

ESSAYS ON THE DETERMINANTS AND IMPLICATIONS OF
ACCESS TO HEALTH CARE

by

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ABSTRACT

An understanding of both the determinants of health care access and the implications of that access is of crucial importance because it enables us to learn about policies and institutions that are welfare enhancing in health outcomes. The first chapter of my dissertation explores how access to sanitarium aided in tuberculosis control in the time before antibiotics. Results indicate that access to an additional sanitarium bed reduced the death rate from tuberculosis for white residents by nearly .695 per 100,000 and had no impact for black residents. The next two chapters investigate the retail health clinic. First, I construct a choice model of clinic location that accounts for both demand and competition effects. I find that clinics are more likely to locate in areas that are populous, wealthy, educated, and white, and that they are less likely to locate in traditionally under-served communities. Second, I combine the results of my predictive model with data on ED visits to determine if clinics help direct patients away from receiving treatment at expensive emergency rooms. I find that access to retail clinics causes a substantial decrease in the number of ED visits for bronchitis and upper respiratory infections. The savings associated with retail clinic induced ED diversion is conservatively estimated to be at least \$88 million in 2012 alone. In California, counterfactual analysis suggests that relaxing the barriers to clinic entry would result in \$10.5 million in annual health care savings.

CHAPTER 1

CONTROLLING TB IN A WORLD WITHOUT ANTIBIOTICS: ISOLATION
AND EDUCATION IN NORTH CAROLINA, 1932-1940

Antimicrobial resistant strains of bacteria are becoming more prevalent both in United States and around the world. According to the Center for Disease Control, every year over 2 million Americans become infected with a strain of drug resistant bacteria (CDC, 2013a). This proliferation of antibiotic resistance coupled with the fact that there has been little recent research and development in field has led many, including Dr. Srinivasan (2013), an associate director at the CDC, to claim that we are now “in the post-antibiotic era.” As our pharmacological weapons against disease become less effective, it is imperative to understand effective mechanisms for controlling the spread of disease in a world without antibiotics.

Roughly one third of the world’s population is currently infected with tuberculosis. In 2012 there were approximately 1,300,000 deaths from tuberculosis worldwide (WHO, 2014). Once thought to be a disease of the past, tuberculosis is making a worldwide resurgence. This recent increase can be attributed to a rising number of immunocompromised individuals due to the HIV/AIDS epidemic as well as the advent of drug resistant tuberculosis (Coker, 2000). Drug resistant tuberculosis is generally classified as either multi-drug resistant (MDR) or extensively drug-resistant (XDR), however recent research by Klopper et al. (2013) in South Africa suggests that strains of totally drug resistant (TDR) tuberculosis are emerging. Drug resistant strains of TB most commonly arise through improper use of the very medicine meant to cure the disease. The disease can buildup immunity from either an interruption of treatment due to a patient skipping doses or from a doctor prescribing similar drug regimens over and over again to a single patient, who has been previously unresponsive to a given drug. While in the United States, the number of reported MDR TB cases is quite low, in India, China, and Eastern

Europe drug-resistant TB is at epidemic proportions (McKay, 2013b).

Under 100 years ago tuberculosis was one of the leading causes of death in the United States and there were no effective drugs with which we were able to combat the disease. Then in the middle of the 20th century miracle drugs were discovered that effectively eliminated the threat of death within one generation (Smith, 1988). However, death rates from tuberculosis were declining prior to the existence of these drugs. Between 1900 and 1940 death rates from tuberculosis dropped from 194.4 per 100,000 to 45.9 per 100,000 (CDC, 2012). There have been multiple explanations offered as drivers for the pre-antibiotic decline. Many scholars attribute this decline to rising standards of living. Others claim that sending infected patients to sanatoria decreased tuberculosis rates by healing those who were infected, by effectively quarantining the infectious away from susceptible members of the population, and by educating the public in hygienic practices (Dheda and Migliori, 2012; Roberts, 2009). Recently, epidemiologists have suggested that transmission dynamics unique to tuberculosis may have resulted in a natural period of incidence decline (Blower et al., 1995).

In the late 1980s a strain of MDR tuberculosis led to an outbreak in New York City that killed 90% of those contracted the strain (Barbanel, 1991). Fears of similar outbreaks due to the recent rise drug resistant tuberculosis highlight the importance of understanding effective policies of isolation and education. These concerns have also caused some medical professionals to call for a return of the sanitarium as Dheda and Migliori did in their 2012 *Lancet* article. However these calls may be premature as there have not yet been any quantitative studies that have estimated the impact of sanatoria on deaths from tuberculosis. This paper attempts to fill this gap. More specifically, using information from North Carolina between 1932 and 1940, this paper will show that access to an additional sanitarium bed reduced the resident pulmonary tuberculosis death rate for whites but had no impact for blacks. These results are then interpreted using an SLIRQ model of tuberculosis transmission that allows for the inference that education was a crucial mechanism through which sanatoria were able to reduce the TB death rate.

1.1 Historical Background

Tuberculosis is caused by *Mycobacterium tuberculosis*. It is a pathogenic infectious agent that invades a host and causes disease. Tuberculosis is an ancient disease and it has plagued man for centuries.¹ There have been traces of tuberculosis found in Neolithic skeletons and in Egyptian mummies (Teller, 1988). Before mankind began to live in large cities tuberculosis survived through mainly through infection from animals. As human population density increased, infections of tuberculosis increased as well. Thus in the early 1600s major tuberculosis epidemics began in Europe and as people migrated to the New World, they brought the infectious disease with them (Blower et al., 1995). Tuberculosis quickly spread throughout the country.² By 1902 tuberculosis was the leading cause of death in the United States at a rate of 174.2 per 100,000. Over the next forty years the death rates gradually dropped and by 1940 rates were only 45.9 per 100,000 (CDC, 2012).

In 1854 Hermann Brehmer, a German physician “asserted that he could cure tuberculosis with a regimen of fresh air, exercise and good nutrition in a sanatorium” (Warren, 2006). In 1872 Edward Trudeau, himself a physician, was diagnosed with pulmonary tuberculosis. Familiar with Brehmer’s open-air hypothesis, he decided to spend the remainder of his life living in the wilderness of the Adirondack Mountains in upstate New York. Trudeau successfully combated the disease and eventually found himself cured, leaving him convinced of the curative powers of the open-air. By 1884 Trudeau opened his own sanitarium in the Adirondacks at Saranac Lake. This was one of the first sanatoria to be opened in the United States. Trudeau’s sanitarium served as model that most other sanatoria would copy as they

¹Tuberculosis is also known as TB, consumption, phthisis, scrofula, and wasting. This paper will use either TB or tuberculosis when referring to the disease. The primary form of tuberculosis is pulmonary tuberculosis, however it is possible to have a tuberculosis infection of almost any body part. Throughout this paper when tuberculosis is mentioned it will be referring to pulmonary tuberculosis unless otherwise stated.

²Pepperell et al. (2011) found that while the indigenous population in Canada contracted tuberculosis from European fur traders starting in 1710, it wasn’t until the late 1800s that epidemics of tuberculosis began. They attribute this 150 year delay to the 19th century creation of reservations, which the authors claim fostered ideal conditions for a more rapid transmission of the disease

began to open across America. The key features of a sanitarium were fresh air, climate, diet and rest. Climate was considered to be a crucial factor in selecting a location for a sanitarium. Both fresh, open air and altitude were considered to be desirable attributes for a location. Altitude was an important enough characteristic that in the *Classified Sanitarium Directory of Eastern United States*, it was listed as the first key attribute for each sanitarium (Harrington, 1906). Echoing this belief, in the late 19th publication, *Cases of Phthisis Treated at High Altitudes*, C. Theodore Williams, a physician, described the physiological influence of a high altitude mountain climate “as intensely stimulating. . . appetite increases, digestion and assimilation improves...The effect on the lungs is chiefly the result of the rarified air (Williams, 1879).³” The thought of even a remotely effective treatment for tuberculosis enticed many to open sanitarium and began the sanitarium movement. This movement reached its peak in the mid-1940s with over 100,000 sanitarium beds in the US (Roberts, 2009).

In 1882 Robert Koch discovered that tuberculosis was caused by a bacterium, which was aptly named *Mycobacterium tuberculosis*. This discovery assisted in convincing a skeptical public that tuberculosis was in fact contagious and that public health measures should be undertaken in order to help combat the disease. However the discovery of tuberculosis’ cause did not lead to a quick discovery of a cure. It was not until well into the peak of the sanitarium movement in the late 1940s that the advent of antibiotics and chemotherapeutic drugs promised an effective solution for the disease.⁴ In the 1950s it was discovered that no single drug was successful in combating tuberculosis in all patients and that a regimen of multiple drugs is necessary for effective control. As a result by the 1970s tuberculosis was effectively

³While the belief of the protective effects of altitude was popular in the late 19th and early 20th centuries, this belief is not widely held today. The majority of the few studies attempting to look at the impact of altitude on tuberculosis disease mortality have ignored confounding factors such as access to health care facilities, population density and income. Saito et al. (2006) found that when controlling for confounding factors there is no protective effect of high altitude on tuberculosis infections in urban communities.

⁴While a vaccine, Bacillus Calmette-Guérin, (BCG) for tuberculosis was synthesized in the 1920s, it was never utilized in the United States as it never federal received approval. It is not extremely effective and it renders simple skin testing for tuberculosis impossible.

eliminated in the United States (Coker, 2000). The elimination of tuberculosis also eliminated the need for the sanitarium and by the late 1980s almost every sanitarium in the US had either closed or switched its primary type of service.

1.2 Theoretical Model of TB Transmission

1.2.1 SLIR Model of Tuberculosis Transmission

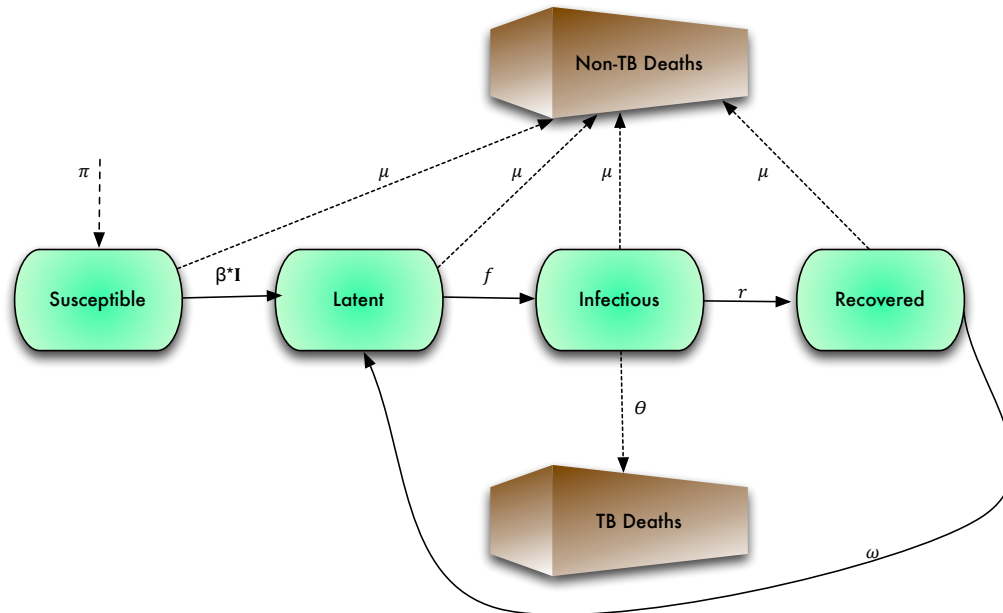
Tuberculosis moves through a population in a nonlinear manner. Individuals are born into a population and are immediately susceptible to contracting the disease. Susceptible members of the population are infected with tuberculosis by breathing in the live bacterium, *Mycobacterium tuberculosis*. As the bacterium tends to locate itself in the lungs of its host, it is spread by infectious individuals primarily through being coughed into the air.⁵ Upon becoming infected with the bacterium, immunocompetent individuals will fight the disease and often succeed in walling it off in the lungs with a layer of white blood cells. Most individuals will be able to successfully combat the disease into a state of dormancy. 90% of individuals who are infected with tuberculosis will remain in this latent state, where they will never become infectious and the disease never activates.⁶ If a latently infected individual becomes immunocompromised, the bacterium may be released from the protective white blood cell layer resulting in endogenous reactivation. 10% of those who are latently infected with tuberculosis will endogenously reactivate the disease during their lifetime. This reactivation can occur at anytime and is the primary way through which individuals become infectious.⁷ While 50% of infectious individuals will die within 5 years if untreated, there is roughly a 25% chance that the

⁵While cross-species infection from animals is possible, this is not considered to be a significant source of infection in the following model. One such example is *Mycobacterium bovis*, which can be spread from cows to humans. For a more detailed explanation bovine tuberculosis and efforts to eradicate it, see Olmstead and Rhode (2004).

⁶These parameters and those throughout the paper are adapted from Blower et al. (1995) unless otherwise noted.

⁷Being infected with tuberculosis may mean that an individual is either latently infected or that that the disease has activated and the individual is infectious. The difference between infectious and infected is important in the exposition of the transmission model.

Figure 1.1: SLIR Model of Tuberculosis Transmission



disease will be naturally cured within 5 years. Of those who have recovered from tuberculosis there is a 12% chance of relapse back into infectiousness.

The first model assumes that there is no treatment attempt and seeks to estimate how the disease will move through a population given its natural transmission dynamic.⁸ This is a deterministic model of four differential equations. At any point a given individual can be in one of four living states, Susceptible (S), Latent (L), Infectious (I) or Recovered (R), hence a SLIR model. A visualization of this transmission model can be seen in Figure 1.1. The set of differential equations characterizing the four living states are Equations 1.1 through 1.4.

⁸More complex models of tuberculosis transmission exist, but this model serves as a generalization of these others (see Dye et al., 1998; Blower et al., 1995; Porco and Blower, 1998).

Table 1.1: Parameters Used in SLIR Model

Parameter	Description	Value
β	Transmission Coefficient	.00005
f	Rate of Endogenous Reactivation	.00256
r	Natural Cure Rate	.058
ω	Relapse Rate	.01
μ	Death Rate from Other Causes	.0222
θ	Death Rate from Tuberculosis	.139

$$\frac{dS}{dt} = \pi + (1 - \beta I - \mu)S \quad (1.1)$$

$$\frac{dL}{dt} = \beta IS + \omega R + (1 - f - \mu)L \quad (1.2)$$

$$\frac{dI}{dt} = fL + (1 - r - \mu - \theta)I \quad (1.3)$$

$$\frac{dR}{dt} = rI + (1 - \omega - \mu)R \quad (1.4)$$

Individuals enter into the susceptible population (S) at a constant rate, π .⁹ π is the only vein through which entry to the model is possible and represents both births and immigration into the otherwise closed population.¹⁰ Exit from the population is possibly only through death, either from tuberculosis or another cause. Infectious individuals are the only subset that is capable of dying from tuberculosis and they die at rate θ . All other individuals die at rate μ . βI represents the rate at which susceptible individuals are infected. β is the per capita rate at which two individual come into effective contact with one another in a given year. Vynnycky and White (2010) define effective contact as contact “that is sufficient to lead to transmission between a susceptible and an infectious person.” Thus individuals enter the population and are subsequently infected with tuberculosis at varying rate

⁹The assumption that π is a fixed number is utilized for simplicity. This can be relaxed such that π is a function of the current population without changing the key results of the SLIR model.

¹⁰Immigrants in this model are considered to be uninfected with tuberculosis. Changing the model to incorporate immigrants who are either L , I or R would not alter the key results.

βI and die at a rate μ . Endogenous reactivation (becoming infectious) occurs at the constant rate f . Natural recovery then occurs at rate r and those who have recovered are reinfected at a constant rate ω . Ultimately we will use this framework, combined with the parameter estimates from Table 1.1, to consider the pathways, including isolation and education, through which sanatoria could reduce tuberculosis deaths.

