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The Impact of Retail Availability on Health Behaviors: Policy Applications for the Prevention
& Management of Chronic Conditions

by

Aryn Z. Phillips

A dissertation submitted in partial satisfaction of the

requirements for the degree of

Doctor of Philosophy

in

Health Policy

in the

Graduate Division

of the

University of California, Berkeley

Committee in charge:

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Spring 2020

Abstract

The Impact of Retail Availability on Health Behaviors: Policy Applications for the Prevention & Management of Chronic Conditions

by

Aryn Z. Phillips

Doctor of Philosophy in Health Policy

University of California, Berkeley

Professor Hector Rodriguez, Chair

Chronic conditions contribute to vast sums of excess morbidity, mortality, and healthcare expenditures. Many leading risk factors for chronic conditions are related to behavior, including poor nutrition, alcohol misuse, and tobacco use. Literature from psychology, neuroscience, and behavioral economics suggests that aspects of the environment can encourage unhealthy behaviors. This dissertation uses natural experiments and new combinations of administrative data to explore the role of retail availability in the consumption of unhealthy foods, alcohol, and tobacco and health outcomes and service utilization for chronic conditions. The first paper assesses whether or not adults with diabetes residing in “food swamps” have higher rates of hospitalizations for complications. The second paper focuses on the privatization of liquor sales that occurred in Washington in 2012, investigating if the increase in liquor availability that followed privatization impacted hospitalizations for acute and chronic alcohol-related disorders and accidental injuries. The third paper analyzes the impact of CVS Health’s 2014 tobacco-free pharmacy policy on cigarette smoking among current smokers. Findings from these papers provide additional insight into how governmental and organizational policies may be used to better prevent and manage chronic conditions.

Table of Contents

Acknowledgements	ii
Curriculum Vitae	iii
Introduction.....	1
References	5
Chapter 1: U.S. County "Food Swamp" Severity and Hospitalization Rates among Adults with Diabetes: A Nonlinear Relationship	8
1.1 Introduction	9
1.2 Methods	10
1.3 Results	13
1.4 Discussion.....	16
1.5 Conclusion	18
1.6 References	19
1.7 Appendix	26
Chapter 2: Washington’s Liquor License System and Alcohol-Related Adverse Health Outcomes	29
2.1 Introduction	30
2.2 Methods	32
2.3 Results	35
2.4 Discussion.....	42
2.5 Conclusion	44
2.6 References	45
2.7 Appendix	50
Chapter 3: The Impact of CVS Health’s Tobacco-free Pharmacy Policy on Tobacco Use Among Current Smokers	55
3.1 Introduction	56
3.2 Methods	59
3.3 Results	65
3.4 Discussion.....	70
3.5 Conclusion	71
3.6 References	72
3.7 Appendix	80
Conclusion	82

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Introduction

Background

Preventing and managing chronic conditions have become major areas of focus in the recent era of health care payment and delivery system reform. Defined broadly as any condition that lasts for one or more years and either requires ongoing medical attention or limits one's daily activities, chronic conditions are the leading contributors to death and disability throughout the world.^{1,2} Within the United States, rates of chronic conditions have been steadily increasing and are projected to continue to increase in coming years with the aging of the population.³ Recent estimates suggest that, as of 2014, 60 percent of all American adults had at least one chronic condition and 40 percent had more than one.⁴ In 2016, chronic conditions made up seven of the ten leading causes of death, which accounted for 74 percent of all deaths in that year.⁵ It is estimated that almost 90 percent of health care expenditures in the U.S. are incurred by patients with chronic conditions.^{4,6}

Among the most prevalent chronic conditions are cardiovascular disease, diabetes, chronic lung diseases, cancer, and stroke, and leading risk factors for these diseases include poor nutrition, tobacco use, and excessive alcohol use.¹ Governments and health systems around the world have recognized the growing need to address these behavioral risk factors. However, to date, most of their efforts have involved providing information to individuals, often through mass media campaigns or individual counseling for at-risk patients. The purpose of such efforts is to educate individuals about the risks they face and how to improve their behaviors, with the intention of changing behavior by encouraging goal setting or improving self-efficacy.

However, these information-driven approaches are often ineffective at producing sustained behavior change, even if they are effective at changing intentions.⁷⁻⁹ This lack of impact is likely due to fact that these approaches are based on the assumption that people make fully rational choices about eating, alcohol use, and tobacco use, and that a simple lack of information is behind our penchants to make unhealthy choices.¹⁰ However, the fields of psychology, neuroscience, and behavioral economics have all suggested that these behaviors are often beyond the scope of rational decision making, and that we engage in them automatically and without conscious effort or awareness, rather than because we have fully deliberated on their nature and consequences.^{11,12} This idea stems from dual process theory, which claims that the human brain thinks and makes decisions via two separate processing systems.¹³⁻¹⁷ "System 1" relies on intuition and impulses and is quick and automatic. It operates without voluntary control, often triggered by environmental cues. In contrast, "System 2" is aware of our personal goals and makes reasoned deliberate choices, but it is slower and requires effortful calculations. System 2 regulates and overrules the quick impulsive thoughts of System 1, but this regulation requires effort and is impossible to do for each of the countless decisions that we make throughout the day, especially considering that humans are boundedly rational and have limited ability to attend to, process, and remember information.¹⁷⁻¹⁹ As a result, System 1 often triumphs over System 2 in decision making, especially in our smaller decisions such as food choice or whether or not to purchase another package of cigarettes.¹⁷ In the language of dual process theory, public health efforts that attempt to change behavior by delivering information often fail because they target System 2 but do not address System 1, and today's retail environment is one that overwhelmingly triggers System 1 to engage in unhealthy behaviors.

While many aspects of the environment can trigger System 1, one particular environmental cue known to do so is availability, which, according to dual process theories, can influence unhealthy behaviors in a variety of ways. First, extended availability diminishes the likelihood of resisting temptation. Desire for a product can be prompted by a visual cue.²⁰⁻²² When a product is more available, it is more visually salient, meaning it is more frequently encountered, and System 1 is more frequently triggered to feel desire. The more often System 1 is triggered, the higher the odds that it will eventually dominate over System 2 in a decision. This is especially true considering we have a limited amount of willpower and exerting self-control to resist temptation over multiple occasions draws on this resource, ultimately depleting it and making us less able to resist these temptations when they are offered again.^{23,24}

Additionally, the availability of products can trigger habitual behaviors. Habits are automatic behaviors that occur consistently in certain contexts because they are activated by the context itself.²⁵ Habitual actions do not require any goals or intentions for us to perform them; in fact, even when counter-intentions exist, they are often not translated into behavior when we have developed habits around that behavior.⁸ When unhealthy products that we have formed habitual behaviors around are more readily available (i.e. cigarettes are on display behind the counter at the pharmacy in which we have stopped to purchase ibuprofen), we are more frequently exposed to our habits' context cues and, thus, are likely to more frequently activate and engage in these behaviors.

Further, increased availability influences consumption because it makes products more accessible. We are simply unable to make unhealthy decisions when unhealthy products are not available to access and consume. This inability particularly impacts consumption for people with severely present-biased preferences, which occur when we place greater weight on present concerns than on future concerns, prioritizing immediate rewards and gratifications rather than those that are delayed.^{26,27} It has been suggested that limited availability can serve as a commitment device for those with severe present-biased preferences; people may plan to abstain from unhealthy products in the future so they do not purchase them, but when the future becomes the present and they want to consume them, they simply cannot do so if these products are not accessible.²⁸

Shy of this extreme, making products marginally more or less accessible impacts how convenient they are to obtain. Convenience can play a large role in behavior because it decreases the costs in time and effort that must be spent to obtain a product.²⁹ Many studies have suggested that we are less likely to purchase and consume products when doing so requires the additional time and effort of going out of our way than when the product is more readily obtainable or in close proximity.³⁰⁻³³

Finally, availability can influence social norms around the consumption of unhealthy products. When products are more available and more visually salient, they may contribute to a prevalent social norm that the consumption of these products is normal and perhaps even more common than is true.³⁴⁻³⁶ People may choose to engage in behaviors that they know to be unhealthy because they perceive these behaviors to be normatively sanctioned. Conversely, when products are less commonly seen, it may lead to a norm that the behaviors associated with them are not as socially acceptable. For instance, laws that banned smoking in public were meant to

protect passerby from second-hand smoke but have been instrumental in creating a social norm that smoking in general is less acceptable.³⁷

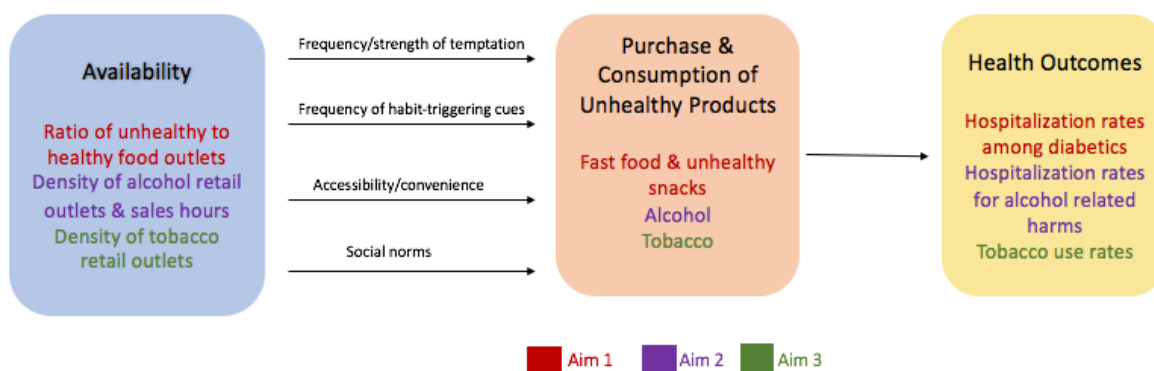
Conceptual Model & Aims

Based on the theoretical and empirical literature discussed in the introduction, this dissertation will focus on the role of retail availability in driving the health behaviors most associated with the exacerbation of chronic conditions. Specifically, it will ask the question of how the availability of unhealthy foods, alcohol, and tobacco impacts their purchase, consumption, and related health outcomes and service utilization, and by, extension, whether or not policies can influence these behaviors by targeting the availability of these products rather than by imparting information.

The dissertation will take the form of three papers, each using a distinct natural experiment or other unique combinations of observational data to analyze the impact of the retail availability of a product on its consumption or health outcomes. These papers include:

1. An analysis of the relationship between the relative rate of unhealthy food outlets to healthy food outlets in a county and hospitalization rates among adults with diabetes (Aim 1).
2. An analysis of changes in county-level rates of hospitalizations for alcohol-related disorders and for accidental injuries following the abolition of the liquor control system in Washington in 2012 (Aim 2).
3. An analysis of the impact of CVS Health’s 2014 tobacco-free pharmacy policy on cigarette consumption among current smokers (Aim 3).

A theoretical model outlining these aims is presented in the figure below.



Importance & Policy Relevance

Given the current trends in the prevalence of chronic conditions, it is becoming increasingly important to understand the roles that environmental factors, including availability, can play in health behaviors so that we may leverage this knowledge to create effective policies, especially considering our previous over-reliance on ineffectual information-based approaches. Several models used in public health, including the ecological model and the health impact pyramid, emphasize the importance of targeting the environment and the context in which we make decisions when designing interventions.^{38,39} The health impact pyramid, for instance, claims that interventions that change the context in order to make default decisions healthier have some of the highest population impacts, second only to efforts that alter socioeconomic factors.³⁹ However, environment and behavior are both very complex and more research is needed to understand the intricacies and detailed mechanisms of their relationship if we are to craft policies that will truly be effective in changing consumer behavior. Many states and localities around the U.S. are experimentally creating policies that alter the environment but that are not evidence-based. While these policies may turn out to be effective, they may also backfire, cause confusion, or simply create legislative chaos. With more research, we may be able to avoid such problems.

It is also useful to understand how the environment can influence behavior around unhealthy foods, alcohol and tobacco in particular. While these products vary widely in addictive properties, social stigma, and other characteristics, they are sometimes compared to one another and regulated in similar ways. For instance, fast food has been called the “new tobacco,” and it has been suggested that lessons learned from regulating the tobacco industry be applied to the food industry. The term “commercial determinants of health” was first used in 2016 to describe the practices used by private companies to promote the consumption of unhealthy products broadly, and this literature stresses that the mechanisms behind unhealthy eating, alcohol use, and tobacco use can be similar but that the study of each is often siloed.⁴⁰ It calls for more boundary-spanning work, as cross-industry comparisons may help us better understand these mechanisms and create effective policies around these products. This dissertation adds to this nascent literature.

Further, this work contributes to the field of natural experiment methodology. This constantly developing field is rife with debates over best practices, and within these papers I test the equivalence of model results using multiple methods and specifications currently under debate. With the mounting availability of big data, natural experiments are likely to become even more prevalent and it is important to continue to innovate and test these methods.

References

1. Centers for Disease Control and Prevention. About Chronic Disease | Chronic Disease Prevention and Health Promotion | CDC. National Center for Chronic Disease Prevention and Health Promotion. <https://www.cdc.gov/chronicdisease/about/index.htm>. Published August 30, 2018. Accessed September 12, 2018.
2. World Health Organization. NCDs | Noncommunicable diseases and their risk factors. World Health Organization. <http://www.who.int/ncds/en/>. Accessed September 12, 2018.
3. Wu S-Y, Green A. Projection of chronic illness prevalence and cost inflation. *Santa Monica, CA: RAND Health*. 2000;18.
4. Buttorff C, Ruder T, Bauman M. Multiple chronic conditions in the United States. *Santa Monica (CA): RAND Corporation*. 2017.
5. Kochanek KD. Mortality in the United States, 2016. 2017;(293):8.
6. Bauer UE, Briss PA, Goodman RA, Bowman BA. Prevention of chronic disease in the 21st century: elimination of the leading preventable causes of premature death and disability in the USA. *The Lancet*. 2014;384(9937):45–52.
7. Randolph W, Viswanath K. Lessons learned from public health mass media campaigns: marketing health in a crowded media world. *Annu Rev Public Health*. 2004;25:419–437.
8. Webb TL, Sheeran P. Does changing behavioral intentions engender behavior change? A meta-analysis of the experimental evidence. *Psychological bulletin*. 2006;132(2):249.
9. Marteau TM, French DP, Griffin SJ, et al. Effects of communicating DNA-based disease risk estimates on risk-reducing behaviours. *Cochrane Database of Systematic Reviews*. 2010;(10).
10. Cawley J. An economic framework for understanding physical activity and eating behaviors. *American journal of preventive medicine*. 2004;27(3):117–125.
11. Marteau TM, Hollands GJ, Fletcher PC. Changing human behavior to prevent disease: the importance of targeting automatic processes. *science*. 2012;337(6101):1492–1495.
12. Cohen DA, Babey SH. Contextual influences on eating behaviours: heuristic processing and dietary choices. *Obesity Reviews*. 2012;13(9):766–779.
13. Shiffrin RM, Schneider W. Controlled and automatic human information processing: II. Perceptual learning, automatic attending and a general theory. *Psychological review*. 1977;84(2):127.
14. Epstein S. Integration of the cognitive and the psychodynamic unconscious. *American psychologist*. 1994;49(8):709.

15. Sloman SA. The empirical case for two systems of reasoning. *Psychological bulletin*. 1996;119(1):3.
16. Metcalfe J, Mischel W. A hot/cool-system analysis of delay of gratification: dynamics of willpower. *Psychological review*. 1999;106(1):3.
17. Kahneman D, Egan P. *Thinking, Fast and Slow*. Vol 1. Farrar, Straus and Giroux New York; 2011.
18. Kahneman D. Maps of bounded rationality: Psychology for behavioral economics. *American economic review*. 2003;93(5):1449–1475.
19. Simon HA. A behavioral model of rational choice. *The quarterly journal of economics*. 1955;69(1):99–118.
20. Laibson D. A cue-theory of consumption. *The Quarterly Journal of Economics*. 2001;116(1):81–119.
21. Milosavljevic M, Navalpakkam V, Koch C, Rangel A. Relative visual saliency differences induce sizable bias in consumer choice. *Journal of Consumer Psychology*. 2012;22(1):67–74.
22. Armel KC, Beaumel A, Rangel A. Biasing simple choices by manipulating relative visual attention. *Judgment and Decision making*. 2008;3(5):396–403.
23. Hagger MS, Wood C, Stiff C, Chatzisarantis NL. Ego depletion and the strength model of self-control: a meta-analysis. *Psychological bulletin*. 2010;136(4):495.
24. Baumeister RF, Vohs KD, Tice DM. The strength model of self-control. *Current directions in psychological science*. 2007;16(6):351–355.
25. Wood W, Neal DT. A new look at habits and the habit-goal interface. *Psychological review*. 2007;114(4):843.
26. Hoch SJ, Loewenstein GF. Time-inconsistent preferences and consumer self-control. *Journal of consumer research*. 1991;17(4):492–507.
27. O'Donoghue T, Rabin M. Doing it now or later. *American Economic Review*. 1999;89(1):103–124.
28. Ben-David I, Bos M. *Impulsive Consumption and Financial Wellbeing: Evidence from an Increase in the Availability of Alcohol*. National Bureau of Economic Research; 2017.
29. Berry LL, Seiders K, Grewal D. Understanding service convenience. *Journal of marketing*. 2002;66(3):1–17.
30. Meiselman HL, Hedderley D, Staddon SL, Pierson BJ, Symonds CR. Effect of effort on meal selection and meal acceptability in a student cafeteria. *Appetite*. 1994;23(1):43–55.

31. Meyers AW, Stunkard AJ. Food accessibility and food choice: A test of Schachter's externality hypothesis. *Archives of General Psychiatry*. 1980;37(10):1133–1135.
32. Engell D, Kramer M, Malafi T, Salomon M, Leshner L. Effects of effort and social modeling on drinking in humans. *Appetite*. 1996;26(2):129–138.
33. Van Kleef E, Otten K, van Trijp HC. Healthy snacks at the checkout counter: A lab and field study on the impact of shelf arrangement and assortment structure on consumer choices. *BMC public health*. 2012;12(1):1072.
34. Alesci NL, Forster JL, Blaine T. Smoking visibility, perceived acceptability, and frequency in various locations among youth and adults☆. *Preventive medicine*. 2003;36(3):272–281.
35. Albers AB, Siegel M, Cheng DM, Biener L, Rigotti NA. Relation between local restaurant smoking regulations and attitudes towards the prevalence and social acceptability of smoking: a study of youths and adults who eat out predominantly at restaurants in their town. *Tobacco control*. 2004;13(4):347–355.
36. Charlesworth A, Glantz SA. Smoking in the movies increases adolescent smoking: a review. *Pediatrics*. 2005;116(6):1516–1528.
37. Roberto CA, Kawachi I. *Behavioral Economics and Public Health*. Oxford University Press; 2015.
38. Richard L, Gauvin L, Raine K. Ecological models revisited: their uses and evolution in health promotion over two decades. *Annual review of public health*. 2011;32:307–326.
39. Frieden TR. A framework for public health action: the health impact pyramid. *American journal of public health*. 2010;100(4):590–595.
40. Kickbusch I, Allen L, Franz C. The commercial determinants of health. *The Lancet Global Health*. 2016;4(12):e895–e896. doi:10.1016/S2214-109X(16)30217-0

Chapter 1:

U.S. County “Food Swamp” Severity and Hospitalization Rates among Adults with Diabetes: A Nonlinear Relationship

Abstract

The relationship between food environments and diabetes morbidity is vastly understudied, despite the well-recognized linkage between dietary quality and diabetes complications. Further, literature demonstrates that attributes of places can have nonlinear relationships with health outcomes. This study examines the extent to which “food swamps” are associated with greater rates of hospitalizations for complications among adults with diabetes over time as well as the linearity of this relationship. A longitudinal county-level analysis of 832 counties across 16 U.S. states in 2010, 2012, and 2014 is conducted using data from the USDA Food Environment Atlas and the AHRQ Health Care Cost and Utilization Project State Inpatient Databases. Food swamp severity is measured as the percentage of food outlets in a county that sell primarily unhealthy foods. Hierarchical linear mixed models with county random intercepts are estimated, controlling for area-level covariates and state and year fixed effects. Curvilinear relationships are explored by additively incorporating quadratic terms. Over the study period, mean food swamp severity remained relatively stable. Mean hospitalization rates decreased from 296.72 to 262.82 hospitalizations per 1,000 diabetic adults ($p < 0.001$). In adjusted models, greater food swamp severity was associated with higher hospitalization rates in a curvilinear manner (severity: $\beta = 2.181$, $p = 0.02$; severity²: $\beta = -0.017$, $p = 0.04$), plateauing at approximately 64% unhealthy outlets, a saturation point observed in 17% of observations. Policies that limit saturation of the environment with unhealthy outlets may help in the prevention of diabetic complications, but more saturated counties will likely require more extensive intervention

1.1 Introduction

Diabetes is one of the most prevalent chronic conditions in the United States; recent estimates suggest that it affects over 30 million or 12 percent of American adults.¹ Individuals with diabetes are at increased risk of developing a variety of serious complications, from acute issues like ketoacidosis to longer term complications such as cardiovascular disease, kidney disease, nerve damage, and problems of the eyes and feet.² Such complications are the source of diabetes-related morbidity and mortality, and they result in high volumes of hospitalizations. In the U.S., it is estimated that 7.2 million hospital discharges were related to diabetes in 2014 and that over 69 billion dollars were spent on diabetes-related inpatient hospitalizations in 2017.^{1,3} Among adults with diabetes, the leading risk factors for developing complications include poor glycemic control, high blood pressure, and high cholesterol.^{1,4-6} These intermediate outcomes of diabetes can be influenced by many factors, such as physical inactivity, stress, and treatment adherence, but they are also greatly affected by unhealthy diet. As a result, individuals with diabetes are advised to adhere to diets that are low in processed carbohydrates, saturated and trans fats, cholesterol, and sodium.^{7,8}

One's ability to adhere to a recommended diet, however, may be prejudiced by contextual influences. A plethora of research on neighborhood characteristics has found that attributes of places may be determinants of health outcomes, independent of the attributes of the individuals who live within these places.^{9,10} With regard to diabetes management and diet, individuals certainly have varying preferences, abilities, and degrees of knowledge, but we are increasingly learning that dietary choices can also be influenced by the surrounding food environment, including the availability of both healthy and unhealthy foods. It is true that previous studies on the relationship between singular aspects of food availability, such as the number of or distance to grocery stores or fast food outlets, and dietary outcomes have yielded mixed results.^{11,12} However, some studies have sought to capture the overall nature of the food environment by focusing on the relative rate of outlets selling mostly unhealthy foods to outlets selling mostly healthy foods, and have more consistently found significant associations with dietary measures such as fruit and vegetable and fast food intake and purchasing¹²⁻¹⁷ and obesity^{11,18-22} in the expected directions. Environments that are considered unhealthy by these relative measures, where outlets selling unhealthy goods predominate over outlets selling healthy goods, have been described as "food swamps".²³ If such environments encourage diets that are disproportionately lower in fruits and vegetables and higher in fast food and processed snacks, they may place adults with diabetes who live and work within them at higher risk of developing complications and exhibit higher complication rates as a result.

Further, it is possible that relationships between the food environment and diet and related outcomes are nonlinear. Previous work on a variety of subjects has shown that neighborhood characteristics, such as the severity of food swamps as well as community socioeconomic status, land use mix, and natural environment availability,²⁴⁻²⁶ can have curvilinear associations with health outcomes. In the food environment context, the addition of a singular healthy or unhealthy outlet may have a dissimilar influence on food choice when more or less of these outlets already exist. For instance, in a relatively healthy food environment, a new fast food outlet would be highly notable, but in an environment overly saturated with unhealthy options, the overall change to the environment would be small and may not shift

behavior. If true, such a pattern would be important to consider when designing intervention strategies.

While the relationship between food environment and diabetes prevalence,^{18,27–33} incidence,^{25,33–36} and glycemic control among diabetic adults has been examined previously,^{37–39} the relationship between food environment and diabetes-related morbidity is almost entirely unstudied. This analysis builds on previous work that examined the association of food swamp severity and hospitalization rates and found that counties with unhealthier food environments have higher all-cause hospitalization rates among adults with diabetes.⁴⁰ However, the study was cross-sectional and did not explore the possibility of a nonlinear relationship between food swamp severity and hospitalization rates. It also used a limited measure of food environment, comprising only fast food outlets and grocery stores. As such, this current study incorporates additional data and aims to assess the extent to which county-level food swamp severity, measured more comprehensively, is associated with higher county-level hospitalization rates among adults with diabetes in the United States over time. Further, it will examine whether this association is constant across all levels of unhealthy outlet saturation. In light of the mechanisms described, the following hypotheses are proposed:

H1: Greater county-level food swamp severity will be associated with higher rates of hospitalizations among adults with diabetes.

H2: The relationship between food swamp severity and diabetic hospitalization rates will be non-linear, stronger in food environments less saturated with unhealthy outlets.

1.2 Methods

Study Sample

Data on the food environment came from the U.S. Department of Agriculture Economic Research Service (USDA ERS) Food Environment Atlas, which provides statistics on a range of food environment indicators for U.S. counties, including counts of outlet types. The USDA classifies outlet types according to North American Industry Classification System (NAICS) codes. The most recent estimates that have been released for the relevant variables are from 2009, 2012, and 2014.⁴¹ Data on the rate of hospitalizations among diabetic adults came from the Agency for Healthcare Research & Quality (AHRQ) Health Care Cost and Utilization Project (HCUP) state inpatient databases and the Centers for Disease Control and Prevention (CDC) for 16 states (AZ, AR, CO, FL, GA, IA, MA, MI, MN, NJ, NM, NY, OR, RI, VT and WA) for years 2010 through 2014. The HCUP state inpatient databases contain the universe of all-payer hospital inpatient records for each participating state.⁴² The Centers for Disease Control and Prevention uses Bayesian multilevel modeling on data from the Behavioral Risk Factor Surveillance System and the U.S. Census Bureau to calculate county and year-specific estimates of diagnosed diabetes.⁴³ Data on relevant county-level covariates were obtained from the U.S. Department of Health and Human Services Area Health Resources Files (AHRF). All data sources were linked using Federal Information Processing System (FIPS) county codes.

The final analytic sample included data for 832 counties across 16 states in years 2010, 2012, and 2014. Counties with populations under 5,000 (n=41) were dropped to ensure large

enough denominators to reliably estimate hospitalization rates,^{44,45} as were four outlier observations from 2010 where hospitalization rates drastically differed from their 2012 and 2014 rates.

Measures

Food swamp severity was assessed using a relative measure that represented the percentage of food outlets in a county that sell primarily unhealthy foods. These outlets included fast food restaurants (NAICS code 722211) and convenience stores (NAICS codes 445120 and 447110). The total outlet count additionally included grocery stores (NAICS code 445110) and full-service restaurants (NAICS code 722110). There are multiple ways of quantifying food swamps, but percentage measures such as this have been utilized by several recent studies.^{20,25,46–48} Using a percentage measure rather than a ratio of unhealthy to healthy outlets allows for the inclusion of counties with zero healthy outlets that would be dropped for having an invalid denominator with a ratio measure.

