Essays in Health Economics

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Abstract

My doctoral thesis examines the broad question of the effect of some recent health policies on health and also tries to measure socioeconomic inequalities. The first essay investigates the effect of public health insurance on people with vulnerable health. The second chapter analyses the effect of the legalization of marijuana on health while the third chapter measures socioeconomic inequalities in health.

In chapter 1, I study the evolution of access to health care for individuals in vulnerable health before and after the Affordable Care Act. I define leakage of health care as the aggregation of accessibility hurdles for individuals in vulnerable health. However, "being in vulnerable health" is a linguistic concept that does not have a sharp mathematical definition. I draw on the fuzzy sets theory and assume a non-dichotomous membership function to capture the linguistic imprecision. However, the task of choosing the "right" membership function remains an issue. To circumscribe this additional issue, I use a stochastic dominance approach to test for changes in leakage. In order to establish causality, I exploit two quasi-experimental settings offered by the dependent coverage and the states in which medicaid expansion took place. In order to use these quasi-experiments in a stochastic dominance framework, I extend Athey and Imbens (2006) changes in changes approach to

a bivariate setting. Using data from the National Health Interview Survey, the results from a before and after analysis show that leakages are much lower in 2015 compared to 2009 in the US. These before and after results hold irrespective of a person's sex or socio-economic status. The causal analysis shows that leakages in not having insurance and access are reduced in medicaid expansion states after the ACA.

Chapter 2 analyzes the implications of recreational marijuana legalization (RML) on Body Mass Index (BMI) and some healthy behaviours. I exploit the quasi experimental nature of marijuana legalization policy in states using changes in changes and difference in difference approaches to identify the effect of these recreational marijuana policies. Using data from the Behavioral Risk Factor Surveillance System (BRFSS), the results show that recreational marijuana legalization reduces BMI for the entire population. The effect is mainly in the mid and top part of the BMI distribution. Subgroup analysis shows that the reduced BMI resulting from RML is significant among women but not among men. For females, the effect is found both at the lower tail (being underweight) and at the upper tail (morbid obesity). While we found evidence of a reduction in being overweight for both whites and non-whites due to RML, the reduction in obesity and morbid obesity was only found for non-whites. In addition, RML reduces obesity for those below 45 years. I also found evidence that RML increases alcohol consumption, has no effect on smoking of tobacco and binge drinking, but reduces the probability of doing any physical activity.

The final chapter (3) explores the measurement of socioeconomic inequality using ordinal variables. Most measures of socioeconomic inequality are developed for ratio scale variables. These measures use the mean as a reference point which is non-robust in the presence of

categorical variables. This chapter extends Allison and Foster (2004) median based approach to measuring inequalities to a bivariate case and provides conditions to robustly rank any two distributions of socioeconomic inequalities in well-being or mental health. Using the Canadian Community Health Survey (CCHS), I provide robust ordering for socioeconomic inequalities in well-being and mental health for different sub-populations in 2015. The results show that there is less socioeconomic inequality in life satisfaction, happiness, mental health and general health status among employed males and females compared to their respective unemployed groups in 2015.

Dedication

This is dedicated to my family and friends; George, Debbie and Conrad.

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General Introduction

Health and health inequality continue to be of importance to health economists and policy makers. In recent years, the concern with health and health inequalities has been rising due to the evolution of new policies. In addition, the recent literature on cost or efficiency analysis have focused on the impact of health policies (Cookson et al. (2020)). My thesis aims to answer some of the implications universal health care policy and marijuana policy have for health. I show that these new policies can significantly affect health care access and health outcomes.

In the first chapter, I analyze the effect of the Affordable Care Act on health care access in the United States. A large literature has documented the effect of health insurance on healthcare accessibility. However, little is known about healthcare accessibility for people with pre-existing conditions. I address this gap in the literature by studying how access to health care has changed for people with pre-existing conditions after the Affordable Care Act. In order to answer my research question, I define leakage of health care as the accumulation of accessible barriers for those in "vulnerable" health. "Vulnerable health " is a verbal notion that lacks a precise mathematical definition. To account for the language imprecision with "vulnerable health", I use fuzzy sets theory and assume a non-dichotomous membership function. The task of selecting the “correct” membership function remains a challenge. I employ a stochastic dominance strategy to test for changes in leakage to avoid this additional problem. To establish causality, I take advantage of two quasi-experimental conditions provided by the dependent coverage and the states Medicaid expansion programs in the US. I extend Athey and Imbens (2006) changes in changes approach to a bivariate context in order to exploit these quasi-experiments in a stochastic dominance paradigm. My findings of a before and after analysis detailed in this chapter demonstrate that leakages in the United States are substantially lower in 2015 than in 2009. These outcomes are consistent regardless of a person’s gender or socioeconomic position. In addition, after the Affordable Care Act, leakages in not having insurance and access were reduced in Medicaid expansion states, according to the causal analysis.

The second chapter contributes to the debate regarding recreational marijuana legalisation and it’s implications on health outcomes. Specifically, the consequences of recreational marijuana legalisation (RML) on body mass index (BMI) and other healthy indicators are discussed. I make use of the quasi-experimental nature of state marijuana legalisation policies by employing change in change and difference in difference methodologies to determine the impact of these recreational marijuana legislation. The results suggest that recreational marijuana legalisation lowers BMI for the overall population, according to data from the Behavioral Risk Factor Surveillance System (BRFSS). The effect is more noticeable in the middle and upper ranges of the BMI scale. A subgroup study reveals that RML reduces BMI in women, non-whites and those above 45 years but not in men. I also find evidence that legalising recreational marijuana increases alcohol consumption. It has no effect on tobacco smoking or binge drinking, but decreases the likelihood of engaging in any physical activity.

The third chapter looks at how to measure socioeconomic inequality using ordinal variables. The majority of socioeconomic disparity metrics are based on ratio scale variables. In the presence of categorical variables, these measures employ the mean as a reference point, which is non-robust (Allison and Foster (2004)). This chapter applies Allison and Foster (2004) median-based technique to evaluating inequalities to the bivariate case and gives requirements for ranking any two distributions of socioeconomic inequality in well-being. I present a strong ranking for socioeconomic inequalities in well-being and mental health for different subpopulations in Canada in 2015 using the Canadian Community Health Survey (CCHS). Interestingly, I find that employed males and females had less socioeconomic inequality in life satisfaction, happiness, mental health, and overall health status than their corresponding unemployed groups.

The empirical findings from my thesis have significant policy relevance. The main messages from the three chapters are that universal health insurance affect healthcare accessibility and marijuana legalisation affect health but not among the vulnerable groups. In addition, socioeconomic inequalities in health still exist.

Chapter 1

Do individuals in vulnerable health have better access to health care after the Affordable Care Act?*

1.1 Introduction

The Affordable Care Act (ACA) was enacted in March 2010 with the primary objective of providing quality and affordable health insurance for Americans (ObamacareFacts, 2016). The Act is described as a "three-legged stool" designed to fix the problems in the health insurance market (Gruber (2011)). These legs are insurance for all in the non-group markets, an individual mandate that requires everyone to purchase insurance and subsidies

*This chapter is based on joint research work with Paul Makdissi and Myra Yazbeck. This chapter is part of a joint research that is funded by Social Sciences and Humanities Research Council (SSHRC).

for individuals at a threshold of the federal poverty line[1]. The debate on repealing and replacing the ACA was revived in the United States Senate after a draft of a replacement, the American Health Care Act (AHCA), passed in the House of Representatives. Although the repeal of the ACA failed, the new tax policy eliminated the individual mandate under the act which affected the core of the structure of the ACA. Nevertheless, the ACA is the first-ever health policy seeking universal coverage in the United States. A detailed analysis of this policy and its effects is timely and relevant.

Empirical evidence of the early effects of the ACA have shown that insurance coverage has increased among the non-elderly (Frean et al. (2017); Courtemanche et al. (2016)). Insurance coverage is expected to facilitate easy access and the use of health care but whether this gain in insurance was transformed into increased accessibility in this case is still under debate. Courtemanche et al. (2018) provide evidence of how the increase in insurance has led to a significant increase in healthcare use and access. In addition, there are many studies that analyze different components of the ACA, specifically the medicaid expansion and young adult coverage. These studies examine different aspects of access, showing that utilization and preventive care have increased on average, but the effects of the ACA on the affordability of care have also been mixed depending on the study group and years used (Antwi et al. (2015); Barbaresco et al. (2015); Wherry and Miller (2016); Sommers et al. (2013); Sommers et al. (2015))[2]. There is no clear consensus in prior literature on the effect of the ACA on unmet health care needs due to the affordability of care, although an insurance policy is expected to decrease out-of-pocket cost. The importance of this question warrants additional

[1]Includes individuals with preexisting conditions
[2]See literature review for more

studies.

Under the ACA, a step was taken to create a high risk pool of individuals who paid higher insurance upon its enactment in 2010. In 2014, most policies under the ACA were implemented, and allowed for the inclusion of people with pre-existing conditions as having such conditions was no longer a basis for denying insurance. The effect of the policy on access for these individuals is relevant to policy makers. Although there are studies on health care accessibility in the US, to the best of our knowledge none of them focuses on "vulnerable health" groups.[3]

The goal of this paper is to assess the impact of the ACA on access for people in "vulnerable health". Specifically we examine whether or not the ACA has contributed to an increase in health care access for people in "vulnerable health". Addressing this question raises a methodological challenge as the answer to who can be described as " being in vulnerable health" is not necessarily well defined. While we are interested in accounting for health vulnerability, vulnerable health is a vague linguistic variable and does not have sharp boundaries. It is, therefore, difficult to deploy this concept in a binary way.

We contribute to the literature in three ways. First, we add to the growing literature on the Affordable Care Act and access. We provide distributional effects of the ACA as opposed to the average effect of the policy. While an average accessibility hurdle estimate provides insight on common problems, it might mask the problem encountered by those in vulnerable health. In addition, policy makers may be interested in the effect of the Affordable Care Act at other points of the distribution other than the mean. Given these factors, providing

[3] Chatterji et al. (2016) examine the effect of the ACA on the employment mobility decisions of parents of children with preexisting conditions but not on their access to care.

a proper assessment of patients' access while taking into account their health status (health distribution) is vital. However, the linguistic nature of vulnerable health is often challenging to deal with. We propose an approach for dealing with such linguistic variables and provide a robust ordering in assessing the accessibility hurdles of people in "vulnerable health". Lastly, we derive dominance conditions for causal effects adapting Athey and Imbens (2006) approach and extend it to a bivariate distribution of access and health (where access is binary).

To account for the issue arising from the linguistic nature of the health vulnerability, we exploit fuzzy sets theory and define health vulnerability as a gradual transition from vulnerable to not vulnerable using a non dichotomous membership function. We then develop a framework to assess whether the ACA decreased leakages (i.e., the access hurdles of those in vulnerable health). This framework requires that the researcher specify a membership function that describes the extent to which an individual can be considered vulnerable. Specifying a membership function is not without a cost, as the results obtained will depend heavily on the choice made. In addition, in the absence of clear guidelines regarding which form is the most suitable, the choice will necessarily be arbitrary. To overcome this issue, and to provide results that are robust to the specific form of the membership function, we use a dominance approach and define a stochastic dominance condition that will allow us to derive robust conclusions that do not depend on the membership function.

These dominance conditions allow us to compare any two joint distributions across time and or treatment status. We first assess the impact of the ACA exploiting the proposed approach in the simplest context: a before and after analysis. However, it is well-known that in a before and after analysis, one cannot attribute any change over time to just the ACA given the presence of confounding factors. This is why, we extend our analysis to a counterfactual causal framework " à la Rubin" and exploit two quasi-experimental designs: the young adult coverage expansion and the medicaid expansion under the ACA. While exploiting these quasi-experiments allows us to tease out the causal impact (under some assumptions) of ACA on leakages, it allows us to do so for a specific part of the population only. A natural way to estimate the treatment effect, in this case, would be to use a difference in difference approach (DID). Indeed, a large body of literature investigated the impacts of the young adult coverage and the medicaid expansion. Most of these studies use a parametric difference in difference approach that focuses on assessing the impact of the average treatment effects.

Using a parametric DID approach may lead to estimates outside of the allowable range when the outcome of interest is binary (as it is in this case). To avoid this issue, we exploit these natural experiments while extending the changes in changes (CIC) approach adopted by Athey and Imbens (2006) to a bivariate setting where we are interested in access and health and access is binary. This enables us to identify the causal effect of the ACA on leakages for young adults and medicaid expansion states. As in Athey and Imbens (2006), we show that with a weakly monotonic access production function and time invariance within groups, the distribution that would have been observed for the treatment group in the absence of the policy (counterfactual distribution) is identified.[4] Using the identified counterfactual distribution, we rewrite our dominance conditions in a changes-in-changes framework and,

[4]Under the CIC the full counterfactual can be derived and compared with the distribution for treatment group after they have been treated.

thereby test for dominance between the observed distribution of the treatment group and its counterfactual distribution in leakages.

We use the National Health Interview Survey (NHIS), 2009, and 2015 public data files to answer the empirical question in a before and after analysis. The NHIS data offers an advantage to researchers in analyzing national health and access problems because of its high response rate and detailed information on respondent's demographic characteristics. Nevertheless, one problem with using data from 2009 is that, the "Great Recession" could be highly correlated with cost-related barriers. To check for the possibility of bias of the results from 2009, we use data from 2010 and exclude those ages 19 to 25 who were affected by the ACA in its first year in effect compared to in 2015. When assessing the effect of the young adult insurance expansion on leakages, we use data from the National Health Interview Survey NHIS (from 2007 to 2013). Since the NHIS data does not include state identifiers, we use data from the Behavioral Risk Factor Surveillance System (BRFSS) when assessing the effect of the ACA in medicaid expansion states.

Results from the before and after analysis show that leakages in health care access in 2015 dominate 2009 for the entire population. Dominance of leakages in 2015 is also achieved for the working-age population as well as those ages 0 to 17 and those 65 and above. All socio-economic and some demographic groups saw a reduction in leakages in access in 2015 compared to 2009. These results are consistent with the hypothesis that the ACA has led to a significant decrease in cost-related barriers to care. The results are no different when a dominance test is performed between leakages in 2010 and 2015 for the entire population.

The results of the analysis of the causal effect of the young adult coverage show that insurance coverage increased for patients in vulnerable health, while accessibility for people in vulnerable health aged 23 to 25 did not change as a result of the ACA.

Using medicaid expansion states as the treatment group and non-medicaid expansion states as the control group and following a similar method applied to young adults, we find that leakage in not having insurance in medicaid states under the ACA statistically dominates the effect in non-medicaid expansion states as well as leakage in cost prevented care, not having a primary doctor and not having checkups.

The rest of the paper is organized as follows. Section 1.2 provides a review of related literature. The theoretical framework applied is explained in Section 1.3. Details regarding estimation and inference are offered in Section 1.4. In Section 1.5, we provide details of the data used, the restrictions applied and summary statistics. The results of the before and after analysis are presented in Section 1.6, along with the causal effects of the ACA. Section 1.7 concludes.

1.2 Literature Review

In this paper, the main empirical question we seek to address is how vulnerable people's unmet healthcare needs changed after the Affordable Care Act? Were some demographic and socio-economic groups made worse off and others better off? Can these effects be causally linked to the ACA?

The questions posed are linked to two strains of empirical studies, the literature on the link between insurance and health care access and the literature on health insurance and health. Here, we provide a brief review of the literature on insurance, access, and health.

The effect of health insurance on accessibility to health care is intuitive. Having insurance makes health care cheaper and as a result increases the likelihood of use and access (Antwi et al. (2015)). This follows the law of demand, under which a reduction in price or out-of-pocket cost should lead to an increase in affordability and utilization.[5] Due to the level of importance attached to health, the literature on the effect of insurance on access uses methods that can best identify causality. These include the use of randomized control trial, often described as the gold standard and the use of quasi experimental design in finding the relationship between health insurance, accessibility and health.

For instance, Manning et al. (1987) analyze the RAND health insurance experiment, the first natural experiment that assessed the impact of insurance on access. The study shows that insurance increased the number of visits to the doctor. Many subsequent studies have focus on the effect of medicaid on health care accessibility and insurance coverage for low-income individuals, children, pregnant women, and people with disabilities. Finkelstein et al. (2012) also investigate the causally interpretable effect of medicaid on access to health care using the Oregon lottery and find that having medicaid increases access to and utilization of health care among low income subgroups. However, experiments are rare, and researchers use observational data to analyze the effect of insurance on accessibility. Using a quasi-experimental design, Epstein and Newhouse (1998) show that medicaid has an inconsistent effect on health care utilization while Sommers et al. (2013) find that self

[5] This is under the assumption that of infinite supply of health care services or that the healthcare market is nowhere close to saturation

reported cost-related access barriers are reduced for individuals with medicaid. Likewise, studies on the effect of medicare (which is insurance for people above the age of 65) support these findings that medicare (universal insurance) leads to more utilization and access (Lichtenberg (2002); Decker and Rapaport (2002); McWilliams et al. (2003); Card et al. (2009)). Similarly, a before-and-after study by Long and Masi (2009) on the Massachusetts health reform (MHR), universal insurance with a similar model to the ACA, finds that access to health care increased among low income households compared to the high-income group. The chances of getting a usual place of care were also higher. Additionally, the chances of seeing a doctor for preventive care increased by 6% in a simple before and after comparison. It should be noted that an analysis using before and after comparison may be biased as the estimates may be affected by confounding factors. However, using a causal identification strategy does not change the before and after results (Miller (2012a); Miller (2012b)). Despite these findings, Kolstad and Kowalski (2012) observe a reduction in the length of stay and the number of people admitted to the emergency department(ED) after the Massachusetts health reform. As stated earlier, when the ACA was enacted in March 2010, one of the first reforms implemented was coverage for young adults. Several studies examine the effect of this reform on that group. Sommers et al. (2013) apply a difference in difference method to analyze the impact of the ACA on young adults access to health care before and after the policy. Using cross-sectional data, the authors show that there is a higher likelihood of having a usual source of care for 19-26 years and a reduction in the probability of the young adults reporting a delay in medical care or not having medical care due to cost. According to Antwi et al. (2015), the policy has increased the utilization of the ED among young adults between 19 and 25 years old. In the same spirit, Barbaresco et al.

(2015) provide more evidence supporting the findings that access to insurance and having a personal doctor increased after the young adult coverage was introduced, although there was no significant change in income related access barriers.

The differences in the results for cost-related access problems in these studies may be due to the selection of the treatment group as Barbaresco et al. (2015) highlight the importance of the choice of the treatment group. Another key aspect of the policy is the effect of a state's medicaid expansion on access. This forms a major part of the ACA, with about 27 states expanding medicaid coverage in 2014 and 32 states, including the District of Columbia, doing so in September 2016 (Kaiser Family Foundation). Sommers et al. (2016) compare low income households in non-medicaid and medicaid expansion states. The authors find that, there was a reduction in delayed medical care in medicaid states. In addition, Wherry and Miller (2016) evaluate the link between having medicaid and access using data from National Health Interview Survey. They concluded that medicaid expansion states experienced an increase in health insurance compared with non-medicaid states in the 3rd quarter of 2014. Adults were much likely to visit physicians, as well as stay overnight, but there was no reduction in either delay or no medical care due to cost. On the contrary, Miller and Wherry (2017) find a significant reduction in income related barriers to cost. Recently, Courtemanche et al. (2018) use data from the Behavioural Risk Factor Surveillance System and exploit a difference in difference in difference approach using variation in time, space, and treatment intensity in medicaid expansion states to find the effect of the ACA on accessibility. The authors show that cost related barriers and other access problems have decreased.

This paper is related to these studies as it looks to provide more evidence on the effect of the ACA on accessibility and focuses on cost related barriers where prior findings are mixed. However, it is distinct in two ways. First, while we focus on the same components of the ACA, we do not aim at assessing the effect of the policy at the mean but we provide distributional effects. We also allow for heterogeneity in the effect of the ACA on unmet health care needs for both young adults and in medicaid expansion states for people in vulnerable health. Second, we consider individuals' health status, thereby accounting for the needs of those who need access the most. If health state are not considered, this could lead to the overestimation of the policy's effect on accessibility hurdles.

A potential benefit of having better access to health care is improvement in health. Therefore, the effect of insurance on health and overall well-being is of interest in the existing literature. A series of studies that analyze the effect of the Massachusetts health reform on health show that there has been significant improvement in health due to the reform (Miller (2012b);Wees et al. (2013)). Finkelstein et al. (2012) investigate the effect of medicaid on health and find a positive effect of insurance on mortality and self-assessed health among medicaid recipients. The results from recent studies after the ACA on insurance and health is ambiguous. Barbaresco et al. (2015) provide evidence that the ACA led to an increase in excellent health for young adults. However, for very good health and excellent health, there was no significant change in health. Recent studies show that self-assessed health status has not significantly improved under the ACA. (Sommers et al. (2015); Courtemanche et al. (2018); Simon et al. (2017); Miller and Wherry (2017)). In this literature, the health variable is described as a dummy, although the health status variable does not have clear boundaries, and moving from a good health state to a bad state is not abrupt.

A limitation of all these studies is that an analysis that separates health from access can lead to overestimation of unmet health needs. Given this limitation, we use a leakage measure that accounts for health status and unmet health care needs in a fuzzy stochastic dominance framework. The use of fuzzy logic and sets introduced by Zadeh (1965) has been adopted mainly in the development economics literature. Cerioli and Zani (1990) describe moving from one state of dispossession to another as gradual instead of abrupt as is in multidimensional measurement of poverty. Following their work, other studies incorporated fuzzy set theory in the measurement of multidimensional poverty and recently in unidimensional measurement of poverty as well as country-specific case studies.[6] Contrary to these studies, we apply the fuzzy set theory to the measurement of well-being, which is self-assessed health status.[7] Fuzzy theory has also been applied in epidemiology where it is used in decision making, possibility measures as well as probability and risk estimation (Massad et al. (2003); Sadegh-Zadeh (2000)). While fuzzy theory is applied in this literature, none of the studies uses stochastic dominance.[8]

[6]See Martinetti (1994) ; Martinetti (2000); Betti et al. (2006); Betti and Verma (2008); Dagum and Costa (2004); Shorrocks and Subramanian (1994)

[7]Bérenger and Verdier-Chouchane (2007) apply fuzzy theory to the measurement of the quality of life and standard of living across countries and compare these measures to the human development index (HDI) and GDP per capita.

[8]But Makdissi and Wodon (2004) use stochastic dominance for robust ordering of fuzzy poverty indices.

1.3 Theoretical Framework

Fuzzy set theory is well-suited for dealing with linguistic variables resulting from an unclear context-dependent premise or concept. The concept, first introduced by Zadeh (1965), explains the importance of graded membership. Unlike classical set theory, where most elements are either in a set or not, fuzzy theory helps in answering to what extent x is in A is true, thereby allowing for "much or less" as opposed to the "yes ($x \in A$) or no ($x \notin A$)" or "true or false". The binary nature of classical set theory makes the classification of linguistic variables without sharp boundaries difficult as individuals at the margins may be classified into different states despite their close similarities. The flexibility of fuzzy set theory lies in providing a degree of belongingness. Under the fuzzy set theory, one assume that there is intermediary membership in a set that ranges from 0 to 1. The membership function assigns values between 0 and 1 to elements in a set with higher values representing a high level of belongingness and vice versa.

In most surveys, there are often linguistic variables such as general health status. The responses to most of these qualitative variables are often expressed in vaguely defined terms, which increases or decreases in severity. Assuming these responses can be transformed with a suitable numerical scale, then under the classical set theory, there is a threshold below and above which one belongs to a specific group. A problem that arises when the boundaries between the two states is not sharp is, classification of individuals on the margin becomes a major challenge. Conveniently, in this case, a membership function can provide a smooth transition between any two states.

The state of being in good or vulnerable health is inherently linguistic in nature; that is, it is intrinsically fuzzy. This means describing individuals as being in a state of good or vulnerable health is context-dependent. As explained above, it becomes difficult to classify individuals at the margins into a specific health state, assuming a threshold that differentiates good and vulnerable health. Given that the state of vulnerable health is less crisp, we define a membership function $e_v(h)$ which is monotonically non-increasing from 0 to 1 which assigns a graded membership to the state of being in vulnerable health. Using the membership function, we measure accessibility hurdles. Let $h = \{1, 2, \ldots, K\}$ be health status which is ordered from the lowest(1) to the highest(K) category with respective membership functions 1 and 0; that is, the degree of belonging to vulnerable health of the lowest health category is 1 while the highest health category does not belong to the set of vulnerable health, therefore, is assigned a membership function of 0. Formally,

A1. $e_v(1) = 1$ and $e_v(K) = 0$

We assume that this membership function is non-increasing in health status. Formally,

A2. $\Delta e_v(h) = e_v(h) - e_v(h-1) \leq 0$

The discrete representation of health is because of the ordinal nature of the health variable used in this paper.[9].

We are interested in measuring leakage of the health system, i.e. the proportion of individuals in vulnerable health having an accessibility hurdle or not having access. Let S be defined as any form of accessibility to health care. S is a dichotomous variable that takes a value of 1 if the individuals has access to health care when needed and a value of 0 when they are facing an access hurdle. In this context, leakage, $L(e_v, F_{H,S})$ is a function of the membership

[9]Note that health status can also be continuous like the HUI3

function e_v and the joint distribution of health and access to health $F_{H,S}$. Mathematically leakage is defined as,

$$L(e_v, F_{H,S}) = \sum_{h=1}^{K} e_v(h) \Pr(h, 0) \tag{1.3.1}$$

where $\Pr(1,0) = F_{H,S}(1,0)$ and $\Pr(h,0) = F_{H,S}(h,0) - F_{H,S}(h-1,0)$ for $h \in \{2,\ldots,K\}$. $\Pr(h,0)$ is the probability of being in health status h and having an accessibility hurdle ($S = 0$). There are infinite number of membership functions that satisfy assumptions A1 and A2. The numerical value of $L(e_v, F_{H,S})$ will depend on the specific mathematical form chosen for e_v. To illustrate how the index relies on the choice of the numerical membership function, let $e_{v1} = [1, 0.7, 0.5, 0.3, 0]$, $e_{v2} = [1, 0.9, 0.8, 0.5, 0]$ be two numerical membership functions. Assume the distribution of health is $[1, 1, 1, 1, 1]$ and distribution of health accessibility for 3 different periods is given by $A = [0, 0, 1, 1, 1]$, $B = [1, 1, 1, 0, 0]$ and $C = [0, 0, 1, 1, 1]$.[10] The leakage indices using the first membership scale, $L(e_{v1}, F_{H,S})$ are, 0.34, 0.06 and 0.3 respectively while using the second membership scale gives the $L(e_{v2}, F_{H,S})$ values of 0.38, 0.1 and 0.44. Clearly, under the first numerical scale, there is much leakages in A than in B and C. For the second membership scale, while leakages are still higher in A than B, the reverse is true for C and A as leakages are much higher in C than in A. However, even if the numerical value of $L(e_v, F_{H,S})$ is contingent to the choice of e_v, ordering of two distributions $F^i_{H,S}$ and $F^j_{H,S}$ can be robust to all e_v. The next section develops the conditions under which we have such robust orderings.

[10]For this example, we assume the distribution of health does not change.

1.3.1 Stochastic Dominance

As mentioned earlier, the leakage index depends on both the joint cumulative distribution of health and access and the membership function. Specifying the exact mathematical form of the membership function is a debatable choice. Any function that satisfies A1 and A2 can be used based on the researcher's preference which requires judgment. When specifying a membership function, one can measure leakage. However, the value of the leakage index depends heavily on the choice of the membership function. One can argue that the choice of the membership function is driving the leakage measure. Here, we avoid making any value judgment and consider any form of the membership function, incorporating the leakage index in a stochastic dominance framework in order to derive robust ordering. The stochastic dominance test can, therefore be used to compare leakage indices at any two time periods. It is important to note that comparing two leakages in health care access reduces to comparing two joint cumulative distributions at two points in time. We develop a stochastic dominance criterion.

Theorem 1. *Let $F^i_{H,S}$ and $F^j_{H,S}$ be two different joint distributions, $L^i(e_v, F^i_{H,S}) \geq L^j(e_v, F^j_{H,S})$ for all membership function e_v obeying assumptions A1 and A2, if and only if*

$$F^i_{H,S}(h,0) - F^j_{H,S}(h,0) \geq 0 \qquad \forall h \in \{1, 2...., K-1\}$$

Intuitively Theorem 1 states that leakage is a function of the joint distribution of health and access. If the distribution before the ACA dominates the distribution after the ACA, then the ACA has successfully reduced leakage (or improved access).

Proof. **Proof.** Expanding equation 1.3.1 :

$$\begin{aligned} L(e_v, F_{H,S}) &= e_v(K)F_{H,S}(K,0) - [e_v(H) - e_v(H-1)]F_{H,S}(K-1,0) \\ &\quad -..... - [e_v(2) - e_v(1)]F_{H,S}(1,0), \qquad (1.3.2) \\ &= e_v(K)F_{H,S}(K,0) - \sum_{h=1}^{K-1} \Delta e_v(h)F_{H,S}(h,0). \qquad (1.3.3) \end{aligned}$$

Assumption A1 implies that $e_v(K) = 0$. The first term on the r.h.s. of the equation is nil. Equation (1.3.3) can be rewritten as

$$L(e_v, F_{H,S}) = -\sum_{h=1}^{K-1} \Delta e_v(h)F_{H,S}(h,0). \qquad (1.3.4)$$

Using equation (1.3.4), the difference in the two leakage measures is

$$L^i(e_v, F^i_{H,S}) - L^j(e_v, F^j_{H,S}) = -\sum_{h=1}^{K-1} \Delta e_v(h)[F^i_{H,S}(h,0) - F^j_{H,S}(h,0)]. \qquad (1.3.5)$$

Assumption A2 implies that Equation (1.3.8) is positive if $F^i_{H,S}(h,0) \geq F^j_{H,S}(h,0)$ for all $h \in \{1, 2, \ldots, K-1\}$. This proves sufficiency of the condition.

To demonstrate necessity, we will argue a contrario. Imagine that at some health category

h_0, $F^j_{H,S}(h_0, 0) \geq F^i_{H,S}(h_0, 0)$. Also consider a membership function of the form

$$e_v(h) = \begin{cases} 1 & \text{if} \quad h < h_0 \\ \\ 0 & \text{if} \quad h \geq h_0 \end{cases}, \tag{1.3.6}$$

then,

$$\Delta e_v(h) = \begin{cases} 0 & \text{if} \quad h \neq h_0 \\ -1 & \text{if} \quad h = h_0 \end{cases}. \tag{1.3.7}$$

For the membership function e_v above, $L^i - L^j$ is negative if $F^j_{H,S}(h_0, 0) \geq F^i_{H,S}(h_0, 0)$. Hence it cannot be that $F^i_{H,S}(h_0, 0) \geq F^j_{H,S}(h_0, 0)$. This establishes necessity. □

Theorem 1 helps in determining a robust ordering of leakages in health care. According to Theorem 1, the main difference between any two leakage indices lies in the difference in the two cumulative distributions which shows the importance of the cumulative distribution in the dominance test. To answer our question about the effect of the ACA on leakages, we can simply compare leakage before the ACA to leakage after the implementation of the policy using the stochastic dominance condition in theorem 1. While a visual comparison of two empirical cumulative distribution gives an idea of the relationship between the two distributions under consideration, in order to statistically test for the difference in distributions, inference for stochastic dominance needs to be used.

1.3.2 Stochastic Dominance: Discrete Changes in Changes

In this section, we explain some intuition for the need of causal analysis rather than a simple before and after comparison for the ACA. We provide assumptions required in such settings and identification under quasi experimental designs such as difference in difference and changes in changes in a univariate setting before extending the idea of changes in changes to a bivariate setting, which is applied in this paper.

Difference in Difference and Changes-in-Changes: Intuition

Our interest is in identifying the impact of insurance coverage expansion under the Affordable Care Act on leakages in access to health care accounting for the degree of health vulnerability. Given that leakages pre and post the expansion of the insurance under the Affordable Care Act can be observed, a before and after analysis may seem tempting. Nevertheless, it will not allow us to attribute a change in leakages to this expansion alone. Some factors, such as the unemployment rate that peaked during the recession years and dropped due to the recovery from the recession, can affect leakages in accessibility. With a lower unemployment rate, one would expect an increase in employer-provided insurance and overall coverage, thereby reducing leakages in access to health care, which could confound results from the before and after approach and produce spurious results.

Ideally, we would like to assess the causal effect of the insurance coverage expansion by comparing a treatment group and a control group that are randomly assigned. Given that randomization did not occur at the time of the expansion, such an ideal setting is not available. A good alternative to randomization is a setting where policy changes mimic randomization by comparing those who are eligible under the policy and those who are not, before and after the expansion of health insurance. Using such an approach will allow us to remove any confounding factors that could be wrongfully attributed to the expansion of insurance. These are often referred to as quasi experiments. A popular tool that is used to analyze such quasi experimental design is the difference in difference approach (DID). The DID approach to analyzing such experimental design relies on the assumption that the two groups have a common trend in the outcome of interest. This means that in the absence of the treatment, the difference in returns to unobserved characteristics between the treatment and control groups remains the same over time. In this case, the control group is selected on the basis that the control group experienced the same changes in returns to unobserved characteristics as the treatment group would have experienced if the change in policy would not have been implemented. The idea behind this quasi experimental design is to find the average change in the outcome that will have been observed for the treatment group, in the absence of the intervention using the information on the control group and the treatment group in the first period.