1.2.2 The Basic Reproductive Number

The basic reproductive number, R_0 , is the expected number of secondary infections caused by a single infectious individual, introduced into a susceptible population. The R_0 of a disease is informative because it reveals basic transmission dynamics of the disease. If $R_0 > 1$, a disease will spread throughout a population creating an epidemic, but when $R_0 < 1$ the disease will simply die out. Calculating R_0 as an analytic expression or a function of just its parameters can illuminate particular aspects of transmission dynamics. This is often helpful in deciding how to most efficiently and effectively combat a disease. One method to calculate the R_0 of a disease is the next generation method. This method involves the creation of a next generation matrix, G , in which the element g_{ij} is the expected number of secondary infections of type i caused by a single infected individual of type j .¹¹ The R_0 of the disease is the spectral radius of this next generation matrix, G . Using this approach, an analytic result for the R_0 of the SLIR model outlined by Figure 1.1 and Equations 1.1 through 1.4 can be calculated. Using the parameter estimates from Table 1.1, the R_0 as defined in Equation 1.5 is 4.7144. This means that conditional on our parameter estimates, a single infectious individual will cause approximately 4.7 secondary infections when introduced into a population of susceptibles.

$$R_0 = \frac{\beta f \pi (\mu + \omega)}{\mu (r \mu^2 + f \mu^2 + \mu^2 \theta + \mu^2 \omega + \mu^3 + r f \mu + r \mu \omega + f \mu \theta + f \mu \omega + f \theta \omega + \mu \theta \omega)} \quad (1.5)$$

¹¹For an expanded discussion of the next generation method, see Heffernan et al. (2005)

1.2.3 Introducing Quarantine to the SLIR Model

Extending the simple SLIR framework to allow for quarantining of infectious individuals will provide us with a theoretical framework with which we can understand why the limited quarantine of the infectious is not effective policy against tuberculosis without also using other types of intervention. Figure 1.2 represents this SLIRQ model, which allows for a fixed percentage of infectious individuals, q , to be quarantined. Those who are quarantined are assumed to be completely isolated from the susceptible population, thus reducing the probability of infecting susceptible individuals through reducing I . Quarantined individuals are supposed to die at rate σ , which is less than or equal to θ . Quarantined individuals recover from tuberculosis at rate ρ , which is greater or equal to r . The construction of the tuberculosis death rate and recovery rate of quarantined individuals in this manner allows for there to be a positive impact of being isolated in a facility (i.e. a sanitarium) on combating tuberculosis. All other parameters are assumed to be identical to the SLIR model. The set of differential equations that represents this SLIRQ model are listed in the Equations 1.6 through 1.10. The R_0 for the SLIRQ model is seen in Equation 1.11.

$$\frac{dS}{dt} = \pi + (1 - \beta I - \mu)S \quad (1.6)$$

$$\frac{dL}{dt} = \beta IS + \omega R + (1 - f - \mu)L \quad (1.7)$$

$$\frac{dI}{dt} = fL + (1 - r - \mu - \theta - q)I \quad (1.8)$$

$$\frac{dR}{dt} = rI + (1 - \omega - \mu)R + \rho Q \quad (1.9)$$

$$\frac{dQ}{dt} = qI + (1 - \sigma - \rho - \mu)Q \quad (1.10)$$

$$R_0 = \frac{\beta f \pi (\mu + \omega) (r + \mu + \theta)}{\mu (r + \mu + q + \theta) (r \mu^2 + f \mu^2 + \mu^2 \theta + \mu^2 \omega + \mu^3 + r f \mu + r \mu \omega + f \mu \theta + f \mu \omega + f \theta \omega + \mu \theta \omega)} \quad (1.11)$$

Figure 1.2: SLIRQ Model of Tuberculosis Transmission

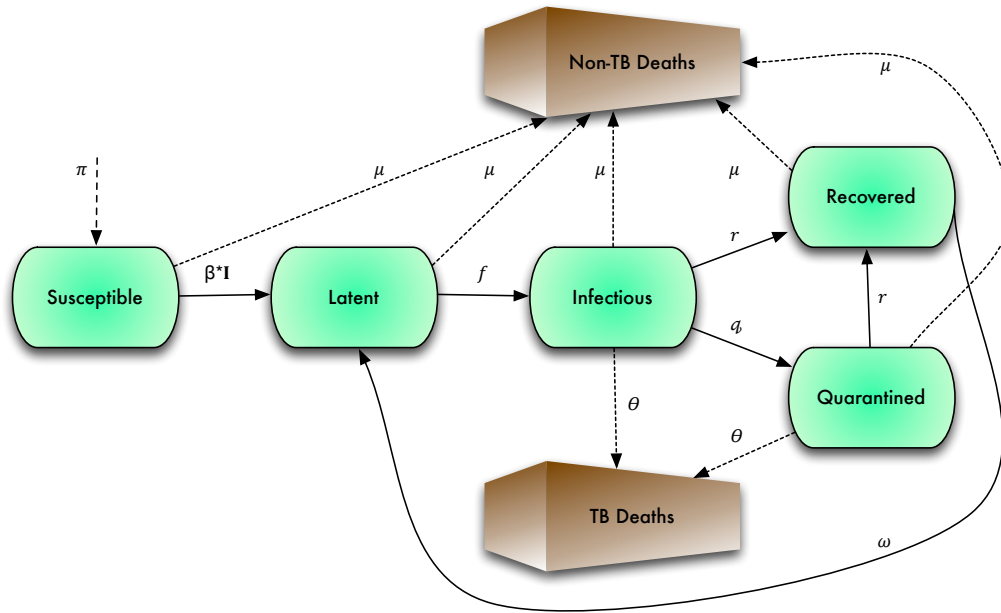
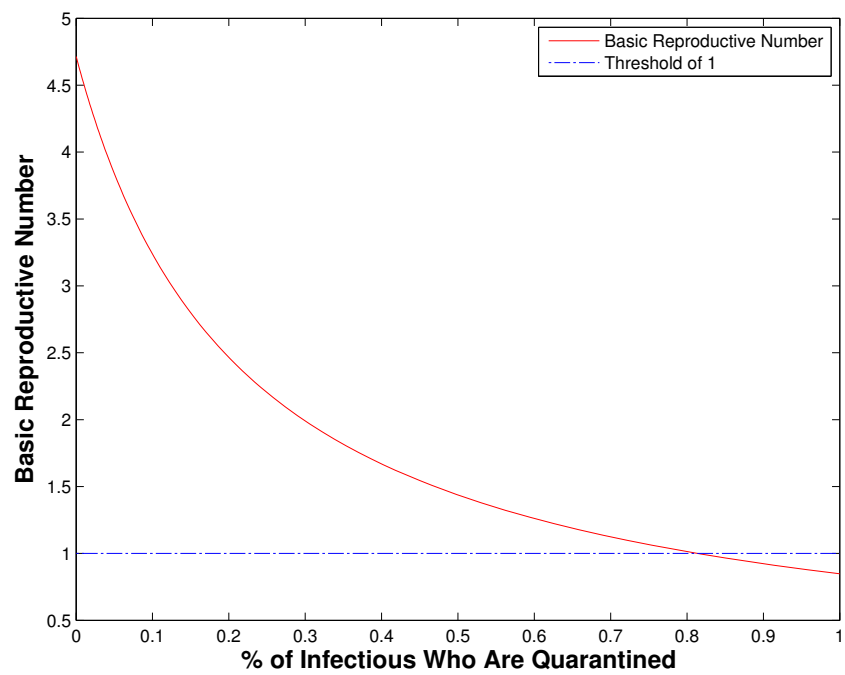


Figure 1.3: Relationship Between Basic Reproductive Number and Quarantine



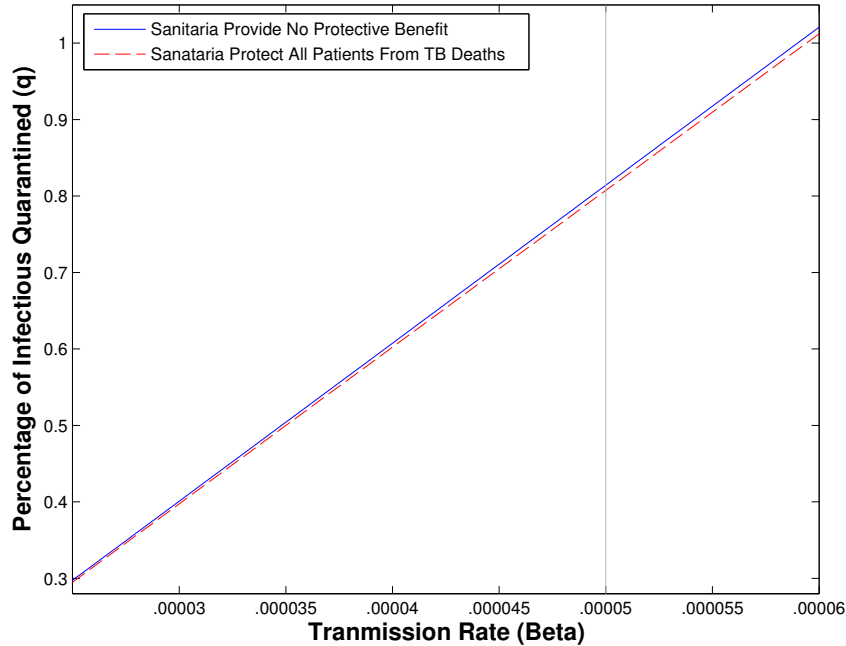
When quarantine is not considered to have a protective benefit, the death rate of quarantined infectious individuals from tuberculosis, σ , is set equal to the death rate of non-quarantined individuals from tuberculosis, θ . Similarly the recovery rate from tuberculosis for the isolated, ρ , is set equal to the recovery rate of the non-isolated infectious, r . Using parameter estimates from Table 1.1, the image of R_0 can be calculated for each $q \in [0, 1]$. When $q = 0$, R_0 is 4.7144 as in Section 1.2.2 and as q increases R_0 decreases in a non-linear manner. A visual representation of this is seen in Figure 1.3. To push R_0 below the threshold level of 1, q needs to be just over 81%. This means that given our parameter choices, over 80% of infectious individuals need to be isolated from the susceptible population in order to foster the conditions necessary for the disease to die out.

When quarantine has a protective effect (i.e. $\sigma < \theta$ and $\rho > r$), reductions in R_0 are primarily driven by increases in q and by decreases in β , the probability of transmission. Extreme values of σ and ρ can be chosen with little impact on the R_0 . Figure 1.4 illustrates this by showing the minimum q and β combinations required to push R_0 to 1. The solid line represents a world where sanitarium have no protective benefit (as in Figure 1.3), while the dashed line represents the extreme case where sanitarium are maximally beneficial for society. These results demonstrate that in order for a policy of isolating infectious individuals to result in a significant decrease in the number of tuberculosis infectious, a large percentage of infectious individuals will need to be quarantined no matter how beneficial sanitarium are for patients. It is important to note that these results are derived from a simple framework and should not be considered predictive. Rather they provide us with an idea of what types of policies will be effective at reducing tuberculosis infections when fundamental transmission dynamics are accounted for.

1.3 How Could Sanitarium Alleviate TB?

There exist two broad pathways through which sanitarium could reduce deaths from tuberculosis. Any reduction in TB deaths that a sanitarium could have on patients

Figure 1.4: Minimum Combinations of % Quarantined and Transmission Rate Such that R_0 is 1 or less



housed within the sanitarium occurs through the direct channel. The primary factors included in the direct channel are reducing the tuberculosis death rate of those in sanatoria (such that $\sigma < \theta$) and increasing the “natural” recovery rate (such that $\rho > r$). The indirect channel encompasses all TB death reductions of the population *not* housed in sanatoria, but still caused by sanatoria.

1.3.1 Direct Channel

While many sanatoria in early 20th century America claimed that those who were treated at a sanitarium died at a lower rate, their claims are generally unsupported and unverifiable. As the key features of sanitarium life were climate, a healthful diet, and rest, any direct impact of sanatoria on tuberculosis would be most likely through these channels. However, if there was any direct benefit of sanatoria it was not through the protective effects of altitude. Saito et al. (2006) found that altitude does not reduce tuberculosis rates in urban areas after controlling for confounding effects. This finding leaves diet and rest as the only two possible direct channels.

While there was variation in the diet among patients in different facilities, there were some unifying features. Most diets were rich in milk and eggs and had a high caloric content, between 2,140 and 4,380 per day (Teller, 1988). While Ramakrishnan et al. (1961), found no impact of a sanitarium diet in patients taking antibiotic and chemotherapeutic treatments for tuberculosis, no similar study exists for the pre-antibiotic era. It is not improbable that a rich diet and increase in bed rest would help many combat a disease more successfully, however as outlined in Figure 1.4, even if tuberculosis death rates at sanatoria decreased substantially and recovery rates increased, the impact on the number of new infections would be minimal.

1.3.2 Indirect Channel

As evidenced in Section 1.2.3, it is possible through the isolation of infectious individuals to reduce the basic reproductive number of a disease by enough to decrease the disease prevalence. Sanatoria offered a system of imperfect quarantine that could have slowed tuberculosis transmission through a nearly identical manner (Roberts, 2009). However in order to cause this type of decline through quarantine alone, a much greater number of individuals would need to be isolated than there were available beds.¹²

There have been few attempts to measure the implications of sanatoria on the population of uninfected individuals and no previous study has been done on this for the pre-antibiotic era. Most of the few studies examined the impact of differences in patient compliance with antibiotic regimens and chemotherapies between patients housed in sanatoria versus in those receiving treatment at home. These studies estimated the impact of these compliance differences on the probability of infection for susceptible individuals whom the infectious were in contact with. One such study, performed by Kamat et al. (1998), showed that the susceptible contacts of

¹²Between 1932 and 1940 in North Carolina, there were an average of 2,500 sanatoria beds and at least 1,300 tuberculosis related deaths in each year. Given the SLIRQ model of tuberculosis transmission, this indicates that approximately 10,000 people were infected with tuberculosis in each year. Therefore the maximum possible percentage capable of being quarantined was around 25%, which is much less than the needed estimate of 81%.

those patients treated in a sanitarium are no worse off than those susceptible contacts of patients who underwent home treatment. However all of the patients examined were being treated with a regimen of Isoniazid plus PAS and all of the contacts were living in an endemic disease environment.

The Role of Education

According to Teller (1988), in the early 20th century the majority of those concerned with the spread of tuberculosis “believed that their first task was educating the public.” In a similar spirit, the central tuberculosis policy of North Carolina as outlined in a 1910 publication by the North Carolina Board of Health was “one of education” in which the sanitarium was embraced “as a sort of central school.” The policy detailed that patients would be taught “how to live with others without infecting them” and that “this training [would] be carried back...into their communities and counties, and its influence [would] be of great benefit to their fellow sufferers at home who [were] unable to attend the training” (North Carolina. State Board of Health, 1910). Graduates of sanitarium were trained against spitting and of proper disposal of their sputa. Local tuberculosis associations often showed videos at sanitarium such as the 1940 production *Goodbye, Mr. Germ* (Ulmer, 1940). The goal of these videos was to educate viewers in a digestible manner about how tuberculosis spread and the virtues of hygienic living.

Thus in North Carolina, sanitarium were essential in the dissemination of information concerning control of tuberculosis. This was reflective of the national viewpoint on the role of sanitarium as was outlined in a 1907 Illinois State Board of Commissioners report:

The educational value of a sanitarium is such that the statement has been frequently made that a tuberculosis sanitarium is primarily an educational institution. Every patient who enters the sanitarium is given the most thorough and enduring training. . . He carries these lessons home with him and because his own future depends upon his faithfulness in obeying them, he enforces their observance upon the other members of his family. His home, in fact, becomes in itself a small educational cen-

ter in the care and prevention of tuberculosis. (Illinois Board of State Commissioners of Public Charities, 1907, pg 270)

In addition to educating their patients, some sanatoria engaged in educational outreach to their local communities. For example, in 1915 the Chicago Municipal Tuberculosis Sanitarium practiced education through the distribution of free leaflets and the regular publication of health bulletins containing advice on preventative hygiene and other information concerning TB. Chicago Municipal also maintained an off-site, educational public tuberculosis exhibit. Thus those who returned from treatment at a sanitarium were educated in hygienic practices that could lead to a decrease in the rate of effective contact. These graduates were also encouraged to share their knowledge with their family members and friends, further decreasing the probability of disease transmission. Consequently a combined effect of reducing β through education and increasing q through isolation could push R_0 below the threshold of 1. This would result in a dramatic decrease in death rates for those with sanatoria access, *ceteris paribus*.

1.3.3 Isolation and Education

In *Water, Race, and Disease*, Troesken (2004) shows that in the early 20th century, despite widespread societal discrimination, African-Americans benefited from water quality improvements intended for white residents. During this time period urban whites and urban blacks lived in close proximity to one another. Troesken explains that this limited segregation made it difficult to provide differential water quality by race. Therefore when cities made efforts to reduce the typhoid and waterborne disease mortality of their white residents through the installation and improvement of higher quality water and sewer systems, nearby African-American residents benefited as well. White residents benefited directly from cleaner water and sewer systems and indirectly from the diminished threat that their African-American neighbors would be propagating waterborne diseases.