The main outcome variable was the inpatient hospitalization rate among adult county residents with diabetes. Individuals were linked to their home counties using the FIPS code of residence listed on the hospitalization record. Rates were calculated by dividing the total number of hospital admissions with any-listed diagnosis of Clinical Classification Software code 49 (“diabetes mellitus without complication”) or 50 (“diabetes mellitus with complications”) incurred by county residents over age 20 in each calendar year by the CDC’s estimated number of diagnosed adults with diabetes within the county in that year. Rates were presented as the number of hospitalizations per 1,000 adult county residents with diabetes.

These rates included admissions for all diagnoses among individuals with diabetes, excluding only admissions for pregnancy and patients transferred from other hospitals. All diagnoses were included because poor glycemic control, high blood pressure, and high cholesterol can result in an array of complications among adults with diabetes, many of which may initially seem unrelated to diabetes. For instance, diabetes affects the blood vessels and nerves that control the heart, and acute myocardial infarction and stroke are by far the most frequent reasons for hospitalization among adults with diabetes.^{1,49} These admissions may be missed by stricter coding definitions of diabetes-related complications.⁵⁰ Further, having diabetes can increase the cost and difficulty of treating one’s co-occurring conditions.³ For example, diabetes can impact immune function, resulting in reduced resistance to influenza and pneumonia, and can damage blood vessels in the lungs, causing further exacerbations in individuals with chronic obstructive pulmonary disease.^{6,51} The intent was to capture complications that result from these interplays as well.

Several other variables were used to capture county-level health systems and sociodemographic characteristics relevant to hospitalization rates and food environment. The percentage of diabetic adult hospitalizations admitted through the emergency room, the percentage of hospitalized diabetic adults that were Medicaid beneficiaries, and the mean number of comorbidities per diabetic patient admitted were created using the HCUP analytic sample. The number of primary care physicians per 1,000 residents, median household income, population density (log transformed), and the percentage of the county population that is non-

Hispanic black, Hispanic, female, and over age 65 were sourced from the AHRF. The number of recreational facilities per 1,000 residents was obtained from the USDA Food Environment Atlas.

Statistical analysis

A hierarchical linear mixed regression model was used to estimate the association of food swamp severity and hospitalization rates among adults with diabetes. A mixed model was chosen because these models, which present weighted averages of the between-cluster and within-cluster effects, allow for an analysis of differing variances between and within clusters and can be used to make inferences about entire populations.⁵² In this context, the biennial observations are considered to be clustered within counties, and the model uses the following specification:

$$Y_{it} = \beta_0 + \beta_1 \text{Severity}_{it} + \beta_2 \mathbf{X}_{it} + \beta_3 \text{Year}_t + \beta_4 \text{State}_i + \zeta_i + \epsilon_{it}$$

where Y represents the hospitalization rate among adults with diabetes for a county i at time t , Severity represents the food swamp severity for the county i at time t , and \mathbf{X} represents a vector of the time-varying health systems-related and sociodemographic covariates previously described. The model also includes indicator values for the years 2012 and 2014 (Year) to account for time trends and state (State) to account for further clustering of counties within states, which may have their own policies, programs, etc. that affect hospitalization rates. Finally, ζ_i represents the county-specific random intercept and ϵ_{it} represents the county and time-specific error term. Standard errors were clustered at the county level.

To allow for a curvilinear relationship, polynomial iterations of the food swamp severity variable were tested in an additive manner, ceasing when an iteration was no longer significant at the 0.05 level. Variance inflation factors (VIF) were examined for model covariates to assess collinearity.

Sensitivity analyses

Several sensitivity analyses were performed to examine the robustness of the main results to alternative measurement and modeling decisions. First, the regression model was estimated using a more restrictive definition of diabetes-related complications. This definition comprised only hospitalizations with a principal diagnosis that met the AHRQ Prevention Quality IndicatorTM Version 6.0 specifications for diabetes with short-term complications, diabetes with long-term complications, uncontrolled diabetes, or lower extremity amputation among patients with diabetes.⁵³ The ICD-9 codes for each qualified diagnosis are included in the appendix, and included such diagnoses as ketoacidosis, and renal, ophthalmic and peripheral circulatory manifestations. Diagnoses such as acute myocardial infarction and stroke were not included in this definition. Second, similar food environments can have different impacts on diet and related outcomes at varying levels of financial security.^{54,55} To examine this possibility, an interaction between food swamp severity and financial resources, measured by median household income and, alternatively, by the percent living in poverty, was assessed. Third, to ensure that any association of food swamp severity and hospitalization rates was not driven by changes in diabetes prevalence estimates (the denominator), the model was run using the log transformed count of hospitalizations as the outcome and additionally controlling for the log transformed number of diabetic county residents. Finally, in an attempt to identify endogeneity from outlets

differentially locating in areas for reasons that influence hospitalizations (i.e. demand for unhealthy foods), the change in food swamp severity between 2010 and 2014 was regressed on a variety of baseline county characteristics, including the diabetes prevalence rate, median household income, logged population density, and the percent of the population that is non-Hispanic black, Hispanic, over age 65, and live in urban areas, as well as state indicators.

1.3 Results

The mean food swamp severity remained relatively stable over the study period; it increased by less than one percentage point from 53.63 percent unhealthy outlets to 54.38 percent unhealthy outlets, but this increase was not statistically significant ($p=0.157$). The mean hospitalization rate decreased from 2010 to 2014, from 296.72 hospitalizations to 262.82 hospitalizations per 1,000 adults with diabetes ($p<0.001$) (Table 1.1). Common primary diagnoses among these hospitalizations included atrial fibrillation, subendocardial infarction, septicemia, pneumonia, obstructive chronic bronchitis, kidney failure, and ketoacidosis. Both variables exhibited far more variation between counties than they did between years for each county. The between-county standard deviation for food swamp severity was 10.56 while the between-year standard deviation was only 3.22. The between-county standard deviation for hospitalization rates was 80.80 while the between-year standard deviation was 34.36.

Table 1.1. Health-Systems Related and Sociodemographic Characteristics of Counties, 2010-2014

Variable	2010 Mean (95% CI) n=828	2012 Mean (95% CI) n=832	2014 Mean (95% CI) n=832
Hospitalization rate (per 1,000 diabetic residents)	296.72 (290.48, 302.96)	269.09 (263.17, 275.02)	262.82 (257.35, 268.30)
Food swamp severity	53.63 (52.92, 54.35)	54.14 (53.37, 54.92)	54.38 (53.63, 55.15)
Percentage admitted in ED	53.08 (51.79, 54.38)	45.83 (43.84, 47.82)	50.09 (48.12, 53.07)
Percentage of patients with Medicaid	18.19 (17.44, 18.94)	18.80 (18.10, 19.51)	21.37 (20.73, 22.01)
Mean comorbidity burden	3.60 (3.57, 3.62)	3.74 (3.71, 3.76)	3.89 (3.87, 3.92)
Primary care physicians (per 1,000 residents)	0.59 (0.57, 3.62)	0.59 (0.57, 0.61)	0.60 (0.58, 0.62)
Recreational facilities (per 1,000 residents)	0.09 (0.08, 0.09)	0.08 (0.7, 0.8)	0.08 (0.07, 0.08)
Median household income (in thousands)	44.45 (43.68, 45.22)	45.91 (45.10, 46.73)	48.08 (47.22, 48.94)
Population density (population/square miles)	348.80 (200.28, 497.33)	353.39 (202.48, 504.30)	359.17 (205.94, 512.40)
Percentage of population non-Hispanic Black	9.36 (8.40, 10.31)	9.73 (8.77, 10.69)	9.85 (8.89, 10.81)
Percentage of population Hispanic	8.86 (8.04, 9.67)	9.25 (8.43, 10.06)	9.56 (8.73, 10.38)
Percentage of population female	50.01 (49.85, 50.17)	49.92 (49.76, 50.08)	49.91 (49.74, 50.07)
Percentage of population over age 65	15.86 (15.56, 16.16)	16.80 (16.49, 17.12)	17.76 (17.44, 18.09)

Note. Boldface indicates statistical significance ($p<0.05$) in t-test of means compared to 2010. a denotes estimate from 2009 rather than 2010.

Source. USDA Food Environment Atlas 2009-2014, AHRQ Health Care Cost and Utilization Project (HCUP) state inpatient files 2010-2014, HHS Area Health Resources File (AHRF) 2010-2014.

Results from the multivariate mixed models with varying quadratic terms indicated that food swamp severity had a significant positive and curvilinear association with hospitalization rates among adults with diabetes at the county level (Table 1.2). A squared food swamp severity

term was significant ($\beta=-0.017$, $p=0.038$), indicating that the strength of the association attenuated as food swamp severity increased. In essence, the association was stronger in environments with lower relative rates of unhealthy food outlets, but the magnitude leveled off after a certain point of saturation by unhealthy outlets (Figure 1.1). This point of saturation was approximately 64 percent, a quantity achieved by only 17 percent of the county-year observations. The mean VIF was 1.58; all variables were below 2.45, with most below 2.00. Cubic and higher polynomial food swamp terms, when added, were not significant and were not included in the final model.

Figure 1.1 Predicted hospitalization rates by food swamp severity (with 95% CI)

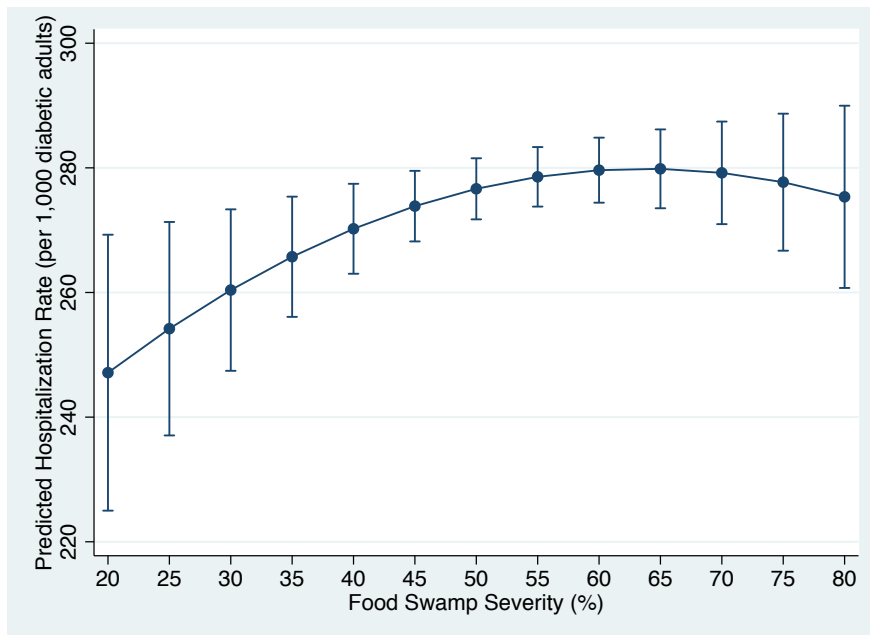


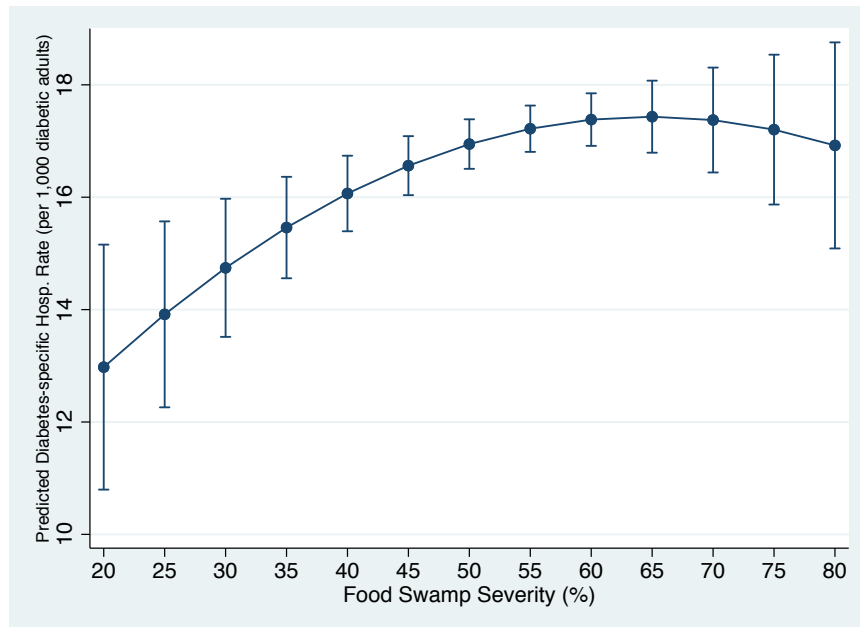
Table 1.2. The Association between County Food Swamp Scores and Hospitalization Rates among Adults with Diabetes, 2010-2014 (n=2,490)

	Coefficient	95% Confidence Interval
Food swamp severity	2.181*	(0.390, 3.972)
Food swamp severity ²	-0.017*	(-0.033, -0.001)
Time trend		
2012	-32.959***	(-37.329, -28.590)
2014	-45.902***	(-52.360, -39.444)
Percentage admitted in ED	0.146	(-0.026, 0.318)
Percentage of patients with Medicaid	0.007	(-0.408, 0.421)
Mean comorbidity burden	53.009***	(39.704, 66.314)
Primary care physicians (per 1,000 residents)	16.843*	(0.786, 32.899)
Recreational facilities (per 1,000 residents)	-33.482	(-94.619, 27.655)
Median household income (in thousands)	-1.673***	(-2.132, -1.214)
Log-transformed population density	8.848***	(4.160, 13.536)
Percentage of population non-Hispanic Black	-0.195	(-0.775, 0.385)
Percentage of population Hispanic	1.502***	(0.913, 2.091)
Percentage of population female	2.996*	(0.480, 5.512)
Percentage of population over age 65	0.802	(-0.450, 2.054)

Note. Table presents estimates from hierarchical linear mixed model with county-level random intercepts and state indicator variables. Standard errors are clustered at county level. Boldface indicates statistical significance (*p<0.05, **p<0.01, ***p<0.001).

The model analyzing hospitalization rates for strictly diabetes-related hospitalizations yielded a consistent pattern of results (food swamp severity: $\beta=0.284$, $p=0.005$; food swamp severity²: $\beta=-0.002$, $p=0.023$). The coefficients were smaller in magnitude, as these particular hospitalizations were relatively rare (baseline mean=17.74 hospitalizations per 1,000 diabetic adults). However, the rate of change across the distribution of food swamp severity was similar to that of the original model, as shown in Figure 1.2. Terms for the interaction of food swamp severity with median household income and with the percent living in poverty were not significant when included in the model. The model decomposing hospitalization rates into hospitalization and prevalence counts was concordant with the original model. No baseline county characteristics were significantly associated with changes in food swamp severity over time, indicating that they were not related to outlet entry and exit.

Figure 1.2. Predicted diabetes-specific hospitalization rates by food swamp severity (with 95% CI)



1.4 Discussion

The results of this study indicate that, in the U.S., food swamp severity is associated with higher rates of hospitalizations for complications among diabetic adults at the county level, even after adjusting for relevant covariates. The results are consistent with previous research that elucidates how the oversaturation of the environment with unhealthy outlets might influence eating behaviors among adults with diabetes. High prevalence of unhealthy foods diminishes the likelihood of resisting temptation to purchase them, as desire for a product can be prompted by visual cues. The more prevalent these products are, the more visually salient they are, meaning desires are more frequently triggered and the odds are higher that we will eventually succumb to them.⁵⁶⁻⁵⁸ This is especially true considering willpower is a limited resource.^{59,60} Further, greater

density of unhealthy outlets makes unhealthy foods more convenient to obtain than healthier foods, and we are more likely to purchase products when doing so does not require additional time and effort than when they are less readily available.⁶¹⁻⁶⁴ In addition, literature on tobacco suggests that when products are more available and visually salient, they may contribute to a social norm that the consumption of these products is ordinary and perhaps even more commonplace than is true.⁶⁵⁻⁶⁷

These results indicate that strategies that limit the oversaturation of counties with unhealthy outlets may help prevent diabetic complications. However, the finding that the relationship plateaus in the most extreme food swamps suggests that more extensive food environment changes may be needed to prevent complications in these counties, perhaps because these areas are so oversaturated with unhealthy outlets that small increases in healthy outlets or decreases in unhealthy outlets do little to impact the overall food environment. Such minor changes in outlet distribution might be imperceptible and thus unlikely to alter the influences that drive food purchasing and consumption decisions. This point of saturation may also help partially explain why studies on the entry of new grocery stores^{55,68-71} or fast food moratoriums⁷² have found null or clinically small results for dietary quality and obesity. These policies are often implemented in the poorest quality food environments in which the introduction of one healthy outlet or the curtailment of further unhealthy outlets could have little impact on the existing degree of saturation. Certainly, these situations are complex and null results could stem from a range of factors (price and quality of foods sold, transportation resources, etc.), but a curvilinear relationship between outlets and outcomes should potentially be explored.

This study is one of the first to assess the relationship between the food environment and diabetes-related morbidity. Previous work similarly identified a positive association, but, as mentioned, was cross-sectional and did not consider nonlinearity.⁴⁰ As such, these findings are consistent yet contribute additional insight into our limited knowledge about this relationship. Given the burden of diabetes, it is important that we understand the diversity of factors that may contribute to complications among diabetic adults, including neighborhood characteristics. Although recent studies have suggested that the rates of complications have decreased in recent years,^{49,73} these rates are still quite high. For instance, in 2010 it was estimated that in the U.S. 45.5 of every 10,000 adults with diabetes was hospitalized for acute myocardial infarction, compared to only 25.8 of every 10,000 adults without diabetes.⁴⁹ Furthermore, with the population aging and diabetes prevalence increasing, more individuals are at risk and the absolute number of complications may continue to rise.⁴⁹ By broadening our understanding of the factors that influence complications, we may be able to recognize and utilize additional avenues to prevent some of these complications.

It might be suggested that counties with higher percentages of unhealthy food outlets are simply the same counties that exhibit other qualities associated with increased rates of hospitalizations among diabetic adults, but this data suggest that food swamps, at least at the county level, represent a wholly separate concept. For instance, food swamp severity is at best moderately correlated with measures of socioeconomic status or deprivation, such as median household income, percent living in poverty, and unemployment rate ($\rho=-0.27, 0.40, 0.16$, respectively) and only weakly correlated with measures of access to preventive care, such as the number of primary care physicians and federally qualified health centers per population and the percent of adults without health insurance ($\rho=-0.28, 0.02, 0.26$, respectively). Thus, if this

analysis' findings can be corroborated, food swamp measures may prove to be additional indicators with which we can identify areas that warrant increased attention and intervention.

This study has important limitations to consider when interpreting the results. First, the observational methods used limit causal inference. However, this study builds upon previous analyses that have used causal methods such as instrumental variables to successfully link food environment with other related outcomes, such as obesity.^{21,74} Highway exits as an instrumental variable, which was used in these studies, was not appropriate for this analysis, as transit is related to health services access and hospital utilization, but these studies bolster confidence in the identified relationship despite the inability to make causal claims. Second, due to data availability, the food swamp severity variable did not include some outlets that may meaningfully contribute to the food environment, such as farmers' markets and specialty stores. While the outlets included likely encompass a sizeable portion of food purchases, future studies may want to consider additional outlet types, when possible. Third, the time period analyzed was chosen based on data availability and may not accurately correspond to the etiologic processes under study. Longer periods in which more change can be observed should be analyzed in the future. Also, due to availability, the observations from 2010 include food environment data from 2009, but this practice of merging data from multiple years has been previously utilized.⁷⁵ As seen in the results, food environment changes vary marginally over short time periods and it is expected that the 2009 estimates are quite close to what would have been observed in 2010. Finally, the unit of analysis is the county level, which forfeits some precision that could have been obtained by using smaller units and masks existing within-county heterogeneity. However, larger units like counties are more likely to capture a greater share of individuals' daily travel routes compared to smaller units. Food shopping often takes place further from home than would be observed with such units. For instance, a Los Angeles-based study found that only approximately 22 percent of those surveyed shopped for groceries within their home census tracts.⁷⁶ However, it remains that county boundaries are arbitrarily drawn and may not truly reflect the space in which people spend their time, risking spatial misclassification. Further, the use of the county unit also makes us unable to incorporate residential selection and other important individual potential confounders. Individual-level analyses would allowed for the consideration of these aspects and exploration the mechanisms behind this association, but unfortunately such analyses were not possible in this study. Hospitalization data does not provide information on adults with diabetes who did not experience hospitalization, rendering no appropriate comparison group at the individual level. Individual-level analyses using alternative outcome data and geographic information systems-based measures should be pursued when such data is available at the national level. However, the aggregate-level conclusions drawn from this study may still be useful for policy discussions because they highlight the challenges faced by communities oversaturated by unhealthy outlets.

1.5 Conclusion

U.S. counties with greater percentages of unhealthy food outlets have higher rates of hospitalizations among adults with diabetes, but this relationship plateaus at a point of extreme saturation by unhealthy outlets. Understanding this food swamp saturation point may provide insight into geographic disparities in diabetes complication rates across the country as well as new ways in which policy makers and practitioners can prevent diabetic complications and the resulting morbidity and mortality.

1.6 References

1. Centers for Disease Control and Prevention. *National Diabetes Statistics Report, 2017*; 2017. <https://www.cdc.gov/diabetes/pdfs/data/statistics/national-diabetes-statistics-report.pdf>. Accessed January 12, 2018.
2. Centers for Disease Control and Prevention. About Chronic Disease | Chronic Disease Prevention and Health Promotion | CDC. National Center for Chronic Disease Prevention and Health Promotion. <https://www.cdc.gov/chronicdisease/about/index.htm>. Published August 30, 2018. Accessed September 12, 2018.
3. American Diabetes Association. Economic Costs of Diabetes in the US in 2017. *Diabetes care*. 2018;41(5):917.
4. Yau JW, Rogers SL, Kawasaki R, et al. Global prevalence and major risk factors of diabetic retinopathy. *Diabetes care*. 2012;DC_111909.
5. Tziomalos K, Athyros VG. Diabetic nephropathy: New risk factors and improvements in diagnosis. *The review of diabetic studies: RDS*. 2015;12(1-2):110.
6. Deshpande AD, Harris-Hayes M, Schootman M. Epidemiology of diabetes and diabetes-related complications. *Physical therapy*. 2008;88(11):1254–1264.
7. Mayo Clinic. Hyperglycemia in diabetes - Symptoms and causes. Mayo Clinic. <http://www.mayoclinic.org/diseases-conditions/hyperglycemia/symptoms-causes/syc-20373631>. Accessed January 12, 2018.
8. Bantle JP, Wylie-Rosett J, Albright AL, et al. Nutrition recommendations and interventions for diabetes: a position statement of the American Diabetes Association. *Diabetes care*. 2008;31 Sup(10.2337/dc08-S061):S61-S78. http://care.diabetesjournals.org/content/31/Supplement_1/S61.full?ijkey=0139447811c08f073edda51cfc6585e745215d6b&keytype2=tf_ipsecsha. Accessed February 26, 2018.
9. Diez Roux AV. Investigating neighborhood and area effects on health. *American journal of public health*. 2001;91(11):1783–1789.
10. Kawachi I, Berkman LF. *Neighborhoods and Health*. Oxford University Press; 2003.
11. Cobb LK, Appel LJ, Franco M, Jones-Smith JC, Nur A, Anderson CA. The relationship of the local food environment with obesity: a systematic review of methods, study quality, and results. *Obesity*. 2015;23(7):1331-1344.
12. Caspi CE, Sorensen G, Subramanian S, Kawachi I. The local food environment and diet: a systematic review. *Health & place*. 2012;18(5):1172-1187.
13. Clary C, Lewis DJ, Flint E, Smith NR, Kestens Y, Cummins S. The local food environment and fruit and vegetable intake: a geographically weighted regression approach in the ORiEL Study. *American journal of epidemiology*. 2016;184(11):837–846.

14. Colón-Ramos U, Monge-Rojas R, Cremm E, Rivera IM, Andrade EL, Edberg MC. How Latina mothers navigate a ‘food swamp’ to feed their children: a photovoice approach. *Public Health Nutrition*. 2017;20(11):1941-1952. doi:10.1017/S1368980017000738
15. Hager ER, Cockerham A, O’Reilly N, et al. Food swamps and food deserts in Baltimore City, MD, USA: associations with dietary behaviours among urban adolescent girls. *Public health nutrition*. 2017;20(14):2598-2607.
16. Mason KE, Bentley RJ, Kavanagh AM. Fruit and vegetable purchasing and the relative density of healthy and unhealthy food stores: evidence from an Australian multilevel study. *J Epidemiol Community Health*. 2013;67(3):231–236.
17. Thornton LE, Kavanagh AM. Association between fast food purchasing and the local food environment. *Nutrition & diabetes*. 2012;2(12):e53.
18. Babey SH, Diamant AL, Hastert TA, Harvey S. Designed for disease: the link between local food environments and obesity and diabetes. 2008.
19. Spence JC, Cutumisu N, Edwards J, Raine KD, Smoyer-Tomic K. Relation between local food environments and obesity among adults. *BMC public health*. 2009;9(1):192.
20. Truong K, Fernandes M, An R, Shier V, Sturm R. Measuring the physical food environment and its relationship with obesity: evidence from California. *Public health*. 2010;124(2):115–118.
21. Cooksey-Stowers K, Schwartz MB, Brownell KD. Food Swamps Predict Obesity Rates Better Than Food Deserts in the United States. *International journal of environmental research and public health*. 2017;14(11):1366.
22. Feng X, Astell-Burt T, Badland H, Mavoa S, Giles-Corti B. Modest ratios of fast food outlets to supermarkets and green grocers are associated with higher body mass index: Longitudinal analysis of a sample of 15,229 Australians aged 45 years and older in the Australian National Liveability Study. *Health & place*. 2018;49:101–110.
23. Rose D, Bodor JN, Swalm CM, Rice JC, Farley TA, Hutchinson PL. Deserts in New Orleans? Illustrations of urban food access and implications for policy. *Ann Arbor, MI: University of Michigan National Poverty Center/USDA Economic Research Service Research*. 2009.
24. Lei L. The impact of community context on children’s health and nutritional status in China. *Social Science & Medicine*. 2017;179:172–181.
25. Mezuk B, Li X, Cederin K, Rice K, Sundquist J, Sundquist K. Beyond access: characteristics of the food environment and risk of diabetes. *American journal of epidemiology*. 2016;183(12):1129-1137.