In the US, there are two policies under the ACA that could be used to answer our question about the unmet health care needs of people in vulnerable health using quasi experimental design. The young adult coverage and states medicaid expansion. Under the Affordable Care Act's dependent coverage provision, people are allowed to stay on their parents' insurance until they are 26 years old. If young adults who qualify for insurance coverage have observed and unobserved characteristics to those just above the eligibility threshold, then we can assess the effect of the intervention using these two groups pre and post intervention. Formally, the assumption is that if people below and above the age threshold have similar observable and unobserved characteristics, then the allocation into treatment and control groups is as good as random. Previous studies have therefore used individuals below the age eligibility cutoff as the treatment group and as a control group, those above the threshold. As mentioned earlier, initially the ACA required all states to expand medicaid. Medicaid provides insurance for people with income at or below the 138% of the federal poverty line, pregnant women, children and people with disabilities. However, in 2012, the Supreme court ruled for states to opt to expand medicaid or not. To analyze the effect of this policy, we can compare medicaid expansion states and non-medicaid expansion states if states that did not expand medicaid are affected by the same observed and unobserved factors and have a similar composition as the medicaid expansion states. The assumption for comparing medicaid and non-medicaid states is that the trend in the outcome of interest in non-medicaid states reflects what will have happened in medicaid states in the absence of the intervention. If this is true, then any differential change can be attributed to the expansion of medicaid. For this reason, several studies which assess the impact of medicaid expansion, compare states that have expanded medicaid as the treatment group to states that have not expanded medicaid before and after the intervention in 2014.

To discuss this approach more formally, we need to introduce some notation. As mentioned earlier, we will compare two groups, $g \in [0, 1]$ observed at two time periods, $t \in [0, 1]$. An observation belongs to the control group when $g = 0$ and to the treatment group when $g = 1$. The pre-treatment period is determined when $t = 0$ and the post-treatment period is observed when $t = 1$, thus the eligible treated group is determined when $g = 1$ and

$t = 1$. In such a setting a traditional way to identify a causal effect would be to use a DID approach. The intuition behind this approach is straightforward, as it assumes that in the absence of treatment the average outcome in the treatment group is assumed to experience the same change that is the same as the one experienced by the control group between period 0 and period 1. While the assumptions behind the DID approach are very clear, it presents two disadvantages. First, it imposes linearity, which may not be a good approximation in nonlinear models as it produces predictions outside the (0,1) range. Second, it assumes point identification and measures the impact of the program at a specific point of the distribution: the mean. While it is possible to evaluate the impact of the treatment at any point of the distribution the mean is by far the most popular choice because the mean of the difference of two marginals (treatment and control) can be found from the difference between the mean of each of the two marginals. This is possible thanks to the linearity of expectation, which is a property that does not apply for other locations in the distribution. This being said, in many cases and especially when the objective is to assess distributive impacts, policymakers may be interested in the entire distribution of treatment effects. Such impacts are not captured by an approach based on a linear approximation of the average impact. Conveniently, Athey and Imbens (2006) propose a changes in changes (CIC) estimator with the necessary properties for identifying these treatment effects through a non-parametric approach that is a generalization of the DID approach. While Athey and Imbens (2006) approach uncovers the distribution of treatment effects, this framework translates to univariate inequality settings that cannot be readily applied to this paper's theoretical framework. Given that we are interested in the joint distribution of access to health care and health status, we first extend Athey and Imbens (2006) approach to allow for a bivariate setting and then exploit this setting to derive

CIC dominance conditions and identify the impact of the insurance expansion on leakages while accounting for vulnerability in health. The underlying intuition behind the CIC is fairly similar to the one for DID. More specifically, the entire distribution of outcomes for the treatment group would experience the same changes over time as the distribution of outcomes for the control group in the absence of the intervention (as opposed to the same assumption on the average of the distribution for the DID). As we discuss later, the CIC approach involves predicting the counterfactual distribution using the observed distributions for the control group and the first-period treatment group.

Univariate Changes-in-Changes: Assumptions

First, we provide the assumptions for a univariate continuous and discrete setting for both the CIC and DID approaches. For a random sample of individuals m=1,.....,M, let S_m be any form of access to health care for individual m (eg. access to doctor, treatment, etc.). We use notations from Neyman (1923, 1990) potential outcome framework. Since we are talking about access to health care, to avoid confusion, it is worth mentioning that we say that an individual is treated when he is subject to the policy change and non-treated if he is not subject to the policy change. Let S_m^I be access if an individual m has been treated (subject to the policy change) and S_m^N if the same individual m has not been treated (not subject to the policy change). Let I be an indicator for the treatment such that $I_m = 1$, if the individual has been treated and $I_m = 0$ if she has not been treated then, the realized outcome is

$$S_m = S_m^N(1 - I_m) + S_m^I I_m \tag{1.3.8}$$

As in Athey and Imbens (2006), we assume that in the absence of the intervention, there is a time dependent production function θ that transforms unobservables characteristics into access to health care:

$$S_m^N = \theta(U_m, T_m), \tag{1.3.9}$$

where U_m is a random variable that represent unobserved characteristics of individual m and T_m is the time period at which m has been observed. We also assume that:

$$\theta(u,t) \text{ is strictly increasing in } u \text{ for } t \in \{0,1\}. \tag{1.3.10}$$

No further assumptions are required on the functional form of θ. Equation (1.3.9) implicitly means that access is the same for an individual m with $U_m = u$ in a given time period regardless of their group membership status.The distribution of U_m can vary from one group to another but is stable within a group over time. More formally we assume that:

$$D(U_m|T_m, G_m) = D(U_m|G_m). \tag{1.3.11}$$

where G_m is group of individual m. Let us denote $U_g \overset{d}{\sim} U|G$ and let us assume that U_g is continuous. To identify the treatment effect on at least part of the distribution, we need to assume that $\Upsilon_1 \subseteq \Upsilon_0$, where Υ_i is the support of the distribution of unobserved characteristics for group i. These assumptions are required to identify the distribution that would have been experienced by the treatment group in the absence of the treatment if health care accessibility is measured as a continuous variable. The assumption in equation (1.3.10) discards the possibility of using discrete responses. Athey and Imbens (2006) argue that it is impossible to assume strict monotonicity for a discrete outcome because the discrete change in the outcome variable is linked with a continuous unobserved variable. In this case, the analyst can only impose a weak monotonicity assumption. With the weak monotonicity assumption and the assumptions in equation (1.3.9) and equation (1.3.11), the authors identify the bounds of the counterfactual distribution for discrete outcomes. However, they show that under additional assumptions that we will discuss later the researcher can recover point identification.

Difference in Difference: Assumptions

Comparably, under DID approach, in the absence of the policy or intervention, access is linearly additive in group and time periods. Formally,

$$S_m^N = \alpha + \lambda T_m + \gamma G_m + \varepsilon_m \tag{1.3.12}$$

where λ represents the time effect, γ captures the time invariant group effect and ε_m is the unobserved characteristics of individual m. The unobserved characteristics of an individual ε_m is assumed to be independent of the time and group effects.

If we compare equation (1.3.9) and equation (1.3.12), it becomes clear that equation (1.3.9) allows for any functional form and embodies equation (1.3.12). if

$$\theta(u_m, t_m) = \vartheta(u_m + \lambda t_m) \tag{1.3.13}$$

where $u_m = \alpha + \gamma G_m + \epsilon_m$ and ϑ is a single index and identity function.

As stated earlier, the main identifying assumption for the DID is the common or parallel trend assumption. The common trend assumption means that in the absence of the intervention, the treatment group will have evolved as in the control group on average. Under the CIC, the time invariance assumption replaces the assumption of common trend used in the DID. Intuitively, the time invariance assumption implies that, there is no change in the rank in the distributions over time. This assumption allows for matching over time within a group. The CIC is, therefore, a generalization of the popular DID approach.

Univariate Difference in Difference and Changes-in-Changes: Identification

We start with identification for a simple DID and then DID for a continuous outcome. The identification under the DID approach is relatively simple. The average treatment effect on the treated is the difference between the average time trend in the control group and the average time trend in the treatment group for a continuous univariate outcome. This estimand eliminates factors that can not be associated with policy or intervention. The counterfactual effect for a simple DID is also given by,

$$E[S^N|G=1,T=1] = E[S|G=1,T=0] + (E[S|G=0,T=1] - E[S|G=0,T=0]) \tag{1.3.14}$$

In the case of CIC, we begin similarly with a univariate outcome where the access variable is continuous (for instance, the time spent at the doctor's office).[11] We briefly discuss how to identify the counterfactual distribution in a univariate continuous setting before we extend

[11]In this paper, we do not consider a continuous utilization variable. This is just to illustrate how the CIC approach works in the case of a continuous variable before we move on to a discrete variable case and the bivariate setting

the idea to a univariate discrete variable and then to a bivariate discrete setting. We omit m for simplicity. Let us denote $F_s^{gt}(s)$ as the cumulative distribution of access for group g at time t and let

$$F_S^{\{g,t\}-1}(q) = \inf\{s : F_S^{\{g,t\}}(s) \geq q\} \tag{1.3.15}$$

where $F_S^{\{g,t\}-1}(q)$ represents the inverse of the cumulative distribution at quantile q.

Identification under the CIC model entails double matching. The first matching is as a result of equation (1.3.9) and equation (1.3.10). From these CIC assumptions, if there is an individual in the first-period treatment group with outcome s, and there exists an individual with the same outcome s in the first-period control group, then these two individuals in the treatment and control groups should have similar unobserved characteristics u.

Athey and Imbens (2006) show that, due to the invertibility of the accessibility production function θ in u because of the strict monotonicity assumption, the second period outcome for the same individual in the control group with the same value of unobserved characteristics is,

$$F_S^{\{0,1\}-1}(F^{\{0,0\}}(s)) = (\theta(\theta^{-1}(s;0),1). \tag{1.3.16}$$

If we apply the same idea to the first period outcome in the treatment group, the entire counterfactual distribution of access is identified as follows.

$$F_{S^N}^{\{1,1\}}(s) = F_S^{\{1,0\}}(F_S^{\{0,0\}-1}(F_S^{\{0,1\}}(s)), \tag{1.3.17}$$

where $F_{S^N}^{\{1,1\}}(s)$ is the counterfactual cumulative distribution of the treatment group in period 1. The counterfactual cumulative distribution is expressed in terms of the cumulative distribution of the control group in both periods and the treatment group in the first period. This expression is only valid when there is a continuous outcome.

As stated earlier, when there is a univariate outcome that is discrete, the difference in difference may give estimates that are not in the allowable range. The assumptions under the continuous CIC approach are also extremely restrictive for discrete outcomes (Athey and Imbens (2006)). The authors relax the assumption of strict monotonicity and allow for weak monotonicity, with discrete outcomes. This leads to a loss of point identification and only bounds are provided. This is because, with weak monotonicity, there is no longer a one-to-one link between the access production function and the distribution of the unobservable characteristics. The bounds follow from Athey and Imbens (2006). We provide details on identification for discrete outcomes in the next section. Although our outcomes of interest are discrete, we can not readily use Athey and Imbens (2006) discrete CIC as it is only applicable to univariate outcomes. We therefore extend the univariate discrete changes in changes to a bivariate setting.

Bivariate Discrete Changes-in-changes: Assumptions and Identification

In this paper, we are interested in identifying the counterfactual joint distribution of health and accessibility hurdles. We define accessibility and health formally and extend Athey and Imbens (2006) to identify the unobserved joint distribution of access and health for the treated group in the second period after the intervention. However, we are concerned with the unmet health care need of individuals in vulnerable health. We are able to derive the counterfactual joint distribution of leakages by using the identified counterfactual joint distribution of access and health by using the idea that the entire joint conditional distribution sums to 1. Before proceeding to identification in the bivariate setting, we provide the assumptions needed for such a bivariate setting. Our outcomes of interest are a binary access variable and a discrete health variable with K categories. The definition of access follows from univariate case. That is, S_m is any form of health care access by individual m. Let S_m^N reflect counterfactual accessibility in the absence of the intervention. We assume that we observe health status of each individual. In this bivariate setting we assume that:

$$S_m^N = \theta_1(U_m, T_m, H_m) \tag{1.3.18}$$

where θ_1 is an access production function, U_m is a random variable that represent unobserved characteristics of individual m, T_m is the time period and H_m is an individual's health status. The only difference between equation (1.3.18) and equation (1.3.9) is that, the access production function in equation (1.3.18) depends not only on unobserved characteristics and the time period but the health of individual m. We relax the strict monotonicity assumption as explained in the univariate discrete case and assume weak monotonicity. Formally,

$$\theta_1(u, t, h) \text{ is non-decreasing in } u \text{ for } t \in \{0, 1\}. \tag{1.3.19}$$

Similarly, using potential outcome notations, let H_m^I be the health status of an individual who receives the treatment and H_m^N be the health status of an individual m who does not receive the treatment.

$$H = H_m^N(1 - I) + H_m^I I \tag{1.3.20}$$

where I is an indicator for treatment. We assume in the absence of the treatment the health production function ψ is given by,

$$H_m^N = \psi(U_m, T_m, S_{-T}) \tag{1.3.21}$$

where $S_{-T} = (S_O, S_1, \ldots, S_{T-1})$ is access to health care in the past periods, U_m is a random variable that represents unobserved characteristics of individual m [12], T_m is the time period. Health status depends on unobserved characteristics, time and one's access to health care in the periods before time T. We also assume that

$$\psi(u, t, s_{-t}) \text{ is non-decreasing in } u \text{ for } t \in \{0, 1\}. \tag{1.3.22}$$

We retain the assumptions in equation (1.3.13) and the common support assumption. To

[12] Individual's capacity to produce health depends on past health which are also related to unobservable characteristics, so, U_m implicitly implies past health

ease notational burden, we introduce the following shorthand

$$
\begin{aligned}
(S^N, H^N)^{\{g,t\}} &\overset{d}{\sim} (S^N, H^N|G = g, T = t) \\
(S^I, H^I)^{\{g,t\}} &\overset{d}{\sim} (S^I, H^I|G = g, T = t) \\
(S, H)^{\{g,t\}} &\overset{d}{\sim} (S, H|G = g, T = t) \\
(S^N|H)^{\{g,t\}} &\overset{d}{\sim} (S, H|G = g, T = t, H = h) \\
(S^I|H)^{\{g,t\}} &\overset{d}{\sim} (S, H|G = g, T = t, H = h) \\
(S|H)^{\{g,t\}} &\overset{d}{\sim} (S, H|G = g, T = t, H = h \\
(H^N)^{\{g,t\}} &\overset{d}{\sim} (H|G = g, T = t) \\
(H^I)^{\{g,t\}} &\overset{d}{\sim} (H|G = g, T = t) \\
(H)^{\{g,t\}} &\overset{d}{\sim} (H|G = g, T = t)
\end{aligned}
$$

The corresponding cumulative conditional distributions are $F^{\{g,t\}}_{H^N,S^N}, F^{\{g,t\}}_{H^I,S^I}, F^{\{g,t\}}_{H,S}, F^{\{g,t\}}_{S^N|H^N}$, $F^{\{g,t\}}_{S^I|H^I}, F^{\{g,t\}}_{S|H}, F^{\{g,t\}}_{H^N}, F^{\{g,t\}}_{H^I}$ and $F^{\{g,t\}}_{H}$ respectively.

Given that it is not possible to invert the joint distribution of access and health, to identify the bivariate counterfactual distribution, we rewrite the joint distribution of access and health as the product of the conditional distribution of access on health and the marginal distribution of health. This joint counterfactual distribution represents the distribution of access and health for the treatment group when they are not affected by the intervention in the second period. The counterfactual joint distribution can be written as,

$$F^{\{g,t\}}_{H^N,S^N}(h, s) = F^{\{g,t\}}_{S^N|H^N}(s|h) \cdot F^{\{g,t\}}_{H^N}(h) \tag{1.3.23}$$

Following Athey and Imbens (2006), we can provide bounds for the counterfactual conditional distribution of health access and the counterfactual marginal distribution of health separately. If the assumptions in equation (1.3.4) and equation (1.3.13) to equation (1.3.17) as well as the common support assumption hold, the bounds for the joint distribution of access and health can be computed.

Let us assume that the support of random unobserved characteristics in the control group U_0 is U[0,1] and let

$$\bar{u}^0(t,h) = \sup(u : \theta_1(u,t,h) = 0) \tag{1.3.24}$$

where $\bar{u}^0(t,h)$ refers to the supremum of unobservable at time t and health status h such that the access production function is 0. For any $u > \bar{u}^0(t,h)$, $\theta_1(u,t,h) = 1$. Define,

$$F^{\{g,t\}}_{S^N|H}(s|h) = \Pr(U_g > \bar{u}^0(t,h)|H = h) \tag{1.3.25}$$

where $F^{\{g,t\}}_{S^N|H}$ is the counterfactual cumulative distribution of accessibility for group g at time t given that health status is h.

Suppose that for health status h, $F^{\{0,1\}}_{S|H}(s|h) > F^{\{0,0\}}_{S|H}(s|h)$, if $U_1 < \bar{u}(0,h)$, there are two cases for the conditional distribution of U_1. Either all the mass is between $[\bar{u}^0(1,h), \bar{u}^0(0,h)]$ or there is no mass between $[\bar{u}^0(1,h), \bar{u}^0(0,h)]$. If all the mass is on the interval $[\bar{u}^0(1,h), \bar{u}^0(0,h)]$, then $\Pr(U_1 > \bar{u}^0(1,h)|H = h) = 1$ or if there is no mass between $[\bar{u}^0(1,h), \bar{u}^0(0,h)]$ then, $\Pr(U_1 > \bar{u}^0(1,h)|H = h) = \Pr(U_1 > \bar{u}^0(0,h)|H = h) = F^{\{1,0\}}_{S|H}(s|h)$. This implies $F^{\{1,1\}}_{S^N|H}(s|h) \in [F^{\{1,0\}}_{S|H}(s|h), 1]$. Following a similar relationship, when $F^{\{0,1\}}_{S|H}(s|h) < F^{\{0,0\}}_{S|H}(s|h)]$, $F^{\{1,1\}}_{S^N|H}(s|h) \in [0, F^{\{1,0\}}_{S|H}(s|h)]$. The possibility for $F^{\{0,1\}}_{S|H}(s|h) =$

$F_{S|H}^{\{0,0\}}(s|h)]$ is almost zero, but in that case, $F_{S^N|H}^{\{1,1\}}(s|h) = F_{S|H}^{\{1,0\}}(s|h)$. These conditions describe the bounds for the conditional counterfactual distribution of access.

To identify the counterfactual marginal distribution of health, we follow Athey and Imbens (2006) discrete changes in changes. Let us define another inverse cumulative distribution function,

$$F_H^{\{g,t\}(-1)}(q) = \sup\left\{h : F_H^{\{g,t\}}(h) \leq q\right\} \tag{1.3.26}$$

The bounds of the counterfactual marginal distribution of health follows from a similar logic from the case where there is a binary outcome.

$$F_{H^N}^{\{1,1\}lb}(h) = F_H^{\{1,0\}}\left(F_H^{\{0,0\}(-1)}(F_H^{\{0,1\}}(h))\right) \tag{1.3.27}$$

$$F_{H^N}^{\{1,1\}ub}(h) = F_H^{\{1,0\}}\left(F_H^{\{0,0\}-1}(F_H^{\{0,1\}}(h))\right) \tag{1.3.28}$$

As Athey and Imbens (2006) point out, there are two main issues with using the bounds of the counterfactual distribution in estimating the treatment effect. First, with a binary outcome, the sign of treatment effect is only determined if the time trend for the treatment and control groups are opposite. Second, the bounds of the counterfactual distribution may be too wide. This is why we impose the conditional independence assumption (CIA) and focus on point identification. The CIA requires that $U \perp G|S,T,H$. Intuitively, the CIA means that if there are a similar distribution of health, access and time, then there are a similar distribution of unobservable characteristics. That is, for a similar distribution of health, access and time, selection into a group is as good as random. The importance of the CIA assumption is to allow for comparison of the level of outcome between groups and comparison over time. For an individual in a given health status with some access in the first period treatment group, one can find the corresponding health care access in the initial period control group and infer the counterfactual distribution using the distribution of the control group in the second period. To show how one can derive the point estimates under the CIA, let the conditional probability of failure of accessibility be $1 - F^{\{g,t\}}_{S^N|H}(s|h) = \Pr(U_g \leq \bar{u}^0(t,h)|H = h)$ for any health status. Consider any $u_h \leq \bar{u}^0(t,h)$ then,

$$\begin{aligned}
\Pr(U_1 \leq u_h|U_1 \leq \bar{u}^0(t,h), H = h) &= \Pr(U_1 \leq u_h|U_1 \leq \bar{u}^0(t,h), T = 0, S = 0, H = h) \\
&= \Pr(U_0 \leq u_h|U_0 \leq \bar{u}^0(t,h), T = 0, S = 0, H = h) \\
&= \frac{u_h}{\bar{u}^0(t,h)}.
\end{aligned}$$

Following the intuition above, the estimates under CIA are:

$$F^{\{1,1\}}_{S^N|H}(s|h) = \begin{cases} 1 - \frac{1-F^{\{0,1\}}_{S|H}(s|h)}{1-F^{\{0,0\}}_{S|H}(s|h)}(1 - F^{\{1,0\}}_{S|H}(s|h)) & \text{if } F^{\{0,1\}}_{S|H}(s|h) > F^{\{0,0\}}_{S|H}(s|h) \\ \\ \frac{F^{\{0,1\}}_{S|H}(s|h)}{F^{\{0,0\}}_{S|H}(s|h)} F^{\{1,0\}}_{S|H}(s|h) & \text{if } F^{\{0,1\}}_{S|H}(s|h) \leq F^{\{0,0\}}_{S|H}(s|h) \end{cases}$$

For each health status, the estimated expected value of the conditional counterfactual distribution is the conditional probability of success of access weighted by the ratio of the conditional probability of success of access over time in the control group when the time trend in the control group is negative. When the time trend in the control group is positive, the estimated distribution of the conditional counterfactual is the difference between 1 and the conditional probability of failure of access in the treatment group weighted by the ratio of the conditional probability of failure in the second-period control group to the conditional probability of failure of access in the control group in the first period.

When there are more than two categories for a discrete outcome, like health status, we get the point identification for the counterfactual marginal distribution of health, which is given by,

$$F_{H^N}^{\{1,1\}}(h) = \begin{cases} F_H^{\{1,0\}}\left(F_H^{\{0,0\}(-1)}(F_H^{\{0,1\}}(h))\right) & \text{if } Z = 0 \\ \\ F_H^{\{1,0\}}\left(F_H^{\{0,0\}(-1)}(F_H^{\{0,1\}}(h))\right) + \Upsilon \cdot \Omega & \text{if } Z > 0 \end{cases}$$

where

$$\begin{aligned} Z &= F_H^{\{1,0\}}\left(F_H^{\{0,0\}-1}(F_H^{\{0,1\}}(h))\right) - F_H^{\{1,0\}}\left(F_H^{\{0,0\}(-1)}(F_H^{\{0,1\}}(h))\right), \\ \Upsilon &= F_H^{\{0,0\}}\left(F_H^{\{0,0\}(-1)}(F_H^{\{0,1\}}(h))\right) - F_H^{\{0,0\}}\left(F_H^{\{0,0\}-1}(F_H^{\{0,1\}}(h))\right), \text{ and} \\ \Omega &= \frac{F_H^{\{0,1\}}(h) - F_H^{\{0,0\}}\left(F_H^{\{0,0\}(-1)}(F_H^{\{0,1\}}(h))\right)}{F_H^{\{0,0\}}\left(F_H^{\{0,0\}-1}(F_H^{\{0,1\}}(h))\right) - F_H^{\{0,0\}}\left(F_H^{\{0,0\}(-1)}(F_H^{\{0,1\}}(h))\right)}. \end{aligned}$$

We have identified the counterfactual conditional distribution of access on health and the marginal distribution of health, the joint distribution of having an accessibility hurdle and health is as follows.

$$F_{H^N,S^N}^{\{1,1\}}(h,0) = \left[1 - F_{S^N|H}^{\{1,1\}}(s|h)\right] \cdot F_{H^N}^{\{1,1\}}(h) \tag{1.3.29}$$

Following the stochastic dominance condition derived under Theorem 1, it is possible to determine a causally robust ranking of leakages for the treatment group by comparing the identified joint counterfactual distribution and the observed joint distribution of accessibility hurdle and health to allow for robust ordering. There is dominance in leakages for the treatment group if the observed bivariate distribution for the treatment is less than or equal to the joint distribution for treatment group in the absence of the policy that has been identified. Formally, if

$$F^{\{1,1\}}_{H^I,S^I}(h,0) \leq F^{\{1,1\}}_{H^N,S^N}(h,0) \tag{1.3.30}$$

1.4 Estimation and Inference

Suppose we have random independent samples of sizes N_1 and N_2 drawn from two independent joint cumulative distribution on health and access, $F^i_{H,S}(h,s)$ and $F^j_{H,S}(h,s)$, with respective sample estimates $\hat{F}^i_{H,S}(h,s)$ and $\hat{F}^j_{H,S}(h,s)$. The natural estimator of the joint empirical distribution is given by

$$\hat{F}_{H,S}(h,s) = \frac{1}{N}\sum_{i=1}^{N} 1(h_i \leq h \wedge s_i \leq s) \tag{1.4.1}$$

where $1(\cdot)$ is an indicator function. The expected value of this estimator is given by

$$\begin{aligned} E[\hat{F}_{H,S}(h,s)] &= E[\frac{1}{N}\sum_{i=1}^{N} 1(h_i \le h \wedge s_i \le s)] \\ &= \frac{1}{N}\sum_{i=1}^{N} E[1(h_i \le h \wedge s_i \le s)] \\ &= \frac{1}{N} N F_{H,S}(h,s) \\ &= F_{H,S}(h,s). \end{aligned}$$

The variance of this estimator is given by

$$\begin{aligned} Var(\hat{F}_{H,S}(h,s)) &= Var(\frac{1}{N}\sum_{i=1}^{N} 1(h_i \le h \wedge s_i \le s)) \\ &= \frac{1}{N^2}\sum_{i=1}^{N} Var(1(h_i \le h \wedge s_i \le s)) \\ &= \frac{1}{N} F_{H,S}(h,s)(1 - F_{H,S}(h,s)) \end{aligned}$$

The asymptotic distribution of the estimator is given by

$$\sqrt{N}(\hat{F}_{H,S}(h,s) - F_{H,S}(h,s)) \xrightarrow{d} \mathcal{N}(0, \frac{1}{N} F_{H,S}(h,s)(1 - F_{H,S}(h,s)))$$

Assume we have four random samples of group and periods, one for each group, g, and time t. Let $F_{S|H}^{\{g,t\}}(s|H)$ be the conditional distribution of access on health with sample estimate $\hat{F}_{S|H}^{\{g,t\}}(s|H)$ and $F_{H}^{\{g,t\}}(H)$ be the marginal distribution of health with sample estimate $\hat{F}_{H}^{\{g,t\}}(H)$. We use $\hat{F}_{H}^{\{g,t\}(-1)}(h) = \sup\{h : \hat{F}_{H}^{\{g,t\}(-1)}(h) \le q\}$ and $\hat{F}_{H}^{\{g,t\}-1}(h) = \inf\{h : \hat{F}_{H}^{\{g,t\}-1}(h) \ge q\}$ as the estimators of the two inverse functions.

Athey and Imbens (2006) show that the observed empirical distribution is efficient. In a bivariate setting like this, we can estimate the counterfactual distribution using sample analogs for changes in changes; that is, we can use the plug-in principle. Since it has been established that the joint counterfactual distribution of leakage is a function of observable elements, we can replace the joint counterfactual distribution with consistent estimates of the observable elements.

Inference in stochastic dominance is popular in the literature on inequality and poverty. The most popular way to statistically test for dominance is to formulate a null of dominance (Mcfadden, 1989; Bishop et al., 1992; Anderson, 1996; Davidson and Duclos, 2000; Barrett and Donald, 2003). We are interested in testing for dominance between any two joint distribution of health care access and health, we follow the popular method of testing for dominance in the literature and pose a null hypothesis of dominance as:

$$H_0: \quad F^i_{H,S}(h,0) - F^j_{H,S}(h,0) \geq 0 \;\; \forall\, h \in \{1,\ldots,K-1\}$$

$$H_1: \quad F^i_{H,S}(h,0) - F^j_{H,S} < 0 \;\; \text{for some } h \in \{1,\ldots,K-1\}$$

where the null represent possible dominance of distribution $F^i_{H,S}(h,0)$ over distribution $F^j_{H,S}(h,0)$ while the alternate implies no incidence in the null exist.

Assuming two independent distributions, Davidson and Duclos (2000) propose that a t-statistics can be used for the test under the null of dominance.

$$t(h,s) = \frac{\hat{\Delta}(h,0)}{\sqrt{Var(\hat{\Delta}(h,0))}}$$

where $\hat{\Delta}(h,0) = \hat{F}^{i}_{H,S}(h,0) - \hat{F}^{j}_{H,S}(h,0)$

For independent distributions:

$$Var(\hat{F}^{i}_{H,S}(h,0) - \hat{F}^{j}_{H,S}(h,0)) = Var(\hat{F}^{i}_{H,S}(h,s)) + Var(\hat{F}^{j}_{H,S}(h,s))$$

The null of dominance is rejected if any of the t-statistics is negative and statistically significant. Significance is determined by the Studentized Maximum Modulus(SMM) distribution with parameter K-1 and infinite number of degrees of freedom.

Under CIC approach discussed here, we are interested in comparing leakages in the observed distribution of the treated group with the distribution of leakages that will have been observed for the treatment group in the absence of the expansion of the Affordable Care Act. Having derived point estimates for the counterfactual distribution of leakages, from Theorem 1 we can formally test for dominance in leakages for the two distributions as

$$\begin{aligned} H_0^{N1}: &\quad F^{\{1,1\}}_{H^I,S^I}(h,0) - F^{\{1,1\}}_{H^N,S^N}(h,0) \leq 0 \;\; \forall\, h \in \{1,\ldots,K-1\} \\ H_1^{N1}: &\quad F^{\{1,1\}}_{H^I,S^I}(h,0) - F^{\{1,1\}}_{H^N,S^N}(h,0) > 0 \;\; \text{for some } h \in \{1,\ldots,K-1\} \end{aligned}$$

Bootstrap standard errors can be used for inference and follow from Athey and Imbens (2006). Given the estimates of the counterfactual distribution and the observed distribution in the second period of the treated group and standard errors, a simple t-test can be used to test for dominance between the counterfactual distribution obtained from the CIC approach and the distribution of observed treatment group access and health. The decision rule follows as the one stated above for the t-statistics.[13]

1.5 Data

1.5.1 National Health Interview Survey (NHIS) Data

Our primary data for a before and after analysis as well as for the young adult coverage under the ACA, is from the National Health Interview Survey(NHIS). Specifically, from the public use data files for individuals and households respondents. The NHIS, conducted by the National Centre for Health Statistics, collects data on non-institutionalized civilians in the US on a quarterly basis.[14] The survey sample is representative of Americans of all ages. Since 1957, the survey's central questions have been categorized in the family core, the household core, sample adult core, and the sample child core. NHIS interviews about 35,000 households yearly with each wave containing information on respondents'

[13] Athey and Imbens (2006) mention that a test of equality of the counterfactual distribution and the observed distribution can be applied after deriving the counterfactual distribution and statistical inference can be obtained using a χ^2 test, which is different from the test of dominance in this paper.
Also, Abadie (2002) proposes using a bootstrap KS method to test for equality, first and second-order stochastic dominance between the distribution for the distribution in the presence of an intervention and the counterfactual distribution using an instrumental variable approach which varies from the changes in changes and first order dominance test used in this paper.
Note we use fuzzy theory to define health status and then use the changes in changes to derive the counterfactual distribution used in this paper. This is not to be confused with the fuzzy changes in changes introduced by de Chaisemartin and d'D'Haultfœuille (2014)

[14] It includes only individuals residing in the USA at the time of the interview. Data on active armed forces members are excluded, with the exception of cases in which some of their family members are civilian. In that case, they are interviewed but their final weights are zero. The survey uses a multi-stage sample design that changes after every decennial census

demographic characteristics, health indicators, injury, limiting activities, health insurance status, and access to care. Although state identifiers are not available in the public use files, the data contains information on the US regions. In addition to these, there are also five imputed income files for all families. The survey is done through a face-to-face interview, so the survey response rate is high, about 70% for all households.

This paper uses data from the "person file" in 2009 and 2015, which is available for public use for the analysis of leakages in a simple before and after comparison. We use these years because the ACA was implemented in 2010 while most of the related policies were put into effect in 2014, a year before and after the ACA's implementation. Survey weights are provided for NHIS data because of the complex multi-stage design of the survey. These weights are used in all analysis in this paper. The NHIS data is suitable for our research question because it provides in depth answers to the questions of interest and detailed information on the households surveyed.

Health Outcome

Self-assessed health status (SAHS) is an ordinal variable that provides information on people's general perception of their health in all dimensions. It is used in the literature because of its simplicity and has been found to be correlated with other objective measures of health such as mortality and morbidity (Idler and Benyamini (1997)). Although SAHS is subjective, it performs better than some objective measures of health and provides a general rating (multidimensional) of health compared to just one-dimensional measures of health. Despite the advantages SAHS has over objective health measures, it is important to acknowledge that an objective measure of health status might be more reliable in some senses. However, the sample sizes for objective measures such as hypertension and diabetes in the NHIS data set are small due to sub-sampling.

In the NHIS data, information on an individual's health status is the response to the question "In general will you say your health is excellent, very good, good, poor or fair". All individuals in our sample were asked this question which provides us with a large sample size for our analysis. The total sample sizes for self reported health in 2009 and 2010 are 88,344 and 103,677 respectively for the before and after analysis.

Access to health care

Access to care has been defined in many ways. It can be defined as the use of health care services. It has also been defined as the potential to use healthcare facilities (Anderson 1995). An individual is considered to have an unmet need if the person did not receive health services when needed. In the NHIS data, this means care is either delayed or no access due to cost (affordability).[15] We measure an unmet health care need using the response to the following question "During the past 12 months, was there any time when you needed medical care, but did not get it because you couldn't afford it?" and "During the past 12 months, has medical care been delayed because of worry about the cost?". Therefore, these access responses are binary outcomes. Note that our accessibility variables are defined as 1 when respondents did not have any difficulty accessing health care and 0 otherwise. However, the summary statistics are shown for the proportions that have accessibility hurdles. The total

[15] We acknowledge the multidimensional nature of defining accessibility (such as, availability, affordability, quality, use, etc.). However, in this study, we associate accessibility with affordability of healthcare.

sample includes ages from 0 to 85 years (A knowledgeable adult in the household answered the question for respondents below the age of 18), but we exclude missing responses for age, race, gender and educational level. The total sample is 88,310 and 103,719 for unmet medical care due to cost in 2009 and 2015 respectively. Similarly, the sample sizes for delay in medical care due to costs are 88,329 and 103,730 in 2009 and 2015, respectively. We will be using these data sets for the before and after analysis.