The early 20th century prescription for tuberculosis was quite different than that

for typhoid. Where a city could expect reductions in typhoid mortality from improving the water quality of a few central mains, there was no analogous point source for tuberculosis. Pre-antibiotic treatment for tuberculosis consisted of an often-costly move from the urban environment to a health center that would ideally be a remote mountainous location. Jim Crow legislation had institutionalized a system in which almost no public sanitariums were established for African-Americans and the small number of private black sanitariums that were established were, as described by Roberts (2009), “small affairs, often short lived.” Thus even though white residents would have certainly benefited from reducing the number of infectious African-Americans, it would have required a concerted, governmental effort.

This endeavor was politically infeasible as both the public and many governmental officials supported this form of institutionalized racism. The belief that blacks would be unable or unwilling to learn and perform hygienic practices necessary to stop the spread of the disease was widely held. Echoing this idea in 1910, the assistant health commissioner of Baltimore City asserted that blacks “are harder to teach, and it is more difficult to make them understand that they must do certain things to prevent the spread of the disease or check its ravages in individuals cases.” A 1915 committee appointed by the governor of Maryland to study black tuberculosis in the state concluded that “it would be futile to emphasize” isolation and education in a sanitarium setting to mitigate black mortality from the disease. The committee based these inferences on the racially charged beliefs that blacks possessed “a much lower resistance to the advance of the disease than the white population” (Roberts, 2009, pg 180-184).

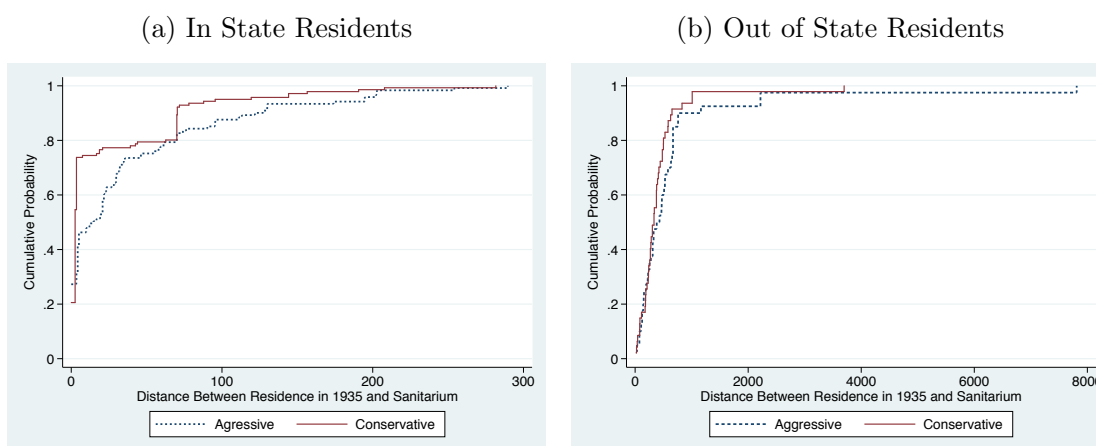
Therefore as public officials thought that blacks could not be helped by sanitariums, there was minimal support behind establishing black only facilities around the country. In addition, under the “separate, but equal” auspices of the Jim Crow south, African-Americans had a difficult if not impossible time utilizing white facilities. Consequently, a racially segregated system of sanitariums was established throughout the south and access to sanitariums was unfortunately, almost exclusively a white privilege. This atmosphere of segregation allows for inference into the mechanisms

through which sanitariums were able to benefit nearby residents. If sanitariums were primarily beneficial through their ability to isolate infectious individuals then both black and white residents should see gains from access to these facilities. However if sanitariums are primarily beneficial through the education of their patients then it is possible that only whites would benefit from access as black residents had very limited access to the services of sanitariums.

1.4 Why North Carolina?

This paper examines the impact of sanitariums on the death rate of residents of North Carolina from pulmonary tuberculosis in the early 1900s. Any results obtained from the analysis of North Carolina can easily be extended to the United States as a whole. This extension is warranted because the internal dynamics of tuberculosis transmission in North Carolina mimic those of the United States. North Carolina has the ideal climate in which to establish a sanitarium as the western portion of North Carolina is quite mountainous. In addition North Carolina collected and has maintained a large collection of vital statistics making quantitative analysis possible. This fact is not true for every other state during this time period.

Figure 1.5: Distance Traveled to NC Sanitariums by:



It was not uncommon for individuals to move across the United States in order

to find a climate deemed suitable for recovery from infection (Teller, 1988). As North Carolina was a net importer of infectious individuals during this time period, it is argued that North Carolinians were more likely to move within their own state rather than across the country.¹³ In order to explore the patient composition and distance traveled, one would ideally construct a dataset at the sanitarium-patient-year level. Given that this data do not exist, the next best alternative is to approximate a patient dataset using the US Census.¹⁴ The complete version of the 1940 census for North Carolina contains information for 1503 individuals, living in 28 distinct sanitariums. When the sample is restricted to only those who had moved since 1935 and whose relationship to the head of household was either “patient” or “lodger,” the dataset is reduced to 188 individuals. This conservative approach provides a database of individuals whom we are absolutely certain are patients in sanitariums at the expense of losing many observations that are only probable sanitarium patients, as they could be employees. When a more lenient criteria that only keeps individuals who had moved since 1935 and whose occupational response did not indicate that could have been an employee in a sanitarium, there is data retained for 347 individuals. For the both conservative and aggressive data, out of state patients respectively represent 25% and 24.5% of the sample. As evidenced in Figure 1.5a, almost 80% of North Carolinians who were patients in a sanitarium in 1940, had lived within 75 miles of that sanitarium five years earlier, with roughly 20% still living in the same city. Nearly all of those living in a sanitarium in 1940 had lived within 150 miles of that facility in 1935.

1.5 Model

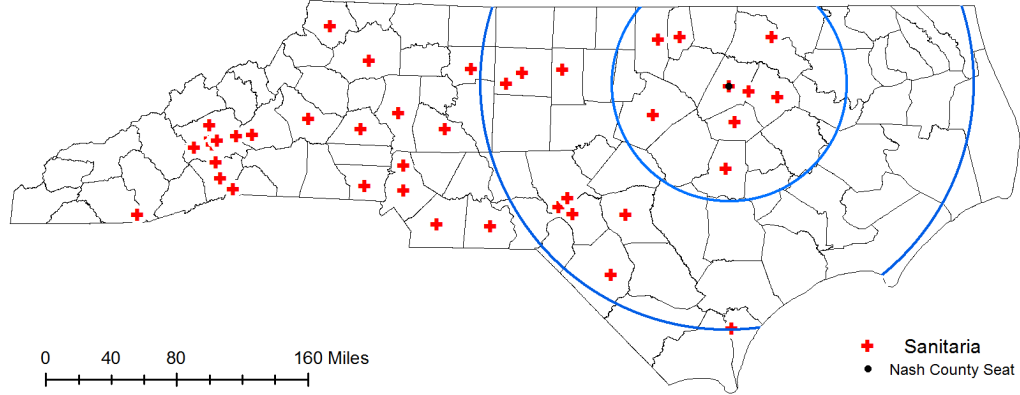
$$TB_{it} = \sum_{j=0}^2 \phi_j^1 SanBeds_{ijt} + \eta^1 R_{it} + \beta^1 X_{it} + \alpha_i^1 + \gamma_t^1 + \lambda^1(U \cdot t) + \varepsilon_{it}^1 \quad (1.12)$$

In order to identify the causal impact of access to sanitariums on tuberculosis rates

¹³Roughly $\frac{1}{4}$ of sanitarium patients were from out of state and in every year there were more deaths from pulmonary TB in NC than there were resident deaths from pulmonary TB.

¹⁴As provided in its complete form via Ruggles et al. (2010)

Figure 1.6: Sanitaria and Distance Rings around Nash County



we would ideally use Equation 1.12. However, there is a potential endogeneity problem between the tuberculosis rate in county i in year t and the number of beds placed in county i in year t . The potential endogeneity is that, all else held constant, a bed is more likely to be placed in an area with a higher resident tuberculosis death rate. In order to overcome this bias, an instrumental variables approach will be utilized. The specifications of interest are all least-squares estimations where the coefficient estimates are weighted by county population, thus TB_{it} is the pulmonary tuberculosis mortality rate per 100,000 in county i and time t . The regressors of interest are $SanBeds_{itj}$, which represents the number of sanatoria beds within the j^{th} interval away from county i in year t . This model allows for individuals to have access to all of the sanatoria beds in the j rings around the county seat and allows for both distance and wealth to influence the impact of this access. Therefore access for residents is different for every county in both i and t as the number of beds in all other counties varies across time. It is also expected that beds close to (or within) a county will have a greater impact than those beds that are far away from a given county. Therefore the index $j \in [0, 2]$ has been added to account for the number of sanatoria beds within a j interval of a given county i in year t .

Those observations in $j = 0$ will include all of the beds in county i in year t . Those observations in $j = 1$ will include all of the beds within 75 miles of county i 's county seat in year t , but not in county i . Those observations in $j = 2$ will include

all of the beds between 75 and 150 miles away from county i 's county seat in year t .¹⁵ See Figure 1.6 for a clarifying example that depicts every sanitarium within each j for Nash county in 1932.¹⁶ The inclusion of three distinct distance rings is more flexible than assuming the impact of beds decays linearly with distance. The three rings do not preclude the possibility that the impact of beds on tuberculosis will decay linearly, but they do allow for decay to occur through a variety of methods.

As increased wealth may, *ceteris paribus*, enable individuals to access sanitarium beds that are further away than they otherwise would have been able to access, R_{it} , retail sales per capita is included. Higher levels of wealth, as proxied by retail sales per capita, is expected to have a negative impact on tuberculosis mortality (i.e. $\eta < 0$). X_{it} is a vector of socio-economic correlates containing % urban, % illiterate, % black, % other race, and the number of mine workers. As these values increase it is expected that the TB death rate will also increase, thus the expected sign for each coefficient in the vector β is positive. Unobserved time invariant county-level characteristics are controlled via fixed effects, α_i . State wide time trends and yearly shocks are controlled for via the inclusion of year dummies, γ_t .¹⁷ An urban-specific time trend is also included to control for the fact that urban and rural counties may have heterogeneous time trends.¹⁸ Not controlling for this type of specific time trend would be problematic if over this time period North Carolina had any policies that differentially treated urban and rural counties. An example of a differential policy captured by this specific time trend would be if urban areas were seen as the biggest problem or as areas with the lowest marginal cost per TB death reduced, and because of this a set of unique TB eradication programs was enacted for all counties with large metropolitan areas. Each specification also has standard errors, ε_{it} , that

¹⁵Rings of 75 and 150 miles were chosen to approximate the observed pattern of distance traveled in Figure 1.5a. They also roughly approximate two and four hours driving distance from a county seat. In previous versions of this paper a larger set of 100-mile rings was used. The incorporation of more rings does not change the main results.

¹⁶This figure was created using shape files from Minnesota Population Center (2011) and the open source mapping software QGIS (QGIS Development Team, 2009).

¹⁷With 1932 serving as the base year.

¹⁸Where t is a time trend and U is a dummy variable that takes the value of 1 if the county is more than 10% urban in a given year.

are clustered at the county-level and that are robust to unknown heteroskedasticity, including heteroskedasticity related to differential population size. If there are unobserved factors that evolve over time and are therefore not time invariant, than even in the presence of county level fixed effects there is use for clustering at the county level. Stated another way clustering at the county level will correct for the fact that the error for Nash county 1932 is likely to be correlated with the error for Nash county in 1933. Thus the remaining and identifying variation in the TB death rate is based on within county changes that deviate from urban or rural specific time trends and yearly-state wide shocks.

1.5.1 Identification and Instrument Creation

In order to correct for the potential endogeneity in Equation 1.12 an instrumental variables approach will be exploited. Equation 1.20 assumes potential endogeneity exists only through the choice to locate beds within county i and Equation 1.21 allows for the choice of beds in nearby counties to be endogenous. The method used to construct the instrument is similar between the specifications. Both instruments involve creating a prediction for the number of tuberculosis specific beds for each county-year and both instruments also exploit the altitude of each county. Altitude is exogenous to tuberculosis death rates and correlated with sanitarium placement, as it was considered to be a desirable characteristic for the location of a sanitarium. Thus the interaction of altitude and the predicted number of TB beds in a county's j^{th} ring will be taken to be plausibly exogenous to tuberculosis death rate in a given county-year. This style of instrumental variable is based on the shift-share literature, popularized by Bartik and common in labor economics (see Bartik, 1991; Katz and Murphy, 1991; Schaller, 2012).

There have been some questions raised concerning the relationship between this instrument and the tuberculosis death rate. Specifically the concern is that the lower air pressure found at higher elevations may create a hypoxic environment in the lungs which could plausibly prevent or reverse a tuberculosis infection. Indeed there have been several recent studies supporting this claim that there is negative

association between high altitude and lower rates of tuberculosis infection (see Eisen et al., 2013; Mansoer et al., 1999; Olender et al., 2003; P eres-Padilla and Franco-Marina, 2004; Tanrikulu et al., 2008). However my instrument likely avoids these complications as the altitude of each individual’s county of resident is time invariant. Thus the county fixed effects will sweep away this variation and concerns of own-county altitude biasing the results.

$$\text{Predicted TB Beds}_{NC,t} = \left(\frac{1}{5} \sum_{k=t-5}^t \frac{\text{Pul. TB Deaths}_{SC,k}}{\text{Population}_{SC,k}} \right) \cdot \text{Population}_{NC,t=1932} \quad (1.13)$$

$$\text{County Share of Suitable Locations}_i = \frac{1(\text{Altitude}_i > \text{Altitude Cutoff}) \cdot \text{Population}_{i,1932}}{\sum_j^{100} 1(\text{Altitude}_j > \text{Altitude Cutoff}) \text{Population}_{j,1932}} \quad (1.14)$$

$$\text{Predicted TB Beds}_{it} = (\text{Predicted TB Beds}_{NC,t}) \cdot (\text{County Share of Suitable Locations}_i) \quad (1.15)$$

The predicted number of TB beds in each county-year is created by first calculating a prediction for the number of TB beds in North Carolina, see Equation 1.13, and then weighting this state-wide prediction by each county’s share of suitable locations in a base period, see Equation 1.14. The predicted number of TB beds for the state is based upon a 1928 report of the Duke Endowment stating that the optimal number of tuberculosis beds in a region in time t should be equal to the average number of deaths from tuberculosis in that same region over the previous five years (Reed and Hollingsworth, 1953).¹⁹ Using the five-year average of TB deaths for a region as an instrument for that same region would be problematic, as it would suffer from similar types of endogeneity as simply using beds. Thus statewide pulmonary tuberculosis deaths from South Carolina are used, as they are

¹⁹A simple fixed effects regression of the five year moving average pulmonary tuberculosis deaths (for place of death) at the county year level, including year dummies, supports the idea that counties in North Carolina followed these recommendations. The coefficient on the five-year moving average is 1.24, with a t-statistic of 4.63. The overall R^2 is .7733.

exogenous to any North Carolina specific and county-specific features that would be related between the placement of beds.

$$\text{Instrument for TB Beds}_{it} = \text{Predicted TB Beds}_{it} \cdot (\text{Altitude}_i) \quad (1.16)$$

$$\text{Instrument for TB Beds, i-75mi}_{it} = \left(\sum_{j \neq i}^{100} \text{Predicted TB Beds}_{jt} \cdot 1(\text{distance}_{ij} \leq 75) \right) \cdot \text{Altitude}_i \quad (1.17)$$

The five-year moving average for South Carolina is divided by the population in South Carolina in each year and then scaled to the population of North Carolina by multiplying it by the population of North Carolina in 1932, which is the base year. This results in a predicted number of beds for North Carolina that varies across time and is exogenous to state or county specific features of North Carolina. This state-level prediction is then mapped into a county-level prediction by placing beds into the most suitable climates. A county is considered to have a suitable climate for sanitarium if its maximum elevation exceeds the 85th percentile of elevation. For each county with a suitable climate, its population share of all suitable counties' population is calculated using 1932 as a base year. All counties whose maximum elevation is below the cutoff are given shares of 0 by design.²⁰ Each county's share is time invariant.

$$\text{Instrument for TB Beds}_{it} = \sum_{j=0}^2 \phi_j^2 \text{SanBeds}_{ijt} + \eta^2 R_{it} + \beta^2 X_{it} + \alpha_i^2 + \gamma_t^2 + \lambda^2(U \cdot t) + \varepsilon_{it}^2 \quad (1.18)$$

$$\text{Instrument for TB Beds, i-75mi}_{it} = \sum_{j=0}^2 \phi_j^3 \text{SanBeds}_{ijt} + \eta^3 R_{it} + \beta^3 X_{it} + \alpha_i^3 + \gamma_t^3 + \lambda^3(U \cdot t) + \varepsilon_{it}^3 \quad (1.19)$$

²⁰The main results are robust to the choice of a threshold for suitable climate between 1500 and 6000 feet. Using the 85th percentile results in a cutoff of 5040 feet.

$$TB_{it} = \phi_0^4 \widehat{\text{Instrument for TB Beds}}_{it} + \sum_{j=1}^2 \phi_j^4 SanBeds_{ijt} + \eta^4 R_{it} + \beta^4 X_{it} + \alpha_i^4 + \gamma_t^4 + \lambda^4(U \cdot t) + \varepsilon_{it}^4 \quad (1.20)$$

$$TB_{it} = \phi_0^5 \widehat{\text{Instrument for TB Beds}}_{it} + \phi_1^5 \widehat{\text{Instrument for TB Beds, i-75mi}}_{it} + \phi_2^5 SanBeds_{i3t} + \eta^5 R_{it} + \beta^5 X_{it} + \alpha_i^5 + \gamma_t^5 + \lambda^5(U \cdot t) + \varepsilon_{it}^5 \quad (1.21)$$

The instrument for TB beds in a county-year (Equation 1.16), is the interaction of that county's altitude with the predicted beds for the same county-year. The instrument for TB beds outside county i , but within 75 miles (Equation 1.17), is the sum of the predicted beds within 75 miles multiplied by county i 's altitude. Both instruments will provide for identification at the county-year level and are valid, as they allow for both the time invariant feature of altitude and for varying trends in regional tuberculosis death rates to impact bed placement decisions in a manner exogenous to any particular locale. The relevance of the instruments will be discussed in Section 1.7.