26. Wu Y-T, Prina AM, Jones A, Matthews FE, Brayne C. The built environment and cognitive disorders: results from the Cognitive Function and Ageing Study II. *American journal of preventive medicine*. 2017;53(1):25–32.
27. Ahern M, Brown C, Dukas S. A national study of the association between food environments and county-level health outcomes. *The Journal of Rural Health*. 2011;27(4):367-379.
28. Salois MJ. Obesity and diabetes, the built environment, and the ‘local’ food economy in the United States, 2007. *Economics & Human Biology*. 2012;10(1):35-42.
29. Frankenfeld CL, Leslie TF, Makara MA. Diabetes, obesity, and recommended fruit and vegetable consumption in relation to food environment sub-types: a cross-sectional analysis of Behavioral Risk Factor Surveillance System, United States Census, and food establishment data. *BMC Public Health*. 2015;15(1):491.
30. Haynes-Maslow L, Leone LA. Examining the relationship between the food environment and adult diabetes prevalence by county economic and racial composition: An ecological study. *BMC public health*. 2017;17(1):648.
31. Lee DC, Gallagher MP, Gopalan A, et al. Identifying Geographic Disparities in Diabetes Prevalence Among Adults and Children Using Emergency Claims Data. *Journal of the Endocrine Society*. 2018;2(5):460–470.
32. Richardson AS, Ghosh-Dastidar M, Beckman R, et al. Can the introduction of a full-service supermarket in a food desert improve residents’ economic status and health? *Annals of epidemiology*. 2017;27(12):771–776.
33. Gebreab SY, Hickson DA, Sims M, et al. Neighborhood social and physical environments and type 2 diabetes mellitus in African Americans: The Jackson Heart Study. *Health & place*. 2017;43:128–137.
34. Auchincloss AH, Roux AVD, Mujahid MS, Shen M, Bertoni AG, Carnethon MR. Neighborhood resources for physical activity and healthy foods and incidence of type 2 diabetes mellitus: the Multi-Ethnic study of Atherosclerosis. *Archives of internal medicine*. 2009;169(18):1698–1704.
35. Christine PJ, Auchincloss AH, Bertoni AG, et al. Longitudinal associations between neighborhood physical and social environments and incident type 2 diabetes mellitus: the Multi-Ethnic Study of Atherosclerosis (MESA). *JAMA internal medicine*. 2015;175(8):1311-1320.
36. Polsky JY, Moineddin R, Glazier RH, Dunn JR, Booth GL. Relative and Absolute Availability of Fast Food Restaurants in Relation to the Development of Diabetes: A Population-Based Cohort Study. *Canadian Journal of Diabetes*. 2016;40(5):S17.

37. Berkowitz SA, Karter AJ, Corbie-Smith G, et al. Food Insecurity, Food “Deserts,” and Glycemic Control in Patients With Diabetes: A Longitudinal Analysis. *Diabetes care*. 2018;dc171981.
38. Tabaei BP, Rundle AG, Wu WY, et al. Associations of Residential Socioeconomic, Food, and Built Environments With Glycemic Control in Persons With Diabetes in New York City From 2007–2013. *American journal of epidemiology*. 2017;187(4):736–745.
39. Zhang YT, Mujahid MS, Laraia BA, et al. Association between neighborhood supermarket presence and glycosylated hemoglobin levels among patients with type 2 diabetes mellitus. *American journal of epidemiology*. 2017;185(12):1297–1303.
40. Phillips AZ, Rodriguez HP. Adults with diabetes residing in “food swamps” have higher hospitalization rates. *Health Services Research*. 0(0). doi:10.1111/1475-6773.13102
41. Economic Research Service (ERS), U.S. Department of Agriculture (USDA). *Food Environment Atlas*. <https://www.ers.usda.gov/data-products/food-environment-atlas/>.
42. Agency for Healthcare Research and Quality. Introduction to the HCUP State Inpatient Databases (SID). 2018. https://www.hcup-us.ahrq.gov/db/state/siddist/Introduction_to_SID.pdf. Accessed August 8, 2018.
43. Centers for Disease Control and Prevention. *Methods and References for County-Level Estimates and Ranks and State-Level Modeled Estimates*.; 2013. <https://www.cdc.gov/diabetes/pdfs/data/calculating-methods-references-county-level-estimates-ranks.pdf>. Accessed August 7, 2018.
44. Chauhan P, Cerdá M, Messner SF, Tracy M, Tardiff K, Galea S. Race/ethnic-specific homicide rates in new york city: Evaluating the impact of broken windows policing and crack cocaine markets. *Homicide studies*. 2011;15(3):268–290.
45. Ahern J, Matthay EC, Goin DE, Farkas K, Rudolph KE. Acute changes in community violence and increases in hospital visits and deaths from stress-responsive diseases. *Epidemiology*. 2018;29(5):684–691.
46. Luan H, Law J, Quick M. Identifying food deserts and swamps based on relative healthy food access: a spatio-temporal Bayesian approach. *International journal of health geographics*. 2015;14(1):37.
47. Mui Y, Gittelsohn J, Jones-Smith JC. Longitudinal associations between change in neighborhood social disorder and change in food swamps in an urban setting. *Journal of Urban Health*. 2017;94(1):75–86.
48. Mui Y, Jones-Smith JC, Thornton RL, Pollack Porter K, Gittelsohn J. Relationships between Vacant Homes and Food Swamps: A Longitudinal Study of an Urban Food Environment. *International journal of environmental research and public health*. 2017;14(11):1426.

49. Gregg EW, Li Y, Wang J, et al. Changes in diabetes-related complications in the United States, 1990–2010. *New England Journal of Medicine*. 2014;370(16):1514–1523.
50. Gibbons DC, Soljak MA, Millett C, Valabhji J, Majeed A. Use of hospital admissions data to quantify the burden of emergency admissions in people with diabetes mellitus. *Diabetic Medicine*. 2014;31(8):971–975.
51. Rogliani P, Lucà G, Lauro D. Chronic obstructive pulmonary disease and diabetes. *COPD research and practice*. 2015;1(1):3.
52. Rabe-Hesketh S, Skrondal A. *Multilevel and Longitudinal Modeling Using Stata*. STATA press; 2008.
53. Agency for Healthcare Research and Quality. Prevention Quality Indicators Technical Specifications. October 2016.
https://www.qualityindicators.ahrq.gov/Modules/PQI_TechSpec_ICD09_v60.aspx.
54. Jones-Smith JC, Karter AJ, Warton EM, et al. Obesity and the food environment: income and ethnicity differences among people with diabetes: the Diabetes Study of Northern California (DISTANCE). *Diabetes Care*. 2013;DC_122190.
55. Allcott H, Diamond R, Dubé J-P, Handbury J, Rahkovsky I, Schnell M. *Food Deserts and the Causes of Nutritional Inequality*. National Bureau of Economic Research; 2017.
doi:10.3386/w24094
56. Armel KC, Beaumel A, Rangel A. Biasing simple choices by manipulating relative visual attention. *Judgment and Decision making*. 2008;3(5):396–403.
57. Laibson D. A cue-theory of consumption. *The Quarterly Journal of Economics*. 2001;116(1):81–119.
58. Milosavljevic M, Navalpakkam V, Koch C, Rangel A. Relative visual saliency differences induce sizable bias in consumer choice. *Journal of Consumer Psychology*. 2012;22(1):67–74.
59. Baumeister RF, Vohs KD, Tice DM. The strength model of self-control. *Current directions in psychological science*. 2007;16(6):351–355.
60. Hagger MS, Wood C, Stiff C, Chatzisarantis NL. Ego depletion and the strength model of self-control: a meta-analysis. *Psychological bulletin*. 2010;136(4):495.
61. Berry LL, Seiders K, Grewal D. Understanding service convenience. *Journal of marketing*. 2002;66(3):1–17.
62. Meiselman HL, Hedderley D, Staddon SL, Pierson BJ, Symonds CR. Effect of effort on meal selection and meal acceptability in a student cafeteria. *Appetite*. 1994;23(1):43–55.

63. Meyers AW, Stunkard AJ. Food accessibility and food choice: A test of Schachter's externality hypothesis. *Archives of General Psychiatry*. 1980;37(10):1133–1135.
64. Thorndike AN, Sonnenberg L, Riis J, Barraclough S, Levy DE. A 2-Phase Labeling and Choice Architecture Intervention to Improve Healthy Food and Beverage Choices. *American Journal of Public Health*. 2012;102(3):527-533. doi:10.2105/AJPH.2011.300391
65. Albers AB, Siegel M, Cheng DM, Biener L, Rigotti NA. Relation between local restaurant smoking regulations and attitudes towards the prevalence and social acceptability of smoking: a study of youths and adults who eat out predominantly at restaurants in their town. *Tobacco control*. 2004;13(4):347–355.
66. Alesci NL, Forster JL, Blaine T. Smoking visibility, perceived acceptability, and frequency in various locations among youth and adults☆. *Preventive medicine*. 2003;36(3):272–281.
67. Charlesworth A, Glantz SA. Smoking in the movies increases adolescent smoking: a review. *Pediatrics*. 2005;116(6):1516–1528.
68. Cummins S, Flint E, Matthews SA. New neighborhood grocery store increased awareness of food access but did not alter dietary habits or obesity. *Health affairs*. 2014;33(2):283–291.
69. Elbel B, Moran A, Dixon LB, et al. Assessment of a government-subsidized supermarket in a high-need area on household food availability and children's dietary intakes. *Public health nutrition*. 2015;18(15):2881–2890.
70. Dubowitz T, Ghosh-Dastidar M, Cohen DA, et al. Diet and perceptions change with supermarket introduction in a food desert, but not because of supermarket use. *Health Affairs*. 2015;34(11):1858–1868.
71. Zhang YT, Laraia BA, Mujahid MS, et al. Is a reduction in distance to nearest supermarket associated with BMI change among type 2 diabetes patients? *Health & place*. 2016;40:15–20.
72. Sturm R, Hattori A. Diet and obesity in Los Angeles County 2007–2012: Is there a measurable effect of the 2008 “Fast-Food Ban”? *Social science & medicine*. 2015;133:205–211.
73. Centers for Disease Control and Prevention. Prevalence of self-reported cardiovascular disease among persons aged ≥ 35 years with diabetes—United States, 1997-2005. *MMWR Morbidity and mortality weekly report*. 2007;56(43):1129.
74. Dunn RA. The effect of fast-food availability on obesity: An analysis by gender, race, and residential location. *American Journal of Agricultural Economics*. 2010;92(4):1149-1164.
75. Rundle A, Neckerman KM, Freeman L, et al. Neighborhood food environment and walkability predict obesity in New York City. *Environmental health perspectives*. 2008;117(3):442–447.

76. Inagami S, Cohen DA, Finch BK, Asch SM. You are where you shop: grocery store locations, weight, and neighborhoods. *American journal of preventive medicine*. 2006;31(1):10–17.

1.7 Appendix

Table A1.1 ICD-9 Codes & Diagnoses Included in AHRQ Prevention Quality Indicators

Prevention Quality Indicator 01: Diabetes with Short-term Complications

ICD-9 Code	Diagnosis	ICD-9 Code	Diagnosis
25010	Diabetes with ketoacidosis, type II	25022	Diabetes with hyperosmolarity, type II, uncontrolled
25011	Diabetes with ketoacidosis, type I	25023	Diabetes with hyperosmolarity, type I, uncontrolled
25012	Diabetes with ketoacidosis, type II, uncontrolled	25030	Diabetes with other coma, type II
25013	Diabetes with ketoacidosis, type I, uncontrolled	25031	Diabetes with other coma, type II
25020	Diabetes with hyperosmolarity, type II	25032	Diabetes with other coma, type II, uncontrolled
25021	Diabetes with hyperosmolarity, type I	25033	Diabetes with other coma, type I, uncontrolled

Prevention Quality Indicator 03: Diabetes with Long-term Complications

ICD-9 Code	Diagnosis	ICD-9 Code	Diagnosis
25040	Diabetes with renal manifestations, type II	25070	Diabetes with peripheral circulatory disorders, type II
25041	Diabetes with renal manifestations, type II	25071	Diabetes with peripheral circulatory disorders, type I
25042	Diabetes with renal manifestations, type II, uncontrolled	25072	Diabetes with peripheral circulatory disorders, type II, uncontrolled
25043	Diabetes with renal manifestations, type I, uncontrolled	25073	Diabetes with peripheral circulatory disorders, type I, uncontrolled
25050	Diabetes with ophthalmic manifestations, type II	25080	Diabetes with other specified manifestations, type II
25051	Diabetes with ophthalmic manifestations, type I	25081	Diabetes with other specified manifestations, type I
25052	Diabetes with ophthalmic manifestations, type II, uncontrolled	25082	Diabetes with other specified manifestations, type II, uncontrolled
25053	Diabetes with ophthalmic manifestations, type I, uncontrolled	25083	Diabetes with other specified manifestations, type I, uncontrolled
25060	Diabetes with neurological manifestations, type II	25090	Diabetes with unspecified complication, type II
25061	Diabetes with neurological manifestations, type I	25091	Diabetes with unspecified complication, type I
25062	Diabetes with neurological manifestations, type II, uncontrolled	25092	Diabetes with unspecified complication, type II, uncontrolled
25063	Diabetes with neurological manifestations, type I, uncontrolled	25093	Diabetes with unspecified complication, type I, uncontrolled

Prevention Quality Indicator 14: Uncontrolled Diabetes

ICD-9 <u>Code</u>	<u>Diagnosis</u>	ICD-9 <u>Code</u>	<u>Diagnosis</u>
25002	Diabetes mellitus without mention of complication, type II, uncontrolled	25003	Diabetes mellitus without mention of complication, type I, uncontrolled

Prevention Quality Indicator 16: Lower Extremity Amputation among Patients with Diabetes

Lower Extremity Amputation

ICD-9 <u>Code</u>	<u>Diagnosis</u>	ICD-9 <u>Code</u>	<u>Diagnosis</u>
8410	Lower limb amputation, not otherwise specified	8416	Disarticulation of knee
8412	Amputation through foot	8417	Amputation above knee
8414	Amputation of ankle through malleoli of tibia and fibula	8418	Disarticulation of Hip
8415	Other amputation below knee	8419	Abdominopelvic amputation

Diabetes

ICD-9 <u>Code</u>	<u>Diagnosis</u>	ICD-9 <u>Code</u>	<u>Diagnosis</u>
25000	Diabetes mellitus without mention of complications, type II	25050	Diabetes with ophthalmic manifestations, type II
25001	Diabetes mellitus without mention of complications, type I	25051	Diabetes with ophthalmic manifestations, type I
25002	Diabetes mellitus without mention of complications, type II, uncontrolled	25052	Diabetes with ophthalmic manifestations, type II, uncontrolled
25003	Diabetes mellitus without mention of complications, type I, uncontrolled	25053	Diabetes with ophthalmic manifestations, type I, uncontrolled
25010	Diabetes with ketoacidosis, type II	25060	Diabetes with neurological manifestations, type II
25011	Diabetes with ketoacidosis, type I	25061	Diabetes with neurological manifestations, type I
25012	Diabetes with ketoacidosis, type II, uncontrolled	25062	Diabetes with neurological manifestations, type II, uncontrolled
25013	Diabetes with ketoacidosis, type I, uncontrolled	25063	Diabetes with neurological manifestations, type I, uncontrolled
25020	Diabetes with hyperosmolarity, type II	25070	Diabetes with peripheral circulatory disorders, type II
25021	Diabetes with hyperosmolarity, type I	25071	Diabetes with peripheral circulatory disorders, type I
25022	Diabetes with hyperosmolarity, type II, uncontrolled	25072	Diabetes with peripheral circulatory disorders, type II, uncontrolled
25023	Diabetes with hyperosmolarity, type I, uncontrolled	25073	Diabetes with peripheral circulatory disorders, type I, uncontrolled
25030	Diabetes with other coma, type II	25080	Diabetes with other specified manifestations, type II
25031	Diabetes with other coma, type I	25081	Diabetes with other specified manifestations, type I
25032	Diabetes with other coma, type II, uncontrolled	25082	Diabetes with other specified manifestations, type II, uncontrolled
25033	Diabetes with other coma, type I, uncontrolled	25083	Diabetes with other specified manifestations, type I, uncontrolled

25040	Diabetes with renal manifestations, type II	25090	Diabetes with unspecified complication, type II
25041	Diabetes with renal manifestations, type II	25091	Diabetes with unspecified complication, type I
25042	Diabetes with renal manifestations, type II, uncontrolled	25092	Diabetes with unspecified complication, type II, uncontrolled
25043	Diabetes with renal manifestations, type I, uncontrolled	25093	Diabetes with unspecified complication, type I, uncontrolled

Source. Agency for Healthcare Research and Quality (AHRQ) Prevention Quality Indicators Technical Specifications, Version 6.0

Chapter 2:

Washington's Liquor License System and Alcohol-Related Adverse Health Outcomes

Abstract

In June 2012, Washington implemented Initiative 1183, which privatized liquor sales. As a result, the number of off-premise outlets increased from 330 outlets to over 1,400 outlets and trading hours lengthened. Increased availability of liquor may lead to increased consumption. This study examines the impact of Initiative 1183 on alcohol-related adverse health outcomes, measured by inpatient hospitalizations for alcohol-related disorders and accidental injuries. It further assesses whether the impact differed by urbanicity, because outlets increased most in urban areas. Data are from the AHRQ Healthcare Cost & Utilization State Inpatient Database 2010-2014. County-level difference-in-differences models were used to compare changes in the rates of hospitalizations following Initiative 1183 in Washington to changes in Oregon.

Washington's Initiative 1183 was associated with a significant increase in the rate of accidental injury hospitalizations in urban areas of Washington that was on average 0.289 hospitalizations per 1,000 county residents per quarter greater than the simultaneous increase observed in urban areas in Oregon ($p=0.017$). This result was robust to specifications using a propensity score matched sample and synthetic control methods. Initiative 1183, however, was not significantly associated with differential changes in the rate of hospitalizations for alcohol-related disorders in counties of urban, suburban, or rural counties. Statewide policies that expand the availability of liquor may increase the number of accidental injuries among the population most exposed, perhaps by encouraging increases in consumption that are incremental but not substantial enough to exacerbate chronic conditions in the short-run.

2.1 Introduction

Background

Alcohol misuse is the third leading cause of preventable death in the United States, resulting in over 88,000 deaths per year.¹ Approximately 44 percent of these deaths are attributable to chronic alcohol-related conditions, and another 36 percent are attributable to accidental injuries.² Alcohol misuse further leads to 2.5 billion years of potential life lost annually, 34 percent of which are attributable to chronic conditions and 40 percent of which are attributable to accidental injuries.² Previous research suggests that alcohol consumption is related to retail availability; observational studies have found that the density of off-premise outlets (establishments that sell liquor to be consumed off the premises) is associated with rates of alcohol-attributable deaths and hospitalizations as well as ambulance attended accidental injuries.³⁻⁸

Some jurisdictions have regulated off-premise density with alcohol control systems, in which the government maintains a monopoly over alcohol retail, in contrast to license systems, in which the government licenses private vendors to sell alcohol. Control systems can be applied to all alcohol or only specific types, but they are most commonly applied to liquor. Currently eleven states operate under some type of retail liquor control system.

In November 2011, the voters of Washington approved Initiative 1183 to abolish the state's liquor control system. Prior to this date, beer and wine had been available for purchase in private licensed stores but liquor was only available in government owned or contracted stores. Initiative 1183 privatized liquor sales, allowing any store larger than 10,000 square feet, including supermarkets, drug stores, supercenters, and large alcohol specialty stores, to sell liquor for the first time. It additionally abolished the three-tier system requiring the separation of the production, wholesale, and retail sectors and, in an effort to keep the state's revenue unchanged, significantly raised taxes on liquor.⁹ Overall, it was the most comprehensive statewide change in alcohol policy since the repeal of prohibition in 1933.

The initiative went into effect in June 2012 and drastically increased the availability of liquor. Since this date, the number of off-premise liquor outlets has increased from about 330 outlets to over 1,400 outlets.^{10,11} Many of these outlets also offer later operating hours than the government stores previously had offered; by law, government stores closed at 9:00 pm on weekdays, 10:00 pm on Fridays and Saturdays, and 5:00 pm on Sundays.¹² Under Initiative 1183, stores are only prohibited from selling alcohol between 2:00 am and 6:00 am.

Initiative 1183 represents a unique opportunity to analyze the relationship between liquor availability and health outcomes in a natural policy experiment. The only other instances of retail liquor privatization in the U.S. took place in Iowa and West Virginia, but these policy changes occurred over thirty years ago and studies were inconclusive with regard to their impact on health outcomes. Of the three existing analyses, all focused on Iowa and none included a control group, which means results rely on strong assumptions about post-policy temporal patterns had the policy not occurred. Two studies examined changes in alcohol sales and yielded conflicting results.^{13,14} The third assessed the impact of privatization on changes in self-reported heavy and problem drinking, with liver cirrhosis as a secondary outcome, but a co-occurring wine

privatization policy made it impossible to disentangle the effect of liquor privatization from that of wine privatization.¹⁵ Much of the existing observational work on the correlations between existing outlet density and alcohol related outcomes has been cross-sectional or has focused on jurisdictions outside of United States.¹⁶⁻¹⁸

Previous research examining the impact of Initiative 1183 focused on public opinion^{19,20} and purchasing and price changes.^{12,21} However, some of this work suggests the potential for impacts on health outcomes. An evaluation conducted by the Washington State Office of Financial Management found that sales in off-premise outlets increased significantly after Initiative 1183's implementation, controlling for on-premise outlet (i.e. bars, restaurants) sales, average prices of alcohol, and population change.¹⁰ Moreover, an analysis of Nielsen Homescan data observed that the addition of outlets after implementation was associated with increases in liquor expenditures, the volume of liquor purchased, and the volume of total ethanol purchased, and does not find evidence that consumers are simply substituting liquor for wine or beer.²² Another study used survey data to assess changes in self-reported drinking habits after privatization and found that while reported total liquor consumption decreased, reported mean quantity of liquor consumed per day of use increased.²³ These findings suggest that riskier drinking patterns may have resulted from liquor privatization even when there was no overall increase in consumption.

Hypotheses & Objective

There are a variety of mechanisms through which a sudden increase in the availability of liquor could theoretically increase consumption. Encountering liquor in grocery stores and drug stores where one has stopped to purchase other items can result in more frequent temptation, depleted willpower, and triggering of habits by context cues. Additionally, the greater density of off-premise outlets and their extended operating hours reduces the time, effort, and opportunity costs of acquiring liquor. Finally, the colocation of liquor in retail outlets with everyday products may foster a norm that it is more socially acceptable than when it could only be obtained in specialty stores, which may have made it seem more elicited. This may especially be true considering it is now sold in such stores as pharmacies and Whole Foods markets, which otherwise sell largely health-promoting goods.

If citizens of Washington are consuming more liquor and, thus, more alcohol overall, they are likely to experience adverse health consequences that result in hospitalizations. Binge drinking, the act of consuming four or more drinks in a single occasion for women and five or more for men, as well as heavy drinking, defined as consuming eight or more drinks per week for women and fifteen or more for men, increase one's risk of acute health conditions, such as alcohol poisoning, and chronic conditions, such as cirrhosis or fatty liver disease.¹³ Research from other countries has found that the density of off-premise outlets is associated with rates of alcohol-attributable deaths and hospitalizations,¹⁴⁻¹⁶ even for those chronic conditions that can require years to manifest.^{16,17}

H1: The implementation of Initiative 1183 will be associated with an increase in the rate of hospitalizations for alcohol-related disorders, both acute and chronic.

If greater availability is encouraging Washington residents to consume more alcohol overall, more liquor at the expense of beer and wine, or even simply more liquor per drinking day, it is likely to result in a greater number of accidental injuries. Consuming alcohol can lead to intoxication, which involves impairment of balance, movement, reaction time, and judgement.¹⁸ All of these impairments can increase the risk of unintentional injuries, such as falls, drownings, and motor vehicle crashes. Previous literature from a variety of settings has confirmed that alcohol consumption is associated with higher risk of injury.¹⁹⁻²¹ There is likely an exponential dose response between consumption and risk,²² but risk of injury is substantially higher with even small amounts of consumption. It is estimated that injury risk doubles at just one drink (odds ratio = 2.3-2.7),²³ and that risk is higher at a blood alcohol concentration of 0.05 for just one hour.²⁴ Studies have found that, in comparison to beer and wine, liquor can raise blood alcohol concentration to a higher level and more quickly,²⁵⁻²⁷ which suggests that more injuries would be likely if residents of Washington began preferentially purchasing liquor even if not purchasing more alcohol overall.

H2: The implementation of Initiative 1183 will be associated with an increase in the rate of hospitalizations for accidental injuries.

Initiative 1183 certainly did not increase liquor availability uniformly across the state; it was particularly concentrated in urban areas. If rural areas experienced only minor changes in outlet density as a result of 1183, outlets may still be few and far apart and liquor may only be marginally more available. If so, the effect of Initiative 1183 may be weaker in rural areas. However, due to both contextual and compositional effects of areas, alcohol usage patterns and alcohol use disorder rates differ in urban, suburban, and rural contexts.³⁰ This heterogeneity can be obscured when aggregating counties of all types. For instance, a nationwide study found that the prevalence of drinking in excess of recommended weekly limits was similar among urban and rural residents, but significantly lower among suburban residents.³¹ Such differences could moderate the effect of Initiative 1183 on adverse health outcomes.

H3: The association of Initiative 1183 with the rate of hospitalizations for alcohol-related disorders and accidental injuries will differ in magnitude in urban, suburban, and rural areas.

As such, this paper assesses the influence of Initiative 1183 on inpatient hospitalizations for alcohol-related disorders and for accidental injuries, outcomes that do not rely on self-report or recall. Further, it explores whether the effect of Initiative 1183 varied in urban, suburban, and rural contexts.

2.2 Methods

Data

Hospitalization data are from the AHRQ Healthcare Cost and Utilization Project (HCUP) State Inpatient Databases 2010-2014, which is an encounter level database that contains all discharge records for community hospital inpatient stays, regardless of payer, within participating states.²⁶ County-level contextual data from the HHS Area Health Resource File (AHRF) 2010-2014 and data on off-premise outlets obtained from the Washington State Liquor and Cannabis Board were integrated with the HCUP data.

Comparison Group

The state of Oregon was used as a comparison group for this analysis. Washington and Oregon are demographically similar (see Table 2.2), and Oregon currently operates under a liquor control system, similar to Washington’s prior to Initiative 1183.