Summary statistics

Table 1.1 shows summary statistics for the samples in 2009 and 2015. The working-age population makes up a considerable part of our sample. In both years, females makeup slightly above half of the sample, with the proportion of individuals having an education at a high school level or less being high. Also, the sample is predominantly white and mostly live in the south.

Table 1.1: Summary statistics for entire sample in 2009 and 2015

	2009		2015			
	mean	sd	mean	sd	difference	p
0-17 years	0.24	0.43	0.23	0.42	-0.01***	(0.00)
18-64 years	0.63	0.48	0.62	0.49	-0.01***	(0.00)
65+	0.13	0.33	0.15	0.35	0.02***	(0.00)
Male	0.49	0.50	0.49	0.50	0.00	(0.81)
Female	0.51	0.50	0.51	0.50	0.00	(0.81)
White	0.80	0.40	0.79	0.41	0.01***	(0.00)
Black	0.13	0.34	0.14	0.34	-0.01***	(0.00)
Asian	0.05	0.21	0.06	0.24	0.01	(0.06)
Other	0.01	0.12	0.02	0.12	0.01	(0.00)
Northeast	0.18	0.38	0.17	0.38	-0.01	(0.64)
Midwest	0.23	0.42	0.22	0.42	-0.01***	(0.00)
South	0.36	0.48	0.38	0.48	-0.02***	(0.00)
West	0.23	0.42	0.23	0.42	0.00***	(0.00)
High school or less	0.57	0.49	0.53	0.50	-0.04***	(0.00)
Some college education	0.43	0.49	0.47	0.50	-0.05***	(0.00)
Personal income	39048.71	29828.21	43608.61	33627.20	4264.99***	(0.00)

**Note*: Weighted means of the population 0-85 years. Standard error are in parenthesis.

Table 1.2 shows summary statistics for the reversed form of the samples' accessibility outcomes in 2009 and 2015. Of the respondents, 7% reported not having had medical care due to cost in the base year. There is a 2% reduction in reported accessibility difficulty due to cost in the year after the policy was implemented, and this difference is statistically significant. Similarly, delay in accessing health care due to cost was reduced from 10% to 6% a year after the ACA in 2015, and the difference of 4% is statistically significant. Generally, reported health status in the baseline period and after the policy does not differ for those in poor, good, very good health, but there is a 1% increase in individuals who perceived their health as fair from 2009 to 2015 which is not statistically significant.

Table 1.2: Summary statistics of outcomes in 2009 and 2015

	2009		2015		Difference	
	mean	sd	mean	sd	difference	
Access to care						
No medical care	0.07	0.001	0.05	0.001	-0.02***	0.002
N	88310		103719			
Delay in medical care	0.10	0.002	0.06	0.001	-0 .04***	0.002
N	88329		103730			
Delay or no medical care	0.11	0.002	0.07	0.001	- 0.04***	0.002
N	88446		103789			
Self reported health status						
Poor	0.02	0.001	0.02	0.001	-0.001	0.001
Fair	0.07	0.001	0.08	0.001	0.003	0.002
Good	0.24	0.003	0.24	0.002	-0.0003	0.003
Very Good	0.30	0.003	0.30	0.003	-0.002	0.003
N	88344		103677			

**Note*: Weighted means of the population 0-85 years. Standard error are in parenthesis.

In further analysis of the causal effects of the ACA, we use data from the 2007 to 2013 period, three years before and after the ACA was implemented. The sample is restricted to individuals between 23 to 29 years (excluding 26) with 23 to 25 as treatment groups and 27 to 29 years as the control. [16] The key outcomes analyzed here includes insurance coverage

[16] We exclude individuals who are 26 years because of the uncertainties around this age group as we do not know their exact date of birth.

and the affordability of health care. [17]

Table 1.3: Summary Statistics for young adults from 2007 to 2013

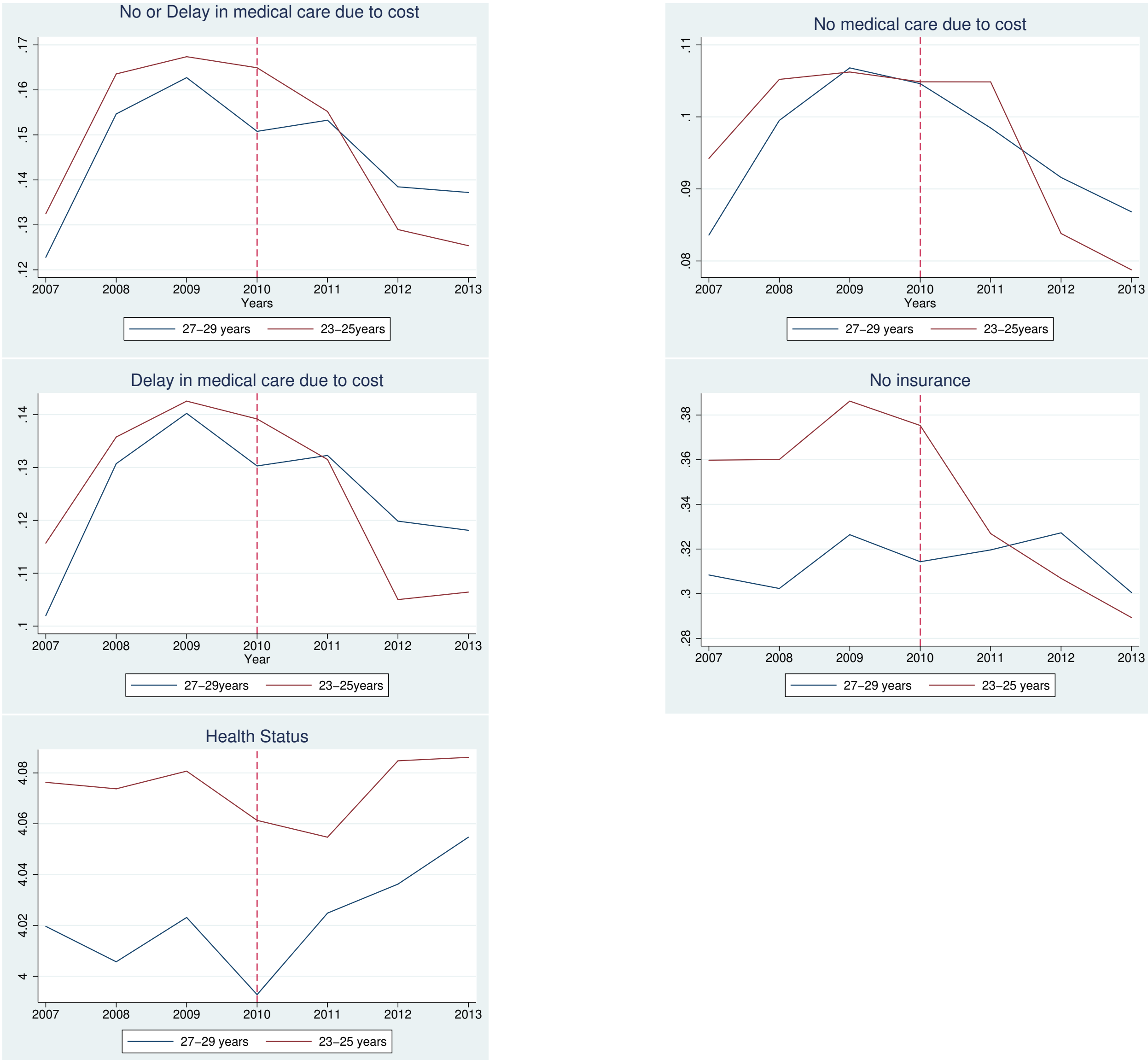
	(Treatment(23 to 25)		Control(27 to 29)		Difference	
	mean	sd	mean	sd	difference	p
23 years	0.34	0.47	-	-	-0.33***	(0.00)
24 years	0.33	0.47	-	-	-0.33***	(0.00)
25 years	0.34	0.47	-	-	-0.34***	(0.00)
27 years	-	-	0.34	0.47	0.34***	(0.00)
28 years	-	-	0.34	0.47	0.34***	(0.00)
Female	0.50	0.50	0.50	0.50	0.00*	(0.01)
Married	0.24	0.43	0.46	0.50	0.22***	(0.00)
Hispanic	0.19	0.39	0.20	0.40	-0.01	(0.22)
Non-Hispanic White	0.61	0.49	0.60	0.49	0.01*	(0.04)
Non-Hispanic Black	0.14	0.35	0.13	0.34	-0.01***	(0.00)
Non-Hispanic Asian	0.05	0.22	0.06	0.24	0.01**	(0.00)
High School or less	0.62	0.49	0.63	0.48	0.01*	(0.01)
Poor health	0.01	0.08	0.01	0.08	0.00	(0.71)
Fair health	0.04	0.19	0.05	0.21	0.01***	(0.00)
Good health	0.21	0.41	0.22	0.42	0.01**	(0.01)
Very Good health	0.33	0.47	0.34	0.47	0.01	(0.06)
No insurance coverage	0.31	0.46	0.29	0.45	-0.03***	(0.00)
No medical care	0.10	0.29	0.10	0.30	0.00	(0.95)
Delay in medical care	0.13	0.33	0.13	0.34	0.00	(0.75)
Do or delay in medical care	0.15	0.36	0.15	0.36	0.00	(0.73)
Observations	25337		25615		51442	

**Note*: Weighted means of the population 0-85 years. Standard error are in parenthesis.

Figure 1.1 shows the trend in both self-reported health, access, and not having insurance for the two groups in the "pre and post" periods. Table 1.3 provides the summary statistics of the treatment and control groups and their sample sizes. Both groups are made up of 50% female with 78% being white and less than half having a high school or less education. About 31% of the treatment group does not have insurance, while only 29% of the control group has no insurance, with the difference being statistically significant at a 1% significance level. There is a slight difference in reported health and access among the groups.

[17] Uninsured is defined in NHIS as a person who reported not having health insurance during the interview under private, medicaid, medicare, state child health insurance program, government health plan and state-sponsored program. We also add health insurance coverage analysis to make our work comparable to what is mostly done in this literature.

Figure 1.1: Trend between treatment and control groups



1.5.2 Behavioural Risk Factor Surveillance System (BRFSS) Data

Given that we do not have detailed information on states in the NHIS, we can present our analysis (with interest in states) using information from the Behavioural Risk Factor Surveillance System (BRFSS). We use data from the public files of the BRFSS from 2011 to 2016 to assess the effect of medicaid expansion under the ACA.[18] The BRFSS is an ongoing phone interview survey by the Centre for Disease Control (CDC) that interviews respondents on health outcomes, use of preventive services and healthy behaviour. It also records demographic characteristics for those 18 years and older. A nationally representative sample of over 300000 individuals is interviewed over the course of the year. In this study we focus on information on cost-related barriers, health insurance and the general health status of respondents. Cost prevented care is a binary outcome, it equals to 1 if the individual did not have a cost related barrier and 0 otherwise. Insurance coverage indicates whether a respondent has insurance, 1 and 0 otherwise (reverse is not having insurance coverage). Similarly, having a personal doctor is the response to having at least one doctor or otherwise. Having a checkup is 1 when a respondent has had a checkup within the last year and 0 otherwise. This way of creating the accessible variables is our way of recoding the variables, which is different from the original coding used in the BRFSS. Like the NHIS, we use self-assessed health status because the small sample sizes for objective measures such as hypertension and diabetes in the BRFSS data sets are due to sub-sampling. Nevertheless, we provide correlations between these objective measures of health and self-assessed health status. In the BRFSS data, using a chi-squared test, we find strong evidence against the null that there is no relationship between hypertension status, diabetes status, and self-assessed health status at a 1% significance level.

In 2015, about 30 states, including the District of Columbia, expanded medicaid under the ACA. These states are specified as the treatment group the 21 other states are the control

[18]We exclude the period before when there were no mobile phone interviews which ended in 2010. The BRFSS data is desirable for evaluation of the medicaid expansion because of its larger sample size as well as state identifiers which are easily accessible in the public data files.

groups. We exclude states that expanded medicaid in 2014 but had prior medicaid expansion similar to the ACA; namely, Delaware, the District of Columbia, Massachusetts, New York and Vermont. The BRFSS only contains income categories, so we restrict the sample to those with income below $15,000 since eligibility under the medicaid expansion was for individuals with income below 138% of the federal poverty line (FPL).

Summary statistics

Table 1.4 provides descriptive statistics for the sample for both medicaid and non-medicaid expansion states. Medicaid states are more likely to report having some form of insurance compared to non-medicaid states. About 28% percent had monetary related accessibility problems and did not use health care when needed in medicaid expansion states.[19] According to Table 1.4, this is lower than the 38% percent in non-medicaid expansion states. However, there is only a 2% difference in persons reporting not having a primary doctor in both areas and it is significant at the 5% level. People in both states are comparable in observed characteristics, as shown in Table 1.4. That is, the proportion of females, individuals who are married, working-age people, non-hispanic white, the unemployed, students and those with income less than $10,000 are similar in the two states being compared.

[19]Note that as explained earlier in the NHIS data, our accessibility variables are defined as 1 when respondents did not have any difficulty accessing health care and 0 otherwise. However, the summary statistics are shown for the proportions that have accessibility hurdles.

Table 1.4: Summary statistics for medicaid and non medicaid states from 2011 to 2016

	Medicaid States		Non- Medicaid states		Difference	
	mean	sd	mean	sd	b	p
18- 64 years	0.84	0.37	0.82	0.38	-0.02***	(0.00)
65+	0.16	0.37	0.18	0.38	0.02***	(0.00)
Married	0.20	0.40	0.20	0.40	0.00	(0.96)
Female	0.58	0.49	0.59	0.49	0.01***	(0.00)
Hispanic	0.10	0.30	0.06	0.23	-0.04***	(0.00)
Non-hispanic white	0.42	0.49	0.46	0.50	0.04*	(0.03)
Non-hispanic black	0.14	0.35	0.26	0.44	0.12***	(0.00)
Income below $10000	0.54	0.50	0.51	0.50	-0.03***	(0.00)
Graduated high school	0.31	0.46	0.33	0.47	0.02***	(0.00)
College	0.26	0.44	0.24	0.43	-0.02***	(0.00)
Graduated College	0.07	0.26	0.06	0.24	-0.01***	(0.00)
Unemployed	0.17	0.38	0.18	0.38	-0.01***	(0.00)
Student	0.10	0.30	0.08	0.27	-0.02***	(0.00)
Poor	0.11	0.32	0.11	0.31	0.00***	(0.00)
Fair	0.18	0.39	0.17	0.37	-0.01***	(0.00)
Good	0.32	0.47	0.32	0.47	0.00	(0.79)
Very Good	0.26	0.44	0.25	0.43	-0.00	(0.42)
Cost prevented care	0.28	0.45	0.36	0.48	0.08***	(0.00)
No insurance	0.28	0.45	0.38	0.48	0.10***	(0.00)
No primary doctor	0.34	0.47	0.36	0.48	0.02*	(0.05)
No checkup	0.38	0.49	0.37	0.48	0.01	(0.16)
Observations	103623		121152		224775	

**Note*: Weighted means of the population 0-85 years. Standard error are in parenthesis.

1.6 Results

This section provides the results for the before and after analysis for the entire vulnerable population (includes the entire sample with different degrees of vulnerability defined on a fuzzy set) and then a comparison for demographic and socioeconomic groups. As mentioned earlier, this study aims to compare leakages before and after the ACA was implemented. Leakage is the proportion of individuals in vulnerable health who have an accessibility hurdle. Trying to compare two years of leakages in health care reduces to comparing two joint cumulative distributions of health and accessibility, in our case, in 2009 and 2015. We also present the results of the causal analysis for the young adult coverage and the medicaid expansion under the ACA where we compare the joint distribution before the implementation of the ACA to a counterfactual distribution that is derived following Athey and Imbens (2006).

1.6.1 Before and After Analysis

We test for dominance using information on the delay in health care use due to cost, information on whether a person had no access to care due to cost, and then for having at least one of the cost related barriers among individuals with vulnerable health as defined earlier. In each of the results tables, each cell represents a dominance test of the same population or subgroup in 2009 and 2015. The main results for these dominance test are presented in Table 1.5, where "D" means leakages (accessibility hurdles for vulnerable population) in 2015 dominates 2009, and "ND" implies non dominance. We use bootstrap standard errors and critical values from the normal distribution.

Table 1.5 shows that leakages in 2015 dominate leakages in 2009. We have strong evidence to support the null of dominance, $H_0 : F_{2015}(h, 0) \leq F_{2009}(h, 0)$ at 1% significance level but we reject the null that $H_0 : F_{2015}(h, 0) \geq F_{2009}(h, 0)$. This implies that there are lower accessibility problems in 2015 compared to 2009 for people in vulnerable health. Individuals who reported a delay in medical care due to cost also reduced in 2015 compared to 2009. Not getting medical care due to cost in 2015 was significantly lower than in 2009. Figure 1.2 shows the difference and a 95% confidence interval for having leakages (having at least one of the cost-related access problems). The figure gives a graphical depiction of the dominance test. As seen in the graph, the difference and the confidence interval all lie below the zero line. The difference in leakages and their respective t statistics are also shown in Table A.1

in the Appendix 1.

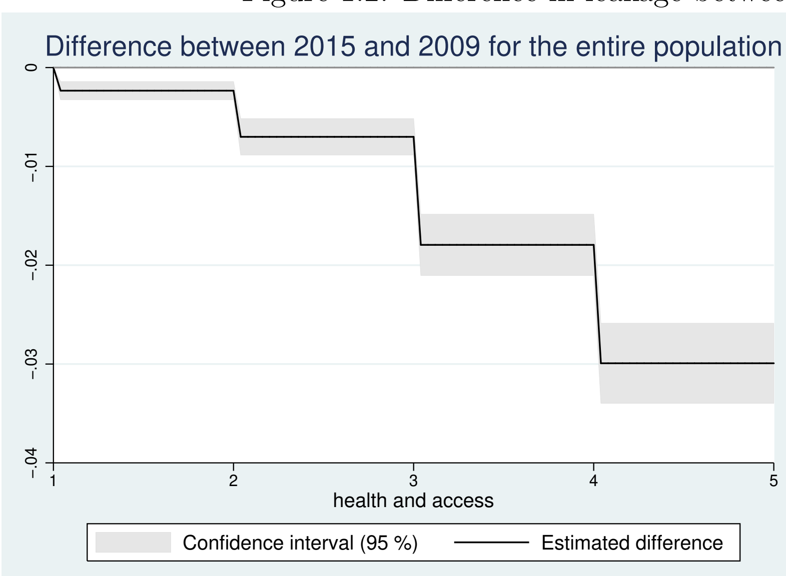
There is a possibility that although the entire population has seen a reduction in access problems, some subgroups among people in vulnerable health might still have higher unmet needs. We decompose the results by demographic, geographic location and socioeconomic factors.

Table 1.5: Results from stochastic dominance test

	Population
No medical care	D^{***}
Delay in medical care due to cost	D^{***}
No or Delay in medical care	D^{***}

Note: D means dominance of 2015 over 2009 and ND means non-dominance *,**,*** significant at the 10%, 5% and 1% respectively.

Figure 1.2: Difference in leakage between 2015 and 2009



Demographic factors

The ACA may have different impact on different age categories. Table 1.6 shows the decomposition of the dominance test by age groups. If we focus on the working age population of 18 to 65, there has been a significant reduction in cost-related barriers for individuals in vulnerable health from 2009 to 2015. Similarly, for 0 to 17 years, there is dominance of leakages in 2015 over 2009. That is, there are lower accessibility hurdles for people in vulnerable health who are 17 years or younger. However, the null of dominance of no medical care due to cost of 2015 over 2009 for the ages 65_+ is rejected at a 10% significance level. This implies that there is no distinguishable difference in cost prevented care for people aged 65 or older in 2009 and 2015. A possible explanation is that individuals who are 65 years and older were covered by medicare before the implementation of the ACA.

Another interesting area to explore is access to health care by gender. There is apparent gender-based inequality concerning access to and use of health care. Although there is no key basis for this inequality, there tend to be access inequalities by sex (Merzel (2000)). Although we do not compare men and women, we test for dominance in each group's leakages separately, before and after the ACA. As seen in Table 1.6, we do not reject the null $H_0 : F_{2015}(h,0) \leq F_{2009}(h,0)$ but have strong evidence against the null $H_0 : F_{2015}(h,0) \geq F_{2009}(h,0)$, which implies leakages for male in 2015 dominates 2009. That men have lower accessibility hurdles in 2015 compared to 2009. The results hold for when we analyze not having accessibility to care due to cost separately from delay to care due to cost. A similar conclusion is found for females, as leakages are much lower in 2015 compared to 2009. Leakages are much lower irrespective of a person's gender after the ACA.

Another disparity commonly noticed in health is by race or ethnicity. According to William(1993), racial disparities in healthcare access exist in the US, which serves as a motivation for a dominance test based on race. For whites, leakages in 2015 significantly dominate leakages in 2009 as shown in Table 1.6 because we find strong evidence to support the null $H_0 : F_{2015}(h,0) \leq F_{2009}(h,0)$ and reject the null $H_0 : F_{2015}(h,0) \leq F_{2009}(h,0)$. Similar results are achieved for just a delay in medical care as well as no medical care due to cost. Blacks in vulnerable health also have lower accessibility hurdles in 2015 than in 2009. However, the dominance of 2015 over 2009 is rejected for Asians and all other races for all measures.

Table 1.6: Demographic Factors

	No medical care	**Delay in medical care**	**No or Delay on medical care**
Age			
0-17	D^{*}	D^{***}	D^{**}
18-64	D^{***}	D^{***}	D^{***}
65+	ND	D^{*}	D^{**}
Sex			
Male	D^{***}	D^{***}	D^{***}
Female	D^{***}	D^{***}	D^{***}
Race			
White	D^{***}	D^{***}	D^{***}
Black	D^{***}	D^{***}	D^{***}
Asian	ND	ND	ND
Other race	ND	ND	ND

Note: D means dominance of 2015 over 2009. ND means non-dominance
*,**,*** significant at the 10%, 5% and 1% respectively.

Since some states expanded medicaid and others did not participate in 2014, there is a possibility that accessibility problems are unbalanced across states. Differences in access per geographic location are highlighted if these differences exist. Since the public use files of the NHIS data do not include states, we test for dominance by regions. In all regions (South, Northeast, Midwest, and West), there is dominance of 2015 over 2009, as shown in Table 1.7. The results show that people in vulnerable health in all regions saw a reduction in accessibility hurdles in 2015 compared to 2009. Although some regions did not expand medicaid, access may have improved due to a federal marketplace exchange set up in these regions where individuals could get subsidies for insurance premiums. Because insurance is a possible channel through which accessibility changes, Frean et al. (2017) estimate that although state exchanges led to a higher increase in insurance, non-federally set-up exchanges in 2013 also had a considerable impact on insurance in 2014 and 2015, and hence on access.

Table 1.7: Region

	No medical care	**Delay in medical care**	**No or Delay on medical care**
Northeast	D^{***}	D^{***}	D^{***}
Midwest	D^{***}	D^{***}	D^{***}
South	D^{***}	D^{***}	D^{***}
West	D^{***}	D^{***}	D^{***}

Note: D means dominance of 2015 over 2009 and ND means non-dominance
*,**,*** significant at the 10%, 5% and 1% respectively.

Socio-economic factors

There is a correlation between income and access to health care (Ettner (1996)). The higher one's income, the higher one's ability to pay for health services; therefore the lower the accessibility problems related to cost. Turning to income, we hypothesize that given universal coverage, a person's income level should not affect health access. We use family income as a measure of socioeconomic status. The imputed income files are adjusted by dividing by the square root of the household size to get individual income, which are divided into five quantiles. Due to the small sample size in each quantile of the bottom of the health and access distribution, we combine the lowest two quantiles and the highest three quantiles. For both the lowest quantiles and highest quantiles, we do not reject the null of dominance of 2015 over 2009 at a 1% significance level. This is shown in Table 1.8, where dominance is achieved for the leakage in having at least one accessibility problem, delay in accessing medical care due to cost and not getting medical care due to cost in the year 2015 over 2009. This implies an individual in the lowest two quantiles and vulnerable health has lower leakage in 2015 than the year 2009. Similarly, individuals in vulnerable health in the highest quantiles have better access to care in 2015 than in 2009.

Education could be a channel for inequality in access. Highly educated individuals usually have easy access to health care due to their access to information which helps them take advantage of any positive changes in health insurance laws. Table 1.8 shows different education status categories, having high school or a lower level of education and having college or higher level of education. There is less leakage in 2015 than period 2009 for patients who have a high school education or less in all cases. The results for those who have achieved a college degree up to the doctoral level are not any different. The highly educated group also experience significantly lower leakages in 2015 compared to 2009.

Table 1.8: Socio-Economic Factors

	No medical care	**Delay in medical care**	**No or Delay on medical care**
Income			
Lowest Quantiles	D^{***}	D^{***}	D^{***}
Highest Quantiles	D^{***}	D^{***}	D^{***}
	No medical care	**Delay in medical care**	**No or Delay on medical care**
Education			
High school or less	D^{***}	D^{***}	D^{***}
Some college education	D^{***}	D^{***}	D^{***}

Note: D means dominance of 2015 over 2009 and ND means non-dominance.
*,**,*** significant at the 10%, 5% and 1% respectively.
Equivalence scale used is $h^{0.5}$

1.6.2 Robustness checks

The recession in 2007/2008 increased the unemployment rate, which decreases income and affects use and access (Schaller and Stevens (2015)). Due to the high correlation of the unmet self-perceived responses with the recession, there is the possibility that the leakage measure for 2009 will be biased upward. To check for robustness, we compare 2010 to 2015 and 2006 and 2015.[20] Although the ACA was enacted in 2010, only the young adult coverage was implemented that year. We exclude individuals aged 19 to 25 years in both 2010 and 2015. Table A.2 in the Appendix provides a difference and the t statistics for the dominance test. 2015 is associated with lower leakage than 2010. Although they are not shown here, we find similar results for 2006 and 2015.[21]

1.6.3 Discrete Changes in changes and Dominance

Table 1.9 presents the statistical test results for a null of dominance of the observed distribution of the treatment group and its counterfactual distribution in leakages for young adults. We also test for the null of dominance of the estimated counterfactual distribution over the treatment group's observed distributions. Among young adults in "vulnerable health", for leakage in not having insurance, we find that there is dominance of the treatment group over the estimated counterfactual group. When we consider the effect of the ACA on other health care access problems among young adults, there is non-dominance for leakage in no medical care and delay in care. Similar results hold when the outcome of interest is either leakage in having no medical care or a delay in medical care. The results implies that

[20]We used the period 2007 or 2008 because of the same issue in 2009. We also believe expectation about subsidies and medicaid expansion may lead to an upward bias in leakage in 2011 to 2013

[21]Result for the dominance test between 2006 and 2015 are available upon request

there is no distinguishable difference between the distribution for the young adults and the estimated counterfactual distribution.

Table 1.9: Dominance test for young adults using NHIS data

No insurance coverage	D^{***}
No medical care	ND
Delay in medical care	ND
No or Delay in medical care	ND

Note: Dominance test between observed leakages in young adults between 23 to 25years and its counterfactual with 27 to 29 as the control group

D means dominance of 2015 over 2009 (i.e. less leakages). ND means non-dominance

*,**,*** significant at the 10%, 5% and 1% respectively.

Individuals in medicaid expansion states experienced a reduction in leakages in not having health insurance coverage, cost prevented care, not having a primary doctor and having no checkup compared to non medicaid states. The results are shown in Table 1.10. The null of dominance (of the observed distribution of the treatment group over the estimated counterfactual distribution) is not rejected because of lack of evidence against the null for not having health insurance and any form of accessibility hurdle. If the aim of the policy was to provide insurance, then these results show that there has been a significant decrease in leakages in not having insurance in medicaid states. The test for dominance is also not rejected for cost prevented care, not having a primary doctor and having no doctor at 1%, 1% and 5% significance level respectively. Unlike the results for the simple before and after results presented earlier, these are causally related to the Affordable Care Act medicaid expansion because we test for dominance between the observed distribution of the treatment group and its counterfactual distribution, not just on average but over the health distribution. This shows that leakages have decreased in medicaid expansion states. These findings aligns with findings by Courtemanche et al. (2018) and Sommers et al. (2015) on the effect of medicaid expansion on access. Both studies show an increase in access after the expansion of medicaid. [22]

Table 1.10: Dominance test for medicaid expansion using BRFSS data

No insurance	D^{***}
Cost prevented care	D^{***}
No checkup	D^{**}
No primary doctor	D^{***}

Note: Dominance test between observed leakages in medicaid expansion states and its counterfactual with non-medicaid expansion states as the control group.
D means dominance of 2015 over 2009 (i.e. less leakages). ND means non-dominance.

*,**,*** significant at the 10%, 5% and 1% respectively.

1.6.4 Placebo Analysis

Our results will be biased if there are unobserved factors that affect health and health care utilization that are not accounted for in our analysis. For instance, in the case of medicaid expansion, if ACA's expectation after its enactment in 2010 forced people to reduce health care use in anticipation of the ACA's implementation in 2014, such a reduction in accessibility

[22] Heterogeneous effect under the policy is relevant both for young adults and medicaid expansion but due to sample sizes, we could not exploit this further.

will bias our estimates. Due to this possibility, we conduct a falsification test to test the validity of our estimates further. As a robustness check, we create an "artificial" medicaid expansion policy that is implemented in 2010 . Following the same approach as earlier, we use data from BRFSS from 2007 to 2010 as the "pre" period and 2010 to 2013 as the "post" period, three years before and after the policy was implemented respectively. Table 1.11 presents the results for the placebo test. For medicaid and non-medicaid states, we can not robustly rank the states in access to insurance, cost prevented, access to checkup, and accessibility to a primary doctor in the periods before and after the "artificially" created medicaid expansion.

In addressing the demand for health care and how accessibility has changed in the period before and after the ACA, one key assumption this paper implicitly made is that, the supply of health does not change in this period therefore the membership function does not change over time. To test the validity of this assumption, we used a two sample Kolmogorov Smirnov test to test the difference in health distributions across time ("pre" and "post"). Using the NHIS data for 2009 and 2015, we can not reject the null that the health distributions for the two periods are from the same population at the 5% significance level. The results also holds when we compare the distribution for young adults and for medicaid and non-medicaid states. This tends to suggest that there are some arguments in support of the assumption that the health distribution did not change over the periods considered in the paper.

Table 1.11: Placebo test for medicaid expansion using BRFSS data

No insurance	ND
Cost prevented care	ND
No checkup	ND
No primary doctor	ND

Note: Dominance test between observed leakages in medicaid expansion states and its counterfactual with non-medicaid expansion states as the control group. D means dominance of 2015 over 2009 (i.e. less leakages). ND means non-dominance *,**,*** significant at the 10%, 5% and 1% respectively.

1.7 Conclusion

This paper address the question of how health care accessibility has evolved for people in vulnerable health in the period before and after the implementation of the Affordable Care Act. We allow for a gradual transition between vulnerable and non-vulnerable health which mimics what is seen in real life. This is achieved by defining a non-dichotomous membership function. The membership function is then used to derive a leakage index. The choice of the membership function is however arbitrary as a researcher can choose any increasing function between 0 and 1. To overcome this issue, we derive a stochastic dominance criterion that allows for robust conclusion irrespective of the choice of the membership function. This allows us to provide a robust ordering of any two distributions.[23]

Having developed this approach, we apply the method to compare leakages in simple before and after analysis for the Affordable Care Act. We also extend our analysis to allow for causal inference by exploiting two quasi-experiments under the ACA, the young adult coverage, and the medicaid expansion. We extend Athey and Imbens (2006) approach to a bivariate setting and use this identification strategy to establish causality.

Our results indicate that leakages were more pronounced in the period before the ACA compared to the period after based on a before and after comparison. A robustness check using different years validates our results. In addition, using the extended bivariate changes in changes approach, we find that there has been a decrease in leakages in health insurance and access for states that expanded medicaid. This solidifies or asserts the initial findings of simple before and after comparison. Our results are in support of studies that find positive effects of the Affordable Care Act on health care accessibility (e.g. Courtemanche et al. (2018)). These findings are important because they show that an increase in insurance coverage has been accompanied by an increase in access to medical care.

There are many possible extensions to this paper. From a policy perspective, one possible question that can be answered using the theoretical approach in this paper is about, the effect of health insurance on risky behavior and preventive care of people in vulnerable health in Canada and the USA. So far, studies that compare Canada and US focus on either health outcomes or access. It will be interesting to find out if risky and healthy behaviors are

[23]It is worth noting that the leakage index and robust ranking in this paper is not restricted to only comparing different time periods in a specific country but can be used to compare health care systems at different places. Also, although this method has been applied to self-assessed health status, it can be applied to other measures of well-being.

different, especially among people at the lower tail of the health distribution in these two countries.

Chapter 2

Assessing the impact of recreational marijuana legalization on the distribution of BMI and healthy behaviours in the United States *

2.1 Introduction

Over the years, marijuana has moved from being an illicit drug in the United States to being a legal drug for medical and recreational purposes in some states. About 8 states and the District of Columbia had legalized recreational marijuana as at 2017. Policy makers and researchers have been interested in the implications of these legalization laws on health

*This chapter is based on joint research work with Paul Makdissi and Myra Yazbeck outcomes. One of these health outcomes is obesity or weight which is a morbidity issue in the USA.

Empirical evidence on marijuana use following recreational marijuana legalization shows that marijuana use has increased among young adults (Hao and Cowan (2020); Ambrose et al. (2021)). An increase in marijuana use can have both a health deterioration or an improvement effect. One potential health risk is obesity due to a lack of physical activity or an increase in appetite for high calorie foods and sleep. An improvement in health could be a decrease in pain or nausea in the short run.[1]

According to the Centers for Disease Control (CDC), the prevalence of obesity increased from 30.5% in 1999-2000 to 42.2% in 2017-2018. Obesity has been linked to heart diseases (one of the leading causes of death), stroke, diabetes, high blood pressure, unhealthy cholesterol, asthma, sleep apnea, gallstones, kidney stones, infertility and cancer (CDC). The CDC has also recently linked obesity to increased risk of severe illness of COVID-19 and it's mortality. Studies also show some social costs of obesity include discrimination (Spahlholz et al. (2016)), lower quality of life, depression and even lower wages (Baum and Ford (2004)). In terms of its economic cost, obesity was responsible for about 6% of medical costs in 1998, or about $42 billion (in 2008 dollars). By 2006, obesity was responsible for closer to 10% of medical costs or nearly $86 billion a year (Finkelstein et al. (2009)).