1.6 Data

Data on sanitarium are extracted from the annual *Hospital Service in the United States* survey presented in The Journal of the American Medical Association American Medical Association (Various Years, 1921-1943). Data from these the surveys from 1921, 1926 and 1928 through 1943 were originally collected and digitized under the supervision of Thomasson and Treber (2008) and then subsequently cleaned and merged for use in this project.²¹ These surveys contain hospital level information on location, number of beds, type of ownership and type of service. Any observation whose type of service contained tuberculosis, isolation, or whose hospital name contained the word sanitarium or similar derivative was marked as a tuberculosis sanitarium. The city in which sanitarium were located was also reported. As many

²¹The use of this data is greatly appreciated!

city names were systematically misspelled though out the digitized dataset, it was necessary to create a city code that could link cities across time even in the presence of misspellings. Each city's location was determined through the use of a geocoding program, *geocode*, that utilizes Google Maps' API (Ozimek and Miles, 2012). Any errors in the geocoding process were then corrected by hand. County-year and city-year level datasets containing the number of sanitarium and the number of sanitarium beds were calculated by aggregating every sanitarium level observation in a given county or city, into a single county-year or city-year observation for each year.

As described in Section 1.5, this location information was then used to create two rings around each county. In order to determine the number of sanitarium beds within X miles of county i 's seat, but not in county i , a program *geonear* was utilized (Picard, 2012). This program utilizes geocoded location information to determine which cities meet the user defined criteria and produces the set of cities that qualify. This set is then merged with the dataset containing the total number of tuberculosis beds in each city and year. Then for each county-year, the total number of sanitarium beds within the determined ring of radius X is created by summing the total number of beds for all qualifying cities into one number. Using this process, two mutually exclusive ring variables are created, the first is the number of TB beds within 75 miles of county i in year t and the second is the number of TB beds between 150 miles and 75 miles from county i in year t . County level summary statistics these variables are displayed in Table 1.2.

Mortality information for North Carolina comes from the annual publications Vital Statistics by North Carolina Center for Health Statistics. The original data contained information for all county-year deaths by cause and by race as well as more specific data concerning only the deaths of residents by cause and by race. The resident deaths include all deaths of county decedents, regardless of county or state in which they perished. For this analysis, only resident deaths will be used as using place of deaths would bias death rates upwards in counties with hospitals and other health institutions. The North Carolina Bureau of Vital Statistics only began reporting death statistics at the resident level in 1932 and thus this is the first year

Table 1.2: Summary Statistics

	Mean	S.D.	Min	Max
Resident TB Deaths per 100k	44.14	25.35	0.00	150.12
White Resident TB Deaths per 100k	25.26	18.02	0.00	134.64
Black Resident TB Deaths per 100k	102.23	154.17	0.00	2531.65
TB Beds in County i	30.92	149.80	0.00	1480.00
TB Beds, i-75mi	700.12	623.22	0.00	2156.00
TB Beds, 75-150mi	912.80	641.48	40.00	2927.00
Predicted Beds in County i	18.79	70.63	0.00	780.79
Predicted Beds, i-75mi	363.54	591.05	0.00	2131.35
White and Black Population	33907.98	27006.84	5242.40	153902.00
White Population	24345.60	20427.64	3195.00	121751.00
Black Population	9562.38	9191.02	1.20	43295.00
Highest Elevation	1927.57	2130.60	20.00	6684.00
% Urban	0.16	0.19	0.00	0.77
% Other Races	0.00	0.02	0.00	0.22
% Black	0.27	0.18	0.00	0.65
% Illiterate	0.10	0.03	0.05	0.21
Retail Sales Per Capita	118.96	71.63	13.82	494.10
Mine Workers	32.13	75.09	0.00	555.60
Observations	900			

of my analysis. As the vital statistics were also reported by race, it is possible to preform separate analysis to see if access to sanitararia had a heterogeneous impact between white and black communities.²² Table 1.2 illustrates the mean resident death rate from tuberculosis by race per 100,000.

State-level pulmonary tuberculosis mortality data for South Carolina from 1927-1936 was provided by Miller (2008). South Carolina mortality from 1937-1940 was collected from the US Bureau of the Census and appended to the Miller data. Yearly population estimates for South Carolina, county level population in North Carolina, % urban, % foreign born, and % black were interpolated from the decennial U.S.censuses and provided via Haines (2010). While % illiterate was reported in the

²²The vital statistics also recorded deaths from Native Americans (reported as Indian) however; due to the low number of Native American deaths in the sample their inclusion does not provide a meaningful result.

1930 census, it was not collected in either the 1940 or the 1950 census. Thus as in Fishback et al. (2011), % illiterate for 1940 is constructed from the years of schooling measure and then interpolated with the 1930 census estimate.²³ The number of mine workers and maximum elevation in each county were also provided by Fishback et al. (2011). Retail sales per capita was collected by Fishback et al. (2005) from the US Census Bureau for 1929, 1933, 1935, and 1939. Fishback et al. interpolated retail sales per capita between these years using information on state-level per capita income. Table 1.2 provides summary statistics for all of the variables used in the analysis.

1.7 Results

Table 1.3 displays the regression estimates when the dependent variable is the county resident pulmonary tuberculosis death rate per 100,000, this includes both black and white deaths. In (1) on Table 1.3 a simple pooled OLS regression of sanitarium beds on tuberculosis rates and weighted by population is considered. (1) indicates that sanitarium beds in county i are associated with an increase in tuberculosis rates. This association highlights the concerns of endogeneity outlined in Section 1.5. As both the coefficients on the ring variables are negative, it is revealed that in a pooled setting an increase in nearby tuberculosis beds is associated with lower resident tuberculosis death rates. If in these two variables, there is a bias created by endogeneity, the bias is working against the sign of the coefficients and thus in our favor. In other words, if one is concerned that the decision to place beds near to, but outside of a county i , is positively related to the tuberculosis death rates of county i , the finding that there is a negative and statistically significant association between nearby beds and resident death rates can be treated as an upper bound of the association, meaning that after correction for the bias the true relationship is

23

$$\% \text{ Illit}_{i,1940} = \frac{78.2 \cdot (\text{M } 25+, 0 \text{ yr school}_{1940}) + 22.53 \cdot (\text{M } 25+, 1-4 \text{ yr school}_{1940}) + 80.7 \cdot (\text{F } 25+, 0 \text{ yr school}_{1940}) + 16.67 \cdot (\text{F } 25+, 1-4 \text{ yr school}_{1940})}{(\text{M Population } 25+_{1940}) + (\text{F Population } 25+_{1940}) - (\text{M Pop } 25+ \text{ Unknown yr School}_{1940}) - (\text{F Pop } 25+ \text{ Unknown yr School}_{1940})}$$

still negative, but larger in absolute value. The specifications grow gradually more complex, with (2) adding the set of control variables, and (3) adding year dummies along with an urban-specific time trend. The sign, magnitude and significance of the coefficients for the number of beds do not meaningfully change between (1), (2) and (3). Specifications (4), (5) and (6) incorporate county fixed effects, where (4) is Equation 1.12, (5) is Equation 1.20, and (6) is Equation 1.21.

The specifications of interest are (5) and (6), both of which utilize instrumental variables to address endogeneity concerns between the placement of beds in county i and the death rate in county i or nearby counties. The first stage Equation for (5), Equation 1.18, yields a Kleibergen-Paap rk Wald F statistic of 18.77, setting aside concerns of a weak instrument when compared against the relevant Stock and Yogo (2005) critical value for 10% maximal IV Size of 16.38.²⁴ Therefore the instrument is both valid (as outlined in Section 1.5.1) and relevant. The interpretation of this coefficient is that adding one sanitarium bed to county i in year t will reduce the resident tuberculosis death rate per 100,000 by approximately 1.16. Specification (6) has been included to address any remaining endogeneity concerns regarding the number of nearby tuberculosis beds. As outlined in Equation 1.21, (6) uses the predicted number of beds in each ring interacted with altitude as an instrument for the number of beds within this ring in addition to the instrument used in (5). The first stage for specification (6), Equation 1.19, produces a Kleibergen-Paap F-statistic for the instrument is 15.32, dismissing concerns of a weak instrument when compared against the relevant Stock and Yogo (2005) critical value for 10% maximal IV Size of 7.03. Thus again, the instruments are valid and relevant.

Given the nature of the data, resident pulmonary tuberculosis deaths per 100,000 is capable of being separated into death rates by race. Tables 1.4 and 1.5 display the two sets of regression results when the dependent variable is separated by race. The two specifications are identical to those outlined in the above exposition of Table 1.3. The initial striking result is that sanatoria had no impact for black residents in any

²⁴The Kleibergen-Paap rk Wald F statistic is used instead of the Angrist and Pischke (2009) F-statistic as it is recommended by Baum et al. when there are non-i.i.d. errors. The Kleibergen-Paap statistic becomes the Cragg-Donald statistic when the errors are indeed i.i.d.

of the fixed effect regressions, (4), (5) or (6). This means that the driving force behind virtually all of the significance of the results in Table 1.3 is through the benefit derived from sanatoria exclusively obtained by white residents. The most conservative specification of interest, Equation 1.21, shows that an increase of one sanitarium bed in a county-year will reduce white resident pulmonary tuberculosis mortality by approximately .695 per 100,000 and will have no impact for black residents. This is not a surprising result as medical facilities in the south were often segregated, thus access to these facilities would have been quite limited for those of color. As outlined earlier, there was an atmosphere such that “the tuberculosis movement rarely reached [blacks]. Blacks in the south were excluded or segregated from the few tuberculosis facilities that existed” (Teller, 1988).

1.7.1 Interpretation of Results

Econometric estimates have shown that sanatoria have a causal impact on reducing tuberculosis deaths, but these estimates do not specifically identify the mechanism through which sanatoria are reducing these deaths. When these coefficient estimates are interpreted within the context our theoretical model of tuberculosis transmission (SLIRQ), the set of most important feasible mechanisms can be isolated. As earlier outlined, it is unlikely that sanatoria provided a large direct benefit to their patients. However as in Figure 1.4, even if sanatoria were able to provide a large direct benefit to their patients, this would not be a primary driver in the reduction of secondary tuberculosis infections. Therefore it is improbable that sanatoria were reducing the aggregate tuberculosis death rate through direct benefits obtained by their patients. This leaves the indirect channel as the primary mechanism through which sanatoria could reduce tuberculosis deaths. As explained in Section 1.2.3, the key indirect ways a sanatoria could reduce tuberculosis deaths are through increasing the percentage of the infectious who are quarantined or housed in a sanitarium (an increase in q), and/or through a reduction in the probability of transmission by increasing the exposure time required to contract the disease (a decrease in β). It has also been shown that sanatoria engaged their patients in a variety of educational programs that

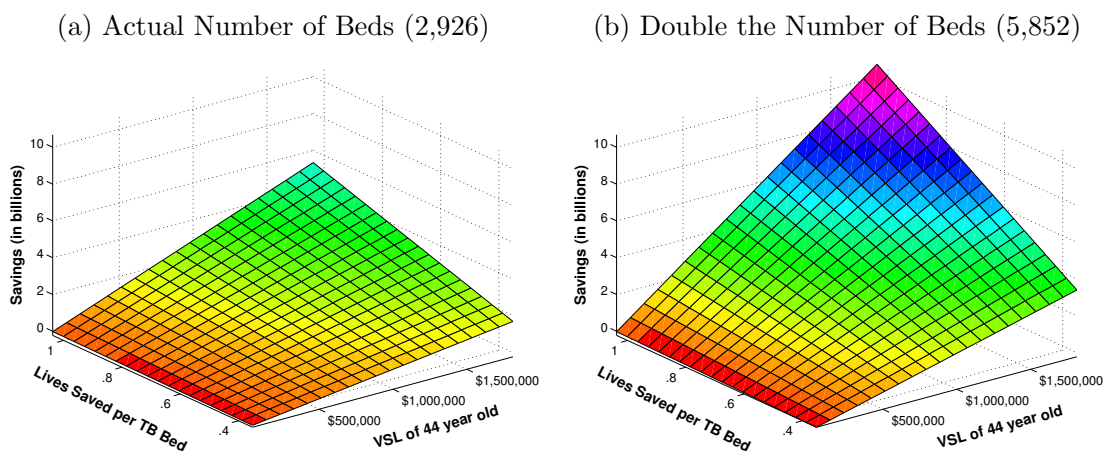
were intended to reduce the likelihood of transmitting the disease, i.e. a reduction in β .

If isolation were the only driver of this reduction, then nearly 80% of the infectious population would have been needed to be quarantined in sanitarium for the reduction to be consistent with our economic results. In order for 80% of the infectious population to be isolated, there would have needed to be well over 5,000 more sanitarium beds than were in existence in any given year. In addition if isolation were the only factor in the mortality reduction, we would have expected for there to have been at least some mortality reduction for black residents. As noted earlier, the maximum number of beds in North Carolina during this time period was on average 2,500, when this information is combined with yearly TB death rates and the SLIRQ model, it can be estimated that there were approximately 10,000 people infected with tuberculosis in any given year. Thus the quarantine rate was likely near 25%. As shown in Figure 1.4, even if the quarantine rate was over twice this estimate, there would also need to occur a significant reduction in β in order to cause such a reduction in TB mortality. Thus when both the maximum number of potentially isolated individuals and the racially stratified econometric results are taken into consideration, it becomes clear that the sanitarium were reducing tuberculosis mortality not solely through isolation, but also through causing a large reduction in the probability of transmission, likely by increasing the level of education.

1.8 Cost-Benefit Analysis

This section outlines a rough estimate of the cost effectiveness of sanitarium treatment for white North Carolinians using estimates of the Value of a Statistical Life (VSL), the average age of death from pulmonary tuberculosis in North Carolina, the cost to open a sanitarium, the yearly per bed operating cost of treatment, and our econometric estimates of the impact of a sanitarium bed. The value of a statistical life for an 18-30 year old in 1940, as calculated by Costa and Kahn (2004), was

Figure 1.7: Savings with:



between \$1,300,218.51 and \$1,816,294.02 in 2014 dollars.²⁵ The mean age of death from pulmonary tuberculosis in North Carolina in 1940 was 44.09; this value was constructed via a weighted mean using age of death data from the 1940 publication of Vital Statistics by the North Carolina Center for Health Statistics (Various Years, 1914-1944). As the weighted-mean age of death is significantly above the age range used to calculate the VSL, a range of values between \$50,000 and \$1,816,294.02 is considered as potential values of the true VSL. Per patient cost information for 1940 was found for the North Carolina Sanitarium and the Western North Carolina Sanitarium in their 1940 Biennial Report (State of North Carolina, 1942). These per capita cost estimates are inclusive of administrative, treatment, custodial, operational, maintenance, agricultural and facility improvement costs. This information was then combined with data on the number beds for these two facilities found in the JAMA surveys. Thus a weighted-mean value of \$8,554 is calculated to be the cost of operating a single sanitarium bed for the year of 1940. Thus the 2,926 sanitarium beds in North Carolina are estimated to have cost roughly \$25 million to operate each year. Data was also collected on the cost to open a sanitarium of 220

²⁵All dollar values in this section are in 2014 dollars as calculated using the Bureau of Labor Statistics' historical Consumer Price Index (U.S. Bureau of Labor Statistics U.S. Department of Labor, 2014).

beds from a 1939 North Carolina state bill, which established the Eastern North Carolina Sanatorium. This building cost \$10,212,000 to construct (State of North Carolina (1942)). Lastly, while the point estimate of the impact of one sanitarium bed on the reduction in the white tuberculosis death rate per 100,000 is estimated to be .695 as in Section 1.7, the 95% confidence interval (.35 to 1.04) will be used for this analysis in order to avoid sensitivity on any particular point estimate.