Measures

The analysis was conducted at the county level, with one observation per county per quarter in the calendar year. The outcomes were the rates of hospitalizations for alcohol-related disorders and for accidental injuries. Hospitalized patients were matched to their counties of residence using the Federal Information Processing System (FIPS) county code on the hospitalization record, and hospitalizations of interest were summed for each county and quarter. Hospitalizations for alcohol-related disorders were defined as inpatient records with any listed ICD-9 Clinical Classification Software code of 660, which denotes “alcohol-related disorders.” Diagnoses that fall under this classification include alcohol-induced mental disorders as well as both chronic and acute physical health conditions. Hospitalizations for accidental injuries were defined according to the recommended framework put forth by the Centers for Disease Control and Prevention, which classifies injuries according to the intent and mechanism by which they occurred.²⁷ According to this framework, relevant hospitalizations include records with any listed External Cause of Injury Code (“Ecode”) of E800-E869 and E880-E929. This range of codes denotes injuries that occurred by accident and includes a broad scope of mechanisms, including falls, fires, cuts, and motor vehicle accidents. A full list of diagnoses included under each classification is included in the appendix. Hospitalization rates were expressed as the number of hospitalizations per 1,000 county residents. Quarterly population estimates were obtained by interpolation from yearly estimates.

Statistical Analysis

The main analysis specified a difference-in-difference model, comparing the changes in the rate of hospitalizations for each alcohol-related adverse health outcome following the implementation of Initiative 1183 in Washington to changes in the rate of such hospitalizations in Oregon over the same time period.

The analysis utilized the following model:

$$Y_{it} = \beta_0 + \beta_1 \text{Post}_t + \beta_2 (\text{Post}_t \times \text{WA}_i) + \beta_3 \mathbf{X}_{it} + \epsilon_{it}$$

In which Y represents the rate of hospitalizations (for alcohol-related disorders or accidental injuries) in county i at time t, Post represents an indicator for time t being after Initiative 1183 was implemented (1) or before (0), and PostxWA, the parameter of interest, represents the interaction of Post and an indicator variable for county i being within Washington (1) or Oregon (0). The model additionally included a vector of fixed effects X, which contains fixed effects for year (to account for time trends), quarter (to account for seasonality in alcohol consumption), county (to account for time-invariant county attributes that may influence hospitalizations) and a beer tax that was implemented and expired in Washington during this period. The inclusion of the county fixed effect eliminated the original WA indicator variable

from the model due to collinearity. ϵ_{it} represents the county and time-specific error term. Errors were clustered at the county level to account for non-independence.

After analyzing counties of all types together in a single model, the analyses were stratified by the urban/suburban/rural classification of each county. The strata were: metropolitan-urban (Rural Urban Continuum Codes 1-3), nonmetropolitan-urban (Codes 4-5), and rural/less urbanized (Codes 6-9).²⁸

Analysis of Pre-Intervention Trends

For difference-in-difference models to be unbiased, they require the assumption that outcome trends for the treated and comparison groups would have followed parallel trajectories over time were it not for the intervention. While this assumption is untestable, whether or not trends were parallel prior to the intervention can be observed. If they were not parallel, the assumption is violated. Thus, differences in trends in both hospitalization rates within each urban-rural stratum were assessed both visually using graphs and statistically by regressing the outcome on the time trend and an interaction of the time trend with a state variable in the pre-implementation period. A coefficient for the interaction term that is close to zero or not statically significant would suggest that there is no significant difference in the states' pre-period trends.^{29,30}

Sensitivity Analyses

The robustness of the study results to alternative model specifications was assessed using several sensitivity analyses. First, although hospitalization rates for each outcome and within each urban-rural stratum were normally distributed, all models were run using negative binomial regression, modeling the counts of hospitalizations with population offset.

Second, the injury models were run using a more restrictive outcome definition. The main models included all accidental injuries because it is common for alcohol-related encounters to not be marked as such in diagnostic codes,³¹⁻³⁵ so stricter definitions would likely fail to capture all true alcohol-related injuries. Nonetheless, results using a narrower definition should mirror those of the wider definition. This stricter definition included only those accidents that also had a listed diagnosis code of alcohol intoxication, either acute alcohol intoxication in alcoholism (ICD-9 codes: 303.00-303.03) or nondependent alcohol abuse (ICD-9 codes: 305.00-305.03). These hospitalizations were extremely rare; twenty percent of county-quarter observations had zero such hospitalizations recorded. Due to the high number of zero values, the model was run using negative binomial regression with hospitalization counts as the outcome and population offset rather than linear regression with rates as the outcome, as linear regression of count data with many zeros can be biased even with various types of transformation.^{36,37}

Third, the models were estimated with the exclusion of certain counties to ensure that the study results were not being driven by a small number of influential counties. The models were run without King County, the home of Seattle, where approximately 28 percent of the population resides. Conversely, the models were run excluding any counties with a population under 5,000 residents, as these counties may not have large enough populations to reliably estimate hospitalization rates.^{38,39}

Finally, for any statewide analysis or urban-rural strata for which there was evidence that pre-intervention outcome trends in Washington and Oregon were not parallel, additional sensitivity analyses were conducted using two alternative comparison groups. First, propensity score matching was used to match counties in Washington to counties similar in key characteristics and pre-intervention outcomes from eleven other states for which HCUP data was available. Second, a comparison group was created from the eleven other states using synthetic control methods, which generate an artificial comparison group by weighting observations from a “donor” group to best approximate the treated unit in pre-intervention outcomes and other relevant covariates.^{40,41} Further details on these methods and their utility as robustness checks in these circumstances are included in the appendix.

2.3 Results

Descriptive Statistics

The number of off-premise outlets changed substantially in Washington after the implementation of Initiative 1183, but the change was primarily concentrated in metropolitan-urban counties (see Figures 2.1 and 2.2). These counties experienced, on average, an increase of 46.43 outlets after implementation. As expected, the largest increase took place in King county, where outlets increased by 467 percent from 72 outlets to 408 outlets. Nonmetropolitan-urban and rural counties experienced much smaller changes. On average, nonmetropolitan-urban counties gained 9.38 outlets while rural counties gained 2.70 outlets. Two rural counties experienced no change in outlets after 1183 implementation and one rural county lost its only existing outlet.

Figure 2.1. Off-premise outlets in WA before (2012) and after (2014) Initiative 1183 Implementation

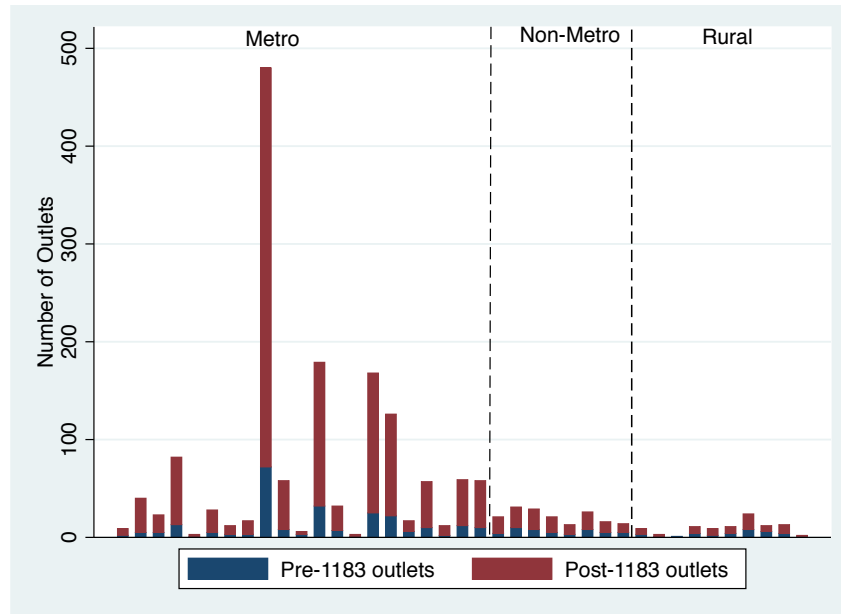
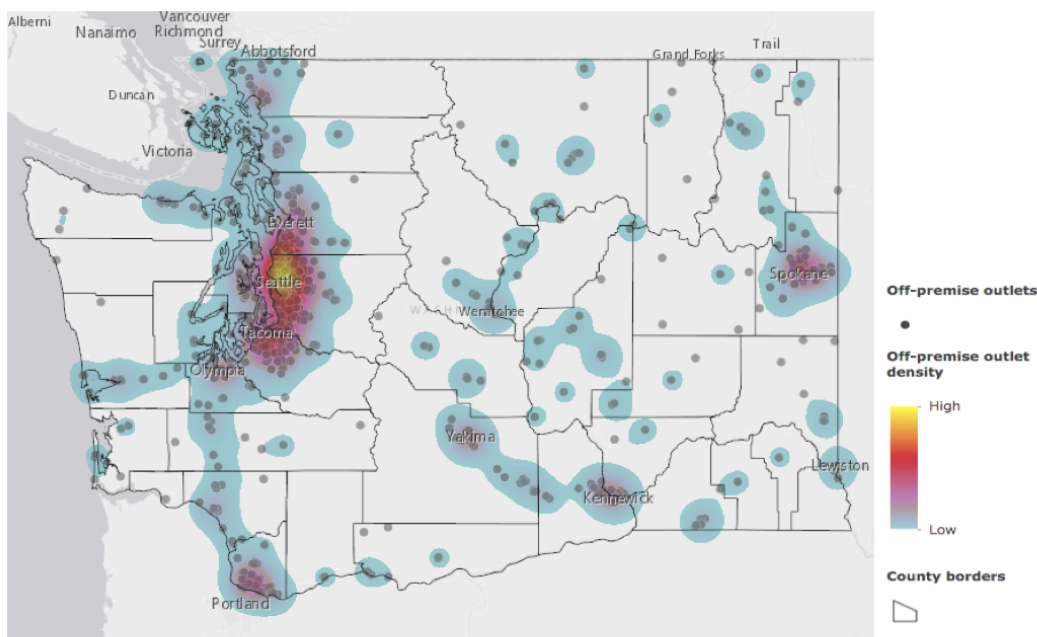


Figure 2.2. Off-premise outlet location & Density in WA after Initiative 1183 Implementation



Data source: Washington State Liquor and Cannabis Board 2019

Hospitalizations for both alcohol-related disorders and accidental injuries were relatively rare in the inpatient data in comparison to other diagnoses; the mean hospitalization rates in Washington prior to 1183 implementation were 1.07 alcohol-related disorder hospitalizations per 1,000 county residents per quarter (SD=0.46) and 1.65 accidental injury hospitalizations per 1,000 county residents per quarter (SD=0.60). The most frequent principal diagnoses for hospitalizations for alcohol-related disorders were alcohol withdrawal, acute pancreatitis, septicemia, alcoholic cirrhosis of the liver, and alcohol withdrawal delirium. The most frequent principal diagnoses for hospitalizations for accidental injuries were falls, most commonly from slipping, tripping, or stumbling, falling from stairs, or in other or unspecified manners.

Rates of both types of hospitalization increased slightly after implementation of 1183 (by 0.05 hospitalizations for alcohol-related disorders and by 0.16 hospitalizations for accidental injuries), but two-sided t-tests reveal that the increase was only statistically significant for accidental injuries. Hospitalization rates for both outcomes showed a similar pattern within each urban-rural stratum, increasing some after implementation, but the increase was only significant for accidental injuries in metropolitan-urban counties (see Table 2.1).

Table 2.1. Washington Hospitalization Rates Before and After 1183 Implementation

<i>Type of Hospitalization</i>	<i>Before 1183 Mean (SD)</i>	<i>After 1183 Mean (SD)</i>
Alcohol-related disorders		
All counties (n=39)	1.07 (0.46)	1.12 (0.44)
Metropolitan-urban (n=21)	1.06 (0.34)	1.12 (0.34)
Nonmetropolitan-urban (n=8)	1.16 (0.54)	1.19 (0.47)
Less urbanized (n=10)	1.02 (0.574)	1.07 (0.58)
Accidental injuries		
All counties (n=39)	1.65 (6.0)	1.81 (0.88)*
Metropolitan-urban (n=21)	1.58 (0.52)	1.75 (0.80)*
Nonmetropolitan-urban (n=8)	1.94 (0.67)	2.07 (1.00)
Less urbanized (n=10)	1.56 (0.65)	1.71 (0.92)

Note. * denotes $p < 0.05$ significance in test of means compared to pre-implementation period. Rates are expressed as the number of hospitalizations per 1,000 residents per county per quarter of the calendar year

Source. AHRQ Health Care Cost and Utilization Project (HCUP) state inpatient files 2010-2014

Mean hospitalization rates were slightly higher in Oregon and also increased over this time period. The mean rate of hospitalizations for alcohol-related disorders increased from 1.16 (SD=0.57) to 1.30 (SD=0.64) hospitalizations per 1,000 county residents per quarter and the mean rate of hospitalizations for accidental injuries increased from 1.73 (SD=0.62) to 1.93 (SD=1.02) hospitalizations per 1,000 county residents per quarter. Both increases were statistically significant. Alcohol-related disorder hospitalization rates increased in all urban-rural strata, but the increase was not significant in nonmetropolitan-urban counties. Accidental injury hospitalization rates increased in both nonmetropolitan-urban and rural counties, but the increase again was not significant in nonmetropolitan-urban counties. The greatest departure from the trends in Washington was that accidental injury hospitalization rates decreased in metropolitan-urban counties, although this decrease was not statistically significant.

Table 2.2 displays the baseline socioeconomic and demographic characteristics of Washington and Oregon counties, including those that have been associated with alcohol use and/or hospitalization rates.^{24,42-46} Tests of means reveals few significant differences, supporting Oregon's utility as a comparison group.

Table 2.2. Demographic and Socioeconomic Characteristics of Counties by State at Baseline

<i>Variable</i>	<i>Washington (n=39) Mean (SD)</i>	<i>Oregon (n=36) Mean (SD)</i>
Median age	40.90 (7.10)	42.66 (5.74)
Percentage of population age 15-24	13.40 (5.30)	12.25 (3.16)
Percentage of population age 65+	16.19 (4.89)	17.69 (4.72)
Median household income	\$46,621.33 (7,814.28)	\$42,680.97* (6,600.10)
Unemployment rate	10.47 (2.19)	11.29 (2.32)
Percent uninsured	67.31 (4.61)	63.67* (4.70)
Percent urban	55.21 (31.69)	55.63 (27.25)
Percentage of population white	83.70 (8.99)	87.78* (6.56)
Percentage of population Non-Hispanic Black	1.24 (1.44)	0.69 (0.91)
Percentage of population Hispanic	12.73 (13.91)	10.58 (8.19)
Percentage of population Asian	2.41 (2.87)	1.50 (1.79)

Note. * denotes $p < 0.05$ significance in test of means compared to Washington.

Source. AHRQ Health Care Cost and Utilization Project (HCUP) state inpatient files 2010-2014, HHS Area Health Resources File (AHRF) 2010-2014

Pre-Intervention Trends

Figures 2.3 and 2.4 show the trends of each outcome over the study period and demonstrate that trends for both outcomes were roughly parallel in Washington and Oregon prior to the implementation of 1183, with approximately matching seasonal peaks and troughs. The same is true within each urban-rural stratum, permitting some leniency considering the lower precision of estimates resulting from smaller sample sizes. Further, when statistically testing for pre-implementation trend differences, the coefficients for the interaction terms of time and state are not significant for all outcomes in the full sample and within all strata, with the exception of accidental injuries in metropolitan-urban counties. This regression results in a statistically significant coefficient of 0.03, which suggests a difference in the pre-existing trends in hospitalization rates in these counties in Washington compared to Oregon, albeit a small difference.

Figure 2.3. Alcohol-related Disorder Hospitalization Rates in Washington & Oregon Before and After 1183 Implementation

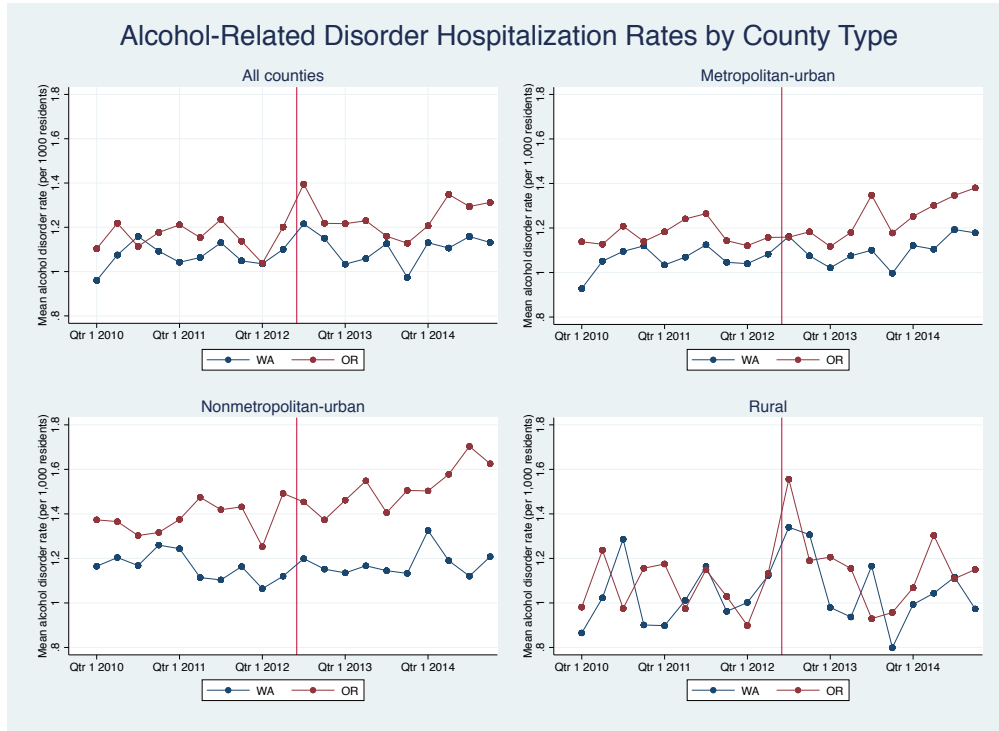
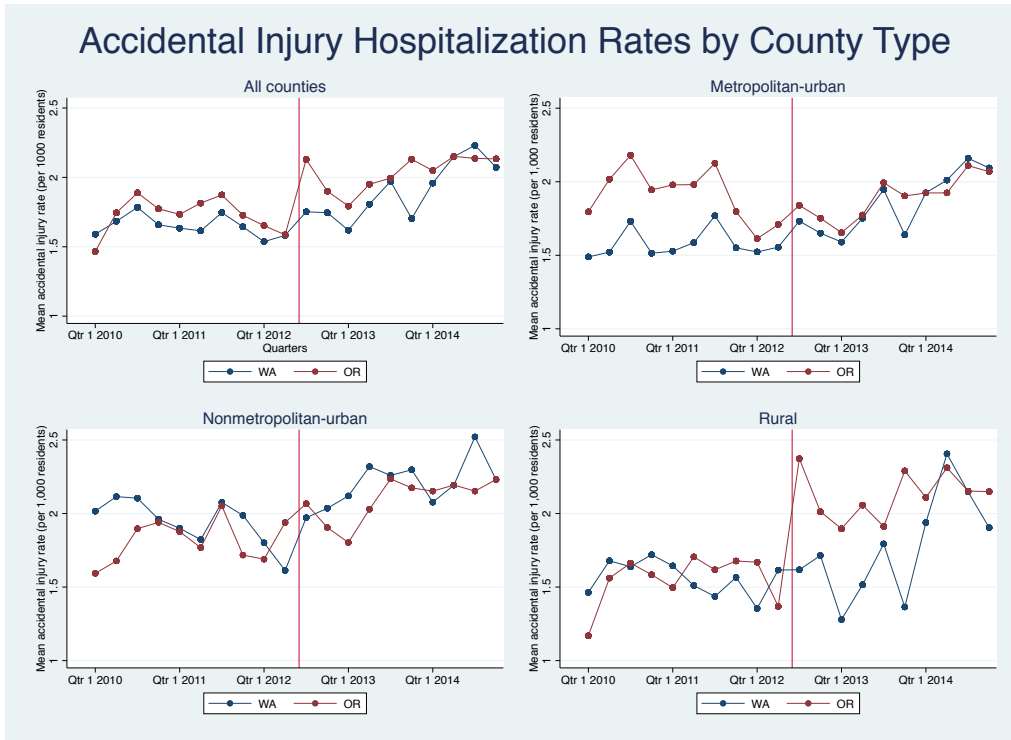


Figure 2.4. Accidental Injury Hospitalization Rates in Washington & Oregon Before and After 1183 Implementation



Model Results

In adjusted difference-in-difference models, the implementation of Initiative 1183 in Washington was not significantly associated with a differential change in hospitalization rates for alcohol-related disorders nor for accidental injuries compared to contemporaneous changes in Oregon (see Table 2.3). Stratified models similarly suggest no significant effect of 1183 for all county types and all outcomes, except with regard to accidental injuries in metropolitan-urban counties. In these counties, 1183 was significantly associated with an increase in the rate of accidental injury hospitalizations in Washington that was on average 0.289 hospitalizations per 1,000 county residents per quarter greater than the simultaneous increase observed in Oregon ($p=0.017$).

Table 2.3. Difference-in Difference Models of County Hospitalization Rates for Alcohol-Related Harms following Implementation of 1183 by Urban-Rural Classification

	<i>Alcohol-Related Disorders</i>	<i>Accidental Injuries</i>
<u>All counties (n=75)</u>		
Post 1183xWA	-0.038 (0.065)	-0.092 (0.144)
<u>Metropolitan-urban (n=34)</u>		
Post 1183xWA	-0.004 (0.056)	0.289* (0.115)
<u>Nonmetropolitan-urban (n=14)</u>		
Post 1183xWA	-0.110 (0.074)	-0.048 (0.193)
<u>Less urbanized (n=27)</u>		
Post 1183xWA	-0.023 (0.146)	-0.453 (0.273)

Note. Presented are coefficient estimates from ordinary least squares regression. Post 1183xWA = interaction between indicator for pre-1183 enactment (0) or post-enactment (1) and indicator for county being in Oregon (0) or Washington (1). Models include indicators for post-enactment and WA beer tax and county, year, and quarter fixed effects. County clustered standard errors are in parentheses. Boldface indicates statistical significance (* $p<0.05$, ** $p<0.01$, *** $p<0.001$).

Source. AHRQ Health Care Cost and Utilization Project (HCUP) state inpatient files 2010-2014.

Sensitivity Analyses

The pattern of results was consistent in sensitivity analyses. Negative binomial regression models found statistically significant increases only in accidental injury hospitalization rates in metropolitan-urban counties (IRR=1.170, $p=0.003$). The models analyzing specifically alcohol-related accidental injuries found a statistically significant and even slightly stronger effect in metropolitan-urban counties (IRR=1.229, $p=0.002$) but no effect in nonmetropolitan-urban or rural counties. The models that excluded King county as well as those that excluded small counties were consistent with the main models (results not shown).

Propensity score and synthetic control methods were used to assess accidental injuries in metropolitan-urban counties, because analyses suggested the parallel trends assumption may not be satisfied for this outcome within this stratum. Results of both alternative methods were consistent, and slightly stronger in magnitude (see Table 2.4). When the adjusted difference-in-difference model was run using the matched control group, Initiative 1183 was significantly associated with an increase in the rate of accidental injury hospitalizations in Washington that was on average 0.375 hospitalizations per 1,000 county residents per quarter greater than the increase observed in the matched counties ($p=0.004$). Similarly, Washington counties had an average increase in the hospitalization rate that was 0.407 hospitalizations per 1,000 county residents per quarter greater than the increase in the hospitalization rate in the synthetic control group ($p=0.016$). Figures 2.5 and 2.6 display these differences in mean hospitalization rates in Washington counties compared to the matched counties and the synthetic control.

Table 2.4. Estimated Treatment Effect of Initiative 1183 on Accidental Injury Hospitalization Rates in Washington Urban Counties across Model Specifications

	<i>DID with Oregon as Control</i>	<i>DID with Propensity Score Matched Control</i>	<i>Synthetic Control</i>
Treatment Effect (ATT)	0.289	0.375	0.362
P-value	0.017	0.004	0.001

Note. DID=Difference-in-difference; ATT=Average treatment effect on the treated.
 Source. AHRQ Health Care Cost and Utilization Project (HCUP) state inpatient files 2010-2014.

Figure 2.5. Accidental Injury Hospitalization Rates in Urban Counties in Washington & Matched Sample Before and After 1183 Implementation

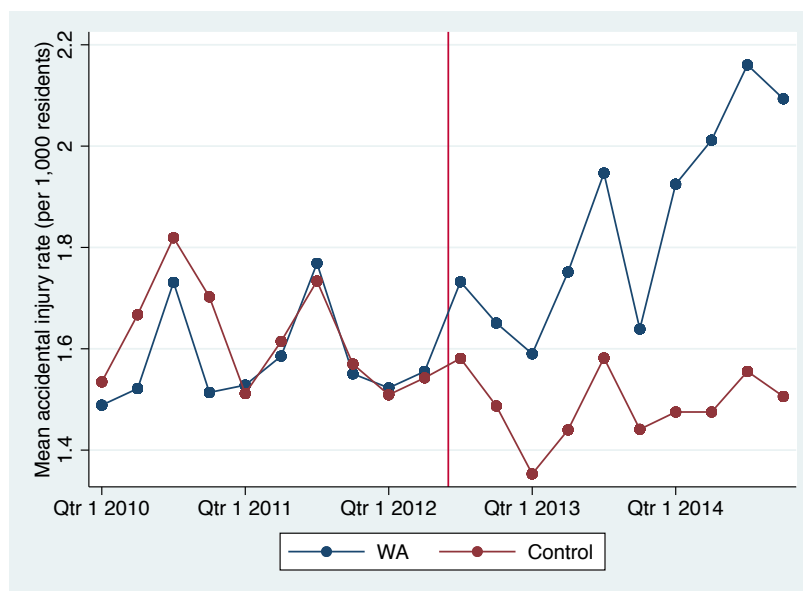
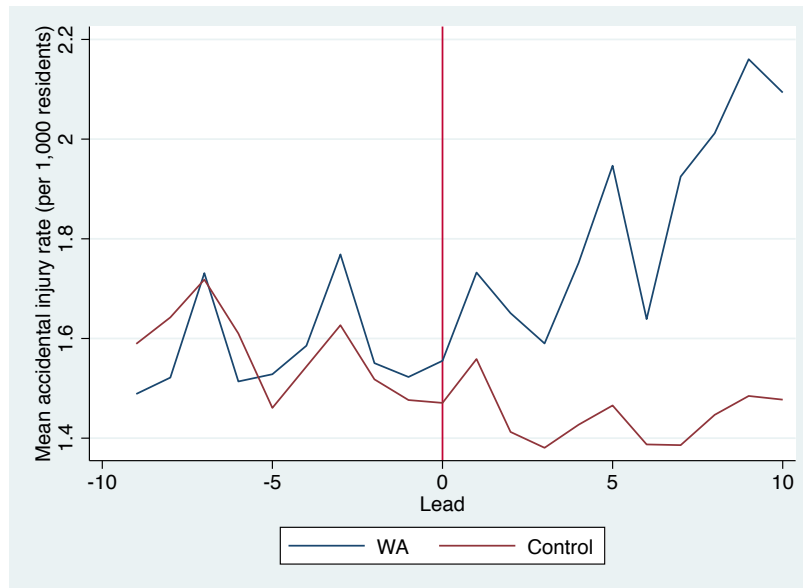


Figure 2.6. Accidental Injury Hospitalization Rates in Urban Counties in Washington & Synthetic Control Before and After 1183 Implementation



2.4 Discussion

These findings suggest that the implementation of Initiative 1183 in Washington was associated with an increase in the rate of hospitalizations for accidental injuries in metropolitan-urban counties. Using the adjusted difference-in-difference estimate and taking into account the average population of 288,319 residents in the 21 urban counties in Washington, this finding translates into 17,498 additional hospitalizations in Washington over the 2.5 year post-intervention period under study. The number rises if the estimates from the matched difference-in-difference or synthetic control analyses are used. This escalation in injuries occurred despite price increases for liquor of between five and fifteen percent,¹² which may have offset some additional effects of increased availability. In contrast, there was no evidence of an increase in accidental injuries in nonmetropolitan-urban or rural counties. These results are consistent with expectations, because the off-premise outlet increase after implementation was primarily concentrated in metropolitan-urban counties. In essence, the analysis finds an effect only in those counties strongly affected by Initiative 1183 and no evidence of an effect in those counties in which the initiative did little to alter the availability status quo.