The objective of this paper is to assess the impact of recreational marijuana legalization (RML) on the distribution of the body mass index (BMI) and healthy behaviour. The effect of marijuana laws, and hence its use are ambiguous. On the one hand, recreational marijuana

[1] Although it does not solve the underlying health problem.

use can reduce weight through an increase in physical activity and energy levels or by lethargy (Sabia et al. (2017). On the other hand, marijuana use can induce an increase in weight through substitution towards high caloric foods and drinks (Berry and Mechoulam (2002)). In this paper, we explore the potential effect of RML on BMI and other healthy outcomes. Empirical evidence of the relationship between marijuana policy and its implication for weight and BMI is limited. The only initial study of the relationship between marijuana laws and BMI study found that medical marijuana utilization reduces BMI and obesity on average (Sabia et al. (2017)). Our study is the first to use the CIC approach to estimate the effect of recreational marijuana at different points of the distribution of BMI

We contribute to the literature in two ways. First, we add to the growing literature on the relationship between recreational marijuana laws and health outcomes and healthy behaviour. Second, we are the first to provide distributional effects of the RML as opposed to the average effect of the policy. While an average effect of RML estimate provides insight on common problems, it might mask the problem encountered at different points of the distribution. For instance, while a reduction in BMI is deemed good from a public health perspective, if this reduction comes from the top of the distribution of BMI (obese people being less obese), it may be deemed bad if it comes from the bottom of the distribution (an increase in anorexia) and this difference is unknown when we look at the average effects of the policy. This means the desirability of the change in BMI is opposite depending on the part of the distribution of BMI the change is happening. In addition, policy makers may be interested in the effect of the RML of the distribution other than the mean.

In this paper we exploit the quasi-experimental design of the recreational marijuana legalization laws in states. Exploiting this quasi-experiment allows us to tease out the causal impact of RML on the distribution of BMI and healthy behaviours. A common way to estimate the treatment effect, in this case, would be to use a difference in difference approach (DID). A few studies analyze the effect of marijuana policy on BMI or obesity. These studies use a parametric difference in difference approach that focuses on assessing the impact of the average treatment effects. As stated earlier, the average treatment effect will mask the exact part of the BMI distribution where RML impacts. For instance there might be differences in the effect of marijuana policy at different point of the distribution of BMI. At the lower tail are individuals who are potentially underweight while at the upper tail are those who are obese. It is policy relevant to know the part of the distribution of BMI to make policies suitable for the groups affected accordingly. In addition, using a linear difference in difference approach may lead to estimates outside the allowable range when the outcome of interest is a dummy variable.

To resolve these issues with the DID, we use Athey and Imbens (2006) changes in changes approach to identify the effect of RML on the entire distribution of BMI. Under the assumption that there is a strictly monotonic BMI production function and time invariance within groups we are able to identify the entire distribution of BMI in the absence of RML (counterfactual distribution). We then test for dominance between the observed distribution of BMI in recreational marijuana states and the counterfactual distribution and also at specific part of the distribution. In addition we use DID to estimate the effect of BMI on average as earlier studies. We also estimate the effect of RML on alcohol consumption, smoking and physical activity using a linear DID and a non-linear DID (Puhani (2012)).

The effect of recreational marijuana legalization on BMI are identified using states that had already legalized medical marijuana (MML) in the periods prior to recreational marijuana laws, and that stayed as MML states after RML (that is, MML and stayed MML), as control states for states who had MML and later legalized recreational marijuana (MML to RML). Thus, any differences in BMI can be attributed to recreational marijuana policy.

We use data from the Behavioural Risk Factor Surveillance System (BRFSS) to estimate the effect of recreational marijuana legalization on the entire distribution of BMI. The BRFSS provides information on respondents weight and height. The survey also includes a derived variable for BMI, healthy behaviour, demographic characteristics and state identifier which are essential in answering our research question.

Our results from the CIC approach shows that, recreational marijuana legalization reduces BMI for the entire population. The effect is mainly in the middle and top parts of the BMI distribution. We also find evidence that there is variation in the effect of RML for different groups. The reduction in BMI as a result of RML is seen in females but not in males. For females the effect is found both at the lower tail (being underweight) and at the upper tail (morbid obesity). While we find evidence of a reduction in being overweight for both whites and non-whites due to RML, the reduction in obesity and morbid obesity is only found for non-whites. In addition, RML reduces obesity for non-whites and those below 45 years and it reduces morbid obesity among women and non-whites. The difference in difference approach supports this finding. We also find evidence that recreational marijuana legalization have adverse health effects by increasing alcohol consumption and reducing the probability of participating in any physical activity.

The rest of the paper is structured as follows. Section 1.2 is a review of the recent literature, while Section 1.3 explains the conceptual framework. Section 1.4 describes the DID and CIC approaches to estimating the effect of marijuana on BMI and other outcomes. In Section 1.5 we discuss the data used and how our outcomes of interest are defined, Section 1.6 presents the main results and shows the results of robustness check. Section 1.7 concludes.

2.2 Literature review

This research explores the relationship between the recreational legalization of marijuana in the US and its effect on BMI and healthy behaviour. The process to legalization of marijuana in the US has been lengthy but in 2012, two states (Colorado and Washington) legalized the use of marijuana for recreational purposes. In the period before 2012, many states had medical marijuana (MML) policies that allowed for marijuana to be used for medical purposes. Researchers have therefore analyzed the relationship between MML and use and healthy behaviours and most recently, between RML and health outcomes. Anderson and Rees (2021) provide a comprehensive summary of the literature on both medical and recreational marijuana in the US. The authors note that studies in the US on marijuana are still ongoing as policymakers make decisions about legalization in states. We provide a brief summary of the literature on medical marijuana laws and recreational marijuana legalization.

Several studies focus on medical marijuana legalization and usage. For instance, using a DID approach, Pacula et al. (2015) and Mark Anderson et al. (2015) do not find any evidence that medical marijuana legalization affected utilization of marijuana among adolescents using both cross sectional and longitudinal data sets. A recent study in which Coley et al. (2021) analyze the effect of recreational marijuana legalization (RML) using data from Youth Risk Behavior Survey shows that RML is not associated with a significant shift in the likelihood of marijuana use but predicts a small statistically significant decline in the level of marijuana use among users.

On the contrary, using the Youth Risk Behavioural Survey, Choo et al. (2014) find that the legalization of medical marijuana increased the use of marijuana among adolescents. Similarly, using BRFSS data, Ambrose et al. (2021) study the effect of recreational dispensaries on marijuana use among adults in Washington state. The authors find that a 33% reduction in driving time to the nearest recreational dispensary leads to a 5% increase in the probability of past-month marijuana use among individuals ages 18 and over. The effect is slightly higher among those ages 18 to 26. For these young adults, a 33% decrease in driving time is associated with a 9% increase in the probability of past-month marijuana use. The question then becomes if this increase in marijuana utilization translates into non-healthy behaviour, worse health outcomes and an increase in weight.

Findings from the literature on the effect of marijuana laws on alcohol use are mixed. Mark Anderson et al. (2013) examine the effect of medical marijuana legalization(MML) use on alcohol consumption using data from BRFSS and a DID approach and find that MML is associated with a reduction in alcohol consumption. Using a similar data set, Sabia et al. (2017) also find a reduction in binge drinking after the legalization of medical marijuana. Alley et al. (2020) examine the effect of recreational marijuana legalization on college students' drinking behaviour using cross-sectional National College Health Assessment-II survey. They find that RML reduces the likelihood of binge drinking among college students aged 21 and older. Some studies have also used sales data to examine the effect of marijuana policies on alcohol consumption. For instance using data from the Nielsen Retail Scanner data set from 2006 to 2015 and a difference in difference approach at the county level, Baggio et al. (2020) find that MML reduced alcohol sales in these states by 12.4%.

In contrast, Wen et al. (2015) use data from the NSDUH from 2004-2012, leveraging a DID approach and find that MML increases the probability of binge drinking in respondents who were age 21 and above by 10%. However, Veligati et al. (2020) do not find any evidence that any marijuana legalization both (either medical or recreational) has any effect on alcohol use when the authors used data from state tax receipt. These mixed results from authors who mostly use a linear probability model(difference in difference) calls for further studies looking at effect of recreational marijuana legalization on alcohol using a non-linear difference in difference model.

Another strand of studies look at the effect of marijuana legalization laws on tobacco use. These studies either show that marijuana laws have no effect on tobacco use or that they prompt a reduction in use. According to Alley et al. (2020) recreational marijuana legalization has no effect on young adults' use of tobacco. Similarly using National Institute on Alcohol Abuse and Alcoholism's data, AEDS and BRFSS respectively, Veligati et al. (2020) and Andreyeva and Ukert (2019) do not also find any association between medical marijuana legalization and cigarette use.

Nevertheless, a study by Choi et al. (2019) using three data sources, BRFSS, the Current Population Survey Tobacco Use Supplements (CPS-TUS) and the National Survey on

Drug Use and Health, show that MMLs reduces use of any cigarette smoking by 1 to 1.5 percentage-point. The authors do not find any evidence that MMLs are associated with a reduction in the number of cigarettes consumed per day among current smokers. In addition, Anderson et al. (2020) also find evidence that MMLs are associated with a 6% reduction in any teen cigarette use in the past month and a 12% decrease in frequent teen cigarette (20 cigarette in the past 30 days), but the authors do not find any evidence that RML affects cigarette use.

Another healthy behaviour of interest is physical activity. Studies on marijuana laws on physical activity are limited. Among these limited studies, there is evidence that repeated marijuana use reduces athletic performance (Pesta et al. (2013)), and increases lethargy (Delisle et al. (2010); Irons et al. (2014)). Recently, Sabia et al. (2017) use data from the BRFSS and find evidence that medical marijuana legalization is associated with a reduction in physical activity.

A few studies have analyzed the effect of marijuana laws on body weight. For instance, using longitudinal data from the National Longitudinal Study of Adolescent Health and a fixed effect model that is robust to time invariant individual unobserved variables, Beulaygue (2012) find that there is a negative relationship between marijuana use and body weight. However, using individual fixed effects models does not exclude the fact that there is a possibility that these findings are explained, in part or in whole, by time-variant unobserved variables.

A study closely related to our research is a recent one by Sabia et al. (2017). The authors look at the effect of medical marijuana legalization on body weight. Using the Behavioural

Risk Factor Surveillance system (BRFSS) and a DID approach, the authors find that medical marijuana legalization leads to a 2% to 6% reduction in obesity on average. Nevertheless, marijuana legalization may have different impacts at different points of the BMI distribution. Our study is the first to use the CIC approach to estimate the effect of recreational marijuana at different points of the distribution of BMI.

A review of the above mentioned literature shows there are limited studies that look at the effect of RML. There are even fewer studies that examine the association between these RML policies and BMI. This paper contributes to this strand of studies by looking at the entire distribution of BMI and the implication of recreational marijuana legalization.

2.3 Conceptual framework

There are varying ideas about how recreational marijuana use can potentially affect the body mass index. There is growing evidence in medical research on the neurophysiological mechanisms through which marijuana affects appetite. Soria-Gómez et al. (2014) find that the euphoric element in marijuana, tetrahydrocannabinol (THC), is also responsible for its appetite-suppressing effects. THC is one of several cannabinoid-like chemicals known as exogenous cannabinoids because they enter the body through a different route than cannabinoids do (i.e., the consumption of marijuana). Cannabinoid compounds found naturally in the body are known as endocannabinoids. They attach to a receptor known as the CB receptor, which has an impact on regions of the body connected to hunger, such as the gastrointestinal tract, which moderates food intake; the hypothalamus and hind brain, which control food intake; the stomach and intestinal tissue, which ship indicators of satiation to the brain; and the limbic forebrain, which influences the palatability of meals. It is more obvious that the adjustments in marijuana intoxication, or the management of THC, mirror an important function for those endocannabinoid structures in the urge for food, ingestive behavior, power metabolism, and body weight (Kirkham (2005)).

Some evidence from studies that use a randomized control trials show that marijuana use ends in expanded urge for food and caloric intake (Greenberg et al. (1976); Berry and Mechoulam (2002). Foltin et al. (1988) find evidence that THC consumption leads to between meals snacking, and hence to high caloric consumption although calorie consumption for regular meal remains the same. The authors findings support the famous notion of 'muchies' associated with marijuana use. In addition, in a recent study, Lu (2021) shows that recreational marijuana laws lead to an increase in household food expenditure, particularly food consumed outside the home.

Other channels through which marijuana potentially affects diet, and hence BMI is are substitution and the complementary effect of other substances. Some studies show that marijuana and alcohol use are associated. Veligati et al. (2020) find evidence that alcohol consumption reduces due to marijuana laws, which suggest that marijuana and alcohol are substitutes. However, Wen et al. (2015) show that binge drinking increases with marijuana laws, and thus there is a complementary relationship between marijuana and alcohol. The magnitude of the change in weight due to marijuana laws is estimated by some earlier studies as well. For instance, Mark Anderson et al. (2013) estimate that medical marijuana laws reduce alcohol consumption by about 10.6% to 25%. According to Nielsen (2012) alcohol is mostly high calorie drinks; that is, the common serving of beer includes more or less 150 calories and a common serving of wine and spirits is about 120 calories. Hence a reduction in alcohol implies a reduction in calories consumed, and therefore a likelihood of a reduction in weight and vice versa.

In addition, intake of different substances such as cigarettes or illicit pills can be stricken by RMLs, which, in turn, might also have additional effects on weight. If RML affects smoking of tobacco (Coley et al. (2021)) and according to Chen et al. (2005) smoking reduces food intake, then smoking of tobacco can lead to a reduction in weight.

Furthermore, Vidot et al. (2017) report that marijuana users are less likely to participate in both moderate and vigorous physical activity than those who have never used marijuana. Nevertheless, Sabia et al. (2017) find evidence that medical marijuana laws increase physical activity among older adults. This reduction or increase in physical activity by marijuana users affects weight.

In contrast to the channel of appetite, Clark et al. (2018) provide a theoretical mechanism under which marijuana use can affect body weight if the relationship between body weight and food intake move in opposite directions. The authors argue that the mechanism by which marijuana use affects BMI is changes in metabolism and not changes in caloric intake or activity-changing energy level. Clark et al. (2018) explain that "cannabis use causes downregulation of CB_1R, reducing the impact of enhanced N-arachidonoylethanolamide (AEA) and 2-AG production arising from an elevated dietary omega-6/omega-3 ratio" and lowers CB_1R activity reducing obesity and metabolic disruption.

2.4 Empirical Strategy and Model

In this section, we outline some intuitions, assumptions required and identification under quasi experimental designs such as difference in difference (DID) and changes in changes (CIC). We also highlight some issues associated with using a difference in difference approach and some solutions.

2.4.1 Difference in Difference: Intuition, assumptions and identification

We are interested in identifying the effect of the recreational marijuana policy on BMI. Ideally we would like to examine this effect with a treatment group and a control group that are randomly assigned. Given that states that legalize recreational marijuana are not random, this ideal setting is not feasible. A good alternative to randomization is using a quasi-experiment. One of the popular approaches is the difference in difference (one of our empirical strategies). Using this, we can compare changes in BMI in states that have legalized recreational marijuana to states that have not adopted the legalization laws using the difference in difference (DID) approach. The difference in difference approach to analyzing such experimental design relies on the assumption that the two groups have a common trend in the outcome of interest. This means that in the absence of the treatment, the difference in returns to unobserved characteristics between the treatment and control groups remains the same over time.

Let us define some notation to introduce the DID approach. For a random sample of individuals $i = 1, ..., N$, let Y_i be BMI. We will compare two groups, $g \in [0, 1]$, an eligible treatment group ($g = 1$) and a comparison group ($g = 0$) at two time periods, $t \in [0, 1]$, pre treatment ($t = 0$) and post treatment ($t = 1$). Let Y_i^I be BMI if an individual i has been treated and Y_i^N if the same individual i has not been treated (not subject to the policy change).The DID approach assumes that in the absence of treatment the average outcome in the treatment group is assumed to experience the same change as the one experienced by the control group between period 0 and period 1.

We will use some notations from Neyman (1923, 1990) potential outcome framework. Let D be an indicator for the treatment (a state legalizing marijuana) such that $D_i = 1$, if the individual has been treated and $D_i = 0$ if he has not been treated ($D_i = G_i * T_i$).

$$Y_i = Y_i^N(1 - D_i) + Y_i^I D_i \tag{2.4.1}$$

Under DID, we assume that in the absence of the marijuana legalization policy, BMI is linearly additive in group and time periods. Formally,

$$Y_i^N = \alpha + \lambda T_i + \gamma G_i + \varepsilon_i \tag{2.4.2}$$

where λ represents the time effect, γ captures the time invariant group effect and ε_i is the unobserved characteristics of individual i. The unobserved characteristics of an individual ε_i is assumed to be independent of the time and group effects.

Under the DID approach, the average treatment effect on the treated is identified as the difference between the average time trend in the control group and the average time trend in the treatment group for BMI. This estimand eliminates factors that can not be associated with recreational marijuana legalization. The counterfactual effect for a simple DID is given by,

$$E[Y^C|G=1,T=1] = E[Y|G=1,T=0] + (E[Y|G=0,T=1] - E[Y|G=0,T=0]) \tag{2.4.3}$$

The average treatment effect is then given by ,

$$E[Y^I|G=1,T=1] - E[Y^C|G=1,T=1] \tag{2.4.4}$$

2.4.2 Difference in difference model

We will use the variation in timing of adoption of recreational marijuana policy for identification. We will use the DID approach that utilizes non-adopting states as control and differential timing of adoption to estimate the effect. As stated above, the DID assumes that in the absence of the treatment, the average outcome in the treatment group is assumed to experience the same change that is the same as the one experienced by the control group between period 0 and period 1. The DID estimator compares the observed mean outcome in the presence of the treatment to the counterfactual mean.

Formally, We estimate the following model,

$$Y_{ist} = \alpha + X'_{ist}\beta + \tau Group_s * Time_t + \lambda Time_t + \gamma Group_s + \epsilon_{ist} \tag{2.4.5}$$

where Y_{ist} is the outcome for individual i in state s at time t, X is observed characteristics and state unemployment rate, $Time$ is a dummy for post legalization, $Group$ is dummy for states that have legalized marijuana, ϵ is the error term and τ is the coefficient of interest.

Non-linear difference in difference

This research also explores the relationship between the recreational legalization of marijuana on healthy behaviour like drinking and smoking that are binary variables in this case. When the outcome of interest is a dummy, the linear difference in difference does not restrict the probability of success to lie between 0 and 1. Puhani (2012) shows how to estimate the treatment effect in a nonlinear model using either a probit or logit model. The author shows that in the non-linear case, the treatment effect is zero only if the coefficient of the interaction of time and group effect is zero. While the treatment effect in a linear difference in difference "is simply the incremental effect of the coefficient of the interaction, the authors showed that in any other nonlinear "difference-in-differences" model with a strictly monotonic transformation function, the treatment effect is the difference between the cross difference of the conditional expectation of the observed outcome and the cross difference of the conditional expectation of the counterfactual outcome". This is due to the fact that in the non-linear model, the cross difference/derivative in the model without the interaction term is not zero. In the non-linear difference in difference, we estimate the following probit model.

$$Y_{ist} = \Phi(\alpha + X'_{ist}\beta + \tau Group_s * Time_t + \lambda Time_t + \gamma Group_s + \epsilon_{ist}) \tag{2.4.6}$$

2.4.3 Staggered difference in difference

Recently, difference in difference approach application to staggered design or timing of policies have been criticized due to the fact that such approach can lead to bias estimates in the presence of treatment effect heterogeneity (Baker et al. (2021); Sun and Abraham (2020); Goodman-Bacon (2021)). A staggered difference in difference gives estimates that are weighted averages of several treatment effect and these effect can have opposite signs compared to the actual average treatment effect where the treatment effects are dynamic or evolve over time.

One potential solution to this issue is stacked regression, as applied by Cengiz et al. (2019) which entails creating new data sets that are event specific or policy specific and having treatment and "clean control" states. The event specific data sets are then stack to estimate the average effect of a policy. This is the approach we employ in this paper to deal with the issues of the staggered nature of the RML policy.

2.4.4 Changes-in-changes: Intuition, assumption and identification

Our main identification strategy is they change in changes estimator (CIC). Athey and Imbens (2006) change in changes estimator has been gaining some popularity in recent empirical studies but we will provide a brief discussion of the CIC assumptions and how it is a build on from the popular linear difference in difference (DID) estimator. Contrary to DID, under changes in changes, Athey and Imbens (2006) do not put any functional form on the production function which transforms unobservable characteristics to BMI. Formally, they assume

$$Y_i^N = \theta(U_i, T_i), \tag{2.4.7}$$

where U_i is a random variable that represent unobserved characteristics of individual i and T_i is the time period at which i has been observed. The assumption in equation 2.4.7 is that in the absence of the treatment, the production function is a function of unobservables and time. We also assume that $\theta(u, t)$ is strictly increasing (monotonicity) in u for $t \in \{0, 1\}$ [2]. Equation 2.4.7 implies that BMI does not vary for individuals with the same unobservable characteristics ($U_i = u$) in the same time period irrespective of their group. The distribution of U_i can vary from one group to another but is stable within a group over time. More formally we assume that:

$$U \perp\!\!\!\perp T|G$$

Denote $U_g \overset{d}{\sim} U|G$ and let us assume that U_g is continuous. To identify the treatment effect on the distribution, we need to assume that the treatment and control group's unobserved characteristics overlap over some common support. This implies that we want to identify the effect of the treatment on the treated. To do so, we need to assume that $\Upsilon_1 \subseteq \Upsilon_0$, where Υ_i is the support of the distribution of unobserved characteristics for group i. These assumptions are required to identify the distribution that would have been experienced by the treatment group in the absence of the treatment.

Athey and Imbens (2006) identification of the entire distribution of the counterfactual involves a double matching approach. From these CIC assumptions, if there is an individual

[2]The single index θ allows for the invertibility of the production function

in the first-period treatment group with outcome y, and there exists an individual with the same outcome y in the first-period control group, then these two individuals in the treatment and control groups should have similar unobserved characteristics u.

Let us define the cumulative distribution for the four groups as $F_{Y_{10}}$ be the treatment group in pre period, $F_{Y_{11}}$ be the treatment group in post period, $F_{Y_{00}}$ be the comparison group in pre period and $F_{Y_{01}}$ be the comparison group in post period. The CIC identifies the treatment effect at each point of the support of the distribution. Like the DID, it uses the observed outcome of the treatment group in the pre period and the observed change in the control group over time to estimate the counterfactual (the outcome for the treatment group in the absence of the treatment) cumulative distribution under the assumptions listed above. Due to the invertibility of the production function θ in u because of the strict monotonicity assumption, the second period outcome for the same individual in the control group with the same value of unobserved characteristics. If one applies the same idea to the first period outcome in the treatment group, the entire counterfactual distribution is identified. Formally,

$$F_{Y_{11}^N}(y) = F_{Y_{10}}(F_{Y_{00}}^{-1}(F_{Y_{01}}(y))) \tag{2.4.8}$$

where $F_{Y_{00}}^{-1}(p) = \inf\{y \in Y : F_{Y_{00}}(y) \geq p\}$

We can use the empirical cumulative distribution function as an estimate of the counterfactual distribution function $F_{Y_{11}^N}$.

With covariates

According to Kottelenberg and Lehrer (2017), the applicability of Athey and Imbens (2006) has been a few because of the nature of the proposed estimation with covariates. Hence, there is a need to identify the counterfactual distribution when we have covariates. With observable covariates (X), we make the following assumptions in this case:

The assumption we make for the CIC with covariates are:

- **A1.** Model: $Y_i^N = \theta(U_i, T_i, X_i) \qquad Y_i^I = \theta^I(U_i, T_i, X_i)$.

- **A2.** Strict monotonicity of $\theta(\cdot)$ and $\theta^I(\cdot)$ in u.

- **A3.** Time invariance conditional on group and covariates: $U \perp\!\!\!\perp T|G, X$.

- **A4.** $\mathscr{U}_1 \subseteq \mathscr{U}_0$.

Let us introduce some notation:

- $F_{Y^N,<gt,g't'>}$ is the distribution function of group g at period t if it had the distribution of covariates of group g' at period t' if non-treated.

- $F_{Y^I,<gt,g't'>}$ is the distribution function of group g at period t if it had the distribution of covariates of group g' at period t' if treated.

We are interested in estimating the counterfactual $F_{Y^N,<11,11>}$. Building on the result of Athey and Imbens (2006), one can show that

Theorem 2. *Under assumptions A1-A4, we would have*

$$F_{Y^N,<11,11>}(y) = F_{Y^N,<10,11>}\left(F^{-1}_{Y^N,<00,11>}\left(F_{Y^N,<01,11>}(y)\right)\right)$$

Proof. **Proof of Theorem 2:** Following Athey and Imbens (2006) closely, we can first write that

$$\begin{aligned} F_{Y^N,<gt,g't'>}(y) &= \Pr\left(\theta(u,t) \leq y | G = g, T = t, F_X = F_{X,g't'}\right) \\ &= \Pr\left(u_{<gt,g't'>} \leq \theta^{-1}(y,t)\right) \\ &= F_{U,<gt,g't'>}\left(\theta^{-1}(y,t)\right) \end{aligned} \tag{2.4.9}$$

Applying the result of equation (2.4.9), we have:

$$F_{Y^N,<11,11>}(y) = F_{U,<11,11>}\left(\theta^{-1}(y,1)\right) \tag{2.4.10}$$

Since $U \perp\!\!\!\perp T | G, X$, we know that

$$F_{U,<11,11>}\left(\theta^{-1}(y,1)\right) = F_{Y^N,<10,11>}\left(\theta(u,0)\right) \tag{2.4.11}$$

Introducing the result of equation (2.4.11) into equation (2.4.10) yields

$$F_{Y^N,<11,11>}(y) = F_{Y^N,<10,11>}\left(\theta\left(\theta^{-1}(y,1),0\right)\right) \tag{2.4.12}$$

We now need to find expressions for $\theta^{-1}(y, 1)$ and $\theta(u, 0)$. From equation (2.4.9), we know that

$$F_{Y^N,<01,11>}(y) = F_{U,<01,11>}\left(\theta^{-1}(y,1)\right) \tag{2.4.13}$$

This implies that

$$\theta^{-1}(y,1) = F^{-1}_{U,<01,11>}\left(F_{Y^N,<01,11>}(y)\right) \tag{2.4.14}$$

We also know that

$$F_{U,<01,11>}(u) = F_{Y^N,<00,11>}\left(\theta(u,0)\right) \tag{2.4.15}$$

This implies that

$$\theta(u,0) = F^{-1}_{Y^N,<00,11>}\left(F_{U,<01,11>}(u)\right) \tag{2.4.16}$$

Introducing equations (2.4.14) and (2.4.16) into equation (2.4.12) yields

$$F_{Y^N,<11,11>}(y) = F_{Y^N,<10,11>}\left(F^{-1}_{Y^N,<00,11>}\left(F_{U,<00,11>}\left(F^{-1}_{U,<01,11>}\left(F_{Y^N,<01,11>}(y)\right)\right)\right)\right) \tag{2.4.17}$$

Since $U \perp\!\!\!\perp T|G, X$, we know that

$$F_{U,<01,11>}\left(\theta(u,0)\right) = F_{U,<00,11>}\left(\theta(u,0)\right) \tag{2.4.18}$$

Introducing equation (2.4.18) into equation (2.4.17) yields

$$F_{Y^N,<11,11>}(y) = F_{Y^N,<10,11>}\left(F^{-1}_{Y^N,<00,11>}\left(F_{Y^N,<01,11>}(y)\right)\right) \tag{2.4.19}$$

□

The result of Theorem 2 indicates that the counterfactual $F_{Y^N,<11,11>}(y)$ can be estimated using the counterfactuals $F_{Y^N,<10,11>}(y)$, $F_{Y^N,<00,11>}(y)$, $F_{Y^N,<01,11>}(y)$. These last three counterfactuals can be estimated using the reweighting approach of DiNardo et al. (1996). Using the law of iterated expectations, these counterfactuals can be written as

$$\begin{aligned} F_{Y^N,<gt,11>}(y) &= \int_{\mathscr{X}} F_{Y^N|X,<gt,gt>}(s|X=x)F_{X,11}(x) \\ &= \int_{\mathscr{X}} F_{Y^N|X,<gt,gt>}(s|X=x)\Psi(x)F_{X,gt}(x) \end{aligned} \tag{2.4.20}$$

where $\mathscr{X}$ is the domain of X and

$$\Psi(x) = \frac{F_{X,11}(x)}{F_{X,gt}(x)} = \frac{\Pr\left[X=x|G=g,T=t\right]}{\Pr\left[X=x|G=1,T=1\right]} \tag{2.4.21}$$

Using Bayes' Law, DiNardo et al. (1996) show that

$$\Psi(x) = \frac{F_{X,11}(x)}{F_{X,gt}(x)} = \frac{\Pr\left[G=g \wedge T=t|X=x\right]}{\Pr\left[G=1 \wedge T=1|X=x\right]} \tag{2.4.22}$$

This last equation can be estimated by pulling the groups gt ($D=0$) and 11 ($D=1$) together and to estimate the unconditional probability of $D=1$ (proportion of observations in group 11) and a binary econometric model estimation of the conditional probability of $D=1$ on $X=x$[3].

$$\widehat{\Psi}(x) = \frac{\widehat{\Pr}[D=1|X=x]/\widehat{\Pr}[D=1]}{\widehat{\Pr}[D=0|X=x]/\widehat{\Pr}[D=0]}. \tag{2.4.23}$$

We use the predicted values $\widehat{\Psi}(x_i)$ and the survey weights, ω_i to reweight each observation

[3] These conditional probabilities are estimated with a logit model.

to produce a new weight for each observation $\widetilde{\omega}_i = \widehat{\Psi}(x_i) \times \omega_i$. The counterfactuals are then estimated with a simple Hájek

$$\widehat{F}_{Y^N,<gt,11>}(y) = \frac{1}{\sum_{i \in gt} \widetilde{\omega}_i} \sum_{i \in gt} \widetilde{\omega}_i 1(y_i \leq y), \tag{2.4.24}$$

and

$$\widehat{F}^{-1}_{Y^N,<gt,11>}(p) = inf \left\{ y_i, i \in \ gt | p \leq \widehat{F}_{Y^N,<gt,11>}(y_i) \right\} \tag{2.4.25}$$

2.4.5 Stochastic dominance

We are interested in statistically testing for the difference in the distribution of the counterfactual and observed distributions of BMI under the null that these distributions come from the same underlying density and/or dominance of one distribution over the other.

We are also interested in “overweightedness”, $o(bmi, z_b)$. This $o(.)$ function takes a 0 value if $bmi < z_b$ and values that are $>= 0$ if $bmi >= z_b$. Here, we also assume that $o(bmi)/bmi >= 0$. We do not know the exact form of this $o(.)$ function and unsure of the critical value z_b. However, the official values for being overweight is 25, that for being obese is 30, and that for being morbidly obese is 35. In our stochastic dominance test we use all values of z_b between z_b^{min} and ∞, where $z_b^{min} = 25$ or 30 or 35.

In addition, we also look at “underweightedness”, $a(bmi, z_a)$ where $a = 0$ if $bmi > y_a$ and $a >= 0$ if $bmi <= y_a$. We also assume that $a(bmi)/bmi <= 0$(inverted sign compared to $o(.)$). We then test for all y between 0 and y_a^{max} using 18.5 as y_a^{max}.

We use the directional Kolmogorov-Smirnov (KS) proposed by Barrett and Donald (2003) where we can test for dominance of distributions 1 over distribution 2. Assuming we have estimated two distributions, $\hat{F_1(y)}$ and $\hat{F_2(y)}$ from two independent samples of size n_1 and n_2, we test:

$H_0 : F_2(y) - F_1(y) \geq 0$ for all $y \in [0, y_b]$

$H_1 : F_2(y) - F_1(y) < 0$ for some $y \in [0, y_b]$

Under the null of dominance, Barrett and Donald (2003) suggest using a test statistic which is based on the supremum of individual differences. Formally:

$$\tau = \sup_{y \in [0, y_b]} \sqrt{\frac{n_1 n_2}{n_1 + n_2}} (\hat{F}_1(y) - \hat{F}_2(y)) \tag{2.4.26}$$

In practice, this test is a directional test where we test for null of dominance of 1 over 2 and 2 over 1. Specifically,

$H_0 : F_2(y) - F_1(y) \geq 0$ for all $y \in [0, y_b]$

and

$H_0 : F_1(y) - F_2(y) \geq 0$ for all $y \in [0, y_b]$

with their respective alternative hypothesis.

We can use the recentered version of the bootstrap proposed by Linton et al. (2005) to obtain p-values for the null hypothesis above. This is a two step process. The first step should be repeated for several times ($b = 1, \ldots, B$). Draw a sample of size n_1 from the first distribution with replacement. Compute the non-parametric estimator $\widehat{F_{1b}}$. Repeat the same process for the second distribution. That is, draw a sample of size n_2 from the second distribution with replacement. Compute the non-parametric estimator $\widehat{F_{2b}}$. After compute the test statistic:

The second step involves using the sample $\hat{\tau_1}, \ldots, \hat{\tau_B}$, you compute the bootstrap p-value as follows:

$$\frac{1}{B}\sum_{b=1}^{B} \not\Vdash(\hat{\tau_b} > \hat{\tau}) \tag{2.4.27}$$

where $\hat{\tau}$ is the test statistic from the initial sample.

Table 2.1 provides the decision rule for when we can reject the null hypothesis or do not reject the null hypothesis.