Estimates of the dollar amount (in billions) saved from the 2,926 beds in 1940 are shown in Figure 1.7a. These estimates incorporate both fixed and variable sanitarium costs including construction, operation, and maintenance. Figure 1.7a shows a positive amount saved in VSL for virtually all realistic values of remaining life and for almost the entirety of the 95% confidence interval for the avoided TB deaths per sanitarium bed.²⁶ Conservatively, choosing a VSL of \$500,000 and varying the estimate of deaths avoided per bed between the extreme values of the 95% confidence interval, the number of TB beds in North Carolina in 1940 saved the state between \$347 million and \$1.35 billion. This framework allows for an estimate of the impact of have twice as many beds in NC in 1940. A doubling in the number of sanitarium beds in NC in 1940 would have resulted in between 1,000 and 3,000 less white resident deaths from pulmonary tuberculosis. As in the earlier exercise the VSL is considered, these costs pale in comparison to the benefits, as the net benefits are between \$703 million and \$2.7 billion.

1.9 Conclusion

Tuberculosis is no longer a disease of the past. With the recent discoveries of multiple, extensively and total drug-resistant strains of tuberculosis, a complete understanding of how sanitarium impact tuberculosis transmission and death rates has never been more important. Using an instrumental variables approach and data from North Carolina in a time before antibiotics it has been shown that access to an additional sanitarium bed reduced the death rate from tuberculosis for white

²⁶The only combinations when TB beds are not a positive investment occur when the VSL is \$50,000 and when the lives saved per bed are below .38.

residents by .695 per 100,000 and had no impact for black residents. When these results are examined through the lens of a SLIRQ model of transmission, the intrinsic transmission dynamics of the disease allow us to deduce the likely mechanisms through which sanitararia were able to reduce resident deaths. These mechanisms were indirect in nature and operated through a combination of imperfect quarantine and through increasing the public's knowledge of hygienic practices. A simple cost-benefit analysis indicates that sanitararia were extremely cost effective. Thus in the pre-antibiotic era increasing the education level of the general public combined with limited quarantine, served to cause large reductions in tuberculosis deaths from those groups who had preferential access to these services. These sanitararia of the past were able to provide such a great benefit because they were able to educate the public in basic hygienic practices in a world afflicted by endemic tuberculosis. The result of this research leads to the conclusion that if we again find ourselves in a world without effective antibiotic treatments for tuberculosis, a return to the use of sanitararia should not be expected to produce a similar decline in the TB death rate unless we believe that the public would similarly gain from learning basic hygienic practices.

Table 1.3: Dependent Variable: Resident Pulmonary TB Death Rate per 100,000

	(1)	(2)	(3)	(4)	(5)	(6)
TB Beds in County <i>i</i>	0.0323*** (0.00411)	0.0351*** (0.00401)	0.0352*** (0.00427)	0.0105 (0.0275)	-1.155*** (0.331)	-0.823*** (0.173)
TB Beds, <i>i</i> -75mi	-0.0212*** (0.00285)	-0.00962** (0.00311)	-0.00772* (0.00316)	0.0439* (0.0217)	-0.0484 (0.0552)	0.0199 (0.0394)
TB Beds, 75-150mi	-0.00414 (0.00358)	-0.00585* (0.00279)	-0.00423 (0.00285)	0.0323 (0.0167)	-0.0413 (0.0506)	-0.00336 (0.0270)
% Urban		58.94*** (12.07)	46.04** (13.75)	62.30 (91.88)	-89.19 (123.6)	-41.69 (88.37)
% Other Races		-94.40*** (25.75)	-99.00*** (24.79)	-104.7 (312.7)	1715.1 (881.2)	1059.0 (558.1)
% Black		27.24* (12.88)	30.33* (12.90)	290.9 (208.2)	-650.2 (559.9)	-352.4 (348.9)
% Illiterate		265.2** (84.19)	243.3** (80.47)	-155.5 (111.7)	164.3 (306.2)	108.2 (212.6)
Retail Sales Per Capita		-0.0465 (0.0257)	0.00645 (0.0240)	-0.109* (0.0428)	0.112 (0.152)	0.0382 (0.107)
Mine Workers		0.0101 (0.00941)	0.00426 (0.00981)	-0.0126 (0.0281)	0.0426 (0.0471)	0.0255 (0.0297)
Year Dummies	No	No	Yes	Yes	Yes	Yes
County Fixed Effects	No	No	No	Yes	Yes	Yes
Specific Time Trends	No	No	Yes	Yes	Yes	Yes
Observations	900	900	900	900	900	900
F-Stat	113.3	79.54	48.00	7.556	82.32	320.6
Kleibergen-Paap F-Stat					18.77	15.32
Stock-Yogo Critical Value for 10% Maximal IV Size					16.38	7.030

Cluster Robust Standard Errors Reported in Parentheses

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table 1.4: Dependent Variable: White Resident Pulmonary TB Death Rate per 100,000

	(1)	(2)	(3)	(4)	(5)	(6)
TB Beds in County i	0.0386*** (0.00169)	0.0396*** (0.00195)	0.0397*** (0.00207)	-0.00409 (0.0289)	-1.291*** (0.348)	-0.695*** (0.175)
TB Beds, i-75mi	-0.00392** (0.00138)	-0.00461* (0.00181)	-0.00332 (0.00182)	0.0406 (0.0217)	-0.0612 (0.0609)	0.0615 (0.0400)
TB Beds, 75-150mi	-0.00398*** (0.00110)	-0.00565*** (0.00108)	-0.00436*** (0.00102)	0.0281 (0.0159)	-0.0532 (0.0546)	0.0150 (0.0262)
% Urban		17.57* (7.278)	3.555 (6.553)	105.7 (83.57)	-61.67 (124.5)	23.74 (68.63)
% Other Races		-83.96*** (14.22)	-78.77*** (15.34)	-102.0 (266.1)	1908.1 (1047.0)	728.4 (566.6)
% Black		-16.32 (8.542)	-12.71 (8.810)	510.6* (206.6)	-528.9 (634.8)	6.552 (321.4)
% Illiterate		124.3* (48.24)	93.58* (44.98)	-94.03 (128.5)	259.2 (327.1)	158.2 (177.9)
Retail Sales Per Capita		-0.0371 (0.0218)	0.00466 (0.0134)	-0.0615 (0.0397)	0.182 (0.166)	0.0503 (0.0870)
Mine Workers		0.0172 (0.00971)	0.0124 (0.0101)	0.0132 (0.0253)	0.0741 (0.0550)	0.0435 (0.0289)
Year Dummies	No	No	Yes	Yes	Yes	Yes
County Fixed Effects	No	No	No	Yes	Yes	Yes
Specific Time Trends	No	No	Yes	Yes	Yes	Yes
Observations	900	900	900	900	900	900
F-Stat	285.9	304.2	190.4	4.424	37.36	302.3
Kleibergen-Paap F-Stat					18.77	15.32
Stock-Yogo Critical Value for 10% Maximal IV Size					16.38	7.030

Cluster Robust Standard Errors Reported in Parentheses

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table 1.5: Dependent Variable: Black Resident Pulmonary TB Death Rate per 100,000

	(1)	(2)	(3)	(4)	(5)	(6)
TB Beds in County i	0.0639*** (0.00649)	0.0390*** (0.00979)	0.0393*** (0.0101)	0.00875 (0.0698)	-0.807 (0.744)	-2.064 (1.245)
TB Beds, i-75mi	-0.0125 (0.00925)	-0.0237* (0.00947)	-0.0199* (0.00991)	-0.0288 (0.0648)	-0.0934 (0.0985)	-0.352 (0.300)
TB Beds, 75-150mi	0.00765 (0.00821)	-0.00741 (0.00899)	-0.00544 (0.00909)	-0.00262 (0.0413)	-0.0541 (0.0685)	-0.198 (0.173)
% Urban		115.8*** (28.84)	125.7*** (36.20)	53.00 (170.5)	-52.99 (188.7)	-233.0 (327.0)
% Other Races		-111.1 (131.5)	-174.4 (140.7)	1965.8 (2344.2)	3239.0 (2783.6)	5725.1* (2769.1)
% Black		-103.8* (45.38)	-107.2* (47.54)	-130.8 (416.5)	-789.3 (794.9)	-1917.6 (1220.6)
% Illiterate		439.7 (227.8)	499.4 (252.9)	-246.4 (313.6)	-22.68 (408.5)	190.1 (587.4)
Retail Sales Per Capita		-0.0914 (0.0514)	-0.0286 (0.0599)	-0.193 (0.122)	-0.0385 (0.208)	0.239 (0.368)
Mine Workers		0.112 (0.0931)	0.106 (0.0912)	-0.175 (0.150)	-0.136 (0.145)	-0.0716 (0.125)
Year Dummies	No	No	Yes	Yes	Yes	Yes
County Fixed Effects	No	No	No	Yes	Yes	Yes
Specific Time Trends	No	No	Yes	Yes		Yes
Observations	900	900	900	900	900	900
F-Stat	46.70	51.09	19.38	3.768	2.817	10.56
Kleibergen-Paap F-Stat					18.77	15.32
Stock-Yogo Critical Value for 10% Maximal IV Size					16.38	7.030

Cluster Robust Standard Errors Reported in Parentheses

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

CHAPTER 2

RETAIL HEALTH CLINICS: ENDOGENOUS LOCATION CHOICE

Over 20% of Americans do not have adequate access to primary care services due to a lack of available primary care physicians (PCPs) (HRSA, 2014b). This problem has been compounded by the Affordable Care Act's recent extension of health insurance coverage to over 10 million individuals (Sommers et al., 2014). Given that the short-run supply of physicians is inflexible, it is unclear how the already stretched U.S. health care system will be able to provide adequate primary care for this large number of newly insured. It will likely become more difficult for new patients to find accepting physicians and for existing patients to schedule timely appointments. Cowling et al. (2013) show that an increased difficulty in accessing traditional primary care services can lead to an increase in unnecessary emergency department (ED) visits. Specifically, they find that British patients who were unable to see a PCP within two weekdays were more likely to self-refer to an ED. To avoid similar undesirable outcomes in the United States, many have called for an increased role of the Nurse Practitioner (NP) (Institute of Medicine, 2010). For many treatments, NPs are able to provide the same quality of care as a PCP, but at a 20-35% lower cost and with a higher level of reported patient satisfaction (Naylor and Kurtzman, 2010). In addition, while the number of PCPs has been recently decreasing, the number of NPs equipped for primary care service has been growing, adding to what is already the largest group of health care providers in the country (American College of Physicians, 2006).

The retail health clinic, a recent innovation, is well suited to serve as a conduit for the expanded role of the NP. These clinics are located in pre-existing retail locations and are primarily staffed by nurse practitioners. They provide low cost treatment for a limited set of acute, episodic ailments. As they enjoy both lower startup and operating costs than traditional primary care facilities, many have hoped that

they would bolster access to primary care in traditionally under-served communities (Howard, 2011; Malvey and Fottler, 2006). A 2009 RAND Commission report on health care spending in Massachusetts recommended that to help contain costs, the state should promote the entry of retail clinics. This recommendation was based upon the belief that patients without adequate primary care access would replace trips to the more expensive emergency rooms and urgent cares, with visits to retail clinics, staffed by cost-effective NPs (Eibner et al., 2009, pg. 16).

In this paper, I model the determinants of retail clinic location. I construct a structural discrete choice model of clinic location that explicitly accounts for both demand and competition effects. As CVS and Walgreens operate 75% of retail clinics in the United States, I model their location decisions as the outcome of an entry game of incomplete information with oligopolistic competition. Each firm maximizes its expected profit, which is a function of known demand characteristics and the probability of rival entry. The structural model allows for both counterfactual analysis and for the estimation of competition effects, which would be otherwise unobservable. While this framework closely follows both Seim (2006) and Zhu and Singh (2009), my model allows for a richer set of unobservables by incorporating an error structure that permits spatial correlation and market level unobservable heterogeneity.

I find that clinics are more likely to locate in areas that are more populous, wealthy, educated, and white, and that they are less likely to locate in under-served communities, where access to primary care is typically most limited. I also find suggestive evidence that there are profit complementarities in proximity to a rival. This finding is robust to numerous controls for unobserved market level heterogeneity. In states where there are minimal barriers to retail clinic operation, the predicted number of clinics from my model fits the actual distribution well. Counterfactual predictions show that states with legislation prohibiting non-physician ownership of clinics, like California, suffer from a significant under-entry of clinics.

In Chapter 3, I combine the results of my predictive model with data on emergency department visits to determine if clinics help direct patients away from re-

ceiving treatment at expensive emergency rooms. To establish a causal relationship I must deal with the potential endogeneity that the true impact of retail clinics on emergency room diversion may be muted by two sources of endogeneity. The first is that clinics may find it more profitable to locate in areas where there are more emergency room visits to divert. The second is that urgent care centers may be opening near to retail clinics and may be the true drivers of any reduction in ED visits. To account for the first source of potential bias, the location predictions from the entry model serve as an instrument for actual clinic locations. Predicted clinic locations are created using the structural model found in this chapter and are not a function of ED visits. Therefore by construction they are exogenous to the concern that a clinic locations might be responding to changes in ED visits. To account for the second source of endogeneity, I use the set of potential clinic locations (CVS and Walgreens drugstores) that existed prior to the advent of retail clinics. This instrument removes concerns that urgent cares are driving any findings as it is unlikely that urgent cares are responding differentially to the age of the building that a retail clinic was built in. The analysis shows that access to retail clinics causes a substantial decrease in the number of emergency department visits for bronchitis and upper respiratory infections, but has no effect for visits related to other illnesses. In 2012, for the 40 states and territories with minimal barriers to retail clinic operation, the savings associated with retail clinic induced ED diversion is conservatively estimated to be over \$88 million. In California, counterfactual analysis suggests that relaxing the barriers to clinic entry would result in \$10.5 million in health care savings. While these are large and significant efficiency spillovers from retail clinics into the health care system, these efficiency gains will only be provided for hospitals in relatively more affluent areas, where the clinics are most likely to locate.

2.1 Institutional Background

Retail clinics provide a standardized suite of mostly episodic, acute health care services. Retail clinics treat a significantly more limited set of medical conditions than

urgent cares, EDs or PCPs. Mehrotra et al. (2008) found that 76% of retail clinic visits are for 7 medical conditions; upper respiratory infections, sinusitis, middle and outer ear infections, pharyngitis, conjunctivitis, urinary tract infections, and immunizations. As the clinics treat a largely homogenous set of simple ailments, NPs and Physicians Assistants (PAs) are often the only employees. This is in stark contrast to urgent cares, EDs, and doctor's offices, who employ PAs, NPs and physicians. Their limited scope of practice and small workforce enables retail clinics to be established in small portions of pre-existing retail stores as, unlike their counterparts, they have no need for large medical equipment, such as X-Rays. The most common locations are within drug-stores, grocery stores or mass merchandise stores. A patient does not typically need to schedule an appointment because the wait time is generally less than 20 minutes. Over 50% of visits to retail clinics occur during the hours in which doctor offices are normally closed. Virtually all retail clinics accept major insurance, but a close approximation to the out of pocket costs generated from an average visit is \$75 (Mehrotra et al., 2008).

In 2000 MinuteClinic (then QuickMedx) opened the first retail clinics in the United States. By 2005 they had 18 locations between Baltimore and Minneapolis. In 2006, CVS purchased MinuteClinic and began national expansion. Similarly in 2007, Walgreens purchased the retail clinic provider, Take Care Clinics (now Healthcare Clinic), and also began subsequent national expansion. The number of retail clinics grew rapidly and led many to predict that there would be over 6000 by 2013 (Mehrotra et al., 2008). However in 2013, there were only 1,221 retail clinics in the United States, 74.04% of which are operated by either CVS or Walgreens.

There is a significant variability in the number of retail clinics per state. It is a commonly held belief that this state level variation in clinics is due to differences in Scope of Practice legislation for nurse practitioners. The American Academy of Nurse Practitioners (AANP) has rated each state according to its autonomy of practice for NP's. Table 2.1 displays the percentage of retail locations in which CVS and Walgreens chose to add a retail clinic by the AANP state practice environment. There is no describable relationship between the percentage of stores with a retail

clinic and the state practice environment. This is confirmed by looking at more detailed legislation on required Physician oversight and the legality of nurses prescribing controlled substances provided by the Robert Wood Johnson Foundation and the US Department of Justice and shown in Table 2.2.

