study
Cigarette Use	Never	1 (1 – 1)	Log-normal	PATH Study
	Current	9.89 (8.14 – 11.70)	Log-normal	PATH Study
	Former	1.45 (1.12 – 1.85)	Log-normal	PATH Study
Annual Income	<$10k	1 (1 – 1)	Log-normal	PATH Study
	$10-25k	0.64 (0.51 – 0.79)	Log-normal	PATH Study
	$25-50k	0.68 (0.51 – 0.88)	Log-normal	PATH Study
	$50-100k	0.40 (0.30 – 0.51)	Log-normal	PATH Study
	>$100k	0.50 (0.37 – 0.65)	Log-normal	PATH Study
Income Change from Previous Year	Stayed the Same	1 (1 – 1)	Log-normal	PATH Study
	Decreased	1.00 (0.80 – 1.20)	Log-normal	PATH Study
	Increased	1.94 (1.79 – 2.09)	Log-normal	PATH Study

Table C.6 E-Cigarette Cessation Relative Risks

E-Cigarette Cessation				
Variable	**Level**	**Sampled Distribution Mean (95% I)**	**Distribution**	**Source**
Age	18-24	1 (1 – 1)	Log-normal	PATH Study
	24-35	0.99 (0.90 – 1.10)	Log-normal	PATH Study
	35-44	1.05 (0.90 – 1.22)	Log-normal	PATH Study
	45-54	1.06 (0.94 – 1.19)	Log-normal	PATH Study
	55-64	0.96 (0.77 – 1.18)	Log-normal	PATH Study
	65+	1.06 (0.82 – 1.35)	Log-normal	PATH Study
Sex	Male	1 (1 – 1)	Log-normal	PATH Study
	Female	0.99 (0.88 – 1.09)	Log-normal	PATH Study
Cigarette Use	Never	1 (1 – 1)	Log-normal	PATH Study
	Current	0.91 (0.83 – 0.99)	Log-normal	PATH Study
	Former	0.44 (0.30 – 0.60)	Log-normal	PATH Study
Annual Income	<$10k	1 (1 – 1)	Log-normal	PATH Study
	$10-25k	1.00 (0.91 – 1.09)	Log-normal	PATH Study
	$25-50k	1.00 (0.87 – 1.14)	Log-normal	PATH Study
	$50-100k	0.99 (0.87 – 1.13)	Log-normal	PATH Study
	>$100k	0.83 (0.59 – 1.13)	Log-normal	PATH Study
Income Change from Previous Year	Stayed the Same	1 (1 – 1)	Log-normal	PATH Study
	Decreased	0.97 (0.88 – 1.07)	Log-normal	PATH Study
	Increased	1.02 (0.91 – 1.15)	Log-normal	PATH Study

Table C.7 E-Cigarette Reinstatement Relative Risks

E-Cigarette Reinstatement				
Variable	**Level**	**Sampled Distribution Mean (95% I)**	**Distribution**	**Source**
Age	18-24	1 (1 – 1)	Log-normal	PATH Study
	24-35	0.71 (0.59 – 0.86)	Log-normal	PATH Study
	35-44	0.63 (0.50 – 0.79)	Log-normal	PATH Study
	45-54	0.62 (0.52 – 0.73)	Log-normal	PATH Study
	55-64	0.56 (0.44 – 0.70)	Log-normal	PATH Study
	65+	0.51 (0.24 – 0.93)	Log-normal	PATH Study
Sex	Male	1 (1 – 1)	Log-normal	PATH Study
	Female	1.07 (0.96 – 1.20)	Log-normal	PATH Study
Cigarette Use	Never	1 (1 – 1)	Log-normal	PATH Study
	Current	1.89 (1.55 – 2.26)	Log-normal	PATH Study
	Former	0.99 (0.78 – 1.24)	Log-normal	PATH Study
Annual Income	<$10k	1 (1 – 1)	Log-normal	PATH Study
	$10-25k	1.25 (0.92 – 1.64)	Log-normal	PATH Study
	$25-50k	1.26 (1.01 – 1.54)	Log-normal	PATH Study
	$50-100k	1.17 (0.93 – 1.45)	Log-normal	PATH Study
	>$100k	1.20 (0.89 – 1.55)	Log-normal	PATH Study
Income Change from Previous Year	Stayed the Same	1 (1 – 1)	Log-normal	PATH Study
	Decreased	0.93 (0.74 – 1.14)	Log-normal	PATH Study
	Increased	1.06 (0.91 – 1.23)	Log-normal	PATH Study