Table 2.1: First decision rule of dominance test

P value	Decision rule
≤ 0.05	reject the null of dominance
>0.05	do not reject the null of dominance

Table 2.2 also shows when two distributions intersect, are inferred to be the same, or when there is strong evidence of dominance of one distribution over the other. We follow these decision rules in inferring dominance for our results.

Table 2.2: Second decision rule of dominance test

Decision rule	1 dominates 2 (Test 1)	2 dominates 1 (Test 2)
Two curves intersect	reject the null of dominance	reject the null of dominance
Curves are the same	cannot reject the null of dominance of 1 over 2	can not reject the null of dominance of 2 over 1
Dominance of 1 over 2	cannot reject the null of dominance	reject the null of dominance
Dominance of 2 over 1	reject the null of dominance	cannot reject the null of dominance

2.5 Data

We use data from the public files of the Behavioural Risk Factor Surveillance System (BRFSS) from 2010-2011 and 2018-2019 to assess the effect of recreational marijuana policy on BMI. The BRFSS is an ongoing phone interview survey by the Centers for Disease Control (CDC) that interviews respondents on health outcomes, use of preventive services and healthy behaviour. It also recorded demographic characteristics for those 18 years and above since 1984. Over 300000 nationally representative sample of individuals is interviewed over the course of the year. In this study we focus on information about respondents' body weight and height along with their other health behaviours. The BRFSS data is desirable for evaluation of the RML because of its large sample size as well as its state identifiers which are easily accessible in the public data files. The sample weights provided by CDC are used for all the analyses.

2.5.1 Outcome variables

Respondents to the survey were asked to report their weight and height. The question on weight was "About how much do you weigh without shoes?". For height, respondents were asked “About how tall are you without shoes?”. The primary variable of interest of this study is BMI which was calculated by dividing respondents’ self reported weight in kilograms by their height in metres squared. [4]

As secondary variables our study analyze the effect of marijuana legalization on some health-related behaviours namely alcohol, cigarette, and physical activity. The survey asked respondents about their alcohol consumption behaviour. Specifically respondents were asked “ During the past 30 days, how many days per week or per month did you have at least one drink of any alcoholic beverage such as beer, wine, a malt beverage or liquor? ”, “One drink is equivalent to a 12-ounce beer, a 5-ounce glass of wine, or a drink with one shot of liquor. During the past 30 days, on the days when you drank, about how many drinks did you drink on the average?” and “During the past 30 days, what is the largest number of drinks you had on any occasion?”. Here, we use these three questions to create an indicator of alcohol consumption. We also use the response to the question “ Considering all types of alcoholic beverages, how many times during the past 30 days did you have 5 or more drinks for men or 4 or more drinks for women on an occasion?” to create an indicator for binge drinking if respondents had answered they had5 or more drinks at least once. In addition, respondents describe their smoking behaviour by answering the question “Have you smoked at least 100 cigarettes in your entire life?” and “Do you now smoke cigarettes every day, some

[4] Self reported weight and height are likely associated with measurement errors (that is, systematic under reporting). For instance a study by Bowring et al. (2012) shows that almost half of respondents were less likely to accurately report their height while only 34% were more likely to self report their weight accurately. The BMI has also been criticised as not being a “good” measure for blacks and other minority races. An objective measure of weight and height using proper scales or other measure such as body fat would have been preferable. Nevertheless, if the error in our variable of interest is not related to the enforcement of recreational marijuana legalization that is, then this measurement error (balanced between the treatment and control groups) should not generate biased estimates. In addition, Athey and Imbens (2006) also explain that, the unobservable component of BMI are not required to be the same but only the distributions remain the same.

days, or not at all? ". The answers to these questions was used to create an indicator for smoking behaviour. Finally participants answered a question about their physical activity. The question on physical activity that we use in the analysis is " During the past month, other than your regular job, did you participate in any physical activities or exercises such as running, calisthenics, golf, gardening, or walking for exercise?".

As control variables, we use information on gender, race, age , marital status, education and state unemployment rate[5].

By 2017, about 8 states, including the District of Columbia, had legalized marijuana. Table 2.3 shows treatment states' marijuana policies and the length of exposure to recreational marijuana and states used as controls. These control states had legalized medical marijuana before the first recreational marijuana year and did not legalize recreational marijuana from 2012 to 2019. Recreational marijuana legalization did not occur in all states at the same time. States adopted the policy at different points in time so there are differences in the length of exposure to RML, which produces a staggered design for these recreational marijuana laws. We group the data based on length of exposure to marijuana policy (treatment states), stack the dataset, and use the control group to identify the causal effect of RML.

Table 2.4 shows some demographic characteristics of the treatment and control groups. 48.21% of treatment states are females, while 48.60% of females are in the control states and the difference is statistically significant. of the treatment group, 79.53% are 18 to 54 years compared to 78.70% of the control groups in that age range. The control group is slightly more white than the treatment group (2.1% more). The treatment group is

[5] We use data on state unemployment rate from the US Bureau of Labor Statistics.

Table 2.3: States marijuana policies

State	Medical marijuana	Recreational marijuana
Panel 1: Treatment states		
Colorado	June 2001	December 2012
Washington	November 1998	December 2012
District of Columbia	July 2010	February 2015
Alaska	March 1999	February 2015
California	November 1996	November 2016
Oregon	December 1998	March 2016
Massachusetts	January 2013	December 2016
Maine	December 1999	January 2017
Nevada	October 2001	January 2017
Panel 2: Control states		
Arizona	April 2011	
Delaware	July 2011	
Hawaii	December 2000	
Montana	November 2004	
New Jersey	July 2010	
New Mexico	July 2007	
Rhode island	January 2006	

Dates source: Sabia et al. (2017). We used https://medicalmarijuana.procon.org/legal-medical-marijuana-states-and-dc/ for state marijuana laws after Sabia et al. (2017) study ended for medical marijuana policy. For recreational marijuana policy we used https://marijuana.procon.org/legal-recreational-marijuana-states-and-dc/#california

5.12% blacks compared to 12.53% in the control group. In contrast, the treatment group comprise of 41.36% of people other races compared to 31.32% in the control group. There is no statistically significant difference between married and never married individuals in the treatment and control states. The state unemployment rate in treatment states is statistically significantly higher than the unemployment rate in the control states.

Table 2.4: Summary statistics of characteristics in the treatment and control states

	Treatment states		Control states			
	mean	sd	mean	sd	Difference	p value
Female	0.4821	0.4997	0.4860	0.4998	0.0039	(0.2550)
18 to 24	0.1503	0.3573	0.1393	0.3462	-0.0110***	(0.0000)
25 to 34	0.2189	0.4135	0.2097	0.4071	-0.0092***	(0.0000)
35 to 44	0.2138	0.4100	0.2095	0.4070	-0.0043	(0.1190)
45 to 54	0.2123	0.4089	0.2285	0.4199	0.0162***	(0.0000)
55 to 65	0.2048	0.4035	0.2130	0.4094	0.0082***	(0.0000)
White	0.5352	0.4988	0.5615	0.4962	-0.0263***	(0.0000)
Black	0.0512	0.2204	0.1253	0.3311	0.0741***	(0.0000)
Other race	0.4136	0.4925	0.3132	0.4638	0.1004***	(0.0000)
Married	0.5832	0.4930	0.5803	0.4935	-0.0029	(0.3970)
Ever married	0.1334	0.3401	0.1393	0.3462	0.0059**	(0.0036)
Never married	0.2834	0.4506	0.2805	0.4492	-0.0015	(0.3860)
More than high school	0.6256	0.4840	0.6352	0.4814	-0.0096***	(0.0050)
State unemployment rate	7.4823	3.6992	6.6919	2.5145	-0.7904***	(0.0000)
N	201414		148846		350260	

Figure 2.1: Distribution of BMI by groups and time

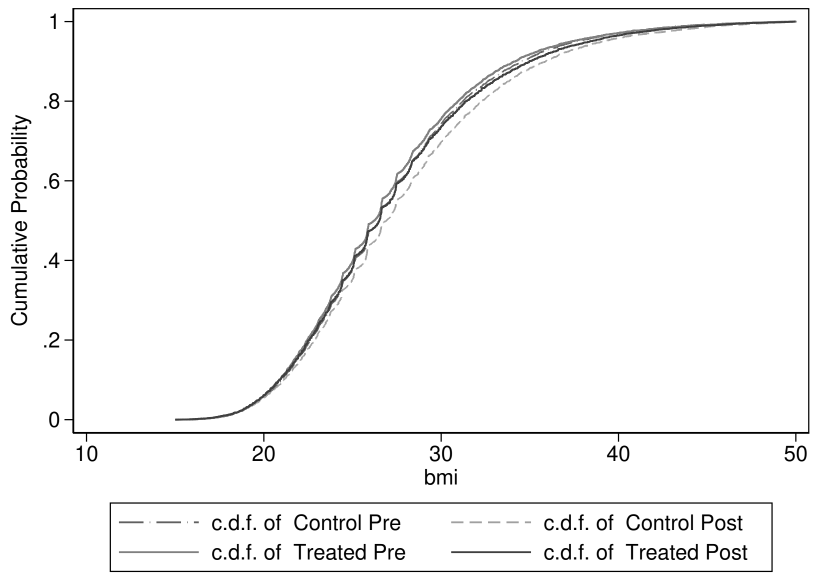
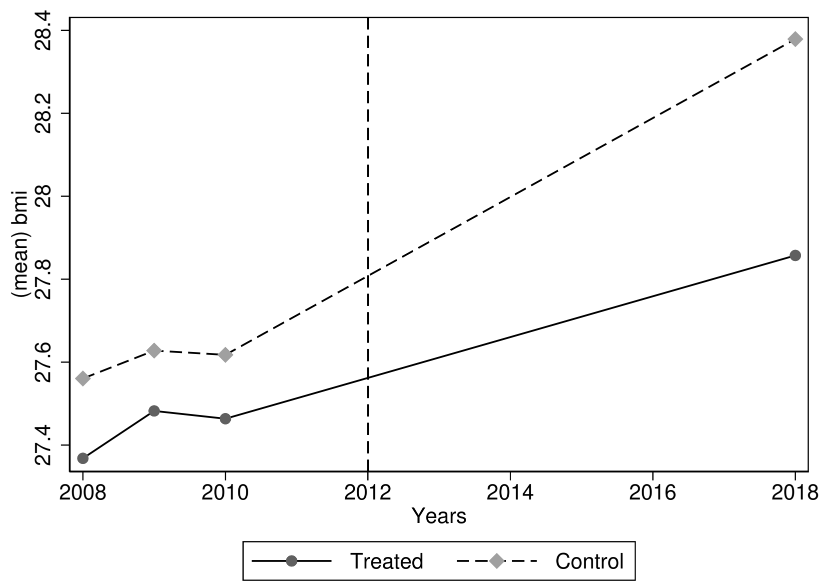


Figure 2.2: Parallel trends between the treatment and control states



in the treatment states with the difference being statistically significant. The percentage of people with bad smoking behaviour in treatment states is higher than in the control states (22% compared to 19%) which is also the case for the percentage of people not performing any form of exercise. The difference are statistically significant at the 1% level.

Table 2.5: Summary statistics of healthy behaviour

	Control states		Treatment states			
	mean	sd	mean	sd	Difference	p value
Binge drinking	0.2014	0.4010	0.1903	0.3926	-0.0111***	(0.0000)
Alcohol	0.2117	0.4085	0.2049	0.4036	-0.0068***	(0.0001)
Smoking	0.1504	0.3575	0.1777	0.3822	0.0247***	(0.0000)
No exercise	0.1903	0.3926	0.2216	0.4153	0.0313***	(0.0000)
N	200648		148292		348940	

2.6 Results

States were exposed to marijuana legalization at different times. Some exposed for longer periods and other for shorter periods but in the results that follow we will be using the entire sample as a pooled sample without accounting for the length of exposure to the treatment and using a clean control group who were only exposed to medical marijuana policy.

2.6.1 Results for changes in changes

We first present the changes in changes results of the effect of recreational marijuana legalization (RML) on BMI without covariates (Figure 2.3). Without accounting for covariates we find that the counterfactual distribution significantly differs (lies below) from the empirical distribution function (EDF). This shows that RML reduces BMI. Note that the evidence of the treatment effect of RML on BMI mainly comes from the middle and top parts of the distribution. To account for uncertainty around these results, we compute p values. Table 2.6 presents the p-values of the statistical test on the entire distribution as well as different parts of the distribution for no effect and dominance. As we explained earlier with these p values, for the entire distribution, we interpret that any function of BMI or obesity that is estimated from the empirical distribution will yield a lower value of obesity than any estimation from the counterfactual distribution. This means that there is obesity dominance (less obesity) after the legalization of recreational marijuana.

To investigate whether this dominance is driven by specific parts of the distribution, we test for dominance at different parts of the distribution. A breakdown of the entire distribution shows that between 15 and 18.5 (underweightedness), we do not reject the null of no effect test and the test of dominance. When we conduct the test at 25 to 50, 30 to 50 (obese) and 35 to 50 (morbidly obese), the p-values on Table 2.6 indicate the empirical distribution is everywhere above the counterfactual distribution in the absence of the legalization of recreational marijuana. This means that there is obesity dominance (less obesity) after legalization.

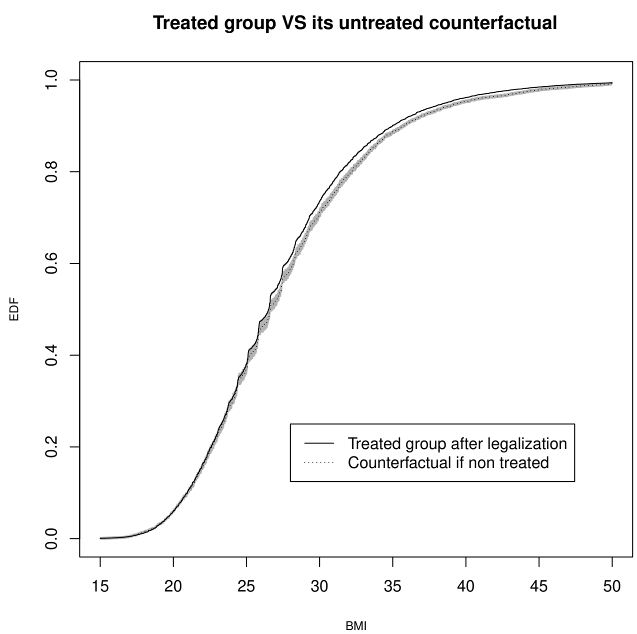


Figure 2.3: Without covariates

Given that these effects are driven by confounding factors, we control for confounding factors that we deem important. Figure 2.4 shows the results for changes in changes

Table 2.6: P values of the test of the effect of recreational legalization of marijuana on BMI using changes in changes without covariates

	Without covariates (1)	Decision (2)
Entire Distribution		
No effect test	0.0000	Reject the null
Dominance test EDF everywhere below Counterfactual	0.0000	Rreject the null
Dominance test Counterfactual everywhere below EDF	0.9798	Do not reject the null
Between 15 to 18.5		
No effect test	0.1717	Do not reject the null
Dominance test EDF everywhere below Counterfactual	0.8586	Do not reject the null
Dominance test Counterfactual everywhere below EDF	0.0000	Reject the null
Between 25 to 50		
No effect test	0.0000	Reject the null
Dominance test EDF everywhere below Counterfactual	0.0000	Reject the null
Dominance test Counterfactual everywhere below EDF	1	Do not reject the null
Between 30 to 50		
No effect test	0.0000	Reject the null
Dominance test EDF everywhere below Counterfactual	0.0000	Reject the null
Dominance test Counterfactual everywhere below EDF	1	Do not reject the null
Between 35 to 50		
No effect test	0.0000	Reject the null
Dominance test EDF everywhere below Counterfactual	0.0000	Reject the null
Dominance test Counterfactual everywhere below EDF	1	Do not reject the nulll
N	350260	

with covariates. As stated earlier, this approach involves using a reweighting to balance observable characteristics between the individuals in states that legalized marijuana and states that did not and identifying the conditional counterfactual distribution using the conditional distributions of the treatment and control groups. After accounting for potential confounding factors, we find that the counterfactual distribution is still statistically different from the empirical distribution function. The results of the statistical test are shown with the p-values in Table 2.7. For the entire distribution, the p-values indicate that the empirical distribution is everywhere above the counterfactual distribution in absence of legalization. This means that there is obesity dominance (less obesity) after legalization. In addition, the p values at different parts of the distribution leads as to interpret that the effect is not found at the lower tail of the distribution (15 to 18.5) but at the middle and upper parts of the distribution (overweightedness, obesity and morbidly obese). The magnitude of these effects are found in the Appendix. We find that the reduction in overweight, obesity, morbidly obese is around $1\% to 6\%$ when we account or do not account for both covariates.

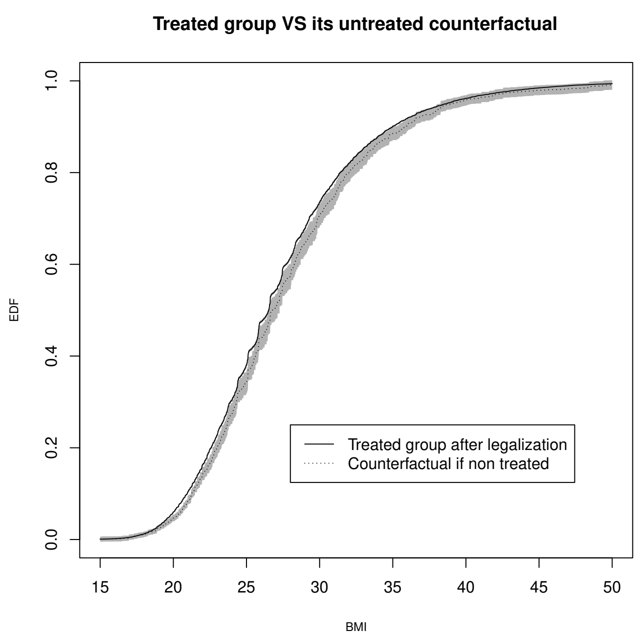


Figure 2.4: With covariates

Table 2.7: P values of the test of the effect of recreational legalization of marijuana on BMI using changes in changes with covariates

	With covariates (1)	Decision (2)
Entire Distribution		
No effect test	0.0404	Reject the null
Dominance test EDF everywhere below Counterfactual	0.0404	Reject the null
Dominance test Counterfactual everywhere below EDF	1	Do not reject the null
Between 15 to 18.5		
No effect test	0.6869	Do not reject the null
Dominance test EDF everywhere below Counterfactual	0.5152	Do not reject the null
Dominance test Counterfactual everywhere below EDF	0.8586	Do not reject the null
Between 25 to 50		
No effect test	0.0404	Reject the null
Dominance test EDF everywhere below Counterfactual	0.0404	Reject the null
Dominance test Counterfactual everywhere below EDF	0.9798	Do not reject the null
Between 30 to 50		
No effect test	0.0303	Reject the null
Dominance test EDF everywhere below Counterfactual	0.0303	Reject the null
Dominance test Counterfactual everywhere below EDF	0.9697	Do not reject the null
Between 35 to 50		
No effect test	0.0505	Reject the null
Dominance test EDF everywhere below Counterfactual	0.0505	Reject the null
Dominance test Counterfactual everywhere below EDF	0.9697	Do not reject the null
N	350260	

Covariates include gender, age, race, marital status, education and state unemployment rate

Heterogeneity effect

Estimating the effect for the treatment group as a whole may conceal important differences in the consequences of RML across subgroups. Consumption of marijuana also varies by different groups. According to MischleyLaurie et al. (2016) men are more likely to use marijuana often and in large quantities compared to women and men also reported an increase in appetite compared to women (most women reported a loss in appetite). In addition, Martins et al. (2021) found evidence of difference in marijuana use after RML. The authors show that there was an increase in past year and month use of marijuana along with changes in marijuana consumption behaviour among blacks. Hence, we estimate the effect of RML on BMI for men, women. whites, non-whites, those above 45 years and those below 45 years.

Combined with the p-values in Table 2.8 , the results of Figure 2.5a suggest that there is no effect of RML on the entire distribution and at the different categories of BMI distribution for men. However, for females, we find that RML reduces BMI. The test at the lower tail (15 to 18.5) and upper tails (25 to 50, 35 to 50) also support the results of the entire distribution for females (Figure 2.5b). These results suggest that RML leads to less obesity among women. These results are similar to finding by Beulaygue (2012) who found that the effect marijuana on BMI is higher in females than in males.

For whites and non-whites, Figure 2.6a and Figure 2.6b in addition to the p-values in Table 2.8 shows that for the entire distribution, there is a reduction in BMI as a result of recreational marijuana laws. At the lower tail (15 to 18.5), we find that RML does not affect underweightedness for both whites and non-whites. Nevertheless, RML reduces overweightedness for whites and non-whites. At the upper end of the distribution (30 to 50 and 35 to 50), RML does not have any effect on whites but reduces obesity among non-whites.

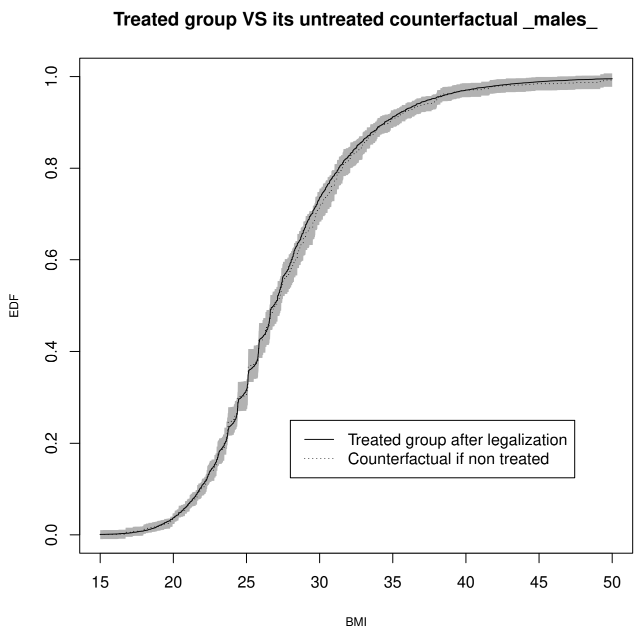
For those above 45 years and those below 45 years, we find evidence that recreational marijuana legalization affects the entire distribution of those above 45 years but not for those below 45 years old as shown on Figure 2.7a and Figure 2.7b in addition to the p-values in Table 2.8. Between 15 to 18.5, we do not find any effect of RML on underweightedness. For those above 45 years, RML reduces being overweight while there is no effect on those below 45 years. The reverse is true when we look at obesity (30 to 50). RML reduces obesity among those below 45 years alone. However, RML has no effect on being morbidly obese for both age groups.

These results show that there is variation in the effect of RML for different groups. We find evidence that RML affects the entire BMI distribution of women, whites and non-whites. Nevertheless, there are no effect for males and those below 45 years. At the lower tail (15 to 18.5 or being underweight), there is only an effect for women. Between 25 to 50 (overweight), there is a reduction for women, non-whites and those above 45 years as a result of recreational marijuana laws. In addition, RML reduces obesity for non-whites and those below 45 years and it reduces morbid obesity among women and non-whites.

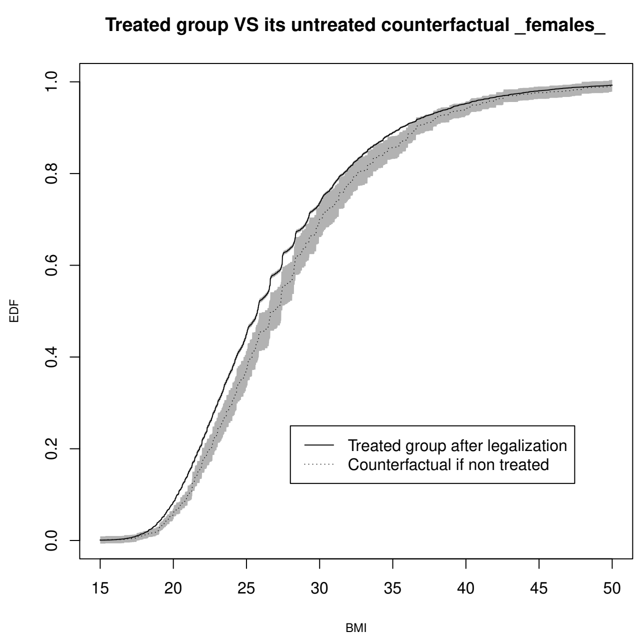
2.6.2 Results for difference in difference

While the CIC approach shows the effect of the legalization of marijuana on the entire distribution of BMI, earlier studies have used a DID approach to examine the effect of

Figure 2.5: With covariates for males and females

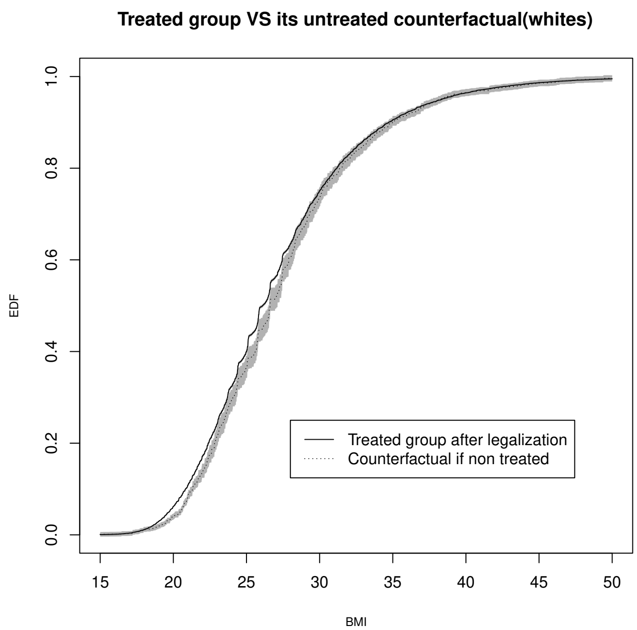


(a) Males

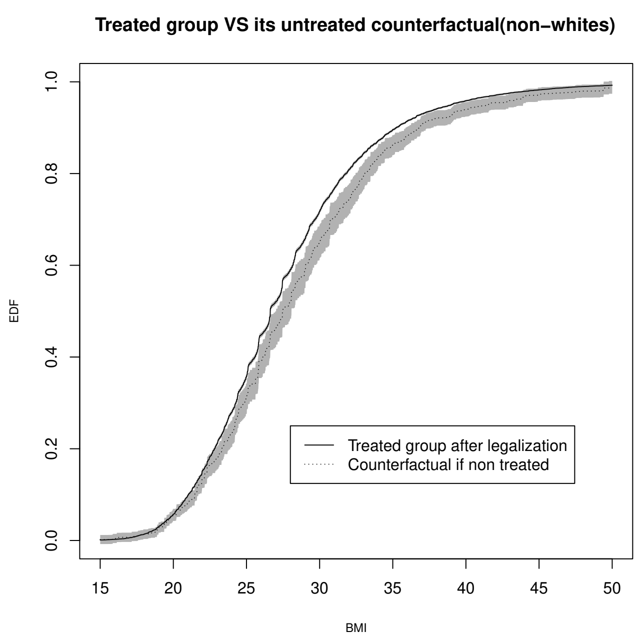


(b) Females

Figure 2.6: With covariates for whites and non-whites

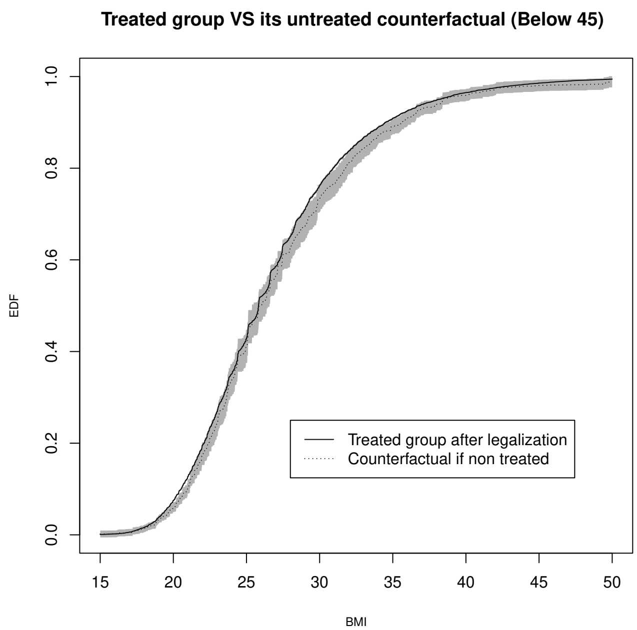


(a) Whites

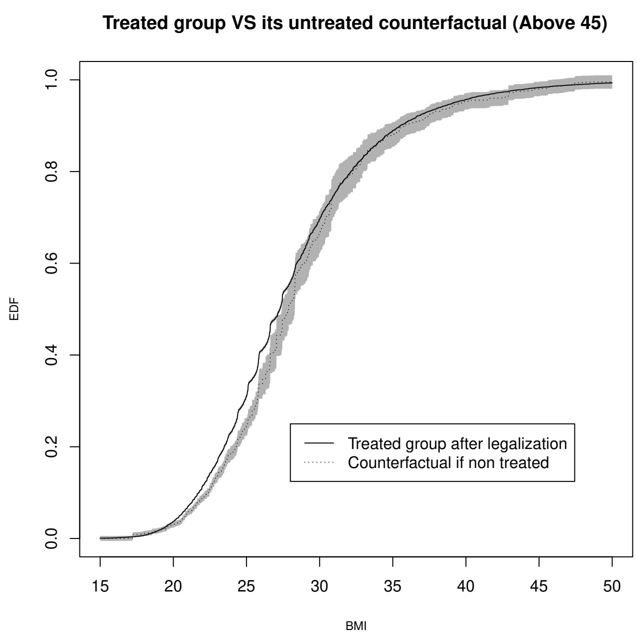


(b) Non whites

Figure 2.7: With covariates for those above and below 45 years



(a) Below 45 years



(b) Above 45 years

Table 2.8: P values of the test of the effect of recreational legalization of marijuana on BMI using changes in changes with covariates for some subgroups

	Male (1)	Female (2)	White (3)	Non-white (4)	Above 45 (5)	Below 45 (6)
Entire Distribution						
No effect test	0.5859	0.0000	0.0202	0.0000	0.0000	0.1414
Dominance test EDF everywhere below Counterfactual	0.4440	0.0000	0.0202	0.0000	0.0000	0.1414
Dominance test Counterfactual everywhere below EDF	0.7677	1	0.9596	0.9798	1	0.9899
Between 15 to 18.5						
No effect test	0.4849	0.0000	0.1212	0.6566	0.2222	0.3333
Dominance test EDF everywhere below Counterfactual	0.7272	0.0000	0.0606	0.5152	0.8889	0.2121
Dominance test Counterfactual everywhere below EDF	0.5353	0.7980	0.7879	0.4849	0.3333	0.7576
Between 25 to 50						
No effect test	0.5051	0.0000	0.0202	0.0000	0.0000	0.1414
Dominance test EDF everywhere below Counterfactual	0.3939	0.0000	0.0202	0.0000	0.0000	0.1414
Dominance test Counterfactual everywhere below EDF	0.6869	1	0.8990	0.9697	0.8889	0.9394
Between 30 to 50						
No effect test	0.2727	0.0606	0.3232	0.0000	0.3333	0.0202
Dominance test EDF everywhere below Counterfactual	0.2424	0.0606	0.3131	0.0000	0.3333	0.0202
Dominance test Counterfactual everywhere below EDF	0.8889	0.9798	0.8182	0.9596	0.7778	0.9596
Between 35 to 50						
No effect test	0.6364	0.0404	0.6970	0.0202	0.5556	0.0707
Dominance test EDF everywhere below Counterfactual	0.4343	0.0404	0.5354	0.0202	0.4444	0.0707
Dominance test Counterfactual everywhere below EDF	0.7071	0.9697	0.6465	0.9596	0.6667	0.9091
N	158,220	192,040	244,541	105,719	205,993	144,267

Covariates include gender, age, race, marital status, education and state unemployment rate

marijuana legalization on average. For these reasons, we complement our CIC analysis with an estimates of the treatment effect using a DID.

Table 2.9 presents the difference in difference results of the effect of recreational marijuana on BMI. Without controlling for demographic characteristics, we find that recreational marijuana legalization reduces BMI by about 0.3374 units on average (Column 1). After adding controls for demographic characteristics and state unemployment rate, the magnitude of the effect of RML on BMI reduces slightly to 0.3179 units (Column 2) but remains quantitatively similar and in the same direction as the CIC results.

Table 2.9: The effect of recreational legalization of marijuana on BMI-Difference in Difference

	BMI (1)	BMI (2)
Group	-0.2379***	-0.1078*
	(0.0569)	(0.0577)
Time	0.6675***	0.6300***
	(0.0717)	(0.0874)
DID	-0.3374***	-0.3179***
	(0.0880)	(0.0873)
Female		-0.6252***
		(0.0409)
Age		0.0652***
		(0.0017)
Black		1.8799***
		(0.0929)
Other race		0.3522***
		(0.0466)
Never married		0.2288***
		(0.0563)
Ever married		-0.3368***
		(0.0580)
More than high school		-1.1833***
		(0.0455)
State unemployment rate		0.0017
		(0.0121)
Constant	27.3855***	25.4506***
	(0.0460)	(0.1394)
N	350,260	350,260

Significance levels are indicated by *, **, *** for 10%, 5%, and 1% respectively

Table 2.10 presents the difference in difference results for different demographic groups. Column 1 shows that for males, RML does not have any effect on BMI on average. However, Column 2 shows that for females RML significantly reduced BMI by 0.4603 units when we control for covariates. Column 3 shows that for whites, RML does not significantly change BMI while in Column 4, RML reduces BMI for non-whites by 0.4796 units. RML does not affect BMI after we control for demographic characteristics and state unemployment rate for people above 45 years. For those below 45 years, RML reduces BMI (Column 6).

In addition to BMI, we also analyzed the effect of RML on drinking alcohol, smoking and physical activity. While uncovering these effects using a distributional approach, would have been interesting, our data does not permit such estimation. However, using a linear DID, Table 2.11 shows that without controlling for covariates, RML has no effect on binge drinking. However, the effect of RML on alcohol consumption is statistically significant after we control for covariates (1.80 percentage points). We also find that RML has no effect on smoking but reduces the probability of exercising (1.47 percentage points). The effect of RML on exercising is statistically significant when we control for covariates or without covariates.