Table 2.1: % Potential Locations with a Retail Clinic by AANP Rating

AANP Rating	Mean	S.D.	Min	Max	N
Full Practice	3.66	5.27	0	13.33	20
Reduced Practice	5.56	6.50	0	25	19
Restricted Practice	8.55	4.57	2.94	16.72	12

Table 2.2: % Potential Locations with a Retail Clinic by Legal Environment

	Mean	S.D.	Min	Max	N
<i>Corporate Practice of Medicine (CPOM)</i>					
<i>Limitations</i>					
Yes	3.74	3.20	0	8.13	7
No	5.80	6.12	0	25	44
<i>Potential Tobacco Limitations</i>					
Yes	0	0	0	0	2
No	5.74	5.83	0	25	49
<i>NP Required Supervision by Physician</i>					
Yes	6.92	5.95	0	25	32
No	3.15	4.88	0	12.56	19
<i>NP Allowed to Rx Controlled Substances Alone</i>					
Yes	5.50	–	6.55	6.55	1
No	5.49	5.83	0	25	50

While NP scope of practice differences may not be responsible for state level variation in the number of retail clinics, it is possible that differences in the interpretation of the Corporate Practice of Medicine Doctrines (CPOM) is related to the variability. CPOM legislation prohibits businesses from being able to profit from the provision of medical services and often prohibits some businesses from employing practicing physicians. States with strict interpretations of these policies have

special exceptions for institutions such as hospitals. States with these strict interpretations make it very difficult for CVS and Walgreens to operate as their typical retail clinic ownership structure is prohibited. Using data from the National Hospice and Palliative Care Organization on CPOM legislation, seen in Table 2.2, there is some correlation between CPOM legislation and retail clinic prevalence. The entry model outlined in Section 2.2 will therefore be restricted to those states that are not limited by CPOM laws. Additionally I will not include any states that have pending legislation to ban the sale of cigarettes in locations that also have retail clinics (WA and RI). This will allow us to compare markets with complex or high barriers to entry with those markets that have less limitation on entry. After these restrictions are imposed, 39 states and D.C. remain in the entry analysis.

2.2 Entry Model

Previous Literature

Predicted entry is taken to be the outcome of a static entry game of oligopolistic competition. In this model profit is explicitly a function of both market characteristics and of the probability of rival entry. This modeling framework relies upon revealed preference analysis. The basic argument is that if a firm is seen operating in a particular location, it can be inferred that this was the most profitable decision the firm could make. This assumes that opening in that particular spot was a better decision than either opening in another location or not opening at all. In this setup, even though profit is not directly observable, we can still learn useful information about a firm's latent profit by looking at the characteristics of places while a firm chooses to locate given their available choice set.

There is a large literature that uses this type of revealed preference analysis to examine endogenous market structure. In their seminal work on firm entry, Bresnahan and Reiss (1991) examine a number of isolated markets and are able to identify the total number of entering firms across a variety of industries. However, their model does not always allow them to tell the identity of entering firms, only the number.

Studying isolated motel locations, Mazzeo (2002) overcame this identification problem by allowing firms to endogenously enter based on their chosen type (i.e. high or low quality). Using this approach, he finds positive returns to firm differentiation in both type and space. He is also able to identify the specific firms that are entering, but this is not without cost. His equilibrium concept is one of complete information and is reliant upon a large and complex number of profit constraints that would be difficult to construct for many other industries and markets. By incorporating incomplete information, Seim (2006) was able to address the identification issue while avoiding the problems of Mazzeo's solution.

Seim examines the entry decisions of video retailers. She frames the choice of location as the endogenous choice of a product characteristic, namely spatial differentiation. Firm profit is impacted by these product characteristics, rival proximity, and idiosyncratic private draws from a known distribution. Incomplete information refers to this private draw, which is revealed before the simultaneous entry decisions. While the actual value of this private draw is known only to the firm itself, all other players know the distribution from which it is drawn. Due to this uncertainty, firms are unable to maximize their ex-post profits, instead they maximize their ex-ante, or expected, profit. Her solution concept is a Bayesian Nash Equilibrium and eliminates the dimensionality problem of Mazzeo's model created by a need for checking a large number of entry conditions to find the equilibria. Framing location as an endogenously chosen characteristic also relaxes the need for isolated markets. Additionally, as firms are maximizing expected rather than actual profits, there could be realizations in some markets that would appear to be ex-post mistakes, but which are ex-ante rational. This concept introduces realistic variation into the model.

Seim's model assumes that firms have homogeneous profit functions and that firms have symmetric and identical competitive interactions with one another. Zhu and Singh (2009) extend her framework to allow for asymmetries in both of these dimensions. Their analysis focuses on oligopolistic competition between discount retailers, K-Mart, Target, and Wal-Mart. They find that the firms have both negative and differential profit impacts on their rivals and that these impacts decrease

with distance. Stated differently, they allow for firms to have differential tastes for exogenous demand characteristics and for heterogeneous competition effects based on the identity of the rival. Zhu and Singh use identification concepts developed by Bajari et al. (2006), who show that a sufficient condition for identification of games of incomplete information in the presence of unobservable heterogeneity is to use exclusion restrictions. Specifically, if there are $I \in \mathbb{N}$ players, an identifying exclusion restriction for player $i \in I$, would be to include a payoff relevant covariate for each $j \in I$ with $j \neq i$ that does not directly impact player i 's profits. These covariates may *indirectly* impact player i 's profits, but only through their effect on the change in probability of entry for each player $j \in I$ with $j \neq i$. Zhu and Singh (2009) use two different sets of exclusion restrictions; distances from each firm's nearest distribution center and distance to each firm's headquarters.

My entry model will closely follow Zhu and Singh but will differ in two key ways. First, Zhu and Singh assume that each firm is a potential entrant in every market. For each firm, I will limit the set of stores in which they can place a retail clinic to those locations that already have a retail store. In other words, MinuteClinics can only be added to pre-existing CVS stores and Healthcare Clinics can only be added to pre-existing Walgreens stores. For most markets, this will mean that CVS and Walgreens have an asymmetric set of potential locations. This assumption yields a set of potential entrants that is both believable and that provides useful variation in potential rival proximity. The second way in which my entry model differs from Zhu and Singh is that I introduce a correlated error structure that exploits additional information from firm location. This error structure will serve to dismiss concerns of proportional substitution common in logit models.

Market and Location Definition

As in Zhu and Singh, there are $i \in \{1, \dots, N\}$ firms with $N \in \mathbb{N}$, who are each trying to maximize their own profit. Each firm can open a retail clinic in any market, m , in which they are a potential entrant. Markets are independently and identically distributed and are indexed by $m \in \{1, \dots, M\}$ with $M \in \mathbb{N}$. In any given market, each firm may choose at most one mutually exclusive location in which to open a

retail clinic. Firms may also choose the outside option, which is to not locate in that market. The outside option also serves to make each market-firm choice set exhaustive. For the same market firms may face different location choice sets. This is due to the assumption that a clinic can only be opened in a pre-existing retail location. Thus, each market is said to have \bar{L} total locations, with any particular location defined as $l \in \{1, \dots, \bar{L}\}$. $l = 1$ refers to the choice of the outside option.

A firm's ideal market level decision can be stated as a profit maximization problem, in which they select the mutually exclusive location within the market that offers the greatest non-negative profit.¹ It is important to note that the outside option, deciding to not open a clinic, does not provide zero profit. As will be outlined later, the outside option will earn profit equal to a draw from the standard type one extreme value distribution. The decision of firm i to open in location l in market m , is represented by a_{iml} and is also subject to the constraint that $\sum_{l=1}^{\bar{L}} a_{iml} = 1$. The constraint prohibits entry of more than one clinic by a single operator in each market. With the firm specific location profit denoted π_{iml} , the decision can be written as:

$$a_{iml} = \begin{cases} 1 & \text{if } \pi_{iml} \geq \pi_{imk}, \forall k = 1, \dots, \bar{L} \\ 0 & \text{otherwise} \end{cases} \quad (2.1)$$

Both CVS and Walgreens provide customers with lists of nearby clinics through their websites and via automated phone information systems. Using either of these services, when a customer wants to begin a query of nearby clinics, they are asked for their zip code of residence. Additionally, it is common practice at both clinics for customers to enter their zip-code of residence into an on-site, electronic system immediately prior to being examined. Thus the natural choice for the market is the zip code. However the zip code is a designation created by the US Postal service. Zip codes are not necessarily contiguous geographic units and thus they are not always composed of mutually exclusive census areas. In order to account for these

¹In the model to be specified, firms do not know the decisions of their rivals with certainty as is the case in this example and are therefore unable to compare ex-post profits in this manner. Thus firms will actually make their entry decisions based on of expected profit, to be defined later.

limitations, I will use the Zip Code Tabulation Area (ZCTA) instead of the zip code.

The ZCTA is developed by the US Census Bureau and is composed of mutually exclusive census blocks. It is a relatively fine and contiguous geographic unit, the United States can be divided into over 30,000 ZCTAs. As ZCTAs are composed of census blocks, they are linkable to other census delineated lines, such as the census block group. To link each census block group to its unique parent ZCTA, I use information on its geographic centroid and QGIS (QGIS Development Team, 2009). This link provides me with the set of distinct block groups that compose each ZCTA. Each ZCTA is said to have \bar{L} locations, even if the potential number of locations for a given firm in a ZCTA is less than \bar{L} . This is done to ensure that comparisons between CVS and Walgreens are being made on the correct locations and for reasons illustrated by the following example.

Consider a market with two potential locations, A and B . Here \bar{L} would be 3. Assume that there is a CVS store in both A and in B , but there is only one Walgreens, which is located in B . In this market, CVS has three options, but Walgreens only has two. CVS can open a clinic in A , in B , or they can choose the outside option to not enter the market. Walgreens may open a clinic in B or not at all. For the sake of exposition, let profits for firm i follow the form $\pi_{i,m} = \{\text{Profit From Not Entering, Profit From Location A, Profit From Location B}\}$. Also assume that profits are known to each firm, such that:

$$\begin{aligned}\pi_{CVS,m} &= \{1.3, 7, 5\} \\ \pi_{WAL,m} &= \{-.2, NaN, 7\}\end{aligned}$$

Based off of the decision rule from Equation 2.1, CVS will choose to enter location A and Walgreens will choose to enter location B . We restrict profit of locations that firms cannot enter to be NaN or “Not A Number.” By incorporating this strategy, we ensure that the second element of each profit vector corresponds to location A and the third element of each profit vector to location B . Correct accounting of location profits in this vector will be of prime importance when rival decisions are

accounted for in the following section. Another benefit of the NaN restriction for locations where a firm has no potential entrants, is that it is superior to alternatively restricting profits to be zero. NaN is neither less than or greater than any other number and will therefore not incorrectly enter a firm’s decision making process. If we were to force profits to be zero at these locations, we may incorrectly predict entry when the action would be impossible. This is due to the fact that the outside option actually receives a non-zero draw from the type I extreme value distribution. In the above example, if profits for Walgreens in location A were 0 rather than NaN , then location A would seem to be a superior choice over the outside option, despite the fact that this choice could not be made.

The minimum number of options a firm may face for any market is two. If a firm does not have any potential locations in a market, then while it is still not entering that market, there is no observed choice and thus no revealed preference inference on latent profits can be made. Again, if a firm does not have at least one pre-existing retail location in a market, it is not considered to be a potential entrant for a clinic in that market. Going back to the example, if instead there were no pre-existing Walgreens in A or B , their profit vector for the market would be $\pi_{WAL,m} = \{NaN, NaN, NaN\}$.

Retail Clinic Data

To account for the supply side of retail clinic entry decisions, data were collected on potential clinic locations and on all of the realized clinic locations. Clinic location information was collected from the Convenient Care Association (CCA), a trade association for retail based health care in the United States (CCA, 2013). As a retail clinic could be added to any CVS or Walgreens location, the set of potential clinic locations for a given retailer is their set of existing retail locations. Thus for both Walgreens and CVS, the location of every retail store was collected from each of their respective corporate websites (CVS, 2013; Walgreens, 2013). For each store, all three of these data sources provided both the street address and a limited set of store-level characteristics. Approximate building level geographic coordinates for

each location was determined by using the fully parsed street address.²³ This level of accuracy permits each store to be reliably linked to enclosing geographic units as fine as the census block level and is a significant improvement over coordinates that are often at the zip code or county centroid. Each company website also provided information on store hours of operation, clinic hours of operation, and whether or not a store contained a drive-through pharmacy.

A crucial feature of the model is that each operator can have no more than one retail clinic per market. There are 8,750 distinct ZCTA's that contain a CVS or Walgreens. Out of this large set of markets, there are only 32 in which an operator has opened more than one retail clinic. Using a simple t-test for mean comparison, this set of 32 is not observably different from the remaining 8,718 locations. Thus it is assumed that there are idiosyncratic features in these markets, such as being near a university, that are driving this multiple entry and these markets are not included in my analysis.

Expected Profit

$$E[\pi_{iml}] = x_{mi}\beta_i + z_{iml}\eta_i - \sum_{i' \neq i} \sum_{b=0}^{B-1} \sum_{k=1}^L \delta_{ii'kl} E[a_{i'mk}] 1(k \in b_l) + \sigma\xi_m + \rho\xi_{ml} + \varepsilon_{iml} \quad (2.2)$$

$$i, i' = \{1, 2\} \quad (\text{firms})$$

$$m = \{1, \dots, M\} \quad (\text{markets})$$

$$l, k = \{1, \dots, \bar{L}\} \quad (\text{locations})$$

Expected clinic profit for firm i in location l and market m expressed in Equation 2.2 is a function of location characteristics, firm specific covariates, the firm's own entry decisions, it's rival's entry decisions, and an idiosyncratic shock. For a

²Geocoding was performed using Texas A&M Geoservices (Goldberg, 2014). Addresses were first parsed, then batch geocoded, and finally, any observations that were still not at the desired level of precision were corrected by hand using their manual correction system.

³The coordinate system used throughout my analysis is World Geodetic System 1984, better known as WGS 84. Any geographic data stored using another coordinate system was reprojected into WGS 84 using QGIS (QGIS Development Team, 2009).

Table 2.3: Summary Statistics for Entry Model

	Mean	Std. Dev.	Min.	Max.	N
<i>Block Group Level Data</i>					
Population	1746.144	1331.247	0	29411	10407
Population w/in 10mi, excluding own block-group	493224.822	503213.709	347	2880493	10407
% Black	0.147	0.216	0	1	10407
% Hispanic	0.139	0.198	0	1	10407
% Some College	0.586	0.191	0	1	10407
% No Car	0.096	0.116	0	1	10407
% Insured	0.851	0.098	0	1	10407
Household Median Income	55839.732	29885.745	2499	251800	10407
# Urgent Care	0.079	0.303	0	4	10407
Distance to nearest Hospital	3.548	3.315	0.025	37.605	10407
# MinuteClinics	0.054	0.226	0	1	10407
# HealthCare Clinics	0.028	0.165	0	1	10407
CVS with Drive-Thru	0.362	0.481	0	1	10407
Wal with Drive-Thru	0.175	0.38	0	1	10407
Distance to nearest Physician Affiliate for CVS	113.206	216.187	0.209	2626.572	10407
Distance to nearest Physician Affiliate for Walgreens	322.731	284.956	0.29	2829.453	10407
<i>Census Tract Level Data</i>					
PCP per 1k	1.57	20.739	0	2000	9620
<i>Market (ZCTA) Level Data</i>					
# CVS	0.972	0.826	0	6	5694
# Walgreens	1.065	0.921	0	10	5694
# MinuteClinics	0.099	0.298	0	1	5694
# HealthCare Clinics	0.051	0.219	0	1	5694
<i>County Level Data</i>					
APRN per capita	0.078	0.282	0	7.27	1374

given market, location characteristics are denoted by the matrix, $X = \{x_1, \dots, x_L\}$ and are common amongst players. Each $x_i \in X$ is a vector of location specific characteristics. Firm specific covariates, or exclusion restrictions, are represented by the matrix $Z_i = \{z_{i1}, \dots, z_{iL}\}$, which is similarly indexed. Rival entry decisions are captured by $a_{i'mk}$ and are allowed to differentially impact profit with distance, which fall into B mutually exclusive bands. As rival entry decisions are not known with certainty, the profit maximizing firm instead focuses on the expectation of rival entry. Prior to entry this is the only source of uncertainty for firm i and is the reason necessitating an expected profit framework. The idiosyncratic shock is composed of three error terms, ξ_m , ξ_{ml} , and ε_{iml} . These error terms respectively represent an i.i.d. market level shock, a spatially correlated location specific shock, and a type 1 extreme value error term. The parameters to be estimated in this model are the represented by $\theta = \{\beta, \eta, \delta, \sigma, \rho\}$. The vector β reflects firm specific tastes for the common variables in X . η captures each firm's preference for their set of firm specific covariates. δ measures the impact of rival competition. σ captures the impact of unobserved market level heterogeneity and ρ measures the extent of spatial correlation between location alternatives within each independent market.