In contrast to findings from observational studies on retail availability, this analysis indicates that there was no influence of Initiative 1183 on hospitalizations for alcohol-related disorders in counties of any type. It is possible that the time period under study was too short to detect a change in chronic alcohol-related conditions such as liver cirrhosis and alcohol dependence that can take many years or decades to manifest, although evidence has suggested that rates of chronic conditions can change alongside changes in per capita consumption.^{47,48} It is also possible that increased availability simply had no impact on the purchasing and

consumption of already habitually heavy drinkers, who make up the population at higher risk of such disorders. However, evidence from other settings again suggests the contrary, that heavy drinkers do increase their consumption in response to changes in alcohol availability, perhaps even more so than lighter drinkers.^{49,50} It is plausible, though, that the increased availability resulted in greater liquor consumption among all drinkers but only by small quantities. As noted in the introduction, injury risk can increase at even low levels of alcohol consumption and intoxication. However, heavy drinkers were presumably already drinking large quantities of alcohol prior to privatization, and a small increase in the amount consumed may have had a negligible effect on the development of chronic conditions. Such conclusions are beyond the scope of this analysis but certainly warrant future study.

This analysis is unique in that it is the first to assess whether the abolition of a statewide liquor control system in the U.S. setting translated into alcohol-related adverse health outcomes. As such, it is difficult to contextualize within existing literature. Wine privatization has occurred in the U.S. and evaluations generally suggest that wine sales increased,^{53,54} but alcohol-related harms were largely unstudied. Further, it is possible that liquor privatization has an entirely different influence on consumption and harms than wine privatization.⁵³ Liquor constitutes a much higher percentage of per capita alcohol consumption than wine in the U.S.,^{55,56} and liquor can raise blood alcohol concentration to a higher level and more quickly than can wine, which may result in different rates of adverse outcomes.⁵⁷⁻⁵⁹ Liquor privatization has taken place in Alberta and British Columbia, but comparisons are challenging because these policy changes were quite different from that of Washington. For instance, sales were allowed in new liquor stores but not in grocery stores or chain stores, precisely opposite to what occurred in Washington. These privatization schemes might have markedly different impacts. As previously mentioned, none of the few evaluations of liquor privatization in Iowa assessed health outcomes. Thus, this analysis generates new knowledge that can hopefully be corroborated with further study. Such knowledge may become increasingly important, as the U.S. is in the midst of a trend toward alcohol liberalization and other states with liquor control systems are increasingly considering license systems. For instance, as recently as April 2019, the North Carolina House of Representatives introduced a bill that would privatize retail and wholesale liquor. Other less aggressive availability-related measures, such as zoning ordinances, are also regularly discussed by states, counties, and cities. As these policy debates continue, it will be important to know whether or not license systems and increased liquor availability in general can have any unintended public health consequences.

A strength of this analysis is its difference-in-difference design, which suggests causality more strongly than observational analyses. Further, the robustness checks using propensity score matching and synthetic control not only bolster confidence in the effect identified with their consistency but account for confounders that a standard difference-in-difference cannot (i.e. differences in baseline covariates with time-varying effects on the outcomes, over-time compositional changes).

Nonetheless, this study has important limitations. First, analyses were performed at the county level, with hospitalizations linked to counties via the FIPS code of residence. While counties are large and likely encompass much of a person's usual travel, the use of this unit of analysis risks spatial misclassification, as individuals may be exposed to outlets outside of their home counties quite frequently. This is particularly problematic for acute outcomes like injuries,

for which it is entirely possible that a person encountered outlets, purchased alcohol, and was hospitalized all within a few hours and all outside of his or her county of residence. Analyses that utilize geographical information systems might be more accurate and should be pursued in the future if such data become available. Further, although they make up less than one percent of the hospitalization records, individuals without a home address on record were excluded and these individuals are not missing at random. A sizeable portion may be homeless, a population with a particularly high burden of alcohol use.

These results suggest several potential areas for future research. This analysis focused strictly on inpatient hospitalizations, which allowed us to avoid what may have been a notable source of bias for the difference-in-difference model: variably rising rates of insurance coverage due to the implementation of the Affordable Care Act during the period of study. As insurance rates rose, it is likely that more people would be visiting the emergency department or other settings for lesser ailments and injuries rather than avoiding care out of fear of large medical bills. Rates of inpatient hospitalizations, however, are likely to remain constant despite coverage changes, as these are the most extreme cases that truly require extensive and immediate care and would presumably prompt a hospital visit regardless of insurance status. However, future work should assess emergency department admissions. Many individuals receive care for injuries in emergency departments but are not admitted to the inpatient setting, so there would be more power to detect effects. Emergency department data would also enable a more detailed examination of alcohol poisonings as an outcome separate from other alcohol-related disorders. Such an acute outcome might respond to changes in availability differently than chronic outcomes, and the majority of healthcare visits for alcohol poisoning take place in the emergency department.⁶⁰ Additionally, longer term analyses of chronic alcohol-related disorders should be done when more years of data are available. As noted, it can take many years if not decades for some of these conditions to develop amongst heavy drinkers. Further, according to the distribution of consumption theory, the proportion of heavy drinkers in a population changes with the average per capita alcohol consumption,^{61,62} meaning that if Initiative 1183 increased average consumption in Washington, the number of heavy drinkers, and thus the number at risk of chronic conditions, may have also increased. A corresponding increase in the number of chronic outcomes would not be evident for many years.

2.5 Conclusion

Despite price increases, the change in liquor availability accomplished by Washington's Initiative 1183 was associated with increased rates of hospitalizations for accidental injuries in urban counties, which were the counties most affected by the policy change. No effect, however, was found for hospitalizations for alcohol-related disorders. This pattern suggests that liquor privatization only impacted those adverse outcomes for which a person is at higher risk from low levels of alcohol consumption, while outcomes that result from heavy drinking did not change over the short-term. These findings indicate that liquor license systems and potentially other availability-related alcohol policies can have unintended yet measurable public health implications, and, in doing so, may help guide policy discussions as the U.S. continues on its trend towards alcohol liberalization and decentralization.

2.6 References

1. National Institute on Alcohol Abuse and Alcoholism. *Alcohol Facts and Statistics.*; 2019. <https://www.niaaa.nih.gov/sites/default/files/AlcoholFactsAndStats.pdf>.
2. Centers for Disease Control and Prevention. *Alcohol and Public Health: Alcohol-Related Disease Impact (ARDI).*; 2013. https://nccd.cdc.gov/DPH_ARDI/Default/Report.aspx?T=AAM&P=f6d7eda7-036e-4553-9968-9b17ffad620e&R=d7a9b303-48e9-4440-bf47-070a4827e1fd&M=8E1C5233-5640-4EE8-9247-1ECA7DA325B9&F=&D=.
3. Stockwell T, Zhao J, Macdonald S, et al. Impact on alcohol-related mortality of a rapid rise in the density of private liquor outlets in British Columbia: a local area multi-level analysis. *Addiction*. 2011;106(4):768–776.
4. Stockwell T, Zhao J, Martin G, et al. Minimum alcohol prices and outlet densities in British Columbia, Canada: estimated impacts on alcohol-attributable hospital admissions. *Am J Public Health*. 2013;103(11):2014–2020.
5. Richardson EA, Hill SE, Mitchell R, Pearce J, Shortt NK. Is local alcohol outlet density related to alcohol-related morbidity and mortality in Scottish cities? *Health Place*. 2015;33:172–180.
6. Livingston M. Alcohol outlet density and harm: comparing the impacts on violence and chronic harms. *Drug Alcohol Rev*. 2011;30(5):515–523.
7. Morrison C, Smith K, Gruenewald PJ, Ponicki WR, Lee JP, Cameron P. Relating off-premises alcohol outlet density to intentional and unintentional injuries. *Addiction*. 2016;111(1):56–64.
8. Gruenewald PJ, Freisthler B, Remer L, Lascala EA, Treno AJ, Ponicki WR. Ecological associations of alcohol outlets with underage and young adult injuries. *Alcohol Clin Exp Res*. 2010;34(3):519-527. doi:10.1111/j.1530-0277.2009.01117.x
9. Washington State Secretary of State. Initiative Measure No. 1183. 2011. <https://www.sos.wa.gov/elections/initiatives/text/i1183.pdf>.
10. Washington State Office of Financial Management. *Privatization of Liquor: The Impact of Initiative 1183*. Seattle, WA: State of Washington; 2015. https://www.ofm.wa.gov/sites/default/files/public/legacy/fiscal/pdf/liquor_privatization_initiative1183.pdf.
11. Washington State Liquor and Cannabis Board. Off-Premises Licensees. 2018. <https://lcb.wa.gov/records/frequently-requested-lists>.
12. Kerr WC, Williams E, Greenfield TK. Analysis of price changes in Washington following the 2012 liquor privatization. *Alcohol Alcohol*. 2015;50(6):654–660.

13. Holder HD, Wagenaar AC. Effects of the elimination of a state monopoly on distilled spirits' retail sales: a time-series analysis of Iowa. *Br J Addict*. 1990;85(12):1615–1625.
14. Mulford HA, Ledolter J, Fitzgerald JL. Alcohol availability and consumption: Iowa sales data revisited. *J Stud Alcohol*. 1992;53(5):487–494.
15. Fitzgerald JL, Mulford HA. Consequences of increasing alcohol availability: the Iowa experience revisited. *Br J Addict*. 1992;87(2):267–274.
16. Babor T, Caetano R, Casswell S, et al. Alcohol: no ordinary commodity: research and public policy. *Rev Bras Psiquiatr*. 2003;26(4):280–3.
17. Campbell CA, Hahn RA, Elder R, et al. The effectiveness of limiting alcohol outlet density as a means of reducing excessive alcohol consumption and alcohol-related harms. *Am J Prev Med*. 2009;37(6):556–569.
18. Gmel G, Holmes J, Studer J. Are alcohol outlet densities strongly associated with alcohol-related outcomes? A critical review of recent evidence. *Drug Alcohol Rev*. 2016;35(1):40–54.
19. Greenfield TK, Williams E, Kerr WC, Subbaraman MS, Ye Y. Washington State Spirits Privatization: How Satisfied were Liquor Purchasers Before and After, and by Type of Retail Store in 2014? *Subst Use Misuse*. 2018;53(8):1260–1266.
20. Subbaraman MS, Kerr WC. Opinions on the privatization of distilled-spirits sales in Washington State: Did voters change their minds? *J Stud Alcohol Drugs*. 2016;77(4):568–576.
21. Ye Y, Kerr WC. Estimated increase in cross-border purchases by Washington residents following liquor privatization and implications for alcohol consumption trends. *Addiction*. 2016;111(11):1948–1953. doi:10.1111/add.13481
22. Illanes G, Moshary S. Market Structure and Product Variety: Evidence from a Natural Experiment in Liquor Licensure. 2018.
23. Kerr WC, Williams E, Ye Y, Subbaraman MS, Greenfield TK. Survey estimates of changes in alcohol use patterns following the 2012 privatization of the Washington liquor monopoly. *Alcohol Alcohol*. 2018;53(4):470–476.
24. Dixon MA, Chartier KG. Alcohol use patterns among urban and rural residents: demographic and social influences. *Alcohol Res Curr Rev*. 2016;38(1):69.
25. Borders TF, Booth BM. Rural, suburban, and urban variations in alcohol consumption in the United States: findings from the National Epidemiologic Survey on Alcohol and Related Conditions. *J Rural Health*. 2007;23(4):314–321.

26. Agency for Healthcare Research and Quality. Introduction to the HCUP State Inpatient Databases (SID). 2018. https://www.hcup-us.ahrq.gov/db/state/siddist/Introduction_to_SID.pdf. Accessed August 8, 2018.
27. Centers for Disease Control and Prevention. Proposed Matrix of E-code Groupings. Injury Prevention & Control. https://www.cdc.gov/injury/wisqars/ecode_matrix.html. Published September 6, 2018. Accessed October 8, 2018.
28. Langlois PH, Jandle L, Scheuerle A, Horel SA, Carozza SE. Occurrence of conotruncal heart birth defects in Texas: a comparison of urban/rural classifications. *J Rural Health*. 2010;26(2):164–174.
29. Ryan AM. Effects of the Premier Hospital Quality Incentive Demonstration on Medicare patient mortality and cost. *Health Serv Res*. 2009;44(3):821–842.
30. Basu S, Meghani A, Siddiqi A. Evaluating the health impact of large-scale public policy changes: classical and novel approaches. *Annu Rev Public Health*. 2017;38:351–370.
31. Indig D, Indig D, Copeland J, et al. Why are alcohol-related emergency department presentations under-detected? An exploratory study using nursing triage text. *Drug Alcohol Rev*. 2008;27(6):584–590.
32. Smothers BA, Yahr HT, Ruhl CE. Detection of alcohol use disorders in general hospital admissions in the United States. *Arch Intern Med*. 2004;164(7):749–756.
33. McKenzie K, Harrison JE, McClure RJ. Identification of alcohol involvement in injury-related hospitalisations using routine data compared to medical record review. *Aust N Z J Public Health*. 2010;34(2):146–152.
34. Samuel AM, Lukasiewicz AM, Webb ML, et al. ICD-9 diagnosis codes have poor sensitivity for identification of preexisting comorbidities in traumatic fracture patients: a study of the National Trauma Data Bank. *J Trauma Acute Care Surg*. 2015;79(4):622–630.
35. Ngo DA, Ait-Daoud N, Rege SV, et al. Differentials and trends in emergency department visits due to alcohol intoxication and co-occurring conditions among students in a US public university. *Drug Alcohol Depend*. 2018;183:89–95.
36. O'hara RB, Kotze DJ. Do not log-transform count data. *Methods Ecol Evol*. 2010;1(2):118–122.
37. Buntin MB, Zaslavsky AM. Too much ado about two-part models and transformation?: Comparing methods of modeling Medicare expenditures. *J Health Econ*. 2004;23(3):525–542.
38. Chauhan P, Cerdá M, Messner SF, Tracy M, Tardiff K, Galea S. Race/ethnic-specific homicide rates in new york city: Evaluating the impact of broken windows policing and crack cocaine markets. *Homicide Stud*. 2011;15(3):268–290.

39. Ahern J, Matthay EC, Goin DE, Farkas K, Rudolph KE. Acute changes in community violence and increases in hospital visits and deaths from stress-responsive diseases. *Epidemiology*. 2018;29(5):684–691.
40. Abadie A, Gardeazabal J. The economic costs of conflict: A case study of the Basque Country. *Am Econ Rev*. 2003;93(1):113–132.
41. Abadie A, Diamond A, Hainmueller J. Synthetic control methods for comparative case studies: Estimating the effect of California’s tobacco control program. *J Am Stat Assoc*. 2010;105(490):493–505.
42. Kerr WC, Greenfield TK, Ye Y, Bond J, Rehm J. Are the 1976–1985 birth cohorts heavier drinkers? Age-period-cohort analyses of the National Alcohol Surveys 1979–2010. *Addiction*. 2013;108(6):1038–1048.
43. Keyes KM, Miech R. Age, period, and cohort effects in heavy episodic drinking in the US from 1985 to 2009. *Drug Alcohol Depend*. 2013;132(1-2):140–148.
44. Collins SE. Associations between socioeconomic factors and alcohol outcomes. *Alcohol Res Curr Rev*. 2016;38(1):83.
45. Chartier K, Caetano R. Ethnicity and health disparities in alcohol research. *Alcohol Res Health*. 2010;33(1-2):152.
46. Keyes KM, Liu XC, Cerda M. The role of race/ethnicity in alcohol-attributable injury in the United States. *Epidemiol Rev*. 2011;34(1):89–102.
47. Holder H, Agardh E, Högberg P, et al. *If Retail Alcohol Sales in Sweden Were Privatized, What Would Be the Potential Consequences?* Statens folkhälsoinstitut; 2007.
48. Corrao G, Ferrari P, Zambon A, Torchio P. Are the recent trends in liver cirrhosis mortality affected by the changes in alcohol consumption? Analysis of latency period in European countries. *J Stud Alcohol*. 1997;58(5):486–494.
49. Mäkelä P. Whose drinking does the liberalization of alcohol policy increase? Change in alcohol consumption by the initial level in the Finnish panel survey in 1968 and 1969. *Addiction*. 2002;97(6):701–706.
50. Ólafsdóttir H. The dynamics of shifts in alcoholic beverage preference: effects of the legalization of beer in Iceland. *J Stud Alcohol*. 1998;59(1):107–114.
51. Cherpitel CJ, Ye Y, Bond J, Borges G, Monteiro M. Relative risk of injury from acute alcohol consumption: modeling the dose–response relationship in emergency department data from 18 countries. *Addiction*. 2015;110(2):279–288.
52. Cherpitel CJ, Ye Y, Kerr WC. Risk of past year injury related to hours of exposure to an elevated BAC and average monthly alcohol volume: data from four national alcohol surveys (2000–2015). *Alcohol Clin Exp Res*. 2017.

53. Cook PJ. Alcohol retail privatization: a commentary. *Am J Prev Med*. 2012;42(4):430–432.
54. Her M, Giesbrecht N, Room R, Rehm J. Privatizing alcohol sales and alcohol consumption: evidence and implications. *Addiction*. 1999;94(8):1125–1139.
55. Martinez P, Kerr WC, Subbaraman MS, Roberts SC. New estimates of the mean ethanol content of beer, wine, and spirits sold in the United States show a greater increase in per capita alcohol consumption than previous estimates. *Alcohol Clin Exp Res*. 2019;43(3):509–521.
56. Kerr WC, Greenfield TK, Tujague J. Estimates of the mean alcohol concentration of the spirits, wine, and beer sold in the United States and per capita consumption: 1950 to 2002. *Alcohol Clin Exp Res*. 2006;30(9):1583–1591.
57. Mitchell Jr MC, Teigen EL, Ramchandani VA. Absorption and peak blood alcohol concentration after drinking beer, wine, or spirits. *Alcohol Clin Exp Res*. 2014;38(5):1200–1204.
58. Smart RG. Behavioral and social consequences related to the consumption of different beverage types. *J Stud Alcohol*. 1996;57(1):77–84.
59. Rehm J, Hasan OS. Is burden of disease differentially linked to spirits? A systematic scoping review and implications for alcohol policy. *Alcohol*. 2019.
60. National Institutes of Health. Alcohol-Related Emergency Department Visits and Hospitalizations and Their Co-occurring Drug-Related, Mental Health, and Injury Conditions in the United States: Findings from the 2006 to 2010 Nationwide Emergency Department Sample (NEDS) and Nationwide Inpatient Sample (NIS). *US Alcohol Epidemiol Data Ref Man*. 2013;9.
61. Skog O-J. Social Interaction and the Distribution of Alcohol Consumption. *J Drug Issues*. 1980;10(1):71-92. doi:10.1177/002204268001000105
62. Skog O-J. The Collectivity of Drinking Cultures: A Theory of the Distribution of Alcohol Consumption. *Addiction*. 1985;80(1):83-99. doi:10.1111/j.1360-0443.1985.tb05294.x

2.7 Appendix

Table A2.1. ICD-9 Codes & Diagnoses Included in Each Hospitalization Type

<i>Hospitalizations for Alcohol-Related Disorders</i>			
<u>ICD-9 Code</u>	<u>Diagnosis</u>	<u>ICD-9 Code</u>	<u>Diagnosis</u>
291.0	Alcohol withdrawal delirium	305.0	Nondependent alcohol abuse
291.1	Alcohol-induced persisting amnesic disorder	305.00	Alcohol abuse, unspecified
291.2	Alcohol-induced persisting dementia	305.01	Alcohol abuse, continuous
291.3	Alcohol-induced psychotic disorder with hallucinations	305.02	Alcohol abuse, episodic
291.4	Idiosyncratic alcohol intoxication	305.03	Alcohol abuse, in remission
291.5	Alcohol-induced psychotic disorder with delusions	357.5	Alcoholic polyneuropathy
291.8	Other specified alcohol-induced mental disorders (withdrawal, sleep disorders, other)	425.5	Alcoholic cardiomyopathy
291.81	Alcohol withdrawal	535.3	Alcoholic gastritis
291.82	Alcohol induced sleep disorders	535.30	Alcoholic gastritis, without mention of hemorrhage
291.89	Other alcohol-induced mental disorders	535.31	Alcoholic gastritis, with hemorrhage
291.9	Unspecified alcohol-induced mental disorders	571.0	Alcoholic fatty liver
303.0	Acute alcoholic intoxication	571.1	Acute alcoholic hepatitis
303.00	Acute alcoholic intoxication in alcoholism, unspecified	571.2	Alcoholic cirrhosis of the liver
303.01	Acute alcoholic intoxication in alcoholism, continuous	571.3	Alcoholic liver damage, unspecified
303.02	Acute alcoholic intoxication in alcoholism, episodic	760.71	Alcohol affecting fetus or newborn via placenta or breast milk
303.03	Acute alcoholic intoxication in alcoholism, in remission	980.0	Toxic effect of ethyl alcohol
303.9	Other and unspecified alcohol dependence		
303.90	Other and unspecified alcohol dependence, unspecified		
303.91	Other and unspecified alcohol dependence, continuous		
303.92	Other and unspecified alcohol dependence, episodic		
303.93	Other and unspecified alcohol dependence, in remission		
<i>Hospitalizations for Accidental Injuries</i>			
<u>ICD-9 Code</u>	<u>Diagnosis</u>	<u>ICD-9 Code</u>	<u>Diagnosis</u>
E800-807	Railway accidents	E880-888	Accidental falls

E810-819	Motor vehicle traffic accidents	E890-899	Accidents caused by fire and flames
E820-825	Motor vehicle nontraffic accidents	E900-909	Accidents due to natural and environmental factors
E826-829	Other road vehicle accidents	E910-915	Accidents caused by submersion, suffocation, and foreign bodies
E830-838	Water transport accidents	E916-928	Other accidents
E840-845	Air and space transport accidents	E929	Late effects of accidental injuries.
E846-849	Vehicle accidents, not elsewhere classifiable		
E850-858	Accidental poisoning by drugs, medicinal substances, and biologicals		
E860-869	Accidental poisoning by other solid and liquid substances, gases, and vapors		

A2.1 Propensity Score Matching & Synthetic Control Methods

Background

Matching using propensity scores or other methods is frequently used in difference-in-difference models to help create a more comparable control group. Treatment and comparison groups may have different values of unobserved covariates that, while time invariant themselves, differentially impact outcomes over time. While standard difference-in-difference models can account for any unobserved covariates with time-constant effects, they cannot account for those with time-varying effects. Matching on these covariates, however, can generate a comparison group that is more similar to the treated group in their values, thereby reducing the bias they cause.^{1,2} Pre-intervention outcome values have also been used for matching to further improve comparability, with the intuition that previous outcomes will proxy for some covariates with time-varying effects that are unobserved in the data.³

Synthetic control methods involve generating an artificial comparison group by weighting observations from a “donor” group to best approximate the treated unit in pre-treatment outcomes and other relevant covariates.^{4,5} A treatment effect is then determined by observing the difference in outcomes between the treated unit and the synthetic control group. Inference is conducted by permutation tests, treating each unit used in the synthetic control group as if it were the treated unit and observing its estimated treatment effect, which allows one to see where in the distribution of effects the treatment effect of interest falls.^{4,5} The synthetic control method has some advantages that may make it preferable to the matched difference-in-difference. Matching based on propensity scores can only balance across treatment and comparison groups those covariates included in the scores, and there may be many covariates relevant to this analysis that are unobserved and not included. Synthetic control methods with a long enough pre-treatment period theoretically achieve balance of unobserved covariates with time-varying effects because only units that are similar in observed and unobserved covariates that impact the outcome will have similar trends in the outcome over a long period of time.⁶

Synthetic control methods were originally developed for analyses with only one treated unit, but these methods have been expanded for circumstances in which there are multiple treated units. The method used for this analysis, developed by Cavallo et al. in 2013, involves

performing a synthetic control analysis for each treated unit and calculating the total treatment effect by averaging each unit's treatment effect. Inference is conducted by performing placebo tests for each treated unit and then, at each post-intervention observation, randomly selecting a placebo estimate for each treated unit and averaging these estimates. This process is repeated for each combination, and the position of the treatment effect of interest within the distribution of these estimates is assessed.⁷

Despite their advantages, these methods were used as robustness checks rather than the primary analytic strategies for this study and only when pre-intervention outcomes trends were not parallel because there is considerable debate over the utility of matching using pre-intervention outcomes in difference-in-difference models. Recent simulation work has suggested that when treatment and control groups have parallel pre-intervention trends but differ in their mean pre-intervention outcomes, matching on these outcomes introduces bias, as the matched controls may be those with extreme values relative to their group means and will regress back to their means in the post-intervention period. This regression to the mean will be misinterpreted as a treatment effect.⁸ Synthetic control methods are subject to this same risk of bias, as they weight using pre-intervention outcomes.⁹ The amount of bias increases with the difference in the group means and decreases with the amount of serial autocorrelation in the outcome. When the parallel trends assumption is violated, however, the authors of this simulation find that matching on pre-intervention outcomes does not introduce additional bias, although it does not overcome the bias implicit in the standard difference-in-difference.⁸ Other work has found that matching based on pre-intervention outcomes can reduce bias in this scenario.^{3,10} Thus, only difference-in-difference models using Oregon as the comparison group were used for all cases in which the parallel trends assumption appears to hold in order to avoid risking biased estimates. Bias could have been quite large considering that, for multiple strata, the mean pre-intervention hospitalization rates differed substantially between counties in Washington and the pool of potential matches and, for all, serial autocorrelation of hospitalization rates was low due to seasonal trends. Propensity score matched and synthetic control models could be used to assess outcomes in counties in which the parallel trends assumption does not appear to hold with less threat of introducing bias, but nevertheless, to be conservative, these models were used only as robustness checks of the original model.