Given that our outcome of interest is a dummy variable, a linear DID approach may lead to predictions of the probability of the success of the outcome outside the $[0, 1]$ range. As stated earlier, a solution to this issue is to use a non-linear difference in difference estimation method.

Using a non-linear DID approach, Table 2.12 shows that RML has no effect on binge drinking, increases the probability of alcohol consumption by 1.20 percentage points without controlling for covariate, with an increase in the magnitude to 1.68 percentage points when we account for covariates. RML does not statistically have any effect on smoking behaviour, however, we find that RML reduces the probability of engaging in any physical activity by 1.29 percentage points. These non-linear difference in difference estimates are not the raw estimates coefficients from the probit model but the estimated average marginal effect from the estimated model. The results from the LPM and non-linear difference in difference are very close although we do not statistically test for differences in the results [6].

[6] Non-linear DID allows for variation in the predicted probability while the estimated effect is fixed in a difference in difference model

Table 2.10: Difference in difference estimates for the effect of RML on BMI for subgroups

	Male	Female	White	Non-white	Above 45	Below 45
	(1)	(2)	(3)	(4)	(5)	(6)
Group	-0.2497***	0.0578	-0.0394	-0.4661***	-0.1986**	-0.0520
	(0.0851)	(0.0766)	(0.0596)	(0.1255)	(0.0873)	(0.0773)
Time	0.2953**	1.0048***	0.3289***	0.7928***	0.7224***	0.6164***
	(0.1232)	(0.1232)	(0.0999)	(0.1545)	(0.1157)	(0.1256)
DID	-0.1866	-0.4603***	-0.1514	-0.4796***	-0.1066	-0.4610***
	(0.1172)	(0.1291)	(0.0985)	(0.1634)	(0.1258)	(0.1209)
Demographic characteristics	Y	Y	Y	Y	Y	Y
State unemployment rate	Y	Y	Y	Y	Y	Y
N	158,220	192,040	244,541	105,719	205,993	144,267

Table 2.10 reports the linear difference in difference results healthy behaviours by subgroups. Significance levels are indicated by *, **, *** for 10%, 5%, and 1% respectively.

Table 2.11: Healthy behaviour outcomes-Difference in Difference

	Binge drinking	Binge drinking	Alcohol	Alcohol	Smoking	Smoking	No exercise	No exercise
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Group	0.0075**	0.0117***	0.0037	-0.0070	-0.0285***	-0.0112***	-0.0408***	-0.0336***
	(0.0038)	(0.0038)	(0.0045)	(0.0045)	(0.0036)	(0.0037)	(0.0037)	(0.0038)
Time	-0.0060	-0.0206***	-0.0612***	-0.0412***	-0.0276***	-0.0659***	-0.0081*	-0.0386***
	(0.0044)	(0.0059)	(0.0048)	(0.0066)	(0.0046)	(0.0058)	(0.0048)	(0.0059)
DID	0.0078	0.0001	0.0114*	0.0180***	0.0078	-0.0049	0.0201***	0.0147**
	(0.0056)	(0.0057)	(0.0061)	(0.0061)	(0.0055)	(0.0056)	(0.0059)	(0.0059)
Demographic characteristics	N	Y	N	Y	N	Y	N	Y
State unemployment rate	N	Y	N	Y	N	Y	N	Y
N	334,108	334,108	296,443	296,443	345,858	345,858	343,241	343,241

Significance levels are indicated by *, **, *** for 10%, 5%, and 1% respectively.

Table 2.12: Healthy behaviour outcomes-Non linear Difference in difference

	Binge drinking	Binge drinking	Alcohol	Alcohol	Smoking	Smoking	No exercise	No exercise
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Average treatment effect	0.0079	0.0002	0.0120***	0.0168***	0.0058	-0.0077	0.0191***	0.0129**
	(0.0000)	(0.0000)	(0.0000)	(0.00002)	(0.0000)	(0.00003)	(0.0000)	(0.00001)
Demographic characteristics	N	Y	N	Y	N	Y	N	Y
State unemployment rate	N	Y	N	Y	N	Y	N	Y
N	334,108	334,108	296,443	296,443	345,858	345,858	343,241	343,241

Significance levels are indicated by *, **, *** for 10%, 5%, and 1% respectively.

Since individual predicted probabilities are allowed to vary in the non-linear difference in difference model, we are able to estimate the effect of marijuana legalization on different groups following the same model. Table 2.13 shows the effect of RML on healthy behaviour for different groups. RML increases alcohol consumption by 1.99 percentage points for males, 1.37 percentage points for females, 1.73 percentage points for whites, 1.56 percentage points for non-whites, 1.35 percentage points for those above 45 years and 2.05 percentage points for those below 45 years.

RML increases physical activity by 1.25 percentage points for males, 1.33 percentage points for females, 1.20 percentage points for whites, 1.50 percentage points among non-whites and 1.38 and 1.19 percentage points respectively among those above 45 years and those below 45 years.

We also analyze the effect of RML on different subgroups using the linear difference in difference approach. Table 2.14, panel 1 presents the results of the effect of RML on binge drinking for different subgroups. We find that RML does not affect binge drinking in any of these groups except for whites among which binge drinking increases by 1.21 percentage points (10% significance level). Nevertheless, the results for alcohol consumption among these subgroups are different (panel 2). For alcohol consumption in general, we find that for males, RML increases the probability of consuming alcohol by 2.79 percentage points when we control for covariates while RML has no effect on the probability of consuming alcohol for females. For whites, the probability of alcohol consumption increases by 3.01 percentage points while for non-whites, RML has no effect on alcohol consumption. In addition, RML increases alcohol consumption by 2.41 percentage points for those above 45 years while marijuana legalization does not affect alcohol intake for those below 45 years.

For smoking for different subgroups, Table 2.14 panel 3, shows that RML does not statistically significantly affect the probability of smoking for males, females, whites and those above 45 years. It increases the probability of smoking by 3.30 percentage points for non-whites and by 1.73 percentage points for those below 45 years.

Finally, Table 2.14 panel 4, presents the results for the lack of exercise for different subgroups. We find that for males, whites and those below 45 years, recreational marijuana reduces the probability of exercising by 1.73, 3.14 and 2.22 percentage points respectively when we account for demographic characteristics and state unemployment rates. However, RML does not affect the probability of exercising for females, whites or those above 45 years.

Table 2.13: Non linear difference in difference estimates for the effect of RML on healthy behaviours by subgroups within the same model

	Male (1)	Female (2)	White (3)	Non-white (4)	Above 45 (5)	Below 45 (6)
Panel 1: Binge drinking						
Average treatment effect	0.0002	0.0001	0.0002	0.0002	0.0001	0.0002
	(0.0000)	(0.0000)	(0.0000)	(0.0000)	(0.0000)	(0.0000)
Panel 2: Alcohol						
Average treatment effect	0.0199***	0.0137***	0.0173***	0.0156***	0.0135***	0.0205***
	(0.00002)	(0.00002)	(0.00002)	(0.00003)	(0.00002)	(0.00002)
Panel 3: Smoking						
Average treatment effect	-0.0055	-0.0044	-0.0051	-0.0046	-0.0049	-0.0050
	(0.00001)	(0.00001)	(0.00001)	(0.00001)	(0.00001)	(0.00001)
Panel 4: No exercise						
Average treatment effect	0.0125***	0.0133***	0.0120***	0.0150***	0.0119***	0.0138***
	(0.00002)	(0.00002)	(0.00001)	(0.00002)	(0.00001)	(0.00002)
Demographic characteristics	Y	Y	Y	Y	Y	Y
State unemployment rate	Y	Y	Y	Y	Y	Y
N	149,200	182,184	230,055	101,329	195,407	135,977

Table 2.14 reports the non-linear difference in difference results of healthy behaviours by subgroups. Significance levels are indicated by *, **, *** for 10%, 5%, and 1% respectively.

Table 2.14: Linear probability model estimates for the effect of RML on healthy behaviours by subgroups

	Male (1)	Female (2)	White (3)	Non-white (4)	Above 45 (5)	Below 45 (6)
Panel 1: Binge drinking						
Group	0.0087	0.0144***	0.0082*	0.0165**	-0.0017	0.0217***
	(0.0062)	(0.0043)	(0.0045)	(0.0073)	(0.0043)	(0.0058)
Time	-0.0208**	-0.0198***	-0.0319***	-0.0053	0.0002	-0.0351***
	(0.0093)	(0.0071)	(0.0076)	(0.0095)	(0.0064)	(0.0089)
DID	0.0055	-0.0047	0.0121*	-0.0120	0.0051	-0.0030
	(0.0088)	(0.0070)	(0.0072)	(0.0094)	(0.0066)	(0.0085)
Panel 2: Alcohol						
Group	-0.0162**	0.0028	-0.0071	-0.0114	-0.0211***	0.0033
	(0.0071)	(0.0053)	(0.0052)	(0.0089)	(0.0052)	(0.0068)
Time	-0.0383***	-0.0459***	-0.0579***	-0.0126	-0.0188***	-0.0591***
	(0.0101)	(0.0081)	(0.0082)	(0.0111)	(0.0071)	(0.0100)
DID	0.0279***	0.0082	0.0301***	0.0125	0.0241***	0.0148
	(0.0094)	(0.0075)	(0.0076)	(0.0106)	(0.0071)	(0.0091)
Panel 3: Smoking						
Group	-0.0060	-0.0150***	-0.0140***	0.0014	-0.0193***	-0.0051
	(0.0060)	(0.0047)	(0.0045)	(0.0073)	(0.0046)	(0.0057)
Time	-0.0712***	-0.0685***	-0.0591***	-0.0922***	-0.0465***	-0.0857***
	(0.0091)	(0.0075)	(0.0076)	(0.0096)	(0.0071)	(0.0088)
DID	-0.0048	-0.0104	0.0086	-0.0330***	0.0074	-0.0173**
	(0.0086)	(0.0074)	(0.0074)	(0.0094)	(0.0072)	(0.0083)
Panel 4: No exercise						
Group	-0.0257***	-0.0418***	-0.0342***	-0.0343***	-0.0356***	-0.0320***
	(0.0057)	(0.0050)	(0.0040)	(0.0081)	(0.0050)	(0.0055)
Time	-0.0501***	-0.0262***	-0.0336***	-0.0490***	-0.0261***	-0.0458***
	(0.0084)	(0.0083)	(0.0069)	(0.0104)	(0.0082)	(0.0082)
DID	0.0173**	0.0121	-0.0007	0.0314***	0.0042	0.0222***
	(0.0082)	(0.0083)	(0.0069)	(0.0106)	(0.0084)	(0.0080)
Demographic characteristics	Y	Y	Y	Y	Y	Y
State unemployment rate	Y	Y	Y	Y	Y	Y
N	150,226	183,882	234,688	99,420	197,200	136,908

Table 2.14 reports the linear difference in difference results healthy behaviours by subgroups. Significance levels are indicated by *, **, *** for 10%, 5%, and 1% respectively. Covariates include gender, age, race, marital status, education and state unemployment rate

Table 2.15: Non linear difference in difference estimates for the effect of RML on healthy behaviours by subgroups

	Male (1)	Female (2)	White (3)	Non-white (4)	Above 45 (5)	Below 45 (6)
Panel 1: Binge drinking						
Average treatment effect	0.0099	-0.0085	0.0111	-0.0128	0.0054	-0.0032
	(0.00001)	(0.00010)	(0.00001)	(0.00002)	(0.00001)	(0.00000)
Panel 2: Alcohol						
Average treatment effect	0.0053***	-0.0049	0.0254***	0.0099	0.0208***	0.0145
	(0.0000)	(0.0000)	(0.0000)	(0.0000)	(0.0000)	(0.0000)
Panel 3: Smoking						
Average treatment effect	0.1265	0.1270	0.0110	-0.0439***	0.0096	-0.0233***
	(0.0004)	(0.0003)	(0.0000)	(0.0000)	(0.0000)	(0.0000)
Panel 4: No exercise						
Average treatment effect	0.1433**	0.1198	-0.0003	0.0350***	0.0057	0.0254***
	(0.0004)	(0.0003)	(0.0000)	(0.0000)	(0.0000)	(0.0000)
Demographic characteristics	Y	Y	Y	Y	Y	Y
State unemployment rate	Y	Y	Y	Y	Y	Y
N	150,226	183,882	234,688	99,420	197,200	136,908

Table 2.14 reports the linear difference in difference results healthy behaviours by subgroups. Significance levels are indicated by *, **, *** for 10%, 5%, and 1% respectively.

Table 2.15 presents the results of the non-linear difference in difference for some healthy behaviours for different subgroups. Panel 1 shows that RML has no effect on binge drinking in all the different groups. Nevertheless, panel 2 shows that RML increases the probability alcohol consumption among males by 0.53 percentage points but does not have any effect on alcohol consumption for females. In addition, we find that RML increases the probability of alcohol consumption by 2.54 percentage points and 2.08 percentage points for whites and people above 45 years respectively while RML does not affect alcohol consumption among non-whites and those below 45 years.

Panel 3 shows that RML does not affect smoking behaviour of males, females, whites or those above 45 years but reduces the probability of smoking for non-whites by 4.39 percentage points and 2.33 percentage points for those below 45 years.

Panel 4 shows that RML reduces the probability of participating in any physical activity for males by 14.33 percentage points, about 3.50 percentage points for non-whites and 2.54 percentage points for those below 45 years. The effect of RML on the probability of exercising is not statistically significant for females, whites and those above 45 years.

Our estimates of the effect of recreational marijuana legalization laws on some healthy behaviours does not necessarily explain the effect we find on BMI. There is no effect of RML on binge drinking and we find an increase in alcohol consumption but a reduction in BMI. Clark et al. (2018) explains that an increase in calorie intake despite a reduction in BMI can be associated with change in metabolism due to marijuana consumption. We also find that RML reduces physical activity which is similar to findings of Vidot et al. (2017). We speculate that an increase in sedentary behaviour can also potentially lead to reduction in muscle mass, and hence, lower BMI over the entire distribution.

2.7 Conclusion

The legalization of marijuana across different states has been increasing over recent years. Yet only one study has investigated the effect of marijuana legalization on weight or BMI. This is one of a few econometric studies that analyze the causal effect of recreational marijuana legalization on the distribution of BMI.

In this study, we employed a changes in changes approach that allows us to identify the entire counterfactual distribution and used a stochastic dominance test to test the dominance between the observed and counterfactual distributions. In addition, following earlier studies of marijuana policy, we use a difference in difference approach to estimate the effect of RML on average. We discuss the issues with the DID approach when the outcome of interest is binary and use a non-linear difference in difference approach to analyze the effect of RML on health behaviours.

Using the Behavioral Risk Factor Surveillance System (BRFSS), this study uncovers that recreational marijuana legalization reduces body mass index. Specifically, using a distributional analysis approach, we found evidence that marijuana legalization reduces BMI, and that these effects are strongest among women and those below 45 years. Both linear and non-difference in difference approaches show that recreational marijuana policy also negatively affects BMI on average. In addition, RML increases alcohol consumption, has no implications related to smoking tobacco, and reduces physical activity.

Chapter 3

Robust ordering of socioeconomic inequalities in well-being and mental health: A median based method for categorical variables*

3.1 Introduction

Inequalities in well-being among groups of various socioeconomic status constitute one of the main challenges for public health, but it is unknown to what extent such inequalities are modifiable. In light of this fact, policy makers seek to address these issues in society by first

*This chapter is based on joint research work with Paul Makdissi and Myra Yazbeck. We used data from the research data centre and received funding from the Canadian Research Data Centre Network (CRDCN) emerging scholar.

assessing the level of socioeconomic inequalities in well-being. Nevertheless, the debate on the right measure of well-being has been ongoing for years.

Earlier studies used the gross domestic product per capita as a measure of well-being. This measure of well-being has been widely criticized because it does not capture the multidimensional aspects of well-being such as health, happiness and life satisfaction (Dutta and Foster (2013)). Consequently, new measures of well-being that are currently commonly used are life satisfaction and happiness (Dutta and Foster (2013); Stevenson and Wolfers (2008); Veenhoven (2007)).[1] It has been argued that these measures capture quality of life despite their subjective nature (Kahneman and Krueger (2006)). Therefore, there has been growing interest in measuring inequalities using subjective well-being. According to Goff et al. (2018), "If life evaluations provide an umbrella measure of the quality of life, it would seem theoretically obvious that well-being inequality would provide a broader measure of inequality than could be derived from the separate measures of inequality in income and wealth, health, education, and friendship".

Subjective well-being like happiness and life satisfaction are often ordinal in nature, similar to self-reported status. Therefore, problems encountered in measuring inequality in these well-being variables are similar to those in measuring inequality in health.[2] Most socioeconomic inequality indices are developed for ratio scale variables. As a result, it is common to impose an arbitrary scale on these ordinal variables to enable them to use the popular measures of socioeconomic inequalities in well-being. In addition, as noted by Allison

[1] According to Kahneman and Krueger (2006) more than 100 papers used happiness and life satisfaction as a measure of well-being from 2001 to 2005 compared to only 4 papers from 1991 to 1995.

[2] Here we are assuming there is a self assessed general health status that is categorical. More on the issues encountered in measuring socioeconomic inequalities in well-being are discussed in the methodological literature review.

and Foster (2004), these popular measures of socioeconomic inequalities in health, which can be extended to other well-being measures like the concentration indices, use the mean as the reference point. The non-robustness of the mean as the reference point is an issue that needs to be addressed as different numerical scales lead to different rankings. Allison and Foster (2004) propose a solution using an alternate reference point, the median. However, their approach applies to only pure or unidimensional inequality in well-being.

Therefore, the current literature has not provided an index for ordinal variables that has a robust reference point to measure socioeconomic inequality in well-being. This paper aims to fill this gap in the literature by proposing an index with a robust reference point and in addition, provide conditions to rank any two distributions. Using insights from Allison and Foster (2004), this paper proposes a median-based index and exploits the idea of total social weights developed by Makdissi and Yazbeck (2017) to provide robust ranking of any two distributions of socioeconomic inequalities in well-being. We show that any two socioeconomic distributions in well-being with equal medians can be ranked using their total social weights function without imposing any numerical scale. A researcher can impose a non-decreasing concave or convex scale to achieve additional rankings of any two distributions if the researcher has reason to believe that the difference between categories of well-being decreases or increases for higher categories, respectively.

Our paper contributes to the literature by proposing an index for measuring socioeconomic inequality in well-being that overcomes the problems associated with the ordinal nature of common well-being variables. In addition, we also provide conditions for unambiguously ranking socioeconomic inequality in any two well-being distributions.

Drawing on the theoretical approach developed in this paper, we use data from the Canadian Community Health Survey (CCHS) in 2015 to rank socioeconomic inequalities in well-being among different demographic groups and regions in Canada. The restricted CCHS data contains information on point estimates of household income, which we use to infer equivalent income. The CCHS also offers information on happiness, life satisfaction, depression, mental health, and general health status, which are important for this study.

We find evidence that there are higher socioeconomic inequalities in well-being and mental health among unemployed females and males than among employed females and males. There is also evidence that subgroups with lower socioeconomic inequality in happiness tend to have higher achievement in happiness. We also provide rankings of socioeconomic inequalities in well-being and mental health in Canada's three classified regions.

The remainder of the paper is organized as follows: Section 1.2 provides an overview of both empirical and theoretical papers closely linked to the question addressed in this paper. Section 1.3 presents the theoretical framework and theorems that allow for robust orderings. Section 1.4 discusses the data. Section 1.5 shows the results of our empirical findings, and Section 1.6 concludes.

3.2 Related Literature

Our study is linked to three strands of literature: first, the literature on the relationship between inequality and well-being; second, the literature on the unidimensional or bidimensional measurement of well-being and, finally, the literature on the measurement of socioeconomic inequality.

A large body of literature analyzes the effect of income on well-being at both the macro and micro levels. The macro and micro approaches used in analyzing the relation between income and well-being yield contradictory findings. At the macro level, most studies conclude that there is no relationship between happiness and income. For instance, according to Clark et al. (2008), in wealthy countries, once basic needs have been satisfied, these countries have been described as being on the "'flat of the curve,' with additional income buying little if any extra happiness". This is the Easterlin paradox, which is the fact that happiness is stationary, although income continues to rise. However, the micro level or within-country analysis of the relationship between happiness and income, show a clear consensus, aptly summarized by Easterlin: " As far as I am aware, in every representative national survey ever done a significant bivariate relationship between happiness and income has been found" (2005, p. 67). While our study does not directly contribute to the Easterlin paradox literature, we look at the link between positions in the income distribution and inequality in subjective well-being.

This paper is linked to the literature that seeks to understand the relationship between income inequality and subjective well-being as well as mental health. Empirical studies in the literature on inequality and well-being focus mainly on the effect of income inequality on well-being using regression analysis. For instance, Morawetz et al. (1977) compares happiness in two communities with different inequality and finds that inequality leads to lower happiness levels. Alesina et al. (2004) also conclude a negative relationship between income inequality and happiness in Europe and the United States. On the contrary, Tomes (1986) uses Canadian data and finds a positive relationship between income inequality and well-being; that is, well-being decreases when income is more concentrated among the bottom 40%.

While the empirical evidence on the relationship between socioeconomic inequality and happiness is mixed, most studies conclude that an individual's socioeconomic status affects their life satisfaction (e.g., Chzhen et al. (2016); Ravens-Sieberer et al. (2013) ; Shek and Lee (2007); Zou et al. (2018)).

There is also a consensus in the empirical literature on the relationship between socioeconomic inequality and mental health. In a recent systematic review, Reiss (2013) shows that, children and adolescents from disadvantaged homes are more likely to have mental health problems. In an earlier scan of the literature on socioeconomic inequalities in mental health, Fryers et al. (2003) concluded that some studies found an inverse relationship between socioeconomic status and mental health while other studies could not establish this positive relationship. However, the authors did not find any studies that found a positive relationship between socioeconomic inequality and mental health.

The literature on socioeconomic inequality and well-being that uses regression methods has some limitations. First, most studies dichotomize the well-being variables (there are a few exceptions, such as Oshio and Kobayashi (2010)). According to Ziebarth (2010), dichotomizing the well-being variable leads to loss of information and reduces the statistical power to detect the relation between socioeconomic inequality and well-being. Second, regression analysis inherently imposes a scale, whereas the scale and the average of a scale are meaningless. Lastly, it focuses on a particular point of the distribution, the marginal effect on the average. While the marginal effect on the average is important, other points of the distributions are also relevant to policymakers. For instance, the relationship between well-being and income may be different at the lower tail of the distribution than at the upper tail, and the marginal effect on the average will not capture these effects. In addition, the average is not robust.

This paper is also closely related to the literature of unidimensional or bidimensional well-being. As stated earlier, measurement tools for inequality are developed for ratio-scale variables like income. However, these tools have been applied to ordinal variables like well-being by imposing an arbitrary scale. Allison and Foster (2004) point to the problem of the non-robustness of the mean as a reference point in the presence of an ordinal variable because of the mean's sensitivity to a chosen numerical scale. The authors suggest using the median as the reference point in ordering inequality in health. This can be extended to happiness and life satisfaction since they have the same ordinal nature than self-assessed health status variables. Naga and Yalcin (2008) introduced a parametric inequality measure based on the median as a reference point and label it *"aversion to median preserving spread"*. Dutta and Foster(2013) use the index proposed by Allison and Foster (2004) and Naga and Yalcin (2008) and data from the General Social Survey (GSS) to measure inequality in happiness across time in the US. They find that happiness inequality decreased from its highest level in the 1970s through the 1980s and 1990s; however, happiness inequality began to rise again in the 2000s.

Jenkins (2019) also provides a concise summary of the literature on unidimensional methods for comparing inequality in well-being. The author also proposes a method to rank inequality between two distributions. Using the World Value Survey(WVS), the author compares the distribution of life satisfaction in New Zealand, Australia, the UK, the US, and South Africa. He finds that South Africa has the highest life satisfaction inequality compared to the other countries.

While some studies in the literature on the unidimensional inequality in well-being have used methods that address the issue with the categorical nature of the well-being variable, these studies do not consider the socioeconomic dimension of inequality in well-being.

The only study that measures socioeconomic inequality in well-being is a recent study by Chen and Zheng (2014). They use a robust ranking approach proposed by Zheng (2011) to empirically measure socioeconomic inequalities in the US from 1994 to 2014 using General Social Survey. Zheng (2011) approach does not require a researcher to impose a numerical scale but rather categorize individuals in different socioeconomic classes and then impose monotonicity of health in socioeconomic ranks. While Zheng (2011) approach addresses the issue of selecting an arbitrary scale, it does not allow for heterogeneity among groups. Chen and Zheng (2014) find that, while they cannot establish a specific trend for socioeconomic inequality in happiness in the US, 1996 showed the lowest level of socioeconomic inequality in happiness with the highest well-being occurring in 2008. This paper is differs from Zheng (2011) because we employ a median-based robust ordering approach to measure socioeconomic inequality in well-being and mental health.

The current study is also related to the literature on measuring socioeconomic inequality when the outcome of interest is ordinal. In this literature, the methodological approach used in measuring socioeconomic inequality in well-being is similar to those used to measure socioeconomic inequality in health, such as the concentration index. The concentration indices measure the intensity of health given socioeconomic status. Nevertheless, these indices have some limitations, which have led to a substantial portion of the literature on socioeconomic inequality proposing related solutions. One such limitation is the argument made by Wagstaff. Wagstaff (2002) argues that the concentration indices do not consider the average level of health. The author explains that while policymakers are interested in inequality measures, they are equally interested in the average level of health or well-being of any specific population: the health system's performance. Wagstaff (2002) suggests that the achievement index, which captures inequalities in health, and the average health of the population, should be used in addition to the concentration indices.

Another issue with the concentration indices, arises when the health measure being used is a cardinal variable (the same as the limitation identified in the unidimensional literature). Since this well-being variable is not ratio-scale (i.e., cardinal with a well defined 0), any numerical scale imposed by a researcher changes the value of the index and might lead to a different ranking of the same population. This issue is commonly referred to as the arbitrariness of the concentration index (Erreygers (2006); Zheng (2008)). A solution to this problem, proposed by Allison and Foster (2004), is the stochastic dominance approach, discussed under the unidimensional studies.

Makdissi and Yazbeck (2017) extend the stochastic dominance approach proposed by Allison and Foster (2004) to identify robust ordering for rank dependent socioeconomic inequality indices and health achievement using "social weights".

This paper's theoretical part is similar to the literature on the measurement of socioeconomic inequality discussed above in that we suggest an index to measure socioeconomic inequality when the variable of interest is ordinal. It is different from other studies in that the proposed index has a robust reference point, the median, for measuring socioeconomic inequality in well-being. We also extend Allison and Foster (2004) and Makdissi and Yazbeck (2017) and provide conditions to robustly rank any two distributions with the same median well-being category.

3.3 Theoretical Framework

Suppose a population of size N has a joint distribution of health or well-being and socioeconomic status as $(h_i, r_i)_{i=1}^{N}$, where $h_i \in \{1, 2, 3, \ldots, K\}$ is the level of well-being/health status and r_i represents the socioeconomic status. The health or well-being variable takes values between 1 (worst outcome) and K (best outcome). Socioeconomic status, r takes values from 1 (lowest) to N (highest). To measure socioeconomic inequality or achievement in well-being or health using the categorical information, the commonly used indices are the extended concentration indices and the achievement index for relative socioeconomic inequality and well-being/health achievement respectively. Given that the variable at hand is ordinal, a numerical scale $c(h)$ which is monotonically increasing in well-being or health, is assigned. Selecting a numerical scale, $c(h) = h$, where h is the value of the ordinal health or well-being variable, allows us to compute a measure for achievement and inequality.

The achievement index measures both the average health and inequality in health between the poor and better of. Essentially, the achievement index, $A(\nu)$, captures inequality and average health or well-being simultaneously and is given by,

$$A(\nu) = \sum_{i=1}^{N} \omega(r_i; \nu)c(h_i) \tag{3.3.1}$$

where the weight is

$$\omega(r_i; \nu) = \frac{(N - r_i + 1)^{\nu} - (N - r_i)^{\nu}}{N^{\nu}}, \tag{3.3.2}$$

The parameter $\nu \geq 1$, captures the aversion to socioeconomic health inequality (Yitzhati, 1983). When $\nu = 1$, there is no aversion to socioeconomic inequality in well-being/health and $A(\nu)$ measures the average health or well-being status.

Following the literature on socioeconomic inequality in well-being/health, we can write the extended health concentration index as given by,

$$C(\nu) = \frac{1}{\mu_c} \sum_{i=1}^{N} \gamma(r_i; \nu)c(h_i) \tag{3.3.3}$$

where the weight is

$$\gamma(r_i;\nu) = \frac{1}{N} - \frac{(N - r_i + 1)^{\nu} - (N - r_i)^{\nu}}{N^{\nu}}, \tag{3.3.4}$$

and μ_c is the average health status based on the scale $c(h)$. For ν=2, we obtain the canonical concentration index and for the rest of the values that ν takes, we get the extended concentration index. The concentration indices, $C(\nu)$ can be interpreted as the loss due to inequalities in health or well-being that is, $(\mu_c - A(\nu))$ relative to the average. Formally,

$$C(\nu) = \frac{\mu_c - A(\nu)}{\mu_c} \tag{3.3.5}$$

The concentration index takes values of -1 (if the poorest has all the health and the others 0) to 1 (if the richer has all the health and the others 0). Normally, negative values are interpreted as a distribution in favour of the poorest or pro-poor and positive values as a distribution in favour of the richest. That is, when health decreases with increasing socioeconomic disadvantage, the index is negative and when health increases with increasing socioeconomic status, the index is positive.

3.3.1 Non-Robustness of the Mean as Reference point

While the concentration index is widely used in the literature for socioeconomic inequality and health, the index's point of reference is the mean. Allison and Foster (2004) argue that the mean is a non-robust reference point to assess inequality in self-reported health statuses or any ordinal variable (for instance, life satisfaction and happiness). The non-robustness of the mean as a reference point can be easily understood. For instance, consider the example of the two distributions of happiness in Table 3.1 and the numerical scales in Table 3.2. The mean of distribution D_1 is 3 under $c_1(h)$ and 4 under $c_2(h)$. The mean of distribution D_2 is 2.9 $c_1(h)$ and 4.4 under $c_2(h)$. In the first case, for distribution D_1 the mean of the distribution corresponds to "somewhat unhappy", in the second case, it is "somewhat happy". Using different numerical scales on the same categorical information changes not only the numerical value of the mean but also the corresponding well-being category. Allison and Foster (2004) argue that the standard inequality principle of aversion to mean-preserving spreads is meaningless and propose to use the median as an alternative reference point. This approach was labeled as an aversion to median preserving spread by Naga and Yalcin (2008). The robustness of the median can also be easily shown using the same distributions in Table 3.1 and the numerical scales in Table 3.2. The median under both $c_1(h)$ and $c_2(h)$ is 3 which corresponds to "somewhat happy". For this reason, following Allison and Foster (2004), Naga and Yalcin (2008), we adopt the median-preserving spread principle when defining the measure of inequality of well-being.

3.3.2 Median Based Socioeconomic Inequality Indices

As shown earlier, the median is robust to the choice of a numerical scale for any ordinal variable. For this reason, Allison and Foster (2004) propose using the statistics that captures the dispersion of the distribution of the ordinal well-being variable from its median. This

Table 3.1: Distributions of happiness

D_1	D_2
so unhappy that life is not worthwhile	so unhappy that life is not worthwhile
very unhappy with little interest in life	so unhappy that life is not worthwhile
somewhat unhappy	so unhappy that life is not worthwhile
somewhat unhappy	very unhappy with little interest in life
somewhat happy	very unhappy with little interest in life
happy and interested in life	somewhat unhappy
somewhat happy	somewhat happy
very unhappy with little interest in life	happy and interested in life
happy and interested in life	happy and interested in life
so unhappy that life is not worthwhile	happy in life

Table 3.2: Numerical scales for happiness and estimated means

	$c_1(h)$	$c_2(h)$	$c_3(h)$
so unhappy that life is not worthwhile	1	1	1
very unhappy with little interest in life	2	2	10
somewhat unhappy	3	3	11
somewhat happy	4	4	12
happy and interested in life	5	10	13
	μ_1	μ_2	μ_3
D_1	3	4	9.4
D_2	2.9	4.4	8.5

is referred to as the *Average Absolute Deviation about the Median*, $E[c(h) - c(h_m)]$. Since this proposed index is an average, and the Wagstaff's Achievement index uses a weighted average, it may seem natural to use the Wagstaff weight and compute a socially weighted absolute deviation about the median. Formally,[3]

$$I(\nu) = \sum_{i=1}^{N} \omega(r_i; \nu)|c(h_i) - c(h_m)| \tag{3.3.6}$$

Wagstaff's social weight can be interpreted as the probability of observing an individual with a particular socioeconomic status. While this index in Equation (3.3.6) can be used to robustly rank bivariate distributions of socioeconomic status and well-being, the index puts higher weight on the deviation from the median well-being status of the "poor" person regardless of whether this deviation is below or above the median. Thus, the underlying ethical judgment only stipulates that a deviation from the median well-being status is socially more costly the poorer the individual is, regardless of whether the deviation is from above or below the median category. As a result, this may not capture the usual ethical concern underlying the measurement of socioeconomic inequality in health or well-being.