Location Specific Data

The common covariates related to demand for retail clinics, are demographic characteristics at the block group level and come from the American Community Survey (ACS) 5-year estimates, 2008-2012. These data were made digitally available by the National Historical Geographic Information System (Minnesota Population Center, 2011). Both total block group population and population within 10 miles (excluding own population) are expected to be positively related with profitability as an increase in either would represent a larger market and a higher demand.

A positive relationship between profit and percent black or percent Hispanic would support the idea that retail clinics were increasing primary care access to these traditionally under-served groups. However there is reason to suspect that this will not be the case. Saha et al. (2000) found that both black and Hispanics were less likely to seek care from health care professionals who were not of their own

race even after controlling for differences in geographic availability. Additionally Hooker and Berlin (2002) show that only 6.4% of NPs are African-American and only 3.6% are Hispanic despite the two groups respectively composing 13.2% and 17.1% of the total US population (Bureau, 2014). As a disproportionate majority of NPs are white and both blacks and Hispanics may be less likely to come to a retail clinic if the NP is white, the profit maximizing retail clinics may be less likely to locate in locations with a high percentage of blacks or Hispanics.

Previous studies have found that patients with low levels of health literacy are less likely to use primary care services than otherwise similar patients (Schumacher et al., 2013). McCormack et al. (2010) developed a survey that quantifies an individual's level of health literacy and they found that those with a higher level of education also have higher levels of health literacy. Thus, I expect that more educated individuals, as measured by the percentage of individuals with some college experience, would be more likely to seek care in a clinic as education serves as a proxy for health literacy.

The remaining demand variables from the ACS are meant to capture the differential ability of some consumers to access a retail clinic's services. Individuals who do not own a car will be less able to access a clinic, as their transportation options will be relatively more limited. Increased insurance rates should increase the likelihood of clinic use by lowering the cost of service. Similarly areas with higher median household income will likely have a higher demand for clinics than otherwise similar areas with less disposable income.

Data on related industries were collected in order to control for both non-strategic competition effects and for unobservables that are correlated with location specific profitability. This information was collected from a wide variety of sources. The American Academy of Urgent Care Medicine (AAUCM) made available address level data on urgent care facilities that was geocoded in an identical manner as outlined earlier (AAUCM, 2013). Hospital locations were extracted from the Centers for Medicare and Medicaid Services (2014) website on general hospital information. Census tract level data on the number and specialties of physicians, including the

number of primary care physicians, were collected from the Health Resources and Services Administration Website (HRSA, 2014a).

The expected relationships between the number of primary care providers, the number of urgent cares, and nearby hospitals with clinic profitability is not theoretically clear. On the one hand, each group may be a non-strategic competitor, siphoning away potential visits from a clinic. But on the other hand, retail clinics may benefit from stealing price- (or time-) sensitive patients away from these higher cost providers. Consider a patient, suffering from a sore throat, driving to an urgent care facility for treatment. Once near to the urgent care, if the patient sees a retail clinic, they may be likely to go there instead due to the clinic's lower cost of service and shorter wait time. All three of these covariates, distance to nearest hospital, number of urgent cares, and the number of PCPs, will also be reflective of unobservable location specific profitability. For example, when comparing two areas whose only observable difference is in the number of primary care providers, it can be inferred that the area with the greater number of PCPs has an unobservable trait that is positively correlated with the profitability of primary care provision.

Information on the main labor input of retail clinics, nurse practitioners, was made available at the county-level via the Area Health Resources Files (USDHHS, 2014). It is expected that areas with more nurses will also be lower cost areas of operation and thus be associated with increased clinic profitability. In many states, another important labor component for retail clinics are physicians who agree to oversee nurses working in the clinics. Therefore areas with more PCPs may enjoy lower costs of entry than otherwise similar areas with less PCPs.

Exclusion Restrictions and Model Identification

Identification of my model will be possible through the use of exclusion restrictions. These arguments follow Zhu and Singh (2009), who rely on work by Bajari et al. (2006). The exclusion restrictions, or firm specific covariates of the rival firm, that I will use can be thought of as shocks that only directly impact a single firm's clinic profitability in a given location. The other firms are impacted by this profitability shock only through the resulting change in the shock-receiving firm's prob-

ability of clinic entry. A firm specific covariate still provides identifying variation for rival retail clinic profitability even if it impacts rival retail *drugstore* profitability, as the model is one of retail clinic profit, rather than drugstore profit.

The first firm specific covariate used for identification will be the number of retail locations each firm has in a given market. For firm j , this exclusion restriction in market m will be the number of stores that firm i has in market m . For example, consider two markets, denoted A and B . In A , both CVS and Walgreens have two pre-existing retail locations. While in B , CVS has five locations and Walgreens only has two locations. Additionally, assume that markets A and B are identical in all other observables. If CVS were to open one retail clinic in both market A and B , it would be expected that CVS would earn more profits from the clinic opened in Market B . This will occur for three reasons. First, clinics are expected to have a positive impact on down-stream profit, as any pharmaceuticals that were prescribed as a result of a patient visit are likely to be ordered by the prescribing store's pharmacy. Similarly, once a patient has experience with a firm's pharmacy network, they may develop pharmacy network inertia and fill future prescriptions in other nearby but more convenient locations. Second, in a market where there are multiple CVS locations, those CVS locations without retail clinics will be able to self refer or recommend to their customers that they visit the nearby location which has a retail clinic. Finally, in the market with more potential locations, CVS would be able to pick from a larger set of options. As I assume that each pre-existing retail store was opened before retail clinics existed, it is likely that many retail stores are in locations that would not be profitable for a clinic. Thus an increased number of stores provides an increased probability that at least one of the stores will provide a location that will be profitable for a retail clinic. Thus Walgreens would be less likely to open a clinic in market B due to the increased likelihood that CVS would enter that market. Identification occurs because the difference in Walgreens' probability of entry between the markets is being driven by changes in expectations about CVS' choice rather than market characteristics.

The second set of exclusion restrictions is the distance to each firm's nearest

group of affiliated physicians. For firm j this will be the distance of block group l to the closest physician affiliate for firm i . In many states physician oversight of nurse practitioners is necessary to operate a clinic. With minimal exceptions, oversight does not mean that the clinic must have an on-site physician, but only that the physician is available to provide guidance if requested. Being less than a few hours drive is often the minimal requirement constituting availability. Both CVS and Walgreens have entered into mutually exclusive agreements with large groups of physicians to economize on the process of finding qualified doctors to fill this role. Lists of these physician groups are available on each of the company's websites (CVS, 2014; Walgreens, 2014). Each physician group's location was determined by geocoding the address of their main office. The distance to the nearest group headquarters serves as one of the firm specific covariates, Z_i , which enter the profit function. It is common for retail clinics to refer patients who enter the clinic seeking care for an ailment outside of the clinic's limited scope of practice to nearby primary care providers. Thus the relationship between the physicians groups and clinics is symbiotic as the group can benefit from being the sole recipient of the potentially large number of referrals generated by the retail clinic.

Ideally I would be able to use two additional firm specific covariates, store size (i.e. square footage) and store-specific foot-traffic. Larger stores have an increased probability of clinic operation as in these stores it is less costly to make the necessary floor space available. Many smaller CVS and Walgreens stores do not have enough floor space to add a clinic without paying to expand the building. Store-specific foot traffic would also ideally be included the model because a store with a larger number of patrons would be a better candidate for a retail clinic than an otherwise similar store with fewer unique customers. As both CVS and Walgreens certainly measure the number of customers that frequent each location, they are able to consider a variable correlated with profitability unobservable to the econometrician. As a proxy for both of these variables, I will use data on store drive-through status. Drive-through can generally only be added to free standing stores. Free-standing drugstores are typically between 11,000 and 14,000 square feet, which is significantly

significantly larger than their strip mall counterparts, which are generally between 6,000 and 8,000 square feet in size (Garbarine, 1997). Thus drive-through status is correlated with store size. Additionally, drive-through windows are typically found in stores that enjoy sufficient demand to support this feature, indicating that the stores with drive-through will likely be busier than similar stores without them. Thus drive-through status serves as an imperfect signal of an operator's beliefs about demand in a particular location. This signal should be useful in controlling for features of the market correlated with profitability that are unobservable to the econometrician, but observable to the both CVS and Walgreens. Drive-through status provides identifying variation as it does not directly affect the profitability of rival retail clinics in any market. Thus for firm j , this exclusion restriction in location m will be if there are any drugstores operated by firm i with a drive through in location l . A potential entrant would be less likely to open a clinic in a block group in which a rival potential entrant location has a drive through only because of the signal this provides as to the increased probability of rival entry, but not because of the drive-through itself.

Distance Bands

Expected profit in location l is affected by the expectation of a rival's entry, $E[a_{i'mk}]$ for every location in a given market, $k \in \{1, \dots, \bar{L}\}$. This impact of a rival is allowed to differentially impact own-profit with distance, allowing for a nearby rival matter more than one farther away. This will be operationalized by allowing for B mutually exclusive bands: one band for rivals in own block group, and one band for rivals within two miles, but not in own block group. The use of the bands allows for a more tractable state space rather than a continuum of distance effects would provide. The bands are also more flexible than assuming that competition decays linearly with distance. As in Zhu and Singh (2009), for location l the indicator variable, $1(k \in b_l)$, will denote when location k 's distance from l falls within band b . When the cutoffs for band b are defined as d_b and d_{b+1} , we have:

$$1(k \in b_l) = \begin{cases} 1 & \text{if } d_b \leq \text{distance}_{lk} < d_{b+1}, \\ 0 & \text{otherwise} \end{cases} \quad (2.3)$$

For the distance calculations, firms are assumed to be located in the centroid of their respective block groups. Block group centroid location coordinates were extracted from the 2010 Tiger/Line shapefiles provided via the National Historical Geographic Information System (Minnesota Population Center, 2011). Distance between all l and k locations was calculated using Vincenty's formula (Vincenty, 1975).

Error Term

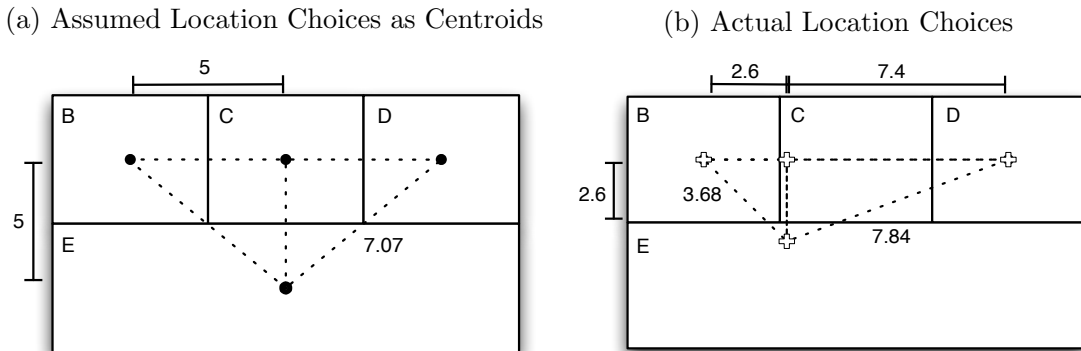
The idiosyncratic error component in Equation 2.2 is composed of three sources of error: ξ_m , ξ_{ml} , and ε_{iml} . This error structure will yield a mixed error components logit model that allows for a rich set of spatially correlated unobservables. The firm's private information is represented by ε_{iml} , which is a draw from the Gumbel, or type I extreme value distribution. This draw is known to firms before entry and its ex-post value is unknown to rivals. However as the distribution of draws is known, ex-ante conclusions can be drawn. For each location where a firm is a potential entrant, they will receive a draw from this error distribution, including the outside option of not entering. Therefore the mean profits of the outside option (when available) are non-zero as the mean of the type I extreme value distribution is the Euler-Mascheroni constant. Conditional on a given error draw for ξ_m and ξ_{ml} , the use of the extreme value distribution allows for the location choice probabilities to follow the closed form expression of the standard logit probabilities. This closed form will be integrated over, or mixed with, the density of the remaining error components, ξ_m and ξ_{ml} , to calculate the simulated conditional choice probabilities.

As this is a cross-sectional analysis, fixed effects cannot be used to control for time invariant unobservables at the market level. In order to account for this unobserved market level heterogeneity, a market level shock, $\sigma\xi_m$, is included. ξ_m is distributed

i.i.d. standard normal and σ is a parameter to be estimated. ξ_m is known and common to both players. It is included to represent market level features that are known to the players, but not to the econometrician. Two examples of such unobserved heterogeneity would be an active local physicians group who would protest the opening of a new retail clinic and thus hurt profitability, or a specific county-level zoning law that would make clinic entry impossible.

Both Seim (2006) and Zhu and Singh (2009) use a logit framework that does not allow for correlation between the location choices within a market. A common issue with this framework is that it exhibits a property known as Independence from Irrelevant Alternatives (IIA), which assumes proportional substitution between alternatives. In other words, when the probability of choosing one alternative increases, the increase in probability will be drawn proportionally from all other alternatives. I address concerns of proportional substitution by allowing for spatial correlation between the location choices in a market.

Figure 2.1: Differences in Market Structure With:



The intensity of correlation between any two alternative locations is determined using differences between assumed store distance and actual store distance. To reduce the state space to a tractable level, it is common to assume that the chosen location of entry is the centroid of enclosing geographic boundary. In other words, no matter where a pre-existing store is located within a particular block group, it is assumed to be located in the centroid of that block group. For example, consider

a market where a potential entrant has five choices: $l \in \{A, B, C, D, E\}$. A is the outside option of not locating and the remaining options are pre-existing retail locations. In Figure 2.1, (a) depicts the assumed building locations, while (b) shows the “actual” building locations. Observationally, it is clear that actual store locations are clustering near alternative B , indicating that there is some characteristic of B that nearby stores find desirable. If due to a store closure, option C suddenly ceases to exist, a standard logit model would assume that all other options would proportionally draw from this decrease. Meaning that A , B , D , and E would equally increase in probability of being chosen. This substitution pattern is unrealistic, as it would have been expected that location B would *disproportionately* increase in probability due its greater desirability. To account for this, my model allows for disproportionate substitutions between those locations whose pre-existing stores are closer together than their enclosing geographic centroids.

In Equation 2.2, the spatial correlation is represented by $\rho\xi_{ml}$, where ρ is a parameter to be estimated and the singleton ξ_{ml} is calculated in the following manner:

$$\xi_{ml} = W_{ml}\zeta_m \quad (2.4)$$

ζ_m , from Equation 2.4 above, is a $(1 \times \bar{L})$ column vector where each element, ζ_{ml} with $l \in \{1, \dots, \bar{L}\}$, is an i.i.d. draw from the standard normal distribution. W_{ml} is a row vector from the larger correlation matrix W_m , from which element w_{mlk} is the intensity of correlation between locations l and k . It is defined as:

$$w_{mlk} = \begin{cases} \frac{\text{centroid distance}_{lk} - \text{mean actual distance}_{lk}}{\text{centroid distance}_{lk}} & \text{if } \frac{\text{centroid distance}_{lk} - \text{mean actual distance}_{lk}}{\text{centroid distance}_{lk}} \geq 0 \\ 1 & \text{if } l = k \\ 0 & \text{if } l = 0, k = 0, \text{ otherwise} \end{cases} \quad (2.5)$$

This pattern of spatial correlation allows for a positive shock in one location, say B from Figure 2.1, to impact locations whose mean store distance is closer than

the assumed, or centroid, distance (C). The closer the pre-existing stores are to one another, relative to their assumed centroid distance, the stronger the correlation will be. The relationship is not permitted to be negative as it is not realistic that a positive shock to a store in one block-group would have a negative impact on another store in that ZCTA.