Methods

For these analyses, matches and donor counties were drawn from the other states for which HCUP data was available. After excluding four states that had changes in policies around alcohol availability or pricing during this period, this pool included eleven states: AZ, AR, CO, FL, MA, MI, NJ, NM, NY, and VT.

Propensity scores were generated using the rate of hospitalizations for accidental injuries in the first quarter of each year prior to the implementation of Initiative 1183 (2010, 2011, and 2012) as well as baseline measures of the unemployment rate, median household income, median age, and percentage of the population that is of a racial/ethnic minority (non-white). All 21 urban counties in Washington were within the common support. Matches were chosen using 1:1 nearest neighbor matching without replacement. The matched control group, as such, included 21 additional counties, of which eleven were from Michigan, five were from Arkansas, two each were from New Jersey and Vermont, and one was from Colorado. Many combinations of

covariates as well as other matching techniques (i.e. 1:2 matching, matching with replacement, caliper matching) were attempted, but this strategy achieved the best balance of included covariates across the treatment and control groups. The standardized difference in means between the treated and the control group was 2.6 percent for the propensity score and less than 15 percent for each of the included covariates (see Table A2.2). These levels of bias were well below the 25 percent threshold suggested by Rubin¹¹ and the 20 percent threshold that determines a “small” effect size as suggested by Cohen.¹² They also represented marked improvements over the differences in means in comparison to Oregon’s urban counties. Figure 5 in the main text displays the mean hospitalization rates in Washington counties and in the matched counties, and the trends are clearly parallel, when not overlapping, in the pre-intervention period. Further, statistical testing of the difference in pre-intervention trends did not result in a statistically significant difference.

Table A2.2 Covariate Balance between Urban Counties in WA, OR, and Propensity Score Matched Sample

Covariate	Washington <i>n=21</i>		Oregon <i>n=13</i>		Matched sample <i>n=21</i>	
	Mean	Mean	Standardized Difference (%)	Mean	Standardized Difference (%)	
Accidental injury rate in Q1 2010	1.49	1.80	61.0	1.53	6.2	
Accidental injury rate in Q1 2011	1.53	1.98	94.3	1.51	2.2	
Accidental injury rate in Q1 2012	1.52	1.61	20.7	1.51	1.8	
Unemployment rate	10.25	11.05	40.1	10.38	5.5	
Median household income	\$49,931.71	\$47,524.08	32.5	\$48,194.00	14.8	
Median age	38.86	38.60	6.1	38.91	1.3	
Percent racial/ethnic minority	17.63	13.65	53.0	16.16	13.4	

Note. Standardized differences in means are in reference to means of Washington. Q1 = quarter 1 of year
Source. AHRQ Health Care Cost and Utilization Project (HCUP) state inpatient files 2010-2014.

The synthetic control was created from the same eleven states as the propensity score matched sample. After attempting many combinations of pre-intervention outcomes and covariates to generate the weights, the best fit (lowest root mean square prediction error in the pre-intervention period) was achieved by using the same outcome lags and covariates as those used in propensity score generation. This specification had a lower pre-intervention RMSPE than 84.62 percent of the placebo specifications. The pre-intervention fit and post-intervention treatment effect can be viewed in Figure 2.6 in the main text. The distribution of the placebo effects suggested that this effect is statistically significant. 19.04 percent of the placebos had an effect size at least as large as this effect, but effect sizes should be considered in relation to their pre-intervention fits, as those with poor fits in the pre-intervention period are likely to have more extreme differences in the post-intervention period.^{5,13} The associated p-value, which is determined by noting where in the distribution the ratio of effect size to pre-intervention fit falls, was 0.016.

A2.2 Appendix References

1. Heckman JJ, Ichimura H, Todd PE. Matching as an econometric evaluation estimator: Evidence from evaluating a job training programme. *The review of economic studies*. 1997;64(4):605–654.
2. Abadie A. Semiparametric difference-in-differences estimators. *The Review of Economic Studies*. 2005;72(1):1–19.
3. O’Neill S, Kreif N, Grieve R, Sutton M, Sekhon JS. Estimating causal effects: considering three alternatives to difference-in-differences estimation. *Health Services and Outcomes Research Methodology*. 2016;16(1-2):1–21.
4. Abadie A, Gardeazabal J. The economic costs of conflict: A case study of the Basque Country. *American economic review*. 2003;93(1):113–132.
5. Abadie A, Diamond A, Hainmueller J. Synthetic control methods for comparative case studies: Estimating the effect of California’s tobacco control program. *Journal of the American statistical Association*. 2010;105(490):493–505.
6. Abadie A, Diamond A, Hainmueller J. Comparative politics and the synthetic control method. *American Journal of Political Science*. 2015;59(2):495–510.
7. Cavallo E, Galiani S, Noy I, Pantano J. Catastrophic natural disasters and economic growth. *Review of Economics and Statistics*. 2013;95(5):1549–1561.
8. Daw JR, Hatfield LA. Matching and Regression to the Mean in Difference-in-Differences Analysis. *Health services research*. 2018;53(6):4138–4156.
9. Daw JR, Hatfield LA. Matching in Difference-in-Differences: between a Rock and a Hard Place. *Health services research*. 2018;53(6):4111–4117.
10. Ryan AM, Burgess Jr JF, Dimick JB. Why we should not be indifferent to specification choices for difference-in-differences. *Health services research*. 2015;50(4):1211–1235.
11. Rubin DB. Using propensity scores to help design observational studies: application to the tobacco litigation. *Health Services and Outcomes Research Methodology*. 2001;2(3-4):169–188.
12. Cohen J. *Statistical Power Analysis for the Behavioral Sciences*. Routledge; 2013.
13. Ferman B, Pinto C. Revisiting the synthetic control estimator. 2016.

Chapter 3:

The Impact of CVS Health's Tobacco-free Pharmacy Policy on Tobacco Use Among Current Smokers

Abstract

In September 2014, CVS Health ceased tobacco sales in all of its 7,700 pharmacies nationwide. This policy change has the potential to reduce cigarette smoking by reducing impulse purchases and increasing social sanctioning against smoking amongst pharmacy shoppers who are also current smokers. This study investigates the impact of the CVS policy change on the number of cigarettes smoked per day among daily and nondaily smokers, who may respond to smoking cues in different manners. Data are from the U.S. Census Bureau Tobacco Use Supplement to the Current Population Survey 2014-2015 and the Blue Cross and Blue Shield Institute Community Health Management Hub. Adjusted difference-in-difference (DID) regressions are used to assess changes in the number of cigarettes smoked per day among daily smokers (n=10,759) and nondaily smokers (n=3,055) after the policy's implementation compared to before, modeling Core-based Statistical Area (CBSA) level CVS pharmacy market share continuously as well as categorically to determine if the policy had non-linear effects across the distribution of CVS market share. CVS' tobacco-free pharmacy policy was not significantly associated with differential changes in the number of cigarettes by daily smokers in either model. However, the policy had a significant beneficial impact on the number of cigarettes smoked by nondaily smokers in the continuous DID (rate ratio=0.985, p=0.022) and a stronger impact among nondaily smokers in CBSAs in the middle and highest thirds of CVS market share in the categorical DID (middle third: rate ratio=0.723, p=0.043; highest third: rate ratio=0.706, p=0.027). These findings suggest that CVS must have a substantial footprint in the pharmacy market for the policy change to have an impact on the number of cigarettes smoked per day among nondaily smokers and that the constant dose response assumed in the continuous DID tempers the larger impact observed at greater degrees of market share. These results were robust to various alternative specifications. Policies that eliminate tobacco from pharmacies may be a promising tactic for reducing smoking and subsequent chronic disease amongst nondaily smokers, a population at risk of adverse health outcomes but that is generally overlooked in clinical attempts to encourage smoking cessation.

3.1 Introduction

Background

Tobacco use is the leading cause of preventable death in the United States, causing a host of chronic conditions including lung disease, chronic obstructive pulmonary disease, heart disease, diabetes, and various cancers as well as immune dysfunction and congenital disorders.¹ Many of these risks have long been acknowledged and tobacco use rates among adults in the U.S. are at a historic low, but many adults still consume tobacco; recent estimates suggest that almost 14 percent of U.S. adults currently smoke cigarettes. Disparities certainly exist and rates are even higher among certain socioeconomic, racial/ethnic, age, and geographic subgroups.²

Pharmacies are just one of many venues in which cigarettes are available for purchase in the United States but represent a promising setting for interventions to reduce smoking rates. Pharmacy sales of cigarettes make up a small percentage of total U.S. cigarette sales (estimated 4.54 percent in 2009), but this percentage has been increasing over time, even while national cigarette sales have been declining. It is estimated that between 2005 and 2009, national pharmacy cigarette sales increased 23 percent while total cigarette sales decreased 17 percent.³ In addition, recent work has suggested that, on average, tobacco products are cheaper in pharmacies than in other tobacco retailers, potentially attracting more sales.⁴

In February 2014, CVS Caremark announced that it would discontinue sales of tobacco products in its pharmacies, claiming the sale of tobacco was inconsistent with the company's purpose to "help people on their path to better health."⁵ The policy went into effect in September 2014 in all CVS pharmacy locations, numbering over 7,700 nationwide.⁶ Prior to this policy change, many CVS locations sold tobacco products, displaying them prominently behind the checkout counter like many other pharmacies, convenience stores, and gas stations in displays that have been termed "power walls." While several local jurisdictions in Massachusetts and California had previously banned the sale of tobacco in pharmacies, this policy marked the first such change to be undertaken by a corporate pharmacy chain.

Hypotheses & Objective

There are multiple mechanisms by which the removal of tobacco products from CVS pharmacies may have impacted cigarette smoking. First, it is likely to have reduced impulse purchases of cigarettes by smokers visiting CVS pharmacies to purchase other items. Impulse purchases of cigarettes are relatively common; surveys of various populations have found that between 11 and 30 percent of cigarette purchases are unplanned,⁷⁻⁹ and these self-reports are likely to be underestimates.¹⁰ Impulse purchases can be spurred by seeing a product on display, as visual cues can prompt desire for a product and the choice to purchase it.¹¹⁻¹³ This relationship is certainly true with regards to cigarettes; several experimental studies have demonstrated that the provision of a cigarette-related visual cue induces cravings¹⁴⁻¹⁶ and increases smoking^{16,17} in comparison to neutral visual cues.

The power walls and tobacco sales at pharmacies present smokers, who may be visiting to purchase other items, with strong visual cues to purchase cigarettes at checkout. One survey, for instance, found that 25.2 percent of smokers reported sometimes purchasing cigarettes on

impulse as a result of seeing cigarettes on display.¹⁸ Qualitative work among former smokers and those trying to quit similarly suggests that encountering cigarettes on display brings on physical and emotions cravings to smoke.¹⁹ Industry research has reported that, on average, individuals make 35 visits to pharmacies annually,²⁰ thus there are many opportunities for such impulse purchases. The removal of tobacco products from CVS pharmacies and, therefore, the visual cues, is likely to have resulted in fewer impulsive cigarette purchases among pharmacy shoppers in areas in which CVS makes up a large portion of the pharmacy market. Evaluations of point-of-sale (POS) display bans in Canada and Australia, which require that stores selling cigarettes keep them out of sight of customers, suggest this to be true, finding that the implementation of these bans was associated with a decrease in reported spontaneous cigarette purchasing.^{7,21}

In addition, it is possible that the removal of tobacco products from CVS pharmacies and the public campaign that accompanied it contributed to greater social sanctioning against smoking. The ubiquitous retail presence of cigarettes can be seen as an indicator that they are popular and widely accepted products, a concept known as the perceived popularity effect.²² Cigarettes' presence in pharmacies may be especially powerful in terms of supporting their normativity, considering pharmacies sell otherwise health-promoting goods. CVS's sales ban may have decreased cigarettes' perceived popularity, as suggested again by evidence from POS display bans. Surveys of youth following a display ban in Ireland found that, after the ban went into effect, a smaller percentage of youth believed that many children their age smoked.²³ Similarly, in qualitative studies following the implementation of a ban in Norway, respondents reported that having to purchase cigarettes from concealed cabinets made them seem more illicit and unhealthy.²⁴ Smokers who live in areas in which CVS has a large market share and frequent CVS locations over other pharmacies are more likely to have experienced these changes in normativity.

H1: CVS Health's tobacco-free pharmacy policy will have reduced the number of cigarettes smoked per day by current smokers in accordance with the CVS pharmacy market share in their areas of residence.

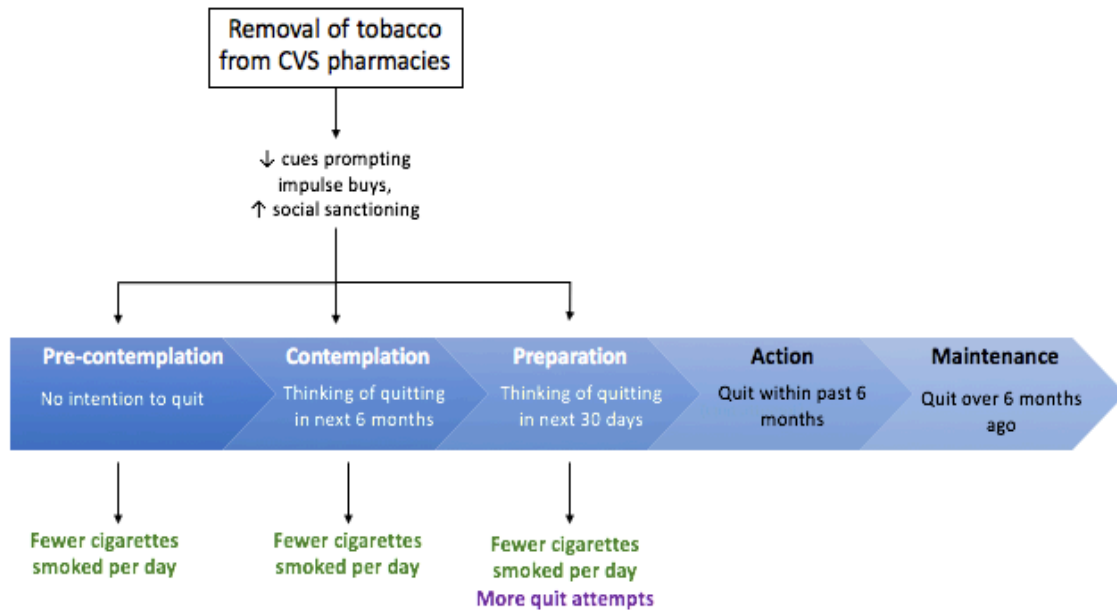
However, it is possible that this policy change had heterogeneous effects on individuals with different smoking behaviors. Most importantly, nondaily smokers are more likely to be impacted than daily smokers, as nondaily smokers are more likely to make impulse purchases than daily smokers.⁸ This is due to the fact that smoking is more strongly related to cues and cravings for nondaily smokers than for daily smokers.²⁵⁻²⁷ Daily and nondaily smokers certainly both experience cravings in response to cues.¹⁶ However, daily smokers often experience moderate cravings to smoke throughout the day,²⁶ suggesting they consistently feel a desire to smoke and have plans to purchase cigarettes and cues only marginally impact their already existing cravings. Conversely, nondaily smokers have few cravings in between smoking episodes.²⁶ Thus, cues are likely to more dramatically influence their cravings, which begin low, and prompt an impulse purchase. In survey work, daily smokers indicated craving, automaticity, and other dependence-related measures as their primary motives for smoking, while nondaily smokers indicated cue exposure and social goals as primary motives.²⁵ The impact of the availability of cigarettes on nondaily smokers' odds of smoking has been found to be almost three times as strong as its impact on daily smokers' odds of smoking.²⁸

H2: The effect of CVS Health's tobacco-free pharmacy policy on the number of cigarettes smoked per day will be stronger in magnitude among nondaily smokers than among daily smokers.

To date, two other studies have analyzed the impact of this CVS's policy change on cigarette purchasing and smoking. One study, conducted by CVS researchers, examined household purchases of tobacco and found that households that purchased cigarettes exclusively at CVS pharmacies were 38 percent more likely to stop purchasing cigarettes after the policy change compared to households that never purchased cigarettes at CVS pharmacies.²⁹ However, the vast majority of smokers purchase cigarettes in locations other than pharmacies; in surveys of current smokers only 5 percent reported that the last place they purchased cigarettes was a pharmacy.³⁰ Even within this particular study, only 2.1 percent of households were CVS exclusive purchasers.²⁹ Thus, findings about CVS-exclusive purchasers are not likely to be representative of a large population or speak to the broad impact of the policy. The study found no significant change in purchasing among households that purchased cigarettes in CVS and in other locations. The study also assessed changes in cigarette sales but did so at the state level, making it difficult to draw conclusions about individual behavior. The second study utilized data from the Behavioral Risk Factor Surveillance Survey to study the impact of the policy change on quit attempts and transitions from daily to nondaily smoking, finding that it significantly increased quit attempts but only among current smokers in urban counties in the highest quartile of CVS density.³¹

Ultimately, both studies had mixed findings. However, neither study considered how the policy change might differentially affect individuals with diverse smoking behaviors, whose varying responses could obscure results when considered together. Further, both studies focused on major behavior modifications (i.e. ceasing purchases for at least six months, quit attempts) that may not be particularly sensitive to the relatively subtle change in cigarette availability accomplished by the policy. The transtheoretical model (TTM) of behavior change suggests that change occurs in five stages: pre-contemplation, contemplation, preparation, action, and maintenance. This model has been used extensively to describe smoking and quit attempts and defines the pre-contemplation stage as when one has no plans to quit smoking, the contemplation stage as when one is thinking about quitting in the next six months, and the preparation stage as when one is thinking about quitting in the next 30 days and has taken some action to do so.³²⁻³⁴ While CVS's removal of tobacco products may have encouraged quitting through increased social sanctioning, a quit attempt requires that an individual be in the preparation stage, and it is estimated that only 20 percent of current smokers are in this stage. Approximately 40 percent of current smokers are estimated to be in the pre-contemplation phase and another 40 percent in the contemplation phase.^{35,36} If the hypothesized mechanisms described above are operating in the population, the impact of the CVS policy may be stronger on more minor behavior changes, such as smoking fewer cigarettes, as impulse buying and social sanctioning may still affect the purchases of smokers who do not plan to quit in the near future (see Figure 3.1). This possibility is supported by recent work on smoke-free home rules that found that such rules reduced the number of cigarettes smoked per day among smokers in the pre-contemplation and contemplation stages as well as the preparation stage.³⁷

Figure 3.1. Influence of CVS tobacco-free pharmacy policy on smokers at various stages of behavior change



Adapted from DiClemente et al. (1991), Prochaska et al. (1992)

This study will examine how the end of tobacco sales in CVS pharmacies impacted a smoking behavior that is pliable among a greater number of current smokers, the number of cigarettes smoked per smoking day, by daily smokers and by nondaily smokers across the spectrum of CVS pharmacy market share.

3.2 Methods

Data

The data on smoking behaviors are from the Tobacco Use Supplement to the Current Population Survey (TUS-CPS), an individual-level nationally representative survey that serves as a primary source of data on tobacco use and cessation behaviors, attitudes, and tobacco-related policies in the United States. The Tobacco Use Supplement is co-sponsored by the National Cancer Institute and the U.S. Food and Drug Administration and has been administered every three to four years since 1992 in conjunction with the U.S. Census Bureau's Current Population Survey, a monthly survey used to collect data on employment. All individuals who are over age 18 and in the civilian non-institutional population that completed the CPS are eligible for the TUS. Most respondents complete the interview for themselves, but some are interviewed by proxy.³⁸ For this analysis, I utilized the 2014-2015 survey, which was administered in three cross-sectional waves that straddled the CVS policy implementation: July 2014, January 2015, and May 2015. The response rates (total and self-respondents only, respectively) were 76.6 and

53.7 percent for July 2014, 77.8 and 55.7 percent for January 2015, and 75.0 and 53.1 percent for May 2015.³⁹

Data on CVS pharmacy market share are from the Blue Cross and Blue Shield (BCBS) Institute Community Health Management Hub (CHM Hub®), a proprietary software application that provides information on health outcomes and physical and socioeconomic characteristics of localities throughout the U.S. The BCBS Institute sources data from Blue Cross and Blue Shield health plan claims as well as the American Community Survey, the North American Industry Classification System, the USDA Economic Research Service, the Bureau of Transportation Statistics, and Nielsen Homescan.⁴⁰ Under a cooperative agreement, BCBS provided us with county-level counts of CVS pharmacies as well as other corporate pharmacies (Walgreens, Rite Aid) and the total number of pharmacies for all counties in the U.S. as of 2014, when the policy was implemented.

Data were linked at the Core-based Statistical Area (CBSA) level using CBSA codes. CBSAs are areas that comprise a county with an urban area of at least 10,000 residents and any surrounding counties in which 25 percent of residents commute to the central county for employment.⁴¹ To link the data at this level, I totaled the number of each type of pharmacy for all the counties within each CBSA that contained more than one county. Only CBSAs with a total population of over 100,000 were used for this analysis because the Census Bureau is prohibited from releasing geographic identifiers for respondents living in areas with fewer than 100,000 residents,⁴² making this the most granular geographic unit possible for analysis. While this restriction does limit the analysis to individuals living in more densely populated metropolitan areas, 85 percent of the U.S. population live in CBSAs with over 100,000 residents.⁴³ CBSAs with over 100,000 residents also have most of the large retail pharmacies. Of all the nonmetropolitan counties not represented in CBSAs, 86 percent had zero CVS locations at all, 13 percent had one location, and 1 percent had two locations. Of counties in micropolitan CBSAs, which include most of those with populations less than 100,000, 53 percent has zero CVS locations and another 32 percent had only one location.

The sample was restricted to self-respondents, as proxy respondents were not asked the full spectrum of tobacco use questions. The data contained records of 163,920 self-respondents. Of these self-respondents, 47,180 individuals (28.78 percent) were excluded for living outside eligible CBSAs; 35,347 of which resided in nonmetropolitan areas and 11,833 of which resided in metropolitan areas of fewer than 100,000 residents. An additional 5,706 individuals (3.48 percent) were excluded because they lived in municipalities that had existing tobacco-free pharmacy laws as of July 2014 (CBSAs of Barnstable, MA, Boston-Cambridge-Newton, MA, Springfield, MA, Worcester, MA, Santa Rosa-Petaluma, CA, San Francisco-Oakland-Fremont, CA, and San Jose-Sunnyvale-Santa Clara, CA). Finally, 503 individuals (0.31 percent) were eliminated for missing data on outcomes.

The resultant sample consisted of 111,034 individuals, 13,814 (12.44 percent) of which were current smokers, defined according to Centers for Disease Control and Prevention recommendations as those who reported that they had smoked at least 100 cigarettes in their lives and that they currently smoked some days or every day. Of these current smokers, 10,759 were daily smokers (77.88 percent) and 3,055 were nondaily smokers (22.12 percent).

Measures

To assess the number of cigarettes smoked per day, I utilized two TUS-CPS questions asked of current smokers. Those who report smoking every day are asked “on the average, about how many cigarettes do you now smoke each day.” Those who report smoking some days are asked “on the average, on those days [you did smoke], how many cigarettes did you usually smoke each day?”

CVS market share was measured as the percentage of all pharmacies in the CBSA that are CVS pharmacies.

A number of covariates were also sourced from the TUS-CPS. These covariates include age, gender, race/ethnicity, highest level of education achieved, and current income. Race/ethnicity was measured using a categorical variable with categories for white, non-Hispanic black, Asian/Pacific Islander, Hispanic, American Indian/Alaska Native, and multiracial. Educational achievement was assessed using a categorical variable with categories for less than high school, high school or GED, some college, and college degree or higher. Income was assessed with categories for less than \$20,000, \$20,000-34,999, \$35,000-49,999, \$50,000-74,999, and above \$75,000.

Statistical Analysis

The analysis utilized multiple difference-in-difference (DID) regressions to assess changes in the number of cigarettes smoked per day among daily and nondaily smokers after the policy’s implementation compared to before. First, I specified continuous DID models, which are similar to conventional DID models except that exposure to the policy is operationalized using a continuous measure that captures the intensity of the exposure rather than a binary variable that categorizes individuals as exposed or unexposed.⁴⁴⁻⁴⁸ Such models are preferable when the treatment is continuous and there is no theoretical reason to specify a particular cut point in the distribution; binary models with arbitrary cut points can obscure the effects of small changes in treatment intensity and risk incorrectly classifying observations that received smaller but still influential amounts of treatment as untreated, resulting in biased estimates.⁴⁹

These DID models were estimated using zero truncated negative binomial regression, as the outcomes were overdispersed count data and likelihood ratio tests that the dispersion parameter was equal to zero revealed that negative binomial models were better fit than poisson models.⁵⁰⁻⁵² Further, no zeros were observed because the questions are only asked to current smokers, making truncation necessary for consistent estimates.^{53,54}

The regressions were estimated according to the following model:

$$Y_{it} = \beta_0 + \beta_1 \text{CVS_percent}_i + \beta_2 \text{Post}_{it} + \beta_3 (\text{CVS_percent}_i \times \text{Post}_{it}) + \beta_4 \mathbf{X}_{it} + \beta_5 \text{State}_{it} + \epsilon_{it} \quad (1)$$

In which Y represents the outcome (the number of cigarettes smoked per day by daily smokers or the number of cigarettes smoked per smoking day by nondaily smokers) for individual i at time t, CVS_percent represents the percentage of pharmacies in individual i’s CBSA that are CVS pharmacies, and Post is an indicator that denotes whether individual i was surveyed prior to the policy implementation (0) or after (1). CVS_percentxPost, the parameter of

interest, represents the interaction of CVS_percent with Post. \mathbf{X} represents a vector of individual-level covariates previously described. The models also included state fixed effects to account for state-wide policies that may influence smoking behaviors (i.e. indoor smoking bans), which were important to include as CBSAs can cross state boundaries. Errors were clustered at the CBSA level.

The models were weighted according to the self-respondent nonresponse weights provided by the TUS-CPS in order to calculate the population average treatment effect rather than the sample average treatment effect.⁵⁵ While there is evidence that the use of sampling weights can result in inefficient estimates when the weights are a function of observed covariates^{56,57} and most of the variables used to generate the weights are included in these models, the TUS-CPS also adjusts the weights by geographical units smaller than CBSAs. In light of this and in an effort to be conservative with regard to type 1 error, I retained the weights in the models.