To solve this issue, we propose an index based on the principle of aversion to median preserving spread, with underlying ethical judgments similar to the inequality indices commonly found in the literature. We propose an index that is built on two censored

[3]The values of the index in Equation (3.3.6) lies between $[0, Z(c(h); \nu)]$, where $Z(c(h); \nu)$ is a constant specific for each scaling function and each value of the parameter of aversion to socioeconomic inequality. It is possible to normalize the index in Equation (3.3.6) to fall between 0 and 1. For details on the normalization, see Appendix A

functions as follows;

$$I^{-}(\nu) = \sum_{i=1}^{N} \omega(r_i;\nu) \max(c(h_m) - c(h_i), 0) \tag{3.3.7}$$

and

$$I^{+}(\nu) = \sum_{i=1}^{N} \tilde{\omega}(r_i;\nu) \max(c(h_i) - c(h_m), 0) \tag{3.3.8}$$

where $\omega(r_i;\nu)$ is defined as in equation (3.3.2) and,

$$\tilde{\omega}(r_i;\nu) = \frac{(r_i)^{\nu} - (r_i - 1)^{\nu}}{N^{\nu}}, \nu \geq 1 \tag{3.3.9}$$

$I^{-}(\nu)$, the first function in equation (3.3.7) focuses on socioeconomic inequality below the median while $I^{+}(\nu)$ in equation (3.3.8) focuses on the distribution above the median. Half of the population lies above the median and the other half lies below the median therefore we can create an index such that,

$$\tilde{I}(\nu) = 0.5I^{-}(\nu) + 0.5I^{+}(\nu) \tag{3.3.10}$$

For a given marginal distribution of well-being, this index $\tilde{I}(\nu)$ reaches a maximum value when well-being increases monotonically with socioeconomic ranks. Conversely, it reaches its lowest value when well-being decreases monotonically with socioeconomic ranks. The value of the index is always larger than 0 because social weights assigned to the absolute deviations are positive. The social weights are higher on deviations of the poor if these deviations are below the median and on deviations of the rich if these deviations are above the median. It is possible to normalize the index in Equation (3.3.10) to lie in the interval $[0, 1]$.[4] Thus, the proposed index, $\tilde{I}(\nu)$ captures the same ethical concern for socioeconomic inequality in health but uses the median as the reference point. When there is no aversion to socioeconomic inequality in well-being, that is, $\nu = 1$, $\tilde{I}(1) = I(1) = E|c(h) - c(h_m)|$. Thus, the average absolute deviation around the median used in Allison and Foster (2004) is a particular case of this index.

3.3.3 Robust rankings using median based indices

Having developed an index that accounts for both the categorical aspect of the data at hand (health or well-being variables) and the necessity of using the median as a reference point, we now turn to our second objective, robust ranking. A robust ranking of socioeconomic inequality in well-being provided by $\tilde{I}(\nu)$ between two distribution that is valid for any scaling functions $c(h)$ can be detected by comparing two social weighted functions. In what follows, we will develop sufficient conditions.

Let $\rho_k := \{i : h_i = k\}$ be the set of individuals in well-being category k and

[4] For more details, see Appendix A.

$\phi(k;\nu) = \sum_{i\in\rho_k}\omega(r_i;\nu)$. Let $\phi^L(s;\nu) = \sum_{i\in\rho_k}\omega(r_i;\nu)$ be the social weights of individuals well-being categories below the median class. Equivalently, let $\phi^R(s;\nu) = \sum_{i\in\rho_k}\tilde{\omega}(r_i;\nu)$ be the social weights of individuals in well-being categories above the median. Define $\Psi^L(k;\nu) = \sum_{s=1}^{k}\phi^L(s;\nu)$ and $\Psi^R(k;\nu) = \sum_{s=1}^{K}\phi^R(s;\nu)$ and let m be the median well-being category. These functions, $\Psi^L(k;\nu)$ and $\Psi^R(k;\nu)$ play an important role in ranking any two distributions. Given these two functions, the index in equation (3.3.10) can also be written as

$$\tilde{I}(\nu) = 0.5\sum_{k=1}^{m-1}\Psi^L(k;\nu)\Delta c(h_k) + 0.5\sum_{k=m+1}^{K}\Psi^R(k;\nu)\Delta c(h_{k-1}) \tag{3.3.11}$$

where $\Delta c(h_k) = c(h_{k+1}) - c(h_k)$

In this context, a ranking of socioeconomic well-being inequality between two distributions that is valid for all scaling functions $c(h)$ can be obtained by comparing their $\Psi^L(k;\nu)$ and $\Psi^R(k;\nu)$ functions.[5]

Theorem 3. *For any two distributions 0 and 1 with the same median category, $\tilde{I}_0(\nu) \geq \tilde{I}_1(\nu)$ for all scaling functions $c(h)$ if and only if;*

$\Psi_0^L(k;\nu) \geq \Psi_1^L(k;\nu), \quad for \quad all \quad k \in \{1, 2, \ldots, m-1\}$

and,

$\Psi_0^R(k;\nu) \geq \Psi_1^R(k;\nu), \quad for \quad all \quad k \in \{m+1, m+2, \ldots, K\}$

[5] A formal proof of theorems 3 to 5 is found in the Appendix

If the conditions for Theorem 3 are not verified, a researcher can impose some level of cardinality on the scale function. If the researcher believes that the importance of the difference between categories of well-being decreases for higher levels of well-being, then a concave scale will be appropriate. For instance a concave scale is suitable for variables such as happiness and life satisfaction. If the researcher has reasons to believe that the difference between adjacent categories of well-being increases for higher levels of well-being, then, a convex scale will be much suitable. For instance, a convex scale is suitable for a variable such as depression.

Robust ranking using concave scale

As mentioned earlier, the concave numerical scale is adequate if the researcher has a strong belief that the difference between adjacent categories becomes less important when we move towards the highest category. For a concave scale, we can define $\Psi^{2+}(k;\nu)$ as

$$\Psi^{2+}(k;\nu) = \begin{cases} \sum_{j=1}^{k} \Psi^{L}(k;\nu) \quad for \quad all \quad k \in \{1,2,\ldots,m-1\} \\ \Psi^{2+}(m-1;\nu) + \sum_{j=m+1}^{k} \Psi_1^{R}(k;\nu) \quad for \quad all \quad k \in \{m+1,m+2,\ldots,K\} \end{cases} \tag{3.3.12}$$

Theorem 4. *For any two distributions 0 and 1 with the same median category,* $\tilde{I}_0(\nu) \geq \tilde{I}_1(\nu)$ *for all concave scaling functions* $c(h)$ *if and only if;*

$$\Psi_0^{2+}(k;\nu) \geq \Psi_1^{2+}(k;\nu), \quad for \quad all \quad k \in \{1,2,\ldots,m-1,m+1,\dot{;}K\}$$

Theorem 4 states that if there is a concave scale, one can obtain a robust ranking if the socially weighted average absolute deviation about the median is higher in distribution 0 than in distribution 1 by comparing the $\Psi^{2+}(k;\nu)$ functions.

Robust ranking using convex scale

As mentioned earlier if the researcher has a strong belief that differences between adjacent categories become more important as one moves towards the highest category, then it is reasonable to assume that numerical scale is convex. For a convex scale, we can define $\Psi^{2-}(k;\nu)$ as

$$\Psi^{2-}(k;\nu) = \begin{cases} \Psi^{2-}(m+1;\nu) + \sum_{j=k}^{m-1} \Psi_1^L(j;\nu) \quad for \quad all \quad k \in \{1,2,\ldots,m-1\} \\ \sum_{j=k}^{K} \Psi^R(k;\nu) \quad for \quad all \quad k \in \{m+1,m+2,\ldots,K\} \end{cases} \tag{3.3.13}$$

Theorem 5. *For any two distributions 0 and 1 with the same median category, $\tilde{I}_0(\nu) \geq \tilde{I}_1(\nu)$ for all convex scaling functions $c(h)$ if and only if;*

$\Psi_0^{2-}(k;\nu) \geq \Psi_1^{2-}(k;\nu), \quad for \quad all \quad k \in \{1,2,\ldots,m-1,m+1,\ldots,K\}$

According to Theorem 5, one can determine if the socially weighted average absolute deviation about the median of a distribution is robustly higher in distribution 0 than in distribution 1 by comparing their $\Psi^{2-}(k;\nu)$ functions. [6]

[6] The next question becomes, what happens to ranking distributions when the median category for any two distributions are not the same. We leave this to future work as it is common to find the same median for categories of well-being or health across subpopulations, across time and across locations

Robust ranking using achievement indices

Given that the concentration index and rank dependent inequality indices in general, do not account for the average health or well-being in the population, Wagstaff (2002) argues that one should complement inequality analysis with an achievement index, as defined earlier in Equation (3.3.1). The achievement index can capture both socioeconomic inequalities in health or well-being and the average health status or well-being. As stated earlier, when the health or well-being variable is categorical in nature, any monotonic non decreasing transformation of the qualitative variable, can be used as a suitable numerical scale. Nevertheless, different numerical scales, give different values for the achievement index, thus giving different rankings for the same population. Makdissi and Yazbeck (2017) propose a solution to this issue and derived conditions that allow for robust ranking of distributions for categorical variables. They proposed using a cumulative "social weight", $\Phi(k) = \sum_{j=1}^{K} \phi(k;\nu)$ which plays a similar role as the cumulative distribution function in first order stochastic dominance. The conditions proposed by the authors are as follows:

For any two distributions 0 and 1, $A_0(\nu) \geq A_1(\nu)$ for all scaling functions $c(h)$ if and only if;

$$\Phi_0^1(k;\nu) \geq \Phi_1^1(k;\nu), \quad \text{for} \quad \text{all} \quad k \in \{1, 2, \ldots, K-1\} \tag{3.3.14}$$

where Φ is the cumulative social weight.

The authors also propose imposing a concave or convex numerical scale if a researcher has a strong belief that the difference in adjacent well-being categories become less important as one moves towards the highest category and vice versa. They defined $\Phi^{2+}(k) = \sum_{j=1}^{k} \Phi^1(j)$ and $\Phi^{2-}(k) = \sum_{j=k}^{K-1} \Phi^1(j)$ and provided the following conditions for robust ranking for concave and convex scales respectively.

For any two distributions 0 and 1, $A_0(\nu) \geq A_1(\nu)$ for all concave scaling functions $c(h)$ if and only if;

$$\Phi_0^{2+}(k;\nu) \geq \Phi_1^{2+}(k;\nu), \quad for \quad all \quad k \in \{1, 2, \ldots, K-1\} \tag{3.3.15}$$

For any two distributions 0 and 1, $A_0(\nu) \geq A_1(\nu)$ for all convex scaling functions $c(h)$ if and only if;

$$\Phi_0^{2+}(k;\nu) \geq \Phi_1^{2+}(k;\nu), \quad for \quad all \quad k \in \{1, 2, \ldots, K-1\} \tag{3.3.16}$$

In the empirical section, we will use Theorems 1 to 3 and the conditions in Equations (3.3.14) to (3.3.16) to rank socioeconomic inequality in well-being and mental health among some sub-groups in Canada.[7]

[7] For details and proofs to the conditions in equation (3.3.14) to (3.3.16), see Makdissi and Yazbeck (2017)

3.4 Data

This paper uses data from the Canadian Community Health Survey (CCHS) in 2015. The CCHS is a cross sectional survey that gathers data on health determinants, health status and health care utilization for Canadians. Data is collected from individuals who are 12 years or older: excluding those living on reserves and other Aboriginal settlements, persons institutionalized, and children between 12 and 17 years who are in foster homes and some health regions in Quebec. The main aim of the CCHS is to monitor the health of Canadians. It uses a multi-stage sampling framework and has a large sample size of 130,000. Samples were collected every other year in 2001, 2003 and 2005[8]. Starting in 2007, the CCHS sample size was decreased to 65000 as data was collected annually. However, the yearly data are also combined biannually to increase the sample size.

The CCHS is suitable for the question we seek to address in this paper because it provides detailed information on life satisfaction and happiness and other health and mental health variables. In addition, demographic factors are recorded for all survey respondents. The socioeconomic status of individuals is of interest in our analysis and the CCHS provides information on both the level of education and point estimates of income.[9] However, due to high missing response on income, starting in 2011, the CCHS provides income data which are imputed using information on tax files and regression analysis. We will be using these imputed income data in all of our analysis which implies that we do not have any missing

[8]The Canadian Community Health Survey was started in 2001

[9]The point estimates of income are available in the restricted access data which are available at Statistics Canada's Research Data Centre.

income data in the years 2015.[10] Our analysis uses the ranks rather than the level of income so these imputations may not necessarily be an issue unless they affect the ranks.

3.4.1 Outcomes

In this paper, our outcomes of interest are subjective well-being measures such as life satisfaction and happiness. Although these measures are subjective and may be prone to measurement errors and interpersonal comparability issues, these measures are widely used (Kahneman et al. (1999); Dutta and Foster (2013); Stevenson and Wolfers (2008); Veenhoven (2007)). In addition, the multidimensional nature of these well-being variables makes them informative (Krueger and Schkade (2008)) and useful in research on well-being (Diener et al. (2013)). Motivated by this, we apply theorems 3 to 5 to rank socioeconomic inequality in life satisfaction and happiness in 2015 in Canada. The CCHS asked respondents questions about their life satisfaction.[11] We use the 5 points categories in all of our analysis for life satisfaction as shown in Table 3.3. Respondents were also asked about their happiness, days they felt depressed, mental and general health. The questions and responses are also shown on Table 3.3.

After missing responses are omitted [12], the sample for life satisfaction is $49,831$ and the sample size for happiness is $51,323$ in 2015. The sample sizes for general mental health and general health status are $50,173$ and $51,441$ respectively, in 2015. For depression, the sample

[10]We exclude one household in 2015 with negative income because including or excluding them in our analysis do not affect our results.

[11]The exact question is, "Using a scale of 0 to 10, where 0 means "very dissatisfied" and 10 means " very satisfied", how do you feel about your life as a whole right now. The 10 points scale and a health health component variable completed by a proxy are used to derive a 5 points category of life satisfaction. For more details, see the CCHS codebook.

[12]We assume that missing information is random

Table 3.3: Description of the some categorical variables centered

Life satisfaction	Would you describe yourself as being usually	Over the last two weeks, how often have you felt down, depressed or hopeless?	In general, would you say your mental health is?	In general, would you say your health is ?
Very Satisfied	happy and interested in life?	not at all	excellent	excellent
Satisfied	somewhat happy?	several days	very good	very good
Neither satisfied nor dissatisfied	somewhat unhappy	more than half the days	good	good
Dissatisfied	unhappy with little interest in life?	nearly everyday	fair	fair
Very Dissatisfied	so unhappy that life is not worthwhile?		poor	poor

is around $32,465$ in 2015.[13]

There is growing evidence that, there are variations in well-being among different subgroups. For instance Clark (1997) explain that there are significant differences in what makes men and women happy and women often report higher life satisfaction than men. Motivated by this, we focus on measuring socioeconomic inequality in well-being for different demographic factors, specifically gender, age, marital status, immigration status, aboriginal status, area of residence and the employment status of each gender. According to Sharpe et al. (2010) geographical variation in well-being exist. In Canada, the close proximity of Quebec and Ontario and the differences in policies across the two jurisdictions, makes a comparison of socioeconomic inequality in well-being between the two provinces policy-relevant. Therefore, we are also interested in the comparison of inequality between these two provinces. We infer the socioeconomic status of individuals using imputed household income available in the CCHS data set. Household income is divided by the square root of household size which is the equivalent scale to get equivalent income. We rank individuals using this

[13] The sample size for the question on depression is 943.

derived equivalent income. Table 3.4 shows the median of life satisfaction and happiness for each sub population. In 2015, the median category for life satisfaction is "satisfied" for all subpopulations, the median for happiness is "happy and interested in life ", that for mental health is "very good", that for general health status is"very good" and that for depression is "not at all". These medians apply for all subpopulations. As the median category does not vary across subgroups, our proposed method is suitable. Table 3.4 also presents the median income of the subgroups. Among these sub population, the median income ranges from $\$30,000$ for unemployed female which is lowest median income to about $\$56,000$ for employed male in 2015. In the CCHS, the median income for the entire population is about $\$42,426$ in 2015.

Table 3.4: Median Categories of well-being in different subpopulations

	Life Satisfaction (2015)	Happiness (2015)	General Mental Health status (2015)	General Health status (2015)	Depression (2015)	Income
Gender						
Male	Satisfied	Happy and interested in life	Very good	Very good	Not at all	45961
Female	Satisfied	Happy and interested in life	Very good	Very good	Not at all	40000
Age						
working age	Satisfied	Happy and interested in life	Very good	Very good	Not at all	46188
65_+	Satisfied	Happy and interested in life	Very good	Very good	Not at all	31819.8
Marital status						
Couple	Satisfied	Happy and interested in life	Very good	Very good	Not at all	35355.34
Non-couple	Satisfied	Happy and interested in life	Very good	Very good	Not at all	49497.48
Immigration status						
Immigrant	Satisfied	Happy and interested in life	Very good	Very good	Not at all	37577.09
Non immigrant	Satisfied	Happy and interested in life	Very good	Very good	Not at all	46188.02
Aboriginal status						
Aboriginal	Satisfied	Happy and interested in life	Very good	Very good	Not at all	35355.34
Non-Aboriginal	Satisfied	Happy and interested in life	Very good	Very good	Not at all	46188.02
Area						
Rural	Satisfied	Happy and interested in life	Very good	Very good	Not at all	42426.41
Population area	Satisfied	Happy and interested in life	Very good	Very good	Not at all	42426.41
Regions						
Atlantic	Satisfied	Happy and interested in life	Very good	Very good	Not at all	40000
Central	Satisfied	Happy and interested in life	Very good	Very good	Not at all	41569.22
Western	Satisfied	Happy and interested in life	Very good	Very good	Not at all	46188.02
Ontario	Satisfied	Happy and interested in life	Very good	Very good	Not at all	54018.63
Quebec	Satisfied	Happy and interested in life	Very good	Very good	Not at all	47965.76

3.5 Results

In our analysis for robust ordering, we use $\nu = 1, 1.5, 2, 2.5$ levels of aversions to socioeconomic inequalities. When $\nu = 1$, we have the case of unidimensional inequality in well-being or health. That is, there is no aversion to socioeconomic inequality, which is in line with Allison and Foster (2004). The other cases where the aversion to socioeconomic inequality is greater than 1, reflect when there is some level of aversion to socioeconomic inequality. We first focus on the case where we use monotonically non-decreasing scale for well-being. After this, we focus on two scales: monotonically non-decreasing convex scales and monotonically non-decreasing concave scales.

Table 3.5 presents the results of a robust ranking of socioeconomic inequality in life satisfaction for all monotonically non-decreasing scales in 2015, allowing for comparison among different groups. On all results tables, D stands for dominance of the row subgroup over the column subgroup for $\nu = 1; 1.5; 2; 2.5$. Similarly, a specific number shows the row subgroup's dominance over the column subgroup when ν (the aversion to socioeconomic inequality) is equal to that particular number(s). For instance, 1 means the row subgroup dominates its corresponding column subgroup when there is no aversion to socioeconomic inequality in well-being. ND means non-dominance. Dominance conditions are established using the theorems in the theoretical framework.[14] We find a dominance of socioeconomic inequality in life satisfaction for males over females for all levels of aversions to socioeconomic inequality in life satisfaction in 2015. This means that males have a lower level of socioeconomic inequality in life satisfaction than females. When we narrow the analysis

[14] We do not statistically test for dominance.

to males only, we cannot robustly rank socioeconomic inequality in life satisfaction among employed males and unemployed males in 2015. On the contrary, females who are employed have a lower socioeconomic inequality in life satisfaction than unemployed females when there is some aversion to socioeconomic inequality. Working-age groups and those aged 65 and older cannot be robustly ranked in socioeconomic inequality in life satisfaction. Similarly, we cannot rank couples and non-couples in socioeconomic inequality in life satisfaction. Comparing whites and non-whites, we find that non-whites have a lower socioeconomic inequality in life satisfaction than whites for 2015. At the highest level of aversion to socioeconomic inequality, $\tilde{I}(2.5)$, non-immigrants have higher socioeconomic inequality in life satisfaction than immigrants. Aboriginals and non-aboriginals cannot be robustly ranked. We also compare the three regions in Canada, namely the Atlantic (Newfoundland and Labrador, Prince Edward Island, Nova Scotia, and New Brunswick), Central (Ontario and Quebec), and Western (British Columbia, Alberta, Saskatchewan, and Manitoba). The Central region has a lower socioeconomic inequality in life satisfaction in 2015 than the Atlantic region. However, the Western region has lower socioeconomic inequality in life satisfaction than the Central region when the aversion to socioeconomic is 1.5. Lastly, in 2015, we compare the two provinces in the Central region, Ontario and Quebec, due to their proximity and policy differences. We find that Ontario has a higher inequality in life satisfaction when pure inequality in life satisfaction is considered. That is, the aversion to socioeconomic inequality in life satisfaction in 2015 is 1. However, we cannot robustly rank the two provinces when there is some level of aversion of socioeconomic inequality in life satisfaction above 1. Finally, we find no dominance in life satisfaction between rural areas and population centers in 2015.

Table 3.5: Robust ordering of socioeconomic inequality in Life satisfaction in 2015

All numerical scales

	Female	65_+	Unemployed male	Unemployed female	Non-couple	White	Non-Immigrant	Aboriginal	Atlantic	Central	Population area	Ontario
Male	D											
Working age		ND										
Employed male			ND									
Employed female				1.5,2,2.5								
Couple					ND							
Non- white						D						
Immigrant							2.5					
Non-aboriginal								ND				
Central									D			
Western										1.5		
Rural area											ND	
Quebec												1

**Note*: 1 means dominance for $\nu = 1$; 1.5 means dominance for $\nu = 1.5$; 2 means dominance for $\nu = 2$; 2.5 means dominance for $\nu = 2.5$; D means dominance for $\nu = 1, 1.5, 2, 2.5$.D also means dominance of the row subgroup over the column subgroup. Finally, ND mean non dominance.

If we impose a non-decreasing concave numerical scale, we obtain additional ranking for employed males and unemployed males as shown in Table 3.6. There is lower socioeconomic inequality in life satisfaction among employed males compared to unemployed males. Also, couples have a lower socioeconomic inequality in life satisfaction than people who are not a couple. Similarly, non-aboriginals have lower socioeconomic inequality in life satisfaction than Aboriginals.

Table 3.6: Robust ordering of socioeconomic inequality in Life satisfaction in 2015

Concave scales

	Female	65_+	Unemployed male	Unemployed female	Non-couple	White	Non-Immigrant	Aboriginal	Atlantic	Central	Population area	Ontario
Male	D											
Working age		ND										
Employed male			D									
Employed female				1.5,2,2.5								
Couple					D							
Non- white						D						
Immigrant							2.5					
Non-aboriginal								D				
Central									D			
Western										1.5		
Rural area											ND	
Quebec												1

**Note*: 1 means dominance for $\nu = 1$; 1.5 means dominance for $\nu = 1.5$; 2 means dominance for $\nu = 2$; 2.5 means dominance for $\nu = 2.5$; D means dominance for $\nu = 1, 1.5, 2, 2.5$.D also means dominance of the row subgroup over the column subgroup. Finally, ND mean non dominance.

Using the theorem derived in Makdissi and Yazbeck (2017) for the achievement index, Table

Table 3.7: Robust ordering of Achievement in Life satisfaction in 2015

All numerical scales A(ν)

	Female	65_+	Unemployed male	Unemployed female	Non-couple	White	Non-Immigrant	Aboriginal	Atlantic	Western	Population area	Ontario
Male	ND											
working age		ND										
Employed male			D									
Employed female				D								
Couple					D							
Non- white						2.5						
Immigrant							ND					
Non-Aboriginal								D				
Central									ND			
Central										ND		
Rural area											D	
Quebec												1.5,2,2.5

**Note*: 1 means dominance for $\nu = 1$; 1.5 means dominance for $\nu = 1.5$; 2 means dominance for $\nu = 2$; 2.5 means dominance for $\nu = 2.5$; D means dominance for $\nu = 1, 1.5, 2, 2.5$.D also means dominance of the row subgroup over the column subgroup. Finally, ND mean non dominance.

3.7 shows that for $\nu = 1, 1.5, 2, 2, 5$, employed males and females show a higher level of life satisfaction achievement than unemployed males and females, respectively. Couples also show a higher life satisfaction achievement than non-couples. Non-whites have higher life satisfaction achievement than whites when the aversion to socioeconomic inequality is 2.5. In addition, non-aboriginals have a higher life satisfaction achievement than aboriginals. People in rural areas also show a higher life satisfaction achievement than those in populated areas. These results hold with and without any aversion to socioeconomic inequality in life satisfaction. For Ontario and Quebec, we cannot rank the two provinces in average life satisfaction, but at any level of aversion to socioeconomic inequality, Ontario has less life satisfaction achievement than Quebec. As shown in Table 3.7, it is not possible to robustly order other sub-populations.

Imposing a non-decreasing concave numerical scale, we obtain additional ranking for the working age group and 65 years and older in Table 3.8. There is a higher level of life satisfaction achievement for the working age group than for those 65 years and older when there is no aversion to socioeconomic inequality. Similarly, the Western region has a higher level of life satisfaction achievement than the Central region when there is no aversion to socioeconomic inequality. In contrast, non-whites have a higher level of life satisfaction achievement than whites when there is aversion for socioeconomic inequality.

If we compare the results for socioeconomic inequality and achievement in life satisfaction among the different subgroups, we find that most groups with lower socioeconomic inequality also have higher achievement in life satisfaction. For instance, we find that employed females have lower socioeconomic inequality and higher achievement in life satisfaction than unemployed females when we consider all numerical scales. Non-whites have lower socioeconomic inequality than whites when aversion to socioeconomic inequality is at its highest point, 2.5, but non-whites have higher achievement in life satisfaction than whites for all aversions to socioeconomic inequality used. On the contrary, we cannot rank some subgroups in socioeconomic inequality in life satisfaction, but we obtain rankings for achievement in life satisfaction while the reverse is true for others. For instance, aboriginals and non-aboriginals cannot be ranked in socioeconomic inequality in life satisfaction, but we find that non-aboriginals have higher achievement in life satisfaction than aboriginals. There are also subgroups that we cannot rank both in socioeconomic inequality or achievement in life satisfaction like the working age group and people aged 65 and older.

Turning to another well-being variable of interest, happiness, Table 3.9 summarizes the result for socioeconomic inequality in happiness for 2015 for different subgroups.[15] While we cannot robustly rank males and females in socioeconomic inequality in happiness for all possible numerical scales, unemployed males have higher socioeconomic inequality in happiness than

[15] Note that for happiness, the median category which is "Happy and interested in life" is also the highest category. In this case, the weight function collapse to ω, therefore we only have $\nu \geq 1$

Table 3.8: Robust ordering of Achievement in Life satisfaction in 2015

Concave scales	A(ν)											
	Female	65_+	Unemployed male	Unemployed female	Non-couple	White	Non-Immigrant	Aboriginal	Atlantic	Central	Population area	Ontario
Male	ND											
Working age		1										
Employed male			D									
Employed female				D								
Couple					D							
Non- white						1.5,2,2.5						
Immigrant							ND					
Non-Aboriginal								D				
Central									ND			
Western										1		
Rural area											D	
Quebec												D

**Note*: 1 means dominance for $\nu = 1$; 1.5 means dominance for $\nu = 1.5$; 2 means dominance for $\nu = 2$; 2.5 means dominance for $\nu = 2.5$; D means dominance for $\nu = 1, 1.5, 2, 2.5$.D also means dominance of the row subgroup over the column subgroup. Finally, ND mean non dominance.

employed males in 2015. This is also true for employed and unemployed females as employed females have lesser socioeconomic inequality in happiness in 2015 than females who are not employed. People who are 65 years or older have a lower socioeconomic inequality in happiness than the working age group. Couples also have lower socioeconomic inequality in happiness than non-couple. Comparing non-whites and whites, we also find that non-whites have lesser socioeconomic inequality in happiness than whites. We establish a similar ranking for aboriginals and non-aboriginals, as non-aboriginals face less socioeconomic inequality in happiness than individuals who identify as aboriginals. Also, rural areas have lower socioeconomic inequality in happiness than population centres. All these results hold for all three cases of aversion to socioeconomic inequality. In addition, immigrants have less socioeconomic inequality than non-immigrants when $\nu > 1$. It is impossible to robustly rank socioeconomic inequality in happiness between the three regions, Atlantic, Western, and Central. However, Quebec has a lower socioeconomic inequality in happiness than Ontario.

Imposing a concave numerical scale, Table 3.10 shows that there is robust ranking of

Table 3.9: Robust ordering of socioeconomic inequality in Happiness in 2015

All numerical scales

	Female	Working age	Unemployed male	Unemployed female	Non-couple	White	Non-Immigrant	Aboriginal	Atlantic	Western	Population area	Ontario
Male	ND											
65_+		D										
Employed male			D									
Employed female				D								
Couple					D							
Non-White						D						
Immigrant							1.5,2,2.5					
Non-Aboriginal								D				
Central									ND			
Central										ND		
Rural area											D	
Quebec												D

**Note*: 1 means dominance for $\nu = 1$; 1.5 means dominance for $\nu = 1.5$; 2 means dominance for $\nu = 2$; 2.5 means dominance for $\nu = 2.5$; D means dominance for $\nu = 1, 1.5, 2, 2.5$.D also means dominance of the row subgroup over the column subgroup. Finally, ND mean non dominance.

Table 3.10: Robust ordering of socioeconomic inequality in Happiness in 2015

Concave scales

	Female	Working age	Unemployed male	Unemployed female	Non-couple	White	Non-Immigrant	Aboriginal	Central	Western	Population area	Ontario
Male	ND											
65_+		D										
Employed male			D									
Employed female				D								
Couple					D							
Non-White						D						
Immigrant							1.5,2,2.5					
Non-Aboriginal								D				
Atlantic									D			
Central										ND		
Rural area											D	
Quebec												D

**Note*: 1 means dominance for $\nu = 1$; 1.5 means dominance for $\nu = 1.5$; 2 means dominance for $\nu = 2$; 2.5 means dominance for $\nu = 2.5$; D means dominance for $\nu = 1, 1.5, 2, 2.5$.D also means dominance of the row subgroup over the column subgroup. Finally, ND mean non dominance.

socioeconomic inequality in happiness between all different groups, except for gender and the Central and Western regions. In addition to the ranking for happiness when we use all numerical scale, we can now rank the Atlantic and Central regions. The Atlantic region has lower socioeconomic inequality in happiness than the Central region.

Using the conditions developed by Makdissi and Yazbeck (2017), Table 3.11 shows that the average happiness for employed males, employed females, couples, non-whites,

Table 3.11: Robust ordering of Achievement in Happiness in 2015

All numerical scales $A(\nu)$

	Female	65+	Unemployed male	Unemployed female	Non-couple	White	Non-Immigrant	Aboriginal	Atlantic	Western	Population area	Ontario
Male	ND											
Working Age		ND										
Employed male			D									
Employed female				D								
Couple					D							
Non- white						D						
Immigrant							2,2.5					
Non-Aboriginal								D				
Central									ND			
Central										ND		
Rural area											D	
Quebec												1.5,2,2.5

**Note*: 1 means dominance for $\nu = 1$; 1.5 means dominance for $\nu = 1.5$; 2 means dominance for $\nu = 2$; 2.5 means dominance for $\nu = 2.5$; D means dominance for $\nu = 1, 1.5, 2, 2.5$.D also means dominance of the row subgroup over the column subgroup. Finally, ND mean non dominance.

non-aboriginals and rural areas is higher than unemployed males, unemployed females, non-couples, whites, aboriginals and populated areas respectively in 2015. These results are robust ordering of pure inequality in well-being derived by Allison and Foster (2004) because the median category of happiness is also the highest category. When we introduce some level of aversion to socioeconomic inequality in happiness, the robust ordering among these groups still hold. In addition, at $\nu = 1.5$, Quebec dominates Ontario in achievement in happiness. At a higher level of aversion to socioeconomic inequality, $\nu = 2, 2.5$, Quebec has a higher achievement in happiness than Ontario. Immigrants show higher achievement in happiness than non-immigrants. However, we cannot rank gender, the three regions, and the different age groups in achievement in happiness in 2015.

To obtain additional rankings, we impose a concave numerical scale. Table 3.12 shows that there is a higher achievement in happiness among immigrants than non-immigrants for all levels of aversion to socioeconomic inequality. The Central region has a higher achievement in happiness than the Atlantic region when aversion to socioeconomic inequality is 2 and 2.5. In addition, the Western region has higher achievement in happiness than the Central region

Table 3.12: Robust ordering of Achievement in Happiness in 2015

Concave scales	$A(\nu)$											
	Female	65+	Unemployed male	Unemployed female	Non-couple	White	Non-Immigrant	Aboriginal	Atlantic	Central	Population area	Ontario
Male	ND											
Working Age		ND										
Employed male			D									
Employed female				D								
Couple					D							
Non- white						D						
Immigrant							D					
Non-Aboriginal								D				
Central									2,2.5			
Western										2.5		
Rural area											D	
Quebec												1.5,2,2.5

**Note*: 1 means dominance for $\nu = 1$; 1.5 means dominance for $\nu = 1.5$; 2 means dominance for $\nu = 2$; 2.5 means dominance for $\nu = 2.5$; D means dominance for $\nu = 1, 1.5, 2, 2.5$.D also means dominance of the row subgroup over the column subgroup. Finally, ND mean non dominance.

at the highest level of aversion to socioeconomic inequality we used, which is 2.5.

We find that most subgroups with lower socioeconomic inequality in happiness also have higher achievement in happiness. Employed males, employed females, couples, non-whites, non-aboriginals, Quebec, and rural areas have lower socioeconomic inequality in life satisfaction and higher achievement in happiness than unemployed males, unemployed females, non-couples, whites, aboriginals, Ontario and populated areas. It is also interesting to mention that although males and females could not be ranked in achievement in happiness, males have lower socioeconomic inequality in happiness than females.

Table 3.13 shows the robust ordering of socioeconomic inequality in depression among different sub-groups in 2015 for all numerical scales. Males have lower socioeconomic inequality in depression than females. For both genders, the unemployed group has a higher socioeconomic inequality in depression than the employed group. The working-age population has higher socioeconomic inequality in depression than 65 or older people. Couples, non-whites, immigrants and non-aboriginals have a lower socioeconomic inequality

Table 3.13: Robust ordering of socioeconomic inequality in Depression in 2015

All numerical scales

	Female	Working age	Unemployed male	Unemployed female	Non-couple	White	Non-Immigrant	Aboriginal	Central	Western	Population area
Male	D										
65_+		D									
Employed male			D								
Employed female				D							
Couple					D						
Non-White						D					
Immigrant							D				
Non-Aboriginal								D			
Atlantic									D		
Central										ND	
Rural area											D

**Note*: 1 means dominance for $\nu = 1$; 1.5 means dominance for $\nu = 1.5$; 2 means dominance for $\nu = 2$; 2.5 means dominance for $\nu = 2.5$; D means dominance for $\nu = 1, 1.5, 2, 2.5$.D also means dominance of the row subgroup over the column subgroup. Finally, ND mean non dominance.