Table C.8 Calibrated Parameters

Calibrated Parameters			
Parameter	**Calibrated Distribution Mean (95% CI)**	**Calibrated To**	**Source**
Population Growth Rate	13.11 (13.11 – 13.11)*	Annual Population Growth from 2014-2015 to 2018-2019	World Bank
Population Carrying Capacity Multiplier	1.59 (1.59 – 1.59)*	Annual Population Growth from 2014-2015 to 2018-2019	World Bank
Cigarette Initiation Scalar	0.64 (0.50 – 0.77)	Annual Current Cigarette Use Prevalence from 2014-2019	NHIS
Cigarette Cessation Scalar	1.66 (1.25 – 2.20)	Annual Ever Cigarette Use Prevalence from 2014 – 2019	NHIS
E-Cigarette Initiation Scalar	0.87 (0.55 – 1.24)	Annual Current E-Cigarette Use Prevalence from 2014 - 2019	NHIS

*Static value for calibration parameters chosen because values did not vary between across model simulations

Imputation, Weighting Procedures, and Statistical Models

The data imputation process for **Chapter 5** is identical to that for **Chapter 4** which is outlined in **Appendix B**. Similarly, **Chapter 5** uses the same weighting procedure as in **Chapter 4.** Similarly, the statistical models for cigarette and e-cigarette initiation and cessation fit to parameterize the model in **Chapter 5** are the same as in **Chapter 4** with an important difference: the statistical models in **Chapter 5** only include the variables corresponding to model strata (i.e., age, sex, cigarette use, e-cigarette use, and income and income change) whereas the models fit in **Chapter 4** also control for race and Hispanic ethnicity. **Chapter 5** includes two additional models to capture the relationship between age, sex, income level, income change, and alternate tobacco product on cigarette/e-cigarette reinstatement (i.e., former use returning to current use).

References

Johnston, Lloyd, et al. *Monitoring the Future National Survey Results on Drug Use, 1975-2019: Overview, Key Findings on Adolescent Drug Use*. 2020.

Miech, Richard, et al. *Monitoring the Future National Survey Results on Drug Use, 1975-2018: Volume I, Secondary School Students*. 2019.

Appendix D: Aim #3 Supplemental Materials

Applying the Potential Outcomes Framework to Mediation

Traditional approaches to mediation (a la Baron and Kenny) (Baron and Kenny) are not appropriate when either the mediator or outcome are categorical (VanderWeele, "Mediation Analysis: A Practitioner's Guide"), and as such we must look to other mediation assessment strategies. Traditional mediation approaches focus on calculating three metrics: the total effect of the exposure on the outcome (i.e., the effect of ACE exposure on COPD/CHD, not accounting for cigarette use); the direct effect of the exposure on the outcome (i.e., the effect of ACE exposure on COPD/CHD not through the mediated pathway of cigarette use); and the indirect effect of the exposure on the outcome (i.e., the effect of ACE exposure on COPD/CHD through the mediated pathway of cigarette use). In traditional applications, the total effect is the sum of the direct and indirect effects (i.e., TE = DE + IE); we can calculate the proportion mediated (i.e., IE/TE) to articulate what percentage of the effect of the exposure is mediated through the mediator. Given that we cannot apply such traditional approaches, it is important to discuss the outcomes we are able to calculate via the potential outcomes approach. The potential outcomes framework leverages counterfactual data to assess causal relationships between variables of interest. A potential outcome is the expected outcome for a participant given a potential treatment. To calculate the mediating role of cigarette use on the relationship between ACE exposure and COPD/CHD, we compare the potential outcome (COPD/CHD) for each participant based on potential treatment exposures (<100 lifetime cigarettes/100+ lifetime cigarettes).

To estimate the total, direct and indirect effects, four assumptions must be considered (VanderWeele, "Mediation Analysis: A Practitioner's Guide"). Calculating the total effect requires only (Assumption 1) that confounding of the exposure-outcome (i.e., ACE-COPD/CHD) relationship be controlled for. We may then calculate the controlled direct effect if (Assumption 2) confounding of the mediator-outcome (i.e., cigarette use-COPD/CHD) relationship is controlled for. The controlled direct effect can be understood as the impact of the exposure (i.e., ACE exposure) on the outcome (i.e., COPD/CHD) at each specific mediator level (i.e., we must calculate a distinct controlled direct effect for both the <100 lifetime cigarettes and 100+ lifetime cigarettes mediator levels). However, to calculate the natural direct and indirect effects (which are interpreted similarly to the results of traditional mediation modeling) two additional assumptions must be satisfied: (Assumption 3) confounding of the exposure-mediator relationship must be controlled for; and (Assumption 4) there cannot be a confounder of the mediator-outcome relationship which is influenced by the exposure. In **Figure 6.1**, we present a model for the relationships between ACEs, cigarette use, COPD/CHD, measured confounders, and measured intermediate confounders. The intermediate confounders in our model violate Assumption 4; therefore, we are unable to calculate the natural direct and indirect effects of ACE exposure on COPD/CHD as mediated through the cigarette use.