Second, in case the policy change had non-linear effects across the distribution of CVS market share, the same models were estimated using a categorical variable for CVS market share rather than a continuous variable. The distribution of CVS market share across CBSAs was divided into thirds, and individuals in CBSAs with zero CVS locations were considered unexposed and compared to individuals in each of the thirds using the following adaptation of Model 1:

$$Y_{it} = \beta_0 + \beta_1 \text{CVS_Third1}_i + \beta_2 \text{Post}_{it} + \beta_3 (\text{CVS_Third1}_i \times \text{Post}_{it}) + \beta_4 \text{CVS_Third2}_i + \beta_5 (\text{CVS_Third2}_i \times \text{Post}_{it}) + \beta_6 \text{CVS_Third3}_i + \beta_7 (\text{CVS_Third3}_i \times \text{Post}_{it}) + \beta_8 \mathbf{X}_{it} + \beta_9 \text{State}_i + \epsilon_{it} \quad (2)$$

In which CVS_Third1 represents an indicator for whether individual i resided in a CBSA in the lowest third of the CVS market share distribution (1) or a CBSA with zero CVS locations (0), CVS_Third2 is an indicator for an individual i residing in a CBSA in the middle third (1) or a CBSA with zero CVS locations (0), and CVS_Third3 is an indicator for an individual i residing in a CBSA in the highest third (1) or a CBSA with zero CVS locations (0).

The lowest third contained CBSAs in which CVS pharmacies made up between 0.31 to 10.09 percent of the pharmacy market, the middle third contained those with 10.17-15.53 percent market share, and the highest third contained those with 15.79-34.78 percent market share. The use of thirds was informed by locally weighted regressions of the number of cigarettes smoked per day on CVS market share in the pre- and post-implementation periods. Cut-offs for thirds more closely matched peaks and troughs than other quantiles. However, as a sensitivity analyses, another categorization was determined using a more data-driven approach in which the first category contained CBSA's in which CVS had between 0.31 and 6.87 percent market share, the second contained CBSAs in which CVS had between 6.87 and 15.53 percent market share, and the last category contained those in which CVS had greater than 15.53 percent market share. These categories more closely mapped to the peaks and troughs of the locally weighted regressions but did not contain equal numbers of CBSAs (see Figures 3.2 and 3.3).

Figure 3.2. Locally weighted regression of cigarettes smoked per day on CVS market share with category cut-off at thirds of market share

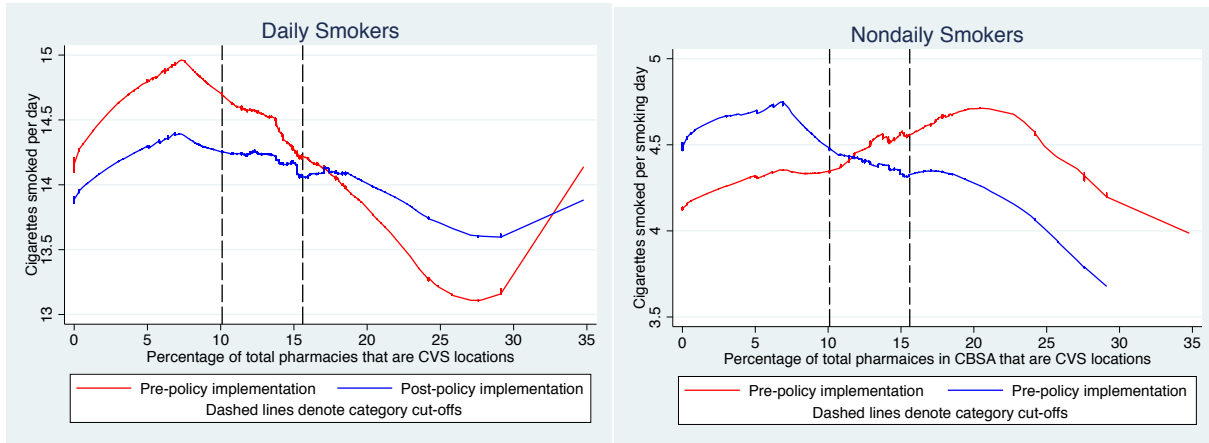
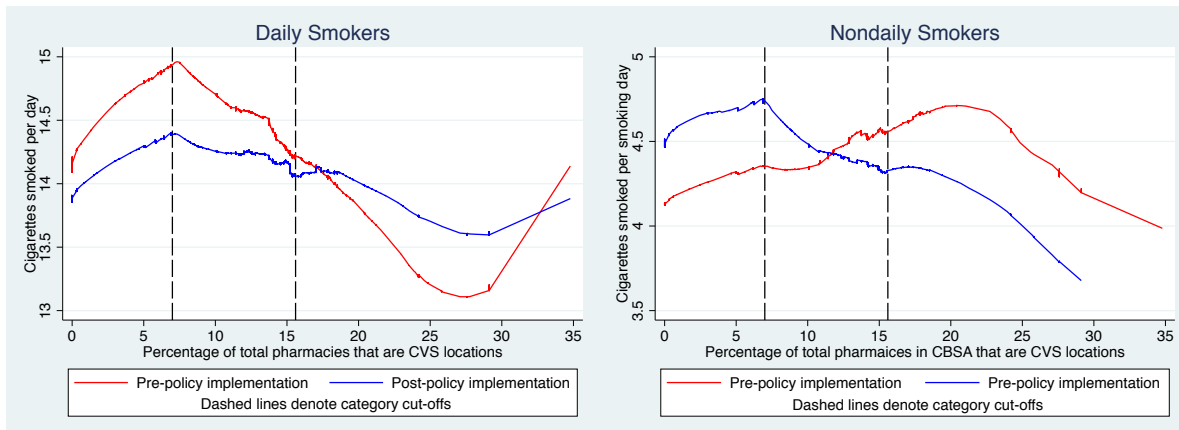


Figure 3.3 Locally weighted regression of cigarettes smoked per day on CVS market share with data-determined category cut-offs



Additional Sensitivity Analyses

Several additional sensitivity analyses were performed to assess the robustness of the models' results. First, in case CVS presence was serving as a proxy for a geographic predictor or correlate of smoking, the models were estimated excluding individuals in states where CVS had no presence at all (Colorado, Idaho, and South Dakota).

Second, the models additionally controlled for the price individuals reported paying for the last pack of cigarettes they purchased, as literature suggests that smokers are price sensitive and the price of cigarettes can be predictive of the number of cigarettes smoked per day.^{58,59} The inclusion of this variable also limited the analysis to smokers who reported usually purchasing their own cigarettes, rather than receiving them from another source, which is the population more likely to be affected by the policy change; those who do not purchase their own cigarettes

cannot make impulse purchases. The inclusion of this variable, however, resulted in reduced sample sizes as not all respondents answered this survey question.

Third, as a negative control, the models were specified using Rite Aid pharmacy market share rather than CVS market share. Theoretically, no effects should be seen on smoking using this exposure because Rite Aid made no changes to its tobacco sales. Rite Aid pharmacies are not as geographically widespread as CVS pharmacies, so these models were estimated using only individuals residing in states with some Rite Aid presence ($n=7,236$ daily smokers, $2,029$ nondaily smokers). Data on Walgreens, which has a broader market presence, was available but not utilized in a negative control because there were very few individuals residing in CBSAs with no Walgreens location, resulting a limited comparison group for the categorical DID. Walgreens market share was also negatively correlated with CVS market share ($\rho=-0.228$), while Rite Aid market share was not ($\rho=0.120$).

Finally, to adjust for selection effects, the models assessing changes among nondaily smokers were specified using propensity score methods. A potential pitfall of DID models, particularly when they rely on repeated cross-sections, is that the groups being evaluated may change in composition over time in characteristics that influence the outcome. In this scenario, simply controlling for the covariates in the regression is generally not effective at removing this selection bias.⁶⁰ When combined, the three waves of the TUS-CPS utilized in this analysis are representative at the national, state, and, to some degree, sub-state level. However, there is no guarantee that individuals surveyed from CBSAs with differing degrees of CVS market share in the first wave are similar in relevant characteristics to those sampled from comparable CBSAs in the second and third waves, especially when the sample is limited to daily or nondaily smokers.

To assess this possibility, I separated CBSAs into categories of CVS market share (again, thirds of the distribution and a separate category for CBSAs with zero CVS locations) and assessed differences in means in all of the included covariates for the analytic sample as a whole, for daily smokers, and for nondaily smokers in the pre-policy period compared to the post-policy period across each of these categories. The standardized differences in means were all below suggested thresholds^{61,62} for the entire sample and for daily smokers, but there was some imbalance for nondaily smokers (see Appendix Table A3.1).

In light of this imbalance, I utilized propensity score methods to create a sample of nondaily smokers in which those interviewed in the post-policy period were more comparable to those interviewed in the pre-policy period within categories of market share. Within each category, I used logistic regression to generate propensity scores for being surveyed in the pre-policy period versus the post-policy period controlling for age, gender, and categories of race/ethnicity, education, and income. I generated these scores by weighting the logistic regression by the non-response survey weight, by including the survey weight as an additional covariate in the regression, and by neglecting the weight entirely, as there is mixed evidence as to how best to incorporate survey weights in propensity scores.⁶³⁻⁶⁷ It has been suggested that whichever method achieves the best covariate balance should be used in estimating the treatment effect, as balance is the best predictor of model performance.⁶⁵ For each of these propensity score formulations, I tested four methods for sample creation: 1:1 nearest-neighbor matching without replacement, 1:1 nearest-neighbor caliper matching without replacement using a caliper of 0.2 times the standard deviation of the logit of the propensity score,⁶⁸ radius matching using

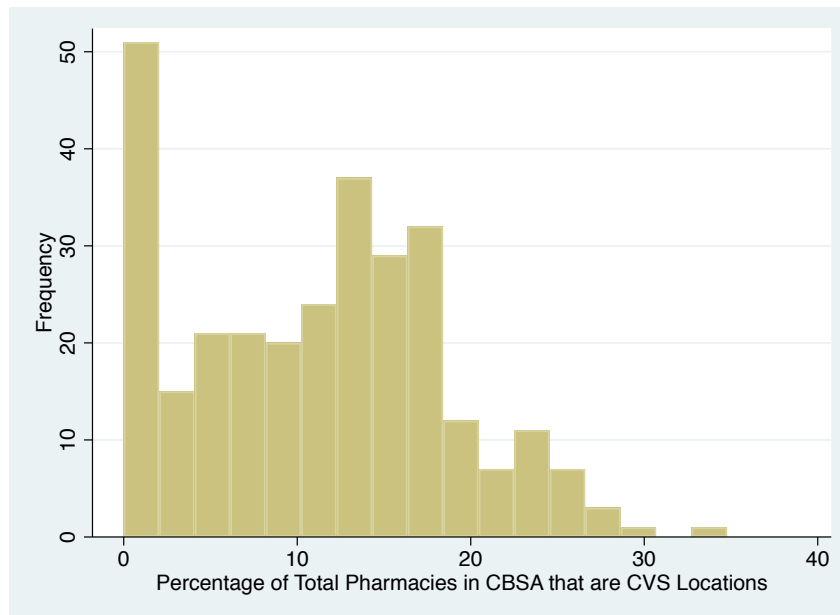
the same caliper, or weighting all post-policy observations to approximate pre-policy observations using treatment-on-the-treated propensity score weights.^{69,70} In this weighting scheme, pre-policy observations are given a weight of one and post-policy observations are given a weight of their propensity score divided by one minus the propensity score. The strategy that achieved the best balance was then used to define the sample for the continuous and categorical DID regressions.

3.3 Results

Descriptive Statistics

CVS made up a sizeable portion of the pharmacy market in 2014. Across the 292 CBSAs in the sample, CVS market share ranged from 0 to 34.78 percent, with a mean of 11.06 percent of a CBSA’s pharmacies. 5.52 and 5.49 percent of daily smokers and nondaily smokers, respectively, lived in CBSAs with zero CVS locations, 30.03 and 30.39 percent lived in CBSA in the lowest third, 39.67 and 41.97 percent lived in CBSAs in the middle third, and 24.77 and 22.15 percent lived in CBSAs in the highest third.

Figure 3.4. CVS pharmacy market share among Core-Based Statistical Areas (n=292)



At baseline, daily smokers reported smoking an average of 14 cigarettes per day (S.D.=7.9) and nondaily smokers reported smoking an average of 4 cigarettes per smoking day (S.D.=3.9). The average number reported declined for both groups in the post-implementation period, but neither of these decreases were statistically significant at the 0.05 level in tests of differences in means. Table 3.1 displays the sociodemographic characteristics of current smokers throughout the study period. Nondaily smokers were on average younger, more likely to be racial/ethnic minorities, more highly educated, and had higher incomes than daily smokers, which is consistent with previous research.⁷¹⁻⁷³

Table 3.1. Demographic and socioeconomic characteristics of current smokers

	<i>All current smokers (n=13,814)</i>	<i>Daily smokers (n=10,759)</i>	<i>Nondaily smokers (n=3,055)</i>
Cigarettes smoked per day	11.7	13.9	4.3
Age (mean)	44.3	45.3	40.9
Female (%)	45.2	45.9	42.8
Race/ethnicity (%)			
White	68.0	71.1	57.7
Black	14.5	13.6	17.5
Asian/PI	3.3	2.8	4.7
Hispanic	11.3	9.6	16.9
American Indian/Alaska Native	0.8	0.8	0.7
Multiracial	2.2	2.1	2.4
Education (%)			
< HS degree	15.7	16.4	13.5
HS degree/GED	38.0	40.1	31.3
Some college	32.9	32.6	34.0
College degree+	13.3	11.0	21.2
Income (%)			
<\$20,000	27.7	27.8	27.2
\$20,000-34,999	21.7	22.2	20.2
\$35,000-49,999	14.8	15.3	13.1
\$50,000-74,999	16.8	16.9	16.5
>\$75,000	19.0	17.8	23.1

Note. Estimates are weighted by self-only non-response weights.

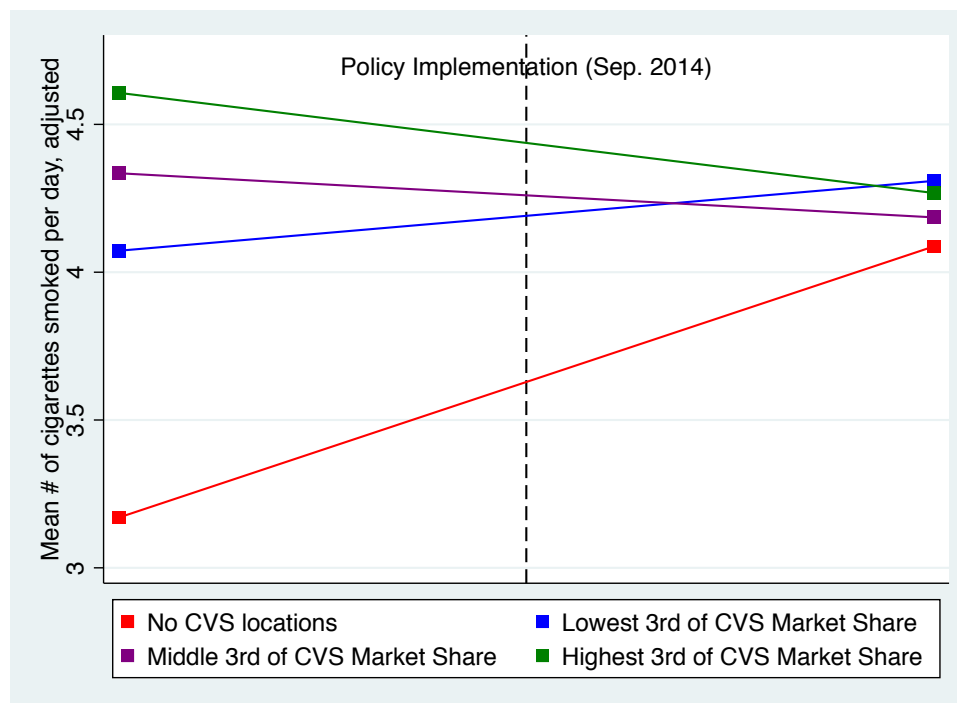
Source. Tobacco Use Supplement to the Current Population Survey (CPS-TUS) 2014-2015.

Model Results

Table 3.2 displays DID parameters for each of the main models. All model estimates are exponentiated and presented as rate ratios, meaning a DID parameter can be interpreted as the ratio of the interaction's two component rate ratios. The table shows that CVS' tobacco-free pharmacy policy was not significantly associated with differential changes in the number of cigarettes smoked by daily smokers in the continuous DID model nor in the categorical DID. However, both models identified a beneficial impact of the policy change on the number of cigarettes smoked by nondaily smokers. The continuous DID results indicated that the effect of a 1 percent increase in CVS pharmacy market share on the number of cigarettes smoked per smoking day was attenuated by 1.5 percent in the period following the policy's implementation (rate ratio=0.985, p=0.022).

Notably, the categorical DID signified a non-linear relationship. The results of this model indicate no differential change in the number of cigarettes smoked by nondaily smokers in CBSAs in the lowest category of CVS market share compared to nondaily smokers in CBSAs with no CVS presence. However, a significant policy impact stronger than that observed in the continuous DID was identified among nondaily smokers in CBSAs in the middle and highest categories (middle category: rate ratio=0.723, p=0.043; highest category: rate ratio=0.706, p=0.027). These results suggest that CVS must have a substantial footprint in the pharmacy market for the policy change to have an impact on the number of cigarettes smoked per day among nondaily smokers and that the constant dose response assumed in the continuous DID tempers the larger impact observed at greater degrees of market share.

Figure 3.5. Changes in mean number of cigarettes smoked per day by nondaily smokers after implementation of CVS tobacco-free pharmacy policy in each category of CVS market share



Note. Mean number of cigarettes per category determined using predictions obtained following adjusted regression.

Table 3.2. Difference-in-difference models of cigarettes smoked per day among current smokers

	<i>Daily smokers</i> (<i>n</i> =10,759)	<i>Nondaily smokers</i> (<i>n</i> =3,055)
<u>Continuous exposure¹</u>		
Post Policy x CVS%	1.002 (0.002) [0.153]	0.985* (-0.006) [0.022]
<u>Categorical Exposure²</u>		
Post Policy x CVS Category 1	0.980 (0.069) [0.774]	0.818 (0.126) [0.192]
Post Policy x CVS Category 2	1.035 (0.070) [0.612]	0.723* (0.116) [0.043]
Post Policy x CVS Category 3	1.042 (0.072) [0.549]	0.706* (0.111) [0.027]

Note. Presented are exponentiated coefficient estimates (rate ratios) of the difference-in-difference parameters using zero truncated negative binomial regression. ¹ denotes estimates from model 1 and ² denotes estimates from model 2, as specified in the text. Clustered standard errors are in parentheses and p-values are in brackets. Boldface indicates statistical significance (*p<0.05, **p<0.01, ***p<0.001).

Source. Tobacco Use Supplement to the Current Population Survey (TUS-CPS) 2014-2015, CBBSA Community Health Management Hub®.

Sensitivity Analyses

The estimation of the categorical DID with the alternative data-driven categories of CVS market share reinforced the use of thirds as categories in the original model, signifying that it does not result in arbitrary cut-points. The alternative categorization led to fewer individuals in the lowest category of CVS share and a greater number of individuals in the middle category. Again, 5.52 and 5.49 percent of daily smokers and nondaily smokers, respectively, lived in CBSAs with zero CVS locations, but 15.35 and 16.82 percent lived in CBSA in the lowest category, 54.36 and 55.54 percent lived in CBSAs in the middle category, and 24.77 and 22.15 percent lived in CBSAs in the highest category. However, the model estimated using these cut-points yielded results very consistent with those of the main models, with no significant effects among daily smokers and estimates of similar magnitude and significance among nondaily smokers (see Table 3.3).

The remaining sensitivity analyses generally supported the original findings. No specification found a significant impact of the policy among daily smokers. The results for nondaily smokers from each of the specifications are presented in Table 3.3. Upon limiting the analysis to states in which CVS has some presence to rule out geographic effects, the models found a significant effect when CVS market share was modeled continuously and in CBSAs in the middle and highest third of CVS market share when modeled categorically, although the effects in these categories were stronger in magnitude than the original estimates. The models that additionally controlled for the price paid for cigarettes in order to adjust for price sensitivity yielded estimates similar in magnitude and significance, with the exception being that the effect for individuals in CBSAs in the middle third of CVS market share was no longer significant at the 0.05 level. The negative control models estimated using Rite Aid market share as the predictor did not identify any statistically significant effects.

Table 3.3 Difference-in-difference estimates from sensitivity analyses assessing cigarettes smoked per day among nondaily smokers

	<i>Main analysis</i> (n=3,055)	<i>Data driven categories</i> (n=3,055)	<i>Only states with some CVS presence</i> (n=2,930)	<i>Controlling for price</i> (n=2,294)	<i>Propensity score weighted</i> (n=3,038)	<i>Rite Aid market share</i> (n=2,029)
<u>Continuous exposure¹</u>						
Post Policy x CVS%	0.985* (-0.006) [0.022]		0.984* (0.007) [0.025]	0.987* (-0.006) [0.035]	0.985* (0.006) [0.022]	1.010 (0.008) [0.165]
<u>Categorical Exposure²</u>						
Post Policy x CVS Category 1	0.818 (0.126) [0.192]	0.902 (0.150) [0.538]	0.676~ (0.153) [0.084]	0.796 (0.134) [0.176]	0.802 (0.131) [0.177]	0.919 (0.259) [0.765]
Post Policy x CVS Category 2	0.723* (0.116) [0.043]	0.719* (0.107) [0.027]	0.598* (0.136) [0.023]	0.733~ (0.131) [0.081]	0.698* (0.118) [0.033]	0.943 (0.271) [0.838]
Post Policy x CVS Category 3	0.706* (0.111) [0.027]	0.704* (0.111) [0.026]	0.584* (0.132) [0.018]	0.702* (0.114) [0.030]	0.695* (0.116) [0.029]	1.053 (0.300) [0.856]

Note. Presented are exponentiated coefficient estimates (rate ratios) of the difference-in-difference parameters using zero truncated negative binomial regression. ¹ denotes estimates from model 1 and ² denotes estimates from model 2, as specified in the text. Clustered standard errors are in parentheses and p-values are presented in brackets. Boldface indicates statistical significance at the 0.05 level (~p<0.10, *p<0.05, **p<0.01, ***p<0.001).

Source. Tobacco Use Supplement to the Current Population Survey (CPS-TUS) 2014-2015, BCBSA Community Health Management Hub®.

With regards to propensity score methods, the best covariate balance was achieved by generating the scores using weighted logistic regression to incorporate the survey weights and using radius caliper matching. All 12 strategies reduced the imbalance substantially, but this

combination was the only one able to achieve standardized differences in means below 0.25 for all covariates. This strategy resulted in a sample of 3,038 observations, with 1,060 pre-period nondaily smokers (13 excluded for being off the common support) matched to 1,978 post-period nondaily smokers. Thus, it had the additional benefit of retaining power compared to the 1:1 matched sample alternatives. The DID models were estimated on this sample, weighting the final models by each observation's frequency weight multiplied by its survey weight.^{63,64} The results of both models were consistent with those of the main models, although the effects identified in the middle and highest third in the categorical model are slightly stronger in magnitude than in the original model.

3.4 Discussion

The findings from this analysis indicate that the removal of tobacco products from CVS pharmacies reduced the number of cigarettes smoked per day among nondaily smokers, particularly in areas in which CVS has a substantial pharmacy market share. While no impact was identified among daily smokers, the impact among nondaily smokers is notable as nondaily smokers comprise an important group that requires more attention. While the overall number of current smokers has declined in recent years, the number of nondaily smokers has increased.⁷⁴ This change does not simply represent numbers of daily smokers reducing their smoking and becoming nondaily smokers. Some nondaily smokers are newly initiated or transitioning from more intensive smoking, but almost half of nondaily smokers are stable in their habits, having been smoking nondaily for at least a year. Of these stable smokers, over 75 percent have been stable for at least five years.⁷¹ Crucially, nondaily smokers tend to be missed by clinical smoking cessation efforts. Many nondaily smokers do not identify as smokers⁷⁵⁻⁷⁷ and they are less likely than daily smokers to be asked by physicians about their tobacco use or be advised to quit.⁷² If they do decide to quit or cut back, pharmacotherapies designed to counter nicotine withdrawal are, for the most part, untested among and have unknown effectiveness for nondaily smokers, who often do not experience nicotine dependence.⁷⁶ Further, with different motivations for smoking²⁵ and different perceptions of health risks than daily smokers,^{78,79} they may not be receptive to standard cessation messages. Thus, any intervention, including removing tobacco from pharmacies, that might assist nondaily smokers in reducing their tobacco use has unique value.

It is important to note that the effects identified in this study are modest in comparison to those of other tobacco control policies⁸⁰⁻⁸² and some pharmacological and behavioral interventions.⁸³⁻⁸⁵ Using the estimates from the categorical DID, nondaily smokers living in CBSAs in the middle third and highest third of CVS market share are predicted to smoke 0.25 and 0.42 fewer cigarettes on smoking days, respectively, after implementation compared to before, holding all other covariates constant at their means or their most populous category. These are certainly small behavioral changes. However, they may still result in improvements in health. There is no safe amount of cigarette consumption; adverse health effects can result from even low levels of smoking and nondaily smokers have higher risk of death than never smokers, even if not quite the risk of daily smokers.^{86,87} However, while evidence is mixed, research indicates that reducing the number of cigarettes smoked per day is associated with reduced risk of lung cancer incidence, lung cancer mortality, and all-cause mortality,⁸⁸⁻⁹⁰ even without fully quitting. Further, reductions in smoking have been associated with future cessation.^{91,92} Certainly, evidence is lacking for diminutions of this small magnitude and for nondaily smokers

specifically, but reductions of any amount are promising. It is also possible that the effect might be more substantial if tobacco were to be removed from all pharmacies, rather than only CVS locations.

This analysis had some important limitations. First, the time period and small number of interview waves included in the TUS-CPS limited the analysis to short-term impacts as well as precluded my ability to assess some DID assumptions, including parallel pre-treatment trends, and adjust for trends over time. Unfortunately, the TUS-CPS is conducted only every three to four years, so the inclusion of prior data would not have been particularly useful for trend analysis, but similar analyses should be conducted if more robust longitudinal data become available. Second, without more nuanced survey questions, the analysis was unable to examine the exact mechanisms underlying the reduction in smoking (i.e. fewer impulse purchases, increased social sanctioning, etc.). Third, as noted, the analysis was restricted to individuals in CBSAs with at least 100,000 residents due to the availability of geographic identifiers, which certainly limits the generalizability of its findings. This limitation was the result of a necessary trade-off in order to be able to study the number of cigarettes smoked per day by current smokers. The Behavioral Risk Factor Surveillance System provides more granular geographic information with specially granted data access, but this survey does not ask about more minor tobacco-related behavior changes, only about current smoking status and quit attempts.⁹³ While the CBSAs under study are the areas most likely to contain CVS pharmacies and be impacted by the policy change, the analysis should be extended to individuals in all areas with any CVS presence if these data are available in the future.