Table 3.14: Robust ordering of socioeconomic inequality in Depression in 2015

Convex scales

	Female	Working age	Unemployed male	Unemployed female	Non-couple	White	Non-Immigrant	Aboriginal	Central	Western	Population area
Male	D										
65_+		D									
Employed male			D								
Employed female				D							
Couple					D						
Non-White						D					
Immigrant							D				
Non-Aboriginal								D			
Atlantic									D		
Central										ND	
Rural area											D

**Note*: 1 means dominance for $\nu = 1$; 1.5 means dominance for $\nu = 1.5$; 2 means dominance for $\nu = 2$; 2.5 means dominance for $\nu = 2.5$; D means dominance for $\nu = 1, 1.5, 2, 2.5$.D also means dominance of the row subgroup over the column subgroup. Finally, ND mean non dominance.

in depression than non-couples, whites, non-immigrants, and aboriginals, respectively. For the three regions, while the Atlantic shows a lower socioeconomic inequality in depression than the Western region, we cannot rank the Central and Western regions. Nevertheless, there is less socioeconomic inequality in depression in rural areas than in populated areas. When we impose a convex numerical scale, there are no additional robust rankings, as shown in Table 3.14.

For depression, the achievement index is described as a "failure" index. Table 3.15 shows

Table 3.15: Robust ordering of Failure in Depression in 2015

All numerical scales A(ν)

	Female	Working age	Unemployed male	Unemployed female	Non-couple	White	Non-Immigrant	Aboriginal	Atlantic	Central	Population area
Male	D										
65_+		D									
Employed male			D								
Employed female				D							
Couple					D						
Non- white						1.5,2,2.5					
Immigrant							D				
Non-Aboriginal								D			
Central									ND		
Western										D	
Rural area											D

**Note*: 1 means dominance for $\nu = 1$; 1.5 means dominance for $\nu = 1.5$; 2 means dominance for $\nu = 2$; 2.5 means dominance for $\nu = 2.5$; D means dominance for $\nu = 1, 1.5, 2, 2.5$.D also means dominance of the row subgroup over the column subgroup. Finally, ND mean non dominance.

there is less failure in self-reported depression among males, people 65 years and above, employed males, employed females, couples, non-aboriginals and immigrants compared to females, working-age group, unemployed males, unemployed females, non-couples, aboriginals and non-immigrants, respectively. Similar results hold for non-whites when compared to whites but at some level of aversion to socioeconomic inequality. At $\nu = 1$, we cannot robustly rank failure in self-reported depression among the different races. For location comparison, we cannot robustly order the Atlantic and Central regions, however, the Western region has less failure in self-reported depression than the Central region. Rural areas also have less failure in self-reported depression than in populated areas.

When we impose a convex numerical scale, we achieve complete ordering for subgroups. In addition to the results on Table 3.16, there is ranking of non-whites and whites even when there is no aversion to socioeconomic inequality, that is $\nu = 1$. The Atlantic region also has less failure in self-reported depression than the Central region.

The results on depression show that males, people 65 years and above, employed

Table 3.16: Robust ordering of Failure in Depression in 2015

Convex scales	A(ν)										
	Female	Working age	Unemployed male	Unemployed female	Non-couple	White	Non-Immigrant	Aboriginal	Central	Central	Population area
Male	D										
65_+		D									
Employed male			D								
Employed female				D							
Couple					D						
Non- white						D					
Immigrant							D				
Non-Aboriginal								D			
Atlantic									D		
Western										D	
Rural area											D

**Note*: 1 means dominance for $\nu = 1$; 1.5 means dominance for $\nu = 1.5$; 2 means dominance for $\nu = 2$; 2.5 means dominance for $\nu = 2.5$; D means dominance for $\nu = 1, 1.5, 2, 2.5$.D also means dominance of the row subgroup over the column subgroup. Finally, ND mean non dominance.

males, employed females, couples, non-aboriginals, immigrants, and rural areas have lower socioeconomic inequality and failure in depression than females, working-age group, unemployed males, unemployed females, non-couples, aboriginals, non-immigrants, and populated areas respectively.

Table 3.17 presents the results for the robust ordering of socioeconomic inequality in mental health in 2015. At $\tilde{I}(2)$ and $\tilde{I}(2.5)$, unemployed females have a higher socioeconomic inequality in mental health than their employed counterparts. Rural areas also dominate populated areas in socioeconomic inequality in mental health at all levels of aversion to socioeconomic inequality. We cannot robustly rank socioeconomic inequality in mental health, among other subpopulations.

When we impose a concave numerical scale, Table 3.18 shows that apart from male and female and age groups, there is robust ordering for all subgroups. When we consider all the levels of aversion to socioeconomic inequality, there is lower socioeconomic inequality in mental health among employed males, employed females, couples, and non-aboriginals

Table 3.17: Robust ordering of socioeconomic inequality in Mental Health in 2015

All numerical scales

	Female	65_+	Unemployed male	Unemployed female	Couple	White	Non-Immigrant	Non-Aboriginal	Atlantic	Western	Populated area	Ontario
Male	ND											
working age		ND										
Employed male			ND									
Employed female				2,2.5								
Non Couple					ND							
Non- white						ND						
Immigrant							ND					
Aboriginal								ND				
Central									ND			
Central										ND		
Rural area											D	
Quebec												ND

**Note*: 1 means dominance for $\nu = 1$; 1.5 means dominance for $\nu = 1.5$; 2 means dominance for $\nu = 2$; 2.5 means dominance for $\nu = 2.5$; D means dominance for $\nu = 1, 1.5, 2, 2.5$. D also means dominance of the row subgroup over the column subgroup. Finally, ND mean non dominance.

than unemployed males, unemployed females, non-couples and aboriginals respectively. For geographic comparison, the Central region has lower socioeconomic inequality in mental health in 2015 than both the Atlantic and western regions. Rural areas also have lower socioeconomic inequalities in mental health than populated areas. Quebec also has less socioeconomic inequality in mental health than Ontario. We also find that, when $\nu = 1$, immigrants have less socioeconomic inequality in mental health than non-immigrants. At $\nu = 2.5$, whites have higher socioeconomic inequality in mental health than non-whites.

For mental health achievement in 2015, in Table 3.19, there is robust ordering for all comparisons among the subgroups except for the comparison between Western and Central, rural areas, and populated areas. Males, working-age group, employed males, employed females, non-couples, non-white, immigrants, aboriginals, Central region and Quebec have higher mental health achievement than females, 65_+ years, unemployed males, unemployed females, couples, whites, non-immigrants, non-aboriginals, the Atlantic region, and Ontario respectively. Imposing a concave scale, 65 years and older have higher mental health

Table 3.18: Robust ordering of socioeconomic inequality in Mental Health in 2015

Concave scales

	Female	65_+	Unemployed male	Unemployed female	Non-couple	White	Non-immigrant	Aboriginal	Atlantic	Western	Populated area	Ontario
Male	ND											
Working age		ND										
Employed male			D									
Employed female				D								
Couple					D							
Non-White						2.5						
Immigrant							1					
Non-Aboriginal								D				
Central									D			
Central										D		
Rural area											D	
Quebec												D

**Note*: 1 means dominance for $\nu = 1$; 1.5 means dominance for $\nu = 1.5$; 2 means dominance for $\nu = 2$; 2.5 means dominance for $\nu = 2.5$; D means dominance for $\nu = 1, 1.5, 2, 2.5$.D also means dominance of the row subgroup over the column subgroup. Finally, ND mean non dominance.

Table 3.19: Robust ordering of Failure in Mental Health in 2015

All numerical scales A(ν)

	Female	65_+	Unemployed male	Unemployed female	couple	White	Non-Immigrant	Non-Aboriginal	Atlantic	Western	Rural area	Ontario
Male	D											
working age		ND										
Employed male			D									
Employed female				D								
Non Couple					D							
Non- white						D						
Immigrant							D					
Aboriginal								D				
Central									D			
Central										D		
Population area											ND	
Quebec												D

**Note*: 1 means dominance for $\nu = 1$; 1.5 means dominance for $\nu = 1.5$; 2 means dominance for $\nu = 2$; 2.5 means dominance for $\nu = 2.5$; D means dominance for $\nu = 1, 1.5, 2, 2.5$. D also means dominance of the row subgroup over the column subgroup. Finally, ND mean non dominance.

achievement than the working-age group, as shown in Table 3.20.

Our mental health results show that employed females and rural areas have lower socioeconomic inequality and higher achievement in mental health than unemployed females and populated areas. For the other subgroups, we can only robustly rank them in achievement in mental health and not socioeconomic inequality in mental health when we use concave numerical scales.

Table 3.21 shows that there is no robust ordering of socioeconomic inequality in health status

Table 3.20: Robust ordering of Failure in Mental Health in 2015

Concave scales A(ν)

	Female	working age	Unemployed male	Unemployed female	Non-couple	White	Non-Immigrant	Aboriginal	Atlantic	Western	Populated area	Ontario
Male	D											
65_+		D										
Employed male			D									
Employed female				D								
Non Couple					D							
Non- white						D						
Immigrant							D					
Aboriginal								D				
Central									D			
Central										D		
Rural area											ND	
Quebec												D

**Note*: 1 means dominance for $\nu = 1$; 1.5 means dominance for $\nu = 1.5$; 2 means dominance for $\nu = 2$; 2.5 means dominance for $\nu = 2.5$; D means dominance for $\nu = 1, 1.5, 2, 2.5$.D also means dominance of the row subgroup over the column subgroup. Finally, ND mean non dominance.

Table 3.21: Robust ordering of socioeconomic inequality in General Health status in 2015

All numerical scales

	Female	Unemployed male	Unemployed female	couple	White	Non-Immigrant	Non-Aboriginal	Atlantic	Western	Populated area	Quebec
Male	ND										
Employed male		ND									
Employed female			ND								
Non Couple				ND							
Non- white					ND						
Immigrant						ND					
Aboriginal							ND				
Central								ND			
Central									ND		
Rural area										ND	
Ontario											ND

**Note*: 1 means dominance for $\nu = 1$; 1.5 means dominance for $\nu = 1.5$; 2 means dominance for $\nu = 2$; 2.5 means dominance for $\nu = 2.5$; D means dominance for $\nu = 1, 1.5, 2, 2.5$. D also means dominance of the row subgroup over the column subgroup. Finally, ND mean non dominance.

among the subgroups in 2015. Imposing a monotonically non-decreasing numerical concave scale, in Table 3.22, we find that unemployed males or females have higher socioeconomic inequality in health status than employed males or females. Couples and non-aboriginals also have lower socioeconomic inequality in health than non-couples and aboriginals, respectively. Similarly, rural areas as well as Quebec, have lower socioeconomic inequalities in health than those in populated areas and Ontario, respectively. We do not obtain robust ordering for the other subgroups.

Table 3.22: Robust ordering of socioeconomic inequality in General Health status in 2015

Concave scales	Female	Unemployed male	Unemployed female	Non-couple	White	Non-Immigrant	Aboriginal	Atlantic	Western	Populated area	Ontario
Male	ND										
Employed male		D									
Employed female			D								
Couple				D							
Non- white					ND						
Immigrant						ND					
Non-Aboriginal							D				
Central								ND			
Central									ND		
Rural area										D	
Quebec											D

**Note*: 1 means dominance for $\nu = 1$; 1.5 means dominance for $\nu = 1.5$; 2 means dominance for $\nu = 2$; 2.5 means dominance for $\nu = 2.5$; D means dominance for $\nu = 1, 1.5, 2, 2.5$.D also means dominance of the row subgroup over the column subgroup. Finally, ND mean non dominance.

Table 3.23: Robust ordering of Achievement in General Health status in 2015

All numerical scales A(ν)	Female	65_+	Unemployed male	Unemployed female	couple	White	Non-Immigrant	Aboriginal	Atlantic	Western	Rural area	Ontario
Male	1.5											
working age		D										
Employed male			D									
Employed female				D								
Non Couple					D							
Non- white						D						
Immigrant							2,2.5					
Non-Aboriginal								D				
Central									D			
Central										ND		
Population area											ND	
Quebec												D

**Note*: 1 means dominance for $\nu = 1$; 1.5 means dominance for $\nu = 1.5$; 2 means dominance for $\nu = 2$; 2.5 means dominance for $\nu = 2.5$; D means dominance for $\nu = 1, 1.5, 2, 2.5$. D also means dominance of the row subgroup over the column subgroup. Finally, ND mean non dominance.

Table 3.23 shows the ranking of health achievement among different subgroups in 2015. employed males, employed females, non-whites, non-aboriginals, Central, Western and Quebec, dominate unemployed males, unemployed females, whites, aboriginals, Atlantic, Atlantic region, and Ontario respectively in health achievement for $\nu = 1, 1.5, 2, 2.5$. Males dominate females in health achievement only when there is some aversion to socioeconomic inequality at 1.5. Also, immigrants have higher achievement in health status when we increase aversion to socioeconomic inequality in health to 2 and 2.5.

Table 3.24: Robust ordering of Achievement in General Health status in 2015

Concave scales	A(ν)											
	Female	working age	Unemployed male	Unemployed female	Non-couple	White	Non-Immigrant	Aboriginal	Atlantic	Western	Population area	Ontario
Male	1, 1.5											
working age		D										
Employed male			D									
Employed female				D								
Non Couple					D							
Non- white						D						
Immigrant							1.5,2,2.5					
Non-Aboriginal								D				
Central									D			
Central										1		
Population area											ND	
Quebec												D

**Note*: 1 means dominance for $\nu = 1$; 1.5 means dominance for $\nu = 1.5$; 2 means dominance for $\nu = 2$; 2.5 means dominance for $\nu = 2.5$; D means dominance for $\nu = 1, 1.5, 2, 2.5$.D also means dominance of the row subgroup over the column subgroup. Finally, ND mean non dominance.

Imposing a non-decreasing concave numerical scale, Table 3.24 shows that we obtain additional ranking for males and females at levels of $\nu = 1$, with males having a higher health achievement than females. Immigrants also dominate non-immigrants in health achievement if aversion to socioeconomic inequality is $\nu = 1.5, 2, 2.5$. The Central region has a higher health achievement than the Western region. It is still not possible to rank the rural and populated areas.

Our results show that while we cannot robustly rank the subgroups in socioeconomic inequality in health, most subgroups can be ranked robustly in health achievement. Imposing a concave scale, the subgroups with lower socioeconomic inequality in health also have higher health achievement.

In general, the ordering for unemployed and unemployed female is robust to the index used and the well-being variable. Employed females have lower socioeconomic inequality in well-being than unemployed females. All other ordering change depending on the well-being variable.

3.6 Conclusion

In this paper, we addressed an important issue of robustly ranking socioeconomic inequality in well-being when the outcome of interest is ordinal. To do so, we follow Allison and Foster (2004), who develop a median-based approach to rank inequality and extend their idea to a multidimensional socioeconomic inequality case for ordinal variables, specifically well-being. Using a median-based index with total social weights, we provide conditions to rank any two distributions with the same median category without imposing a specific numerical scale. However, the method may not provide complete ordering. Nevertheless, a researcher can impose some form of numerical scales, such as non-decreasing concave or convex numerical scale, to allow for further robust ranking of distribution.

Our empirical results using CCHS data support the argument that there are higher socioeconomic inequalities in life satisfaction among females than males. Employed females tend to have lower socioeconomic inequalities in life satisfaction than their unemployed counterparts. Racial immigration and regional differences in socioeconomic inequalities in life satisfaction and other well-being variables also exist. We also observe variations in socioeconomic inequalities in happiness among different demographic groups in Canada.

Appendix A

Appendix for Chapter 1

Table A.1: Difference in leakages in 2009 and 2015 for the entire population. T statistics takes into account the complex survey design

	Population				
		$\hat{F}_{2009,0}$	$\hat{F}_{2015,0}$	$\hat{F}_{2015,0}$-$\hat{F}_{2009,0}$	t -statistics
No medical care due to cost					
	Poor	0.006	0.004	-0.002	-4.946
	Fair	0.018	0.013	-0.005	-6.573
	Good	0.040	0.028	-0.012	-9.774
	Very Good	0.057	0.039	-0.019	-11.563
Delay in medical care due to cost					
	Poor	0.007	0.004	-0.003	-4.944
	Fair	0.022	0.016	-0.006	-7.385
	Good	0.053	0.036	-0.016	-10.886
	Very Good	0.081	0.053	-0.028	-13.862
No or Delay in medical care due to cost					
	Poor	0.007	0.005	-0.002	-4.986
	Fair	0.025	0.018	-0.007	-7.484
	Good	0.059	0.042	-0.018	-11.358
	Very Good	0.091	0.061	-0.03	-14.482

Note: These are cumulative distribution of health and access

Table A.2: Difference in leakages in 2010 and 2015 for the population without age 19-25. T statistics takes into account the complex survey design

	Population				
		$\hat{F}_{2010,0}$	$\hat{F}_{2015,0}$	$\hat{F}_{2015,0}$-$\hat{F}_{2010,0}$	t -statistics
No medical care					
	Poor	0.006	0.004	-0.002	-4.921
	Fair	0.018	0.013	-0.005	-7.124
	Good	0.040	0.025	-0.012	-9.975
	Very Good	0.052	0.035	-0.017	-11.86
Delay in medical care					
	Poor	0.006	0.004	-0.002	-4.377
	Fair	0.021	0.016	-0.006	-6.985
	Good	0.047	0.033	-0.014	-10.299
	Very Good	0.069	0.047	-0.021	-12.491
No or Delay in medical care					
	Poor	0.007	0.005	-0.002	-4.336
	Fair	0.023	0.017	-0.006	-7.179
	Good	0.054	0.038	-0.015	-10.629
	Very Good	0.079	0.055	-0.023	-13.257

Note: These are cumulative distribution of health and access

Table A.3: Sample sizes

	No medical care	**Delay in medical care**	**No or Delay on medical care**
Male	42762	42766	42822
Female	45456	45467	45522
0-17	23786	23796	23823
18-64	54346	54350	54427
65+	10086	10087	10094
White	66444	66458	66529
Black	14368	14360	14390
Asian	6104	6107	6107
Other race	1302	1308	1308
Northeast	14426	14426	14437
Midwest	18023	18029	18069
South	32243	32242	32272
West	23526	23536	23566
Lowest Quantiles	19487	19496	19516
Highest Quantiles	49681	49681	49737
High school or less	34010	34015	34044
Some college Education	52719	52724	52778

Note: These are cumulative distribution of health and access

Appendix B

Appendix for Chapter 2

Table B.1: Magnitude of the effect of recreational legalization of marijuana on BMI using changes in changes without covariates

	Magnitude (1)	Lower bound (2)	Upper bound (3)
Overweight (Between 25 to 50)	-0.0093	0.0008	-0.0194
Obese (Between 30 to 50)	-0.0243	-0.0160	-0.0326
Morbidly obese (Between 35 to 50)	-0.0141	-0.0100	-0.0180
N	350260		

Table B.3: Magnitude of the effect of recreational legalization of marijuana on BMI using changes in changes with covariates

	Magnitude (1)	Lower bound (2)	Upper bound (3)
Overweight (Between 25 to 50)	-0.0362	-0.0148	-0.0575
Obese (Between 30 to 50)	-0.0295	-0.0071	-0.0518
Morbidly obese (Between 35 to 50)	-0.0151	-0.0017	-0.0286
N	350260		

Appendix C

Appendix for Chapter 3

To illustrate the values of the index in Equation(3.3.5), consider the scaling functions $c(h_1), c(h_2), c(h_3)$ on Table 3.2. If we apply a linear scaling function $c_1(h)$, the maximum value of the index would be reached for a population of a very large size such as $N \to \infty$. When 50% of the poorest population minus one person feel "so unhappy that life is not worthwhile", one person feels "somewhat unhappy" and the rest of the population feel "happy in life". Assuming that the value of aversion to socioeconomic inequality, $\nu = 2$, the value of the index in this case is $I(\nu) = 2$ and this the value of the index regardless of the socioeconomic rank of the person in each category. When we use the second numerical scale $c_2(h)$, the maximum value of the index is reached for a population of a very large size and 50% of the poorest population minus one person feel "so unhappy that life is not worthwhile", one person feels "somewhat unhappy" and the rest of the population feel "happy in life", then $I(\nu) = 2.75$. Similarly, when 50% of the poorest population minus one person feel "so unhappy that life is not worthwhile", one person feels "somewhat unhappy" and the rest of the population feel "happy in life", the value of the index is $I(\nu) = 8.5$. We can also restrict the index in the interval $[0, 1]$ by simply using an alternative index which is

$$I(\nu) = \frac{1}{Z(c(h);\nu)} \sum_{i=1}^{N} \omega(r_i;\nu)|c(h_i) - c(h_m)| \tag{C.0.1}$$

Equation (3.3.11) can also be normalized to lie between the values of 0 and 1 as shown in Equation (C.0.2).

$$\tilde{I}(\nu) = 0.5 \frac{1}{\tilde{Z}^{-}(c(h);\nu)} I^{-}(\nu) + 0.5 \frac{1}{\tilde{Z}^{+}(c(h);\nu)} I^{+}(\nu) \tag{C.0.2}$$

C.0.1 Proof of Theorem 3

Proof. **Proof.** Equation (3.3.11) can be rewritten as

$$\widetilde{I}(\nu) = 0.5 \sum_{k=1}^{m-1} \sum_{i \in p_k} \omega(r_i;\nu)(c(h_m) - c(h_k)) + 0.5 \sum_{k=m+1}^{K} \sum_{i \in p_k} \widetilde{\omega}(r_i;\nu)(c(h_k) - c(h_m)). \tag{C.0.3}$$

Decomposing the two terms on the right hand side of equation (C.0.3) yields:

$$\widetilde{I}(\nu) = 0.5 \sum_{k=1}^{m-1} (c(h_{k+1}) - c(h_k)) \sum_{j=1}^{k} \sum_{i \in p_k} \omega(r_i;\nu) + 0.5 \sum_{k=m+1}^{K} (c(h_k) - c(h_{k-1})) \sum_{j=k}^{K} \sum_{i \in p_k} \widetilde{\omega}(r_i;\nu). \tag{C.0.4}$$

Replacing $\Psi^L(k;\nu) = \sum_{j=1}^{k}\sum_{i\in p_k}\omega(r_i;\nu)$, $\Psi^R(k;\nu) = \sum_{j=k}^{K}\sum_{i\in p_k}\widetilde{\omega}(r_i;\nu)$ and $\Delta^1 c(h_k) = c(h_{k+1}) - c(h_k)$ in equation (C.0.4), yields:

$$\widetilde{I}(\nu) = 0.5\sum_{k=1}^{m-1}\Psi^L(k;\nu)\Delta^1 c(h_k) + 0.5\sum_{k=m+1}^{K}\Psi^R(k;\nu)\Delta^1 c(h_{k-1}). \tag{C.0.5}$$

Using equation (C.0.5), we get

$$\widetilde{I}_1(\nu) - \widetilde{I}_0(\nu) = 0.5\sum_{k=1}^{m-1}\left[\Psi_1^L(k;\nu) - \Psi_0^L(k;\nu)\right]\Delta^1 c(h_k) + 0.5\sum_{k=m+1}^{K}\left[\Psi_1^R(k;\nu) - \Psi_0^R(k;\nu)\right]\Delta^1 c(h_{k-1}). \tag{C.0.6}$$

Since $\Delta^1 c(h_k) \geq 0$. This implies that if $\Psi_1^L(k;\nu) \geq \Psi_0^L(k;\nu)$ for all $k \in \{1, 2, \ldots, m-1\}$ and $\Psi_1^R(k;\nu) \geq \Psi_0^R(k;\nu)$ for all $k \in \{m+1, m+2, \ldots, K\}$, then the expression in equation (C.0.6) is non positive, i.e. $\widetilde{I}_0(\nu) \geq \widetilde{I}_1(\nu)$. This proves sufficiency of the condition.

In order to establish necessity, let us consider a particular scaling function that has the following properties:

1. $c(h_1) = c(h_2) = \cdots = c(h_{k^*})$
2. $\Delta^1 c(h_{k^*}) > 0$
3. $c(h_{k^*+1}) = c(h_{k^*+2}) = \cdots = c(h_{K-1})$

Imagine now that $\Psi_0^\lambda(k;\nu) \geq \Psi_1^\lambda(k;\nu)$, $\lambda = L$ or R for all k excepts for k^* for which we have $\Psi_0^\lambda(k^*;\nu) < \Psi_1^\lambda(k^*;\nu)$. For any scaling function having the properties described in the proof of Theorem 3, $\widetilde{I}_0(\nu) < \widetilde{I}_1(\nu)$. Hence it cannot be that $\Psi_0^\lambda(k^*;\nu) < \Psi_1^\lambda(k^*;\nu)$ for any $k \in \{1, \ldots, m-1, m+1, \ldots, K\}$. This proves the necessity of the condition. □

C.0.2 Proof of Theorem 4

Proof. **Proof.** One decomposition of equation (C.0.5) yields:

$$\begin{aligned}\widetilde{I}(\nu) &= 0.5\Delta^1 c(h_{K-1})\left[\sum_{k=1}^{m-1}\Psi^L(k;\nu)+\sum_{k=m+1}^{K}\Psi^R(k;\nu)\right] \\ &+0.5\sum_{k=m+1}^{K}\left[\Delta^1 c(h_k)-\Delta^1 c(h_{k-1})\right]\left[\sum_{j=1}^{m-1}\Psi^L(j;\nu)+\sum_{j=m+1}^{k}\Psi^R(j;\nu)\right] \\ &+0.5\sum_{k=1}^{m-1}\left[\Delta^1 c(h_{k+1})-\Delta^1 c(h_k)\right]\sum_{j=1}^{k}\Psi^L(j;\nu) \qquad \text{(C.0.7)}\end{aligned}$$

Replacing $\Delta^2 c(h_k) = \Delta^1 c(h_{k+1}) - \Delta^1 c(h_k)$ and replacing by $\Psi^{2+}(k;\nu)$ as described in equation (3.3.12) the values in equation (C.0.7), yields to:

$$\widetilde{I}(\nu) = 0.5\Delta^1 c(h_{K-1})\Psi^{2+}(K;\nu) - 0.5\sum_{k=m+1}^{K}\Psi^{2+}(k;\nu)\Delta^2 c(h_{k-1}) - 0.5\sum_{k=1}^{m-1}\Psi^{2+}(k;\nu)\Delta^2 c(h_k) \qquad \text{(C.0.8)}$$

Using equation (C.0.8), we get

$$\begin{aligned}\widetilde{I}_1(\nu) - I_0(\nu) &= 0.5\Delta^1 c(h_{K-1})\left[\Psi_1^{2+}(K;\nu)-\Psi_0^{2+}(K;\nu)\right] \\ &+0.5\sum_{k=m+1}^{K}\left[\Psi_0^{2+}(k;\nu)-\Psi_1^{2+}(k;\nu)\right]\Delta^2 c(h_{k-1}) \\ &+0.5\sum_{k=1}^{m-1}\left[\Psi_0^{2+}(k;\nu)-\Psi_1^{2+}(k;\nu)\right]\Delta^2 c(h_k) \qquad \text{(C.0.9)}\end{aligned}$$

If we consider only concave scaling functions to represent the self-reported health status variable, then $\Delta^2 c(h_k) \leq 0$ in addition to $\Delta^1 c(h_k) \geq 0$. This implies that if $\Psi_0^{2+}(k;\nu) \geq \Psi_1^{2+}(k;\nu)$ for all $k \in \{1, 2, \ldots, m-1, m+1, \ldots, K\}$, then the expression in equation (C.0.9) is non positive, i.e. $\widetilde{I}_1(\nu) \leq \widetilde{I}_0(\nu)$. This proves sufficiency of the condition.

In order to establish necessity, we have to proceed in two steps. First consider a linear scaling function. In this case, $\Delta^2 c(h_k) = 0$ for all k. Imagine now that $\Psi_0^{2+}(k;\nu) \geq \Psi_1^{2+}(k;\nu)$ for all $k \in \{1, 2, \ldots, m-1, m+1, \ldots, K-1\}$ and that for K, we have $\Psi_0^{2+}(K;\nu) < \Psi_1^{2+}(K;\nu)$. For these linear scaling functions, $\widetilde{I}_1(\nu) > \widetilde{I}_0(\nu)$. Hence it cannot be that $\Psi_0^{2+}(K;\nu) < \Psi_1^{2+}(K;\nu)$. Let us now consider a particular scaling function that has the following properties:

1. $\Delta^1 c(h_1) = \Delta^1 c(h_2) = \cdots = \Delta^1 c(h_{k^*}) > 0$

2. $\Delta^1 c(h_{k^*+1}) = \Delta^1 c(h_{k^*+2}) = \cdots = \Delta^1 c(h_{K-1}) = 0$

Imagine now that $\Psi_0^{2+}(k;\nu) \geq \Psi_1^{2+}(k;\nu)$ for all k excepts for k^* for which we have $\Psi_0^{2+}(k^*;\nu) < \Psi_1^{2+}(k^*;\nu)$. For any scaling function having the above mentioned properties, $\widetilde{I}_1(\nu) > \widetilde{I}_0(\nu)$. Hence it cannot be that $\Psi_0^1(k;\nu) < \Psi_1^1(k;\nu)$ for any $k \in \{1, 2, \ldots, m-1, m+1, \ldots, K\}$. This proves the necessity of the condition. □

C.0.3 Proof of Theorem 5

Proof. **Proof.** One decomposition of equation (C.0.5) yields:

$$\begin{aligned}
\widetilde{I}(\nu) &= 0.5\Delta^1 c(h_1) \left[\sum_{k=1}^{m-1} \Psi^L(k;\nu) + \sum_{k=m+1}^{K} \Psi^R(k;\nu)\right] \\
&\quad +0.5 \sum_{k=2}^{m-1} \left[\Delta^1 c(h_k) - \Delta^1 c(h_{k-1})\right] \left[\sum_{j=k}^{m-1} \Psi^L(j;\nu) + \sum_{j=m+1}^{K} \Psi^R(j;\nu)\right] \\
&\quad +0.5 \sum_{k=m+1}^{K} \left[\Delta^1 c(h_{k-1}) - \Delta^1 c_(h_{k-2})\right] \sum_{j=k}^{K} \Psi^R(j;\nu) \qquad \text{(C.0.10)}
\end{aligned}$$

Replacing $\Delta^2 c(h_k) = \Delta^1 c(h_{k+1}) - \Delta^1 c(h_k)$ and replacing by $\Psi^{2-}(k;\nu)$ as described in equation (3.3.13) the values in equation (C.0.10), yields to:

$$\widetilde{I}(\nu) = 0.5\Delta^1 c(h_1)\Psi^{2-}(1;\nu) + 0.5\sum_{k=2}^{m-1}\Psi^{2-}(k;\nu)\Delta^2 c(h_{k-1}) + 0.5\sum_{k=m+1}^{K}\Psi^{2-}(k;\nu)\Delta^2 c(h_{k-2}) \quad \text{(C.0.11)}$$

Using equation (C.0.11), we get

$$\begin{aligned}\widetilde{I}_1(\nu) - \widetilde{I}_0(\nu) &= 0.5\Delta^1 c(h_1)\left[\Psi_1^{2-}(1;\nu) - \Psi_0^{2-}(1;\nu)\right] \\ &+0.5\sum_{k=2}^{m-1}\left[\Psi_1^{2-}(k;\nu) - \Psi_0^{2-}(k;\nu)\right]\Delta^2 c(h_{k-1}) \\ &+0.5\sum_{k=m+1}^{K}\left[\Psi_1^{2-}(k;\nu) - \Psi_0^{2-}(k;\nu)\right]\Delta^2 c(h_{k-2}) \qquad \text{(C.0.12)}\end{aligned}$$

If we consider only convex scaling functions to represent the self-reported health status variable, then $\Delta^2 c(h_k) \geq 0$ in addition to $\Delta^1 c(h_k) \geq 0$. This implies that if $\Psi_0^{2-}(k;\nu) \geq \Psi_1^{2-}(k;\nu)$ for all $k \in \{1, 2, \ldots, m-1, m+1, \ldots, K-1\}$, then the expression in equation (C.0.12) is non positive, i.e. $\widetilde{I}_1(\nu) \leq \widetilde{I}_0(\nu)$. This proves sufficiency of the condition.

In order to establish necessity, we have to proceed in two steps. First consider a linear scaling function. In this case, $\Delta^2 c(h_k) = 0$ for all k. Imagine now that $\Psi_0^{2-}(k;\nu) \geq \Psi_1^{2-}(k;\nu)$ for all $k \in \{2, \ldots, m-1, m+1, \ldots, K\}$ and that for $k = 1$, we have $\Psi_0^{2-}(1;\nu) < \Psi_1^{2-}(1;\nu)$. For these linear scaling functions, $\widetilde{I}_1(\nu) > \widetilde{I}_0(\nu)$. Hence it cannot be that $\Psi_0^{2-}(1;\nu) < \Psi_1^{2-}(1;\nu)$. Let us now consider a particular scaling function that has the following properties:

1. $\Delta^1 c(h_1) = \Delta^1 c(h_2) = \cdots = \Delta^1 c(h_{k^*}) = 0$

2. $\Delta^1 c(h_{k^*+1}) = \Delta^1 c(h_{k^*+2}) = \cdots = \Delta^1 c(h_{K-1}) > 0$

Imagine now that $\Psi_0^{2-}(k;\nu) \geq \Psi_1^{2-}(k;\nu)$ for all k excepts for k^* for which we have $\Psi_0^{2-}(k^*;\nu) < \Psi_1^{2-}(k^*;\nu)$. For any scaling function having the above mentioned properties, $\widetilde{I}_1(\nu) > \widetilde{I}_0(\nu)$. Hence it cannot be that $\Psi_0^1(k;\nu) < \Psi_1^1(k;\nu)$ for any $k \in \{1, 2, \ldots, m-1, m+1, \ldots, K-1\}$. This proves the necessity of the condition. $\square$

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