Given the design of our study and nature of the relationship between variables of interest, we are thus able to use the potential outcomes approach to identify 1) the total effect of ACE exposure on each outcome and 2) the controlled direct effect of ACE exposure – at each level of the mediator (lifetime cigarette smoking) – on each outcome. From these measures, we are then able to calculate a third metric of interest, the proportion eliminated. The proportion eliminated represents the proportion of the impact of the exposure on the outcome that would be eliminated

if the mediator were to be fixed at a given value (it is the difference between the total effect and the controlled direct effect at a given mediator level divided by the total effect – (TE-CDE[m]/TE) (VanderWeele, "Policy-Relevant Proportions for Direct Effects"). We note that, because we are unable to calculate the indirect effect, we cannot ascertain the proportion mediated metric typically presented in mediation analyses. The proportion eliminated is a useful metric for understanding the potential impact of successfully achieving a policy goal (VanderWeele, "Policy-Relevant Proportions for Direct Effects"). In this study, we calculate the proportion of the effect of ACE exposure on COPD/CHD that is eliminated if counterfactual tobacco control measures had successfully prevented the study sample from smoking 100+ lifetime cigarettes. This is intended to present a best-case scenario for the impact of tobacco control measures on the relationship between ACE exposure and both COPD and CHD.

Inverse Probability Weights

The denominator of a given participant's exposure weight was derived by regressing ACE exposure on age, sex, race, and ethnicity using ordinal logistic regression and outputting the resulting conditional predicted probability of a given participant's observed ACE exposure; the numerator was derived from a null ordinal logistic regression. The denominator of each participant's mediator weight was derived by regressing lifetime cigarette use on age, sex, race, ethnicity, ACE exposure, income, education, and marital status using binary logistic regression; the corresponding numerator was similarly derived, after removing the terms for the confounders or intermediate confounders from the model.

Age-Cut Off & Sensitivity Analyses

Due to the rarity of smoking-related outcomes in early adulthood, we restricted our primary analysis to individuals 40 years of age and older. However, we want to make sure that this assumption is valid and that restricting the dataset did not greatly bias the results. Among individuals 40 years and older (our primary study sample), 9.4% (n = 4,203) had ever been diagnosed with COPD and 11.1% (n = 4,918) had ever been diagnosed with CHD. Whereas, among the sample of individuals under 40 years of age (who were excluded for the primary analysis), 2.5% (n = 297) had ever been diagnosed with COPD and 0.9% (n = 111) had ever been diagnosed with CHD. Given the far lower prevalence of both outcomes for those under 40 years of age, we argue that our sample selection is justified.

To confirm that this choice did not meaningfully bias the results, we re-ran the analysis with the full sample (i.e., with no age restrictions) (see **Table D.1**). Given that introducing the younger sample means introducing a set of individuals less likely to have developed the outcomes of interest (even if they have elevated ACE exposure or smoke cigarettes), we expect the introduction of these individuals to attenuate the point estimates extracted. This is exactly the pattern we observe. We note, as well, that the direction of confidence intervals remains positive (except for the CHD CDE for 100+ cigarettes whose lower bound is -0.001) – indicating that the direction of the effects found remain unchanged. Further, we highlight that the proportion eliminated for each outcome and ACE exposure level remains nearly the same as the primary analysis – this indicates that the mediation estimates for our primary analysis are not biased by sub-setting the final sample to be only adults aged 40 and above.

Table D.1 Sensitivity Analysis with No Age Cut-Off. *Sensitivity Analysis replicating the primary mediation analysis from the manuscript – including participants aged under 40. The Total and Controlled Direct Effects of Adverse Childhood Experiences (ACEs) on Ever Being Diagnosed with COPD or Coronary Heart Disease among participants in the Behavioral Risk Factor Surveillance System, 2011 to 2017.*

		CDE (95% CI)			PE
Outcome	ACE Exposure	<100 Cigarettes	100+ Cigarettes	TE (95% CI)	<100 Cigarettes
COPD	1 ACE	0.008 (0.003-0.014)	0.012 (0.002-0.032)	0.017 (0.012-0.022)	52.9%
	2 ACEs	0.014 (0.007-0.021)	0.030 (0.017-0.042)	0.031 (0.024-0.039)	54.8%
	3 ACEs	0.016 (0.007-0.025)	0.053 (0.038-0.068)	0.049 (0.040-0.059)	67.3%
	4+ ACEs	0.050 (0.040-0.059)	0.093 (0.080-0.106)	0.094 (0.85-0.103)	46.8%
CHD	1 ACE	0.009 (0.002-0.016)	0.009 (-0.001-0.019)	0.013 (0.007-0.018)	30.7%
	2 ACEs	0.025 (0.015-0.035)	0.015 (0.003-0.028)	0.026 (0.017-0.034)	3.8%
	3 ACEs	0.016 (0.004-0.027)	0.017 (0.003-0.031)	0.026 (0.016-0.036)	38.4%
	4+ ACEs	0.037 (0.026-0.048)	0.020 (0.008-0.031)	0.036 (0.028-0.045)	-2.8%

References

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