3.5 Conclusion

This analysis suggests that the removal of tobacco products from CVS pharmacies in 2014 resulted in nondaily smokers consuming fewer cigarettes per smoking day, particularly in CBSAs in which CVS has a larger share of the pharmacy market. It is possible this decrease was accomplished by reducing the cues that prompt impulse purchases and by increasing social sanctioning against smoking, which could impact nondaily smokers across multiple stages of behavior change, including those not actively interested in quitting smoking. These findings are noteworthy as they suggest a benefit for a population that is still at high risk of adverse health outcomes but that is generally overlooked in attempts to encourage smoking cessation from the health care system. Similar policies should be considered by other corporate pharmacies that have a substantial market share as well as by governmental policy-makers considering state or municipality-level tobacco free pharmacy policies, as they may be a promising tactic for reducing smoking and subsequent chronic disease amongst this population at relatively little cost to society.

3.6 References

1. U.S. Department of Health and Human Services. *The Health Consequences of Smoking - 50 Years of Progress: A Report of the Surgeon General*. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; 2014.
2. Creamer MR, Wang TW, Babb S, et al. Tobacco product use and cessation indicators among adults—United States, 2018. *Morbidity and Mortality Weekly Report*. 2019;68(45):1013.
3. Seidenberg AB, Behm I, Rees VW, Connolly GN. Cigarette sales in pharmacies in the USA (2005–2009). *Tobacco control*. 2012;21(5):509–510.
4. Henriksen L, Schleicher NC, Barker DC, Liu Y, Chaloupka FJ. Prices for tobacco and nontobacco products in pharmacies versus other stores: results from retail marketing surveillance in California and in the United States. *American journal of public health*. 2016;106(10):1858–1864.
5. PRNewswire. CVS Caremark to Stop Selling Tobacco at all CVS/pharmacy Locations. <https://cvshealth.com/newsroom/press-releases/cvs-caremark-stop-selling-tobacco-all-cvspharmacy-locations>. Published February 5, 2014.
6. CVS Health. We're Tobacco Free. September 2014. <https://www.cvshealth.com/thought-leadership/we-are-tobacco-free>.
7. Carter OBJ, Phan T, Mills BW. Impact of a point-of-sale tobacco display ban on smokers' spontaneous purchases: comparisons from postpurchase interviews before and after the ban in Western Australia. *Tobacco Control*. 2015;24(e1):e81-e86. doi:10.1136/tobaccocontrol-2013-050991
8. Clattenburg EJ, Elf JL, Apelberg BJ. Unplanned cigarette purchases and tobacco point of sale advertising: a potential barrier to smoking cessation. *Tobacco Control*. 2013;22(6):376-381. doi:10.1136/tobaccocontrol-2012-050427
9. Wood L, Gazey A, Murray K, Kennington K. Unplanned purchasing of tobacco products: Beyond point of sale display. *Health Promotion Journal of Australia*. June 2019. doi:10.1002/hpja.260
10. Robertson L, McGee R, Marsh L, Hoek J. A systematic review on the impact of point-of-sale tobacco promotion on smoking. *Nicotine Tob Res*. 2015;17(1):2-17. doi:10.1093/ntr/ntu168
11. Laibson D. A cue-theory of consumption. *The Quarterly Journal of Economics*. 2001;116(1):81–119.

12. Milosavljevic M, Navalpakkam V, Koch C, Rangel A. Relative visual saliency differences induce sizable bias in consumer choice. *Journal of Consumer Psychology*. 2012;22(1):67–74.
13. Armel KC, Beaumel A, Rangel A. Biasing simple choices by manipulating relative visual attention. *Judgment and Decision making*. 2008;3(5):396–403.
14. Carter B, Robinson J, Lam C, et al. A psychometric evaluation of cigarette stimuli used in a cue reactivity study. *Nicotine & Tobacco Research*. 2006;8(3):361-369. doi:10.1080/14622200600670215
15. Engelmann JM, Versace F, Robinson JD, et al. Neural substrates of smoking cue reactivity: a meta-analysis of fMRI studies. *Neuroimage*. 2012;60(1):252-262. doi:10.1016/j.neuroimage.2011.12.024
16. Shiffman S, Dunbar M, Kirchner T, et al. Smoker reactivity to cues: effects on craving and on smoking behavior. *J Abnorm Psychol*. 2013;122(1):264-280. doi:10.1037/a0028339
17. Conklin CA, Vella EJ, Joyce CJ, Salkeld RP, Perkins KA, Parzynski CS. Examining the relationship between cue-induced craving and actual smoking. *Experimental and Clinical Psychopharmacology*. 2015;23(2):90-96. doi:10.1037/a0038826
18. Wakefield M, Germain D, Henriksen L. The effect of retail cigarette pack displays on impulse purchase. *Addiction*. 2008;103(2):322-328. doi:10.1111/j.1360-0443.2007.02062.x
19. Hoek J, Gifford H, Pirikahu G, Thomson G, Edwards R. How do tobacco retail displays affect cessation attempts? Findings from a qualitative study. *Tobacco Control*. 2010;19(4):334-337. doi:10.1136/tc.2009.031203
20. AmerisourceBergen Corporation. *AmerisourceBergen's Inaugural Pharmacy Check-Up Survey Finds That Pharmacists Want & Expect to Expand Role in Patient Care, but Burdens Must Lessen*. Chesterbrook, PA: AmerisourceBergen Corporation; 2018. <https://www.amerisourcebergen.com/newsroom/press-releases/survey>.
21. Li L, Borland R, Fong GT, Thrasher JF, Hammond D, Cummings KM. Impact of point-of-sale tobacco display bans: findings from the International Tobacco Control Four Country Survey. *Health Education Research*. 2013;28(5):898-910. doi:10.1093/her/cyt058
22. Pollay RW. More than meets the eye: on the importance of retail cigarette merchandising. *Tobacco Control*. 2007;16(4):270-274. doi:10.1136/tc.2006.018978
23. McNeill A, Lewis S, Quinn C, et al. Evaluation of the removal of point-of-sale tobacco displays in Ireland. *Tobacco Control*. 2011;20(2):137-143. doi:10.1136/tc.2010.038141
24. Scheffels J, Lavik R. Out of sight, out of mind? Removal of point-of-sale tobacco displays in Norway. *Tobacco Control*. 2013;22(e1):e37-e42. doi:10.1136/tobaccocontrol-2011-050341

25. Shiffman S, Dunbar MS, Scholl SM, Tindle HA. Smoking motives of daily and non-daily smokers: A profile analysis. *Drug and Alcohol Dependence*. 2012;126(3):362-368. doi:10.1016/j.drugalcdep.2012.05.037
26. Shiffman S, Dunbar MS, Li X, et al. Craving in Intermittent and Daily Smokers During Ad Libitum Smoking. *Nicotine & Tobacco Research*. 2014;16(8):1063-1069. doi:10.1093/ntr/ntu023
27. Shiffman S, Ferguson SG, Dunbar MS, Scholl SM. Tobacco Dependence Among Intermittent Smokers. *Nicotine & Tobacco Research*. 2012;14(11):1372-1381. doi:10.1093/ntr/nts097
28. Shiffman S, Dunbar MS, Li X, et al. Smoking Patterns and Stimulus Control in Intermittent and Daily Smokers. Zhang XY, ed. *PLoS ONE*. 2014;9(3):e89911. doi:10.1371/journal.pone.0089911
29. Polinski JM, Howell B, Gagnon MA, Kymes SM, Brennan TA, Shrank WH. Impact of CVS Pharmacy's Discontinuance of Tobacco Sales on Cigarette Purchasing (2012–2014). *American journal of public health*. 2017;107(4):556–562.
30. Kruger J, Jama A, Lee JGL, et al. Point-of-sale cigarette purchase patterns among U.S. adult smokers—National Adult Tobacco Survey, 2012–2014. *Preventive Medicine*. 2017;101:38-43. doi:10.1016/j.ypmed.2017.05.005
31. Ali FRM, Neff L, Wang X, et al. Tobacco-Free Pharmacies and U.S. Adult Smoking Behavior: Evidence From CVS Health's Removal of Tobacco Sales. *American Journal of Preventive Medicine*. 2020;58(1):41-49. doi:10.1016/j.amepre.2019.09.003
32. DiClemente CC, Prochaska JO, Fairhurst SK, Velicer WF, Velasquez MM, Rossi JS. The process of smoking cessation: An analysis of precontemplation, contemplation, and preparation stages of change. *Journal of Consulting and Clinical Psychology*. 1991;59(2):295-304. doi:10.1037/0022-006X.59.2.295
33. Prochaska JO, DiClemente CC, Norcross JC. In search of how people change: Applications to addictive behaviors. *American Psychologist*. 1992;47(9):1102-1114. doi:10.1037/0003-066X.47.9.1102
34. Prochaska JO, DiClemente CC. *The Transtheoretical Approach: Crossing Traditional Boundaries of Therapy*. Homewood, IL: Dow Jones Irwin; 1984.
35. Babb S, Malarcher A, Schauer G, Asman K, Jamal A. Quitting Smoking Among Adults — United States, 2000–2015. *MMWR Morbidity and Mortality Weekly Report*. 2017;65(52):1457-1464. doi:10.15585/mmwr.mm6552a1
36. Velicer WF, Fava JL, Prochaska JO, Abrams DB, Emmons KM, Pierce JP. Distribution of Smokers by Stage in Three Representative Samples. *Preventive Medicine*. 1995;24(4):401-411. doi:10.1006/pmed.1995.1065

37. Owusu D, Quinn M, Wang K, Williams F, Mamudu HM. Smokefree home rules and cigarette smoking intensity among smokers in different stages of smoking cessation from 20 low-and-middle income countries. *Preventive Medicine*. 2020;132:106000. doi:10.1016/j.ypmed.2020.106000
38. National Cancer Institute, Division of Cancer Control and Population Sciences. The Tobacco Use Supplement to the Current Population Survey. November 2019. <https://cancercontrol.cancer.gov/brp/tcrb/tus-cps/>.
39. U.S. Department of Commerce, Census Bureau. Tobacco Use Supplement to the Current Population Survey Technical Documentation, July 2014-May 2015. 2017. <https://cancercontrol.cancer.gov/brp/tcrb/tus-cps/questionnaires.html>.
40. Blue Cross Blue Shield Institute. *Community Health Management Hub.*; 2017. <https://app.chmhub.com/>.
41. Office of Management and Budget. *2010 Standards for Delineating Metropolitan and Micropolitan Statistical Areas.*; 2010. <https://www.govinfo.gov/content/pkg/FR-2010-06-28/pdf/2010-15605.pdf>.
42. National Cancer Institute, Division of Cancer Control & Population Sciences. TUS-CPS Frequently Asked Questions. February 2020. <https://cancercontrol.cancer.gov/brp/tcrb/tus-cps/faq.html>.
43. United States Census Bureau. *American FactFinder.*; 2019. <https://factfinder.census.gov/faces/nav/jsf/pages/index.xhtml>.
44. Acemoglu D, Autor DH, Lyle D. Women, War, and Wages: The Effect of Female Labor Supply on the Wage Structure at Midcentury. *Journal of Political Economy*. 2004;112(3):497-551. doi:10.1086/383100
45. Allen T, Whittaker W, Sutton M. Does the proportion of pay linked to performance affect the job satisfaction of general practitioners? *Social Science & Medicine*. 2017;173:9-17. doi:10.1016/j.socscimed.2016.11.028
46. Card D. Using Regional Variation in Wages to Measure the Effects of the Federal Minimum Wage. *ILR Review*. 1992;46(1):22-37. doi:10.1177/001979399204600103
47. Gaynor M, Moreno-Serra R, Propper C. Death by Market Power: Reform, Competition, and Patient Outcomes in the National Health Service. *American Economic Journal: Economic Policy*. 2013;5(4):134-166. doi:10.1257/pol.5.4.134
48. Nunn N, Qian N. The Potato's Contribution to Population and Urbanization: Evidence From A Historical Experiment. *The Quarterly Journal of Economics*. 2011;126(2):593-650. doi:10.1093/qje/qjr009

49. Han B, Yu H. Causal difference-in-differences estimation for evaluating the impact of semi-continuous medical home scores on health care for children. *Health Services and Outcomes Research Methodology*. 2019;19(1):61-78. doi:10.1007/s10742-018-00195-9
50. Cameron AC, Trivedi PK. Econometric models based on count data. Comparisons and applications of some estimators and tests. *Journal of Applied Econometrics*. 1986;1(1):29-53. doi:10.1002/jae.3950010104
51. Hausman J, Hall BH, Griliches Z. Econometric Models for Count Data with an Application to the Patents-R & D Relationship. *Econometrica*. 1984;52(4):909. doi:10.2307/1911191
52. Johnson S, Kotz N. *Discrete Distributions*. Houghton Mifflin Company; 1969.
53. Grogger JT, Carson RT. Models for truncated counts. *Journal of applied econometrics*. 1991;6(3):225–238.
54. Shaw D. On-site samples' regression: Problems of non-negative integers, truncation, and endogenous stratification. *Journal of Econometrics*. 1988;37(2):211–223.
55. Imai K, King G, Stuart EA. Misunderstandings between experimentalists and observationalists about causal inference. *Journal of the Royal Statistical Society: Series A (Statistics in Society)*. 2008;171(2):481-502. doi:10.1111/j.1467-985X.2007.00527.x
56. Solon G, Haider SJ, Wooldridge JM. What are we weighting for? *Journal of Human resources*. 2015;50(2):301–316.
57. Winship C, Radbill L. Sampling weights and regression analysis. *Sociological Methods & Research*. 1994;23(2):230–257.
58. Cavazos-Rehg PA, Krauss MJ, Spitznagel EL, et al. Differential effects of cigarette price changes on adult smoking behaviours. *Tobacco Control*. 2014;23(2):113-118. doi:10.1136/tobaccocontrol-2012-050517
59. Franz GA. Price effects on the smoking behaviour of adult age groups. *Public Health*. 2008;122(12):1343-1348. doi:10.1016/j.puhe.2008.05.019
60. Stuart EA, Huskamp HA, Duckworth K, et al. Using propensity scores in difference-in-differences models to estimate the effects of a policy change. *Health Services and Outcomes Research Methodology*. 2014;14(4):166–182.
61. Cohen J. *Statistical Power Analysis for the Behavioral Sciences*. Routledge; 2013.
62. Rubin DB. Using propensity scores to help design observational studies: application to the tobacco litigation. *Health Services and Outcomes Research Methodology*. 2001;2(3-4):169–188.
63. Austin PC, Jembere N, Chiu M. Propensity score matching and complex surveys. *Statistical Methods in Medical Research*. 2018;27(4):1240-1257. doi:10.1177/0962280216658920

64. DuGoff EH, Schuler M, Stuart EA. Generalizing Observational Study Results: Applying Propensity Score Methods to Complex Surveys. *Health Services Research*. 2014;49(1):284-303. doi:10.1111/1475-6773.12090
65. Lenis D, Nguyen TQ, Dong N, Stuart EA. It's all about balance: propensity score matching in the context of complex survey data. *Biostatistics*. 2019;20(1):147-163. doi:10.1093/biostatistics/kxx063
66. Ridgeway G, Kovalchik SA, Griffin BA, Kabeto MU. Propensity Score Analysis with Survey Weighted Data. *Journal of Causal Inference*. 2015;3(2). doi:10.1515/jci-2014-0039
67. Zanutto EL. A comparison of propensity score and linear regression analysis of complex survey data. *Journal of data Science*. 2006;4(1):67–91.
68. Austin PC. Some Methods of Propensity-Score Matching had Superior Performance to Others: Results of an Empirical Investigation and Monte Carlo simulations. *Biometrical Journal*. 2009;51(1):171-184. doi:10.1002/bimj.200810488
69. Hirano K, Imbens GW, Ridder G. Efficient estimation of average treatment effects using the estimated propensity score. *Econometrica*. 2003;71(4):1161–1189.
70. Morgan SL, Todd JJ. A Diagnostic Routine for the Detection of Consequential Heterogeneity of Causal Effects. *Sociological Methodology*. 2008;38(1):231-282. doi:10.1111/j.1467-9531.2008.00204.x
71. Hassmiller KM, Warner KE, Mendez D, Levy DT, Romano E. Nondaily smokers: who are they? *Am J Public Health*. 2003;93(8):1321-1327. doi:10.2105/ajph.93.8.1321
72. Tong EK, Ong MK, Vittinghoff E, Pérez-Stable EJ. Nondaily smokers should be asked and advised to quit. *American journal of preventive medicine*. 2006;30(1):23–30.
73. Wang Y, Sung H-Y, Yao T, Lightwood J, Max W. Infrequent and Frequent Nondaily Smokers and Daily Smokers: Their Characteristics and Other Tobacco Use Patterns. *Nicotine & Tobacco Research*. 2018;20(6):741-748. doi:10.1093/ntr/ntx038
74. Jamal A, Homa D, O'Connor E, et al. *Current Cigarette Smoking among Adults - United States, 2005-2014*. Office on Smoking and Health, National Center for Chronic Disease Prevention and Health Promotion, Centers for Disease Control and Prevention; 2015. <https://www.cdc.gov/mmwr/pdf/wk/mm6444.pdf>.
75. Lee JK, Boyle RG, D'Silva J, St. Claire AW, Whittet MN, Kinney AM. Smoker Identity Among Occasional Smokers: Findings From Minnesota. *American Journal of Health Behavior*. 2013;37(4):525-530. doi:10.5993/AJHB.37.4.10
76. Schane RE, Glantz SA, Ling PM. Nondaily and Social Smoking: An Increasingly Prevalent Pattern. *Archives of Internal Medicine*. 2009;169(19). doi:10.1001/archinternmed.2009.315

77. Thompson B, Coronado G, Chen L, et al. Prevalence and characteristics of smokers at 30 Pacific Northwest colleges and universities. *Nicotine & Tobacco Research*. 2007;9(3):429-438. doi:10.1080/14622200701188844
78. Schane RE, Prochaska JJ, Glantz SA. Counseling Nondaily Smokers about Secondhand Smoke as a Cessation Message: A Pilot Randomized Trial. *Nicotine & Tobacco Research*. 2013;15(2):334-342. doi:10.1093/ntr/nts126
79. Schane RE, Glantz SA, Ling PM. Social Smoking. *American Journal of Preventive Medicine*. 2009;37(2):124-131. doi:10.1016/j.amepre.2009.03.020
80. Azagba S, Shan L, Latham K. County Smoke-Free Laws and Cigarette Smoking Among U.S. Adults, 1995–2015. *American Journal of Preventive Medicine*. 2020;58(1):97-106. doi:10.1016/j.amepre.2019.08.025
81. Fichtenberg CM. Effect of smoke-free workplaces on smoking behaviour: systematic review. *BMJ*. 2002;325(7357):188-188. doi:10.1136/bmj.325.7357.188
82. van Hasselt M, Kruger J, Han B, et al. The relation between tobacco taxes and youth and young adult smoking: What happened following the 2009 U.S. federal tax increase on cigarettes? *Addictive Behaviors*. 2015;45:104-109. doi:10.1016/j.addbeh.2015.01.023
83. Ellerbeck EF, Nollen N, Hutcheson TD, et al. Effect of Long-term Nicotine Replacement Therapy vs Standard Smoking Cessation for Smokers With Chronic Lung Disease: A Randomized Clinical Trial. *JAMA Network Open*. 2018;1(5):e181843. doi:10.1001/jamanetworkopen.2018.1843
84. Fagerström KO, Hughes J, Callas P. Long-term effects of the Eclipse cigarette substitute and the nicotine inhaler in smokers not interested in quitting. *Nicotine & Tobacco Research*. 2002;4(4):141-145. doi:10.1080/1462220021000032771
85. Liao Y, Wu Q, Kelly BC, et al. Effectiveness of a text-messaging-based smoking cessation intervention (“Happy Quit”) for smoking cessation in China: A randomized controlled trial. Degenhardt L, ed. *PLOS Medicine*. 2018;15(12):e1002713. doi:10.1371/journal.pmed.1002713
86. Inoue-Choi M, McNeel TS, Hartge P, Caporaso NE, Graubard BI, Freedman ND. Non-Daily Cigarette Smokers: Mortality Risks in the U.S. *American Journal of Preventive Medicine*. 2019;56(1):27-37. doi:10.1016/j.amepre.2018.06.025
87. Schane RE, Ling PM, Glantz SA. Health Effects of Light and Intermittent Smoking: A Review. *Circulation*. 2010;121(13):1518-1522. doi:10.1161/CIRCULATIONAHA.109.904235
88. Godtfredsen NS. Effect of Smoking Reduction on Lung Cancer Risk. *JAMA*. 2005;294(12):1505. doi:10.1001/jama.294.12.1505

89. Inoue-Choi M, Hartge P, Park Y, Abnet CC, Freedman ND. Association Between Reductions of Number of Cigarettes Smoked per Day and Mortality Among Older Adults in the United States. *American Journal of Epidemiology*. 2019;188(2):363-371. doi:10.1093/aje/kwy227
90. Song Y-M, Sung J, Cho H-J. Reduction and cessation of cigarette smoking and risk of cancer: a cohort study of Korean men. *Journal of clinical oncology*. 2008;26(31):5101–5106.
91. Broms U, Korhonen T, Kaprio J. Smoking reduction predicts cessation: Longitudinal evidence from the Finnish adult twin cohort. *Nicotine & Tobacco Research*. 2008;10(3):423-427. doi:10.1080/14622200801888988
92. Klemperer EM, Hughes JR. Does the Magnitude of Reduction in Cigarettes Per Day Predict Smoking Cessation? A Qualitative Review. *Nicotine & Tobacco Research*. March 2015:ntv058. doi:10.1093/ntr/ntv058
93. Centers for Disease Control and Prevention. Behavioral Risk Factor Surveillance System Survey Questionnaire. 2016 2015. <https://www.cdc.gov/brfss/questionnaires/index.htm>.

3.7 Appendix

Table A3.1. Covariate balance among all current smokers, daily smokers, and nondaily smokers in each category of CVS market share

	<i>All current smokers</i> (n=13,814)	<i>Daily smokers</i> (n=10,759)	<i>Nondaily smokers</i> (n=3,055)
Zero CVS Presence			
Age	-0.092	-0.155	0.101
Female	-0.038	-0.027	-0.072
Race/ethnicity			
White	-0.165	-0.055	-0.478
Black	0.075	-0.025	0.293
Asian/PI	0.054	0.008	0.189
Hispanic	0.149	0.069	0.353
American Indian/Alaska Native	0.066	0.070	0.056
Multiracial	-0.048	-0.014	-0.199
Education			
< HS degree	-0.008	-0.102	0.398
HS degree/GED	-0.029	-0.010	-0.094
Some college	0.139	0.149	0.107
College degree+	-0.153	-0.114	-0.263
Income			
<\$20,000	0.006	-0.005	0.044
\$20,000-34,999	-0.016	0.037	-0.214
\$35,000-49,999	0.177	0.132	0.313
\$50,000-74,999	-0.004	-0.018	0.038
>\$75,000	-0.163	-0.157	-0.186
Lowest 3rd of CVS Market Share			
Age	0.018	0.028	0.012
Female	-0.012	-0.013	-0.003
Race/ethnicity			
White	0.010	0.007	0.036
Black	0.014	-0.041	0.161
Asian/PI	-0.026	-0.002	-0.101
Hispanic	-0.011	0.028	-0.125
American Indian/Alaska Native	0.002	0.015	-0.042
Multiracial	-0.012	0.015	-0.091
Education			
< HS degree	-0.061	-0.079	0.012
HS degree/GED	0.082	0.087	0.082
Some college	-0.047	-0.039	-0.076
College degree+	0.012	0.013	-0.014
Income			
<\$20,000	-0.033	-0.024	-0.057
\$20,000-34,999	-0.008	0.011	-0.066
\$35,000-49,999	0.060	0.055	0.079
\$50,000-74,999	-0.003	-0.003	-0.003
>\$75,000	-0.007	-0.032	0.058
Middle 3rd of CVS Market Share			
Age	-0.005	-0.027	0.019
Age	0.058	0.040	0.107
Female			
Race/ethnicity	0.003	0.023	-0.101
White	0.017	0.018	0.027
Black	-0.049	-0.028	-0.094
Asian/PI	-0.001	-0.055	0.161
Hispanic	-0.017	-0.020	-0.008
American Indian/Alaska Native	0.028	0.045	-0.014
Multiracial			
Education	0.032	0.029	0.035
< HS degree	0.001	0.010	-0.057
HS degree/GED	0.001	-0.011	0.045
Some college	-0.037	-0.033	-0.018
College degree+			
Income	-0.003	-0.001	-0.007
<\$20,000	-0.002	0.022	-0.085

\$20,000-34,999	0.049	0.053	0.015
\$35,000-49,999	-0.014	-0.038	0.064
\$50,000-74,999	-0.026	-0.038	0.021
Highest 3rd of CVS Market Share			
Age	0.042	-0.010	0.237
Female	0.044	0.039	0.066
Race/ethnicity			
White	0.023	-0.005	0.109
Black	0.005	0.016	-0.029
Asian/PI	-0.010	-0.004	-0.021
Hispanic	-0.048	-0.014	-0.142
American Indian/Alaska Native	0.068	0.079	0.017
Multiracial	-0.044	-0.065	0.015
Education			
< HS degree	-0.038	-0.014	-0.142
HS degree/GED	-0.106	-0.091	-0.170
Some college	0.131	0.106	0.224
College degree+	0.011	0.001	0.049
Income			
<\$20,000	0.071	0.100	-0.043
\$20,000-34,999	-0.047	-0.039	-0.081
\$35,000-49,999	-0.065	-0.048	-0.132
\$50,000-74,999	0.013	0.026	-0.043
>\$75,000	0.012	-0.058	0.240

Note. Table presents standardized differences in means between pre-policy observations and post-policy observations. Boldface represents a difference above suggested threshold of 0.25. Balance is reported at the population level (weighted by survey non-response weights).

Source. Tobacco Use Supplement to the Current Population Survey (TUS-CPS) 2014-2015.

Conclusion

Each of these papers provides evidence of a relationship between retail availability and health behaviors and related health outcomes. Upon investigation, greater food swamp severity was linked with higher hospitalization rates among adults with diabetes in a curvilinear manner, the relationship plateauing in the counties most saturated with unhealthy food outlets. The abolition of the liquor control system in Washington resulted in a proliferation of liquor outlets in urban counties and was associated with higher rates of hospitalizations for accidental injuries in these counties. CVS Health's removal of tobacco from its pharmacies was associated with a decrease in the number of cigarettes smoked per day by nondaily smokers in areas with substantial CVS market share. While each study certainly has its own limitations, taken together, they suggest that a prolific retail presence can encourage unhealthy behaviors, at least to some extent, across each of these products. These findings support the call to action from literature on the "commercial determinants of health" to study these products and industries in tandem, rather than each in isolation. They also provide important evidence to governmental and organizational policymakers that reducing retail availability of unhealthy products may be an additional tool outside of the health care system with which they can prevent or reduce the burden of chronic conditions in localities and nationwide.