Calculating the market level spatially correlated row vector, ξ_m , for the example market in Figure 2.1, yields:

$$\underbrace{\xi_m}_{(\bar{L} \times 1)} = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 \\ 0 & 1 & .48 & 0 & .4796 \\ 0 & .48 & 1 & 0 & .48 \\ 0 & 0 & 0 & 1 & 0 \\ 0 & .4796 & .48 & 0 & 1 \end{pmatrix} \begin{pmatrix} \zeta_{m,l=A} \\ \zeta_{m,l=B} \\ \zeta_{m,l=C} \\ \zeta_{m,l=D} \\ \zeta_{m,l=E} \end{pmatrix}$$

Each element of ξ_m , ξ_{ml} , captures the relationship between l and all other locations in that same market in a way that addresses concerns of proportional substitution. Returning again to the example from Figure 2.1, the introduction of spatial correlation allows for disproportional substitution to B relative to other alternatives like D . This fact is evidenced by the spatially correlated location-specific error for B and D , in which shocks in B are now explicitly related to shocks in C and E , while D 's shock remains independent.

$$\begin{aligned} \xi_{m,l=B} &= \zeta_{m,l=B} + .48 \zeta_{m,l=C} + .4796 \zeta_{m,l=E} \\ \xi_{m,l=D} &= \zeta_{m,l=D} \end{aligned}$$

2.3 Estimation

Choice Probabilities

Given that ex post profits are uncertain, the choice with certainty posed in Equation 2.1 is not possible. Therefore in every market each firm alternatively maximizes

their expected profit, found in Equation 2.2. Subject to the constraint $\sum_{l=1}^{\bar{L}} a_{iml} = 1$ the choice problem can be rewritten as an ex ante decision rule:

$$a_{iml} = \begin{cases} 1 & \text{if } E[\pi_{iml}] \geq E[\pi_{imk}], \forall k = 1, \dots, \bar{L} \\ 0 & \text{otherwise} \end{cases} \quad (2.6)$$

Equation 2.6 represents a discrete choice of location that is both exhaustive and mutually exclusive. This set-up coupled with the type I extreme value error distribution in the expected profit function allows for a closed form solution for location choice probabilities to be calculated for any given draw of market and location unobservables, ξ_m and ζ_{ml} . I will call the probability of choosing a_{iml} , the location choice probability. In this location choice probability, the expectation of rival entry in each location, $E[a_{i'ml}]$, will be replaced with some belief of the probability for rival entry, $P_{i'ml}$. Thus for a given $P_{i'ml}$, ξ_m and ζ_{ml} , the probability of entry for firm i in location l and in market m can be thought of as a best response function and defined as:

$$P_{iml} = \frac{\exp \left(x_{ml}\beta_i + z_{iml}\eta_i - \sum_{i' \neq i} \sum_{b=0}^{B-1} \sum_{k=1}^{\bar{L}} \delta_{ii'kl} P_{i'mk} 1(k \in b_l) + \sigma\xi_m + \rho\xi_{ml} \right)}{1 + \sum_{j=1}^{\bar{L}} \exp \left(x_{mj}\beta_i + z_{mj}\eta_i - \sum_{i' \neq i} \sum_{b=0}^{B-1} \sum_{k=1}^{\bar{L}} \delta_{ii'kj} P_{i'mk} 1(k \in b_j) + \sigma\xi_m + \rho\xi_{mj} \right)} \quad (2.7)$$

By construction, for each market where a firm is a potential entrant, $\sum_{l=1}^{\bar{L}} P_{iml} = 1$ and $\forall l \in \{1, \dots, \bar{L}\}$, $0 \leq P_{iml} \leq 1$. All firms construct their beliefs on the probability of rival entry in this manner. Let $P_m = (P_{im}, P_{i'm})$ be an $(\bar{L} \times N)$ vector that describes each firm's probability of entry for each location in a given market, along with their beliefs about rival entry. As the space of probabilities in the best response function in Equation 2.7 is compact and continuous in $P_{i'ml}$, the model has at least one Bayesian Nash Equilibrium by Brouwer's fixed point theorem. Thus,

equilibrium is an asymmetric Bayesian Nash Equilibrium, which is reached via a fixed point of the probability vector P_m .

For a given θ , ξ_M and ζ_{ml} , this fixed point is operationalized by using a recursive process: calculating a new P_m for each market and iteration and stopping at a predefined criterion. Let r index the particular iteration for the fixed point giving P_m^r as the choice probabilities for that iteration. Starting with choice probabilities of zero, P_m^0 for all alternatives, the vector P_m^1 is calculated using P_m^0 as the rival probabilities. This process continues until a fixed point is reached. This occurs when the sum of the error for each firm is less than some predetermined epsilon, i.e. $\sum_{i=1}^N error_i^r \leq \varepsilon$. The firm specific error for iteration r is calculated by first finding the absolute value of difference in location choice probability for each location-firm between r and $(r - 1)$, and then selecting the largest value of this difference across all market-locations. Thus, $error_i^r = \max_m |P_{iml}^r - P_{iml}^{r-1}|$.

In order to estimate the parameters of interest, $\theta = \{\beta, \eta, \delta, \sigma, \rho\}$, it is necessary to calculate equilibrium choice probabilities for any guess of θ such that they can be compared with one another. Calculating the equilibrium choice probabilities is only possible once both the market level and the spatially correlated location level error components are accounted for. Ideally, this would be done using closed form integration over the entire distribution of ξ_m and ζ_{ml} . Unfortunately, due to the construction of the choice probabilities and the fixed point, this closed form does not exist. As such, the integral will be simulated using randomized Halton sequences. Randomized Halton draws are well placed random draws which are negatively correlated with one another as the number of observations increases. Train (1999) found that 100 Halton draws had a lower simulation variance than 1000 random draws. This will allow for the calculation of simulated equilibrium choice probabilities, \hat{P}_{iml} for any guess of θ .

The procedure for calculating \hat{P}_{iml} for a given θ guess is as follows. First take two sets of $M \times D + 100$ randomized Halton draws from a standard normal distribution. Each set is generated from a different base prime and the first 100 draws are removed from each to avoid potential correlation (Train, 2009). Thus for each

market, m , there are D draws from the standard normal to represent the market-level unobservables, ξ_m , and D different draws from the standard normal to estimate the correlated location unobservables, ζ_{ml} . Let $d \in \{1, \dots, D\}$ index the order of draws for each market m . Second, for each d , take the corresponding realization of ξ_m^d and ζ_{ml}^d and calculate the fixed point equilibrium location probabilities as in Equation 2.7. Then average the D choice probabilities for each market, yielding \hat{P}_{iml} .

$$\begin{aligned} \hat{P}_{iml} &= \frac{1}{D} \sum_{d=1}^D P_{iml}^d \\ &= \frac{1}{D} \sum_{d=1}^D \frac{\exp\left(x_{ml}\beta_i^d + z_{iml}\eta_i^d - \sum_{i' \neq i} \sum_{b=0}^{B-1} \sum_{k=1}^L \delta_{i'kl}^d P_{i'mk}^d \mathbf{1}(k \in b_l) + \sigma^d \xi_m^d + \rho^d \zeta_{ml}^d\right)}{1 + \sum_{j=1}^{\bar{L}} \exp\left(x_{mj}\beta_i^d + z_{mj}\eta_i^d - \sum_{i' \neq i} \sum_{b=0}^{B-1} \sum_{k=1}^{\bar{L}} \delta_{i'kj}^d P_{i'mk}^d \mathbf{1}(k \in b_j) + \sigma^d \xi_m^d + \rho^d \zeta_{mj}^d\right)} \end{aligned} \quad (2.8)$$

Maximum Simulated Likelihood Estimation

Simulated maximum likelihood estimation (MSLE) will be used to calculate the value of θ that maximizes the simulated log-likelihood (SLL). The SLL is the likelihood of the observed clinic opening decisions given a guess of θ . Let y represent the data on retail clinic locations, such that the $(1 \times \bar{L})$ vector, y_{im} denotes the observed actions taken by firm i in market m . Also let the simulated choice probabilities, \hat{P}_{iml} , be defined as above. Thus for a given θ :

$$SLL(y, X, Z|\theta) = \sum_{m=1}^M \sum_{i=1}^N \sum_{l=1}^{\bar{L}} y_{iml} \ln(\hat{P}_{iml}) \quad (2.9)$$

The value of θ that maximizes the simulated log-likelihood is:

$$\hat{\theta}^{MSLE} = \arg \max_{\theta} SLL(y, X, Z|\theta) \quad (2.10)$$

The MSLE procedure for finding $\hat{\theta}^{MSLE}$ starts with an initial guess of θ , called θ^0 . Given θ^0 , the Bayesian Nash Equilibrium simulated choice probabilities are calculated as earlier outlined in Equation 2.7. These equilibrium probabilities are then combined with the observed data on outcomes, y , to determine an estimate for the Simulated Log-Likelihood conditional on θ^0 . As the calculation of the choice fixed point is nested within the MSLE procedure, a new guess for θ is then chosen and the process is recursively repeated, searching for a θ that maximizes the SSL and meets a pre-determined convergence criteria. Operationally, I use the non-linear optimization algorithm called the subplex method developed by Rowan (1990). The subplex method is a generalization of the Nelder-Mead simplex method. It more efficiently handles larger state spaces than Nelder-Mead and it is more robust to noise in the objective function.⁴ The optimization routine is run multiple times with a variety of initial guesses for θ . This is done because Brouwer's Fixed Point Theorem, while guaranteeing existence of an equilibrium, does not guarantee uniqueness. The use of multiple starting guesses addresses concerns that there may be multiple equilibria. In my model no matter what initial guess is used, the resulting $\hat{\theta}^{MSLE}$ is identical. This suggests that multiple equilibria are likely not a problem within my specification and that $\hat{\theta}^{MSLE}$ is likely the unique equilibrium. Standard errors are estimated using the Berndt, Hall, Hall and Hausman (BHHH) method and centered differencing as outlined in Train (2009). This is necessary as an analytical Hessian cannot be calculated due to the non-differentiability of the fixed point.

2.4 Results

The model of interest finds the parameter values, $\hat{\theta}^{MSLE}$, which maximize the likelihood that the expected profit equation from Equation 2.2 and related variables (X , Z , and W), generated the observed entry data, y for retail clinics. This specification

⁴A variety of non-linear optimization algorithms were used in various stages of this project. When running test estimates on datasets generated from known parameters and whose size and variance closely matched the real data used in this paper, the subplex method (while computationally costly and slow) performed significantly better than every other algorithm in that it always returned the true values no matter the initial guess.

incorporates variables that capture clinic demand, labor inputs, and unobserved heterogeneity. Rival proximity is explicitly captured through a structural framework as it is otherwise unobservable. Concerns of unobserved heterogeneity are addressed by using firm-specific covariates and a rich set of error components. The $\hat{\theta}^{MSLE}$, which results from the estimation procedure earlier outlined can be found in Table 2.4.

The results show that both operators prefer to open clinics in more populous areas, with CVS being more likely to open a MinuteClinic in block-groups that have large populations, and Walgreens preferring entry in those locations with a large surrounding population. While the significant coefficient on own-block population for CVS and the significant coefficient on population within 10-miles for Walgreens have different orders of magnitude, the effect at the mean of each variable is of a similar magnitude. Specifically for CVS at the mean of own population (1,746.144), the impact of its own-population (4.2688) results in 7,453 and for Walgreens at the mean of population within 10 miles (493,224.822), the impact of population within 10 miles (.0295) results in 14,550. Block groups whose population is composed of either a larger percent black or percent Hispanic are less likely to receive a clinic than otherwise similar locations. This is likely due to a concern that minority residents may be unwilling to frequent a retail clinic staffed by a white nurse practitioner. There is some basis for this concern as a disproportionate majority of nurse practitioners are white and minority groups are less likely to seek care from health care providers who are not of their own race (Hooker and Berlin, 2002; Saha et al., 2000). The finding of a negative relationship between profit and percent black and percent Hispanic indicates that retail clinics are not increasing primary care access to these traditionally under-served groups. In fact, these clinics are serving to increase access disparity in health care access between whites and non-whites by disproportionately opening clinics in white areas. This is not to say that retail clinics could not bolster access for these groups, but only that as currently operated, they are not finding it profitable to do so.

Both CVS and Walgreens are more likely to open clinics in areas where a greater percentage of their potential customers own their own cars. While this finding is

Table 2.4: Estimation Results for Specification of Interest

	CVS	Walgreens
Constant	-5.2709*** (0.5896)	-3.0943*** (0.6512)
Block Group Population (in 100k)	4.2688*** (1.4998)	-0.6199 (2.2788)
Population w/in 10 mi. (in 100k)	0.0167 (0.0137)	0.0295* (0.0165)
% Black	-1.1573*** (0.318)	-0.5633* (0.3278)
% Hispanic	-0.7955** (0.3563)	-0.5081 (0.4001)
% Some College	2.2935*** (0.2985)	0.1182 (0.3958)
% No Car	-1.3493*** (0.4823)	-0.9179 (0.6272)
% Insured	1.3396** (0.5963)	0.2778 (0.7579)
Household Median Income (in 10k)	0.0718*** (0.0159)	0.0457* (0.0239)
APRN per capita	0.0105 (0.0551)	0.1339 (0.0814)
Primary Care Physicians per 1000	-0.0086 (0.0157)	-0.0062 (0.0198)
# Urgent Care	0.2865** (0.1233)	0.3229** (0.1558)
Distance to Nearest Hospital	0.0091 (0.0172)	-0.011 (0.0305)
Distance to Nearest Affiliate	-2.6275* (1.4674)	-0.3118*** (0.0438)
# of stores in Market	-0.1228* (0.0653)	0.0509 (0.0619)
Drive-Through	0.6147*** (0.1369)	1.0251*** (0.1193)
Competitor in Block Group	1.7803 (1.1849)	0.9991 (0.9321)
Competitor within 2 miles	1.3898** (0.5815)	-0.0411 (0.4547)
ρ	0.0242 (0.8601)	
σ	0.0322 (0.1133)	
LL at Convergence	2790	
Pseudo R^2	.5865	
N	5738	

Standard Errors Reported in Parentheses

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

statistically significant for CVS, it is not for Walgreens, although it is of the same sign and of a similar magnitude. Both operators are also more likely to open clinics in wealthier areas as measured by median household income. Interesting differences between the two firms appear in their preference for both an educated and an insured customer base. CVS prefers their clinics to be located in block groups where the population has at least some college education. CVS also prefers to open clinics where customers have some form of insurance. Walgreens's clinic profitability is insensitive to both of these variables.

Neither CVS', nor Walgreens' clinic profitability is sensitive to the number of nurses per capita, the number of primary care physicians per 1000, or the distance to the nearest hospital. Despite having insignificant coefficients, these variables are still important to include as they control for unobserved features in each location that are related to underlying profitability of health care provision. However, the number of urgent cares in a specific block group is positively related with the probability of clinic entry for both CVS and Walgreens. Even though the urgent care may act as a competitor for a retail clinic, it is considered to be a non-strategic competitor. As retail clinics enjoy a lower operating cost than urgent cares, they are likely benefiting by siphoning away price sensitive patients from these institutions.

An increase in distance from each respective firm's nearest physician affiliate group reduces the probability that a retail clinic will be opened for both CVS and Walgreens. This distance influences CVS' decisions by an order of magnitude greater than it does for Walgreens'. This difference in effect is likely due to the difference in the number of clinical affiliations that each firm has. CVS has engaged in 32 of these agreements, while Walgreens has only 6. Therefore CVS has more thorough coverage of overseeing physicians in their most desirable locations for clinics than Walgreens does. As a result, there are fewer locations for CVS that are "uncovered" by a physicians group than there are for Walgreens and this small subset of uncovered locations for CVS is more likely to be considered undesirable for the placement of a clinic than similarly uncovered locations for Walgreens. The coefficients for the exclusion restrictions of drive-through status are both statistically significant in the

expected direction. This indicates that market and store level unobservables that would lead a firm to add a drive through are also positively related to retail clinic profitability. Illustrative examples of two such unobservables are store-size and store specific daily foot-traffic. Drive through windows are more likely present in both larger stores and in busier stores and retail clinics are more likely to be present in both bigger and busier stores.

Competition Effects

For CVS, the coefficient on the probability of a rival clinic entry within 2 miles of a potential location is both positive and statistically significant. This positive competition effect initially appears to be counter-intuitive. A natural critique of this finding would be that the positive coefficient is not being driven by rival proximity, but instead by unobserved market level heterogeneity. In other words, for a given market there may exist factors that are observed to CVS and Walgreens and are correlated with retail clinic profitability, but are unobserved by the econometrician. While I cannot use fixed effects to completely dismiss concerns of unobserved heterogeneity as I have cross-sectional data, I have used two main approaches to address these unobservables. I have included exclusion restrictions which have been shown to provide identifying variation in the presence of unobservables by Bajari et al. (2006) and I have included a rich error structure that allows for spatially correlated unobservables to impact location specific profit. After numerous attempts to control for unobservables, the positive competition effect is robust enough that it should be considered to be viable.

Consider the results displayed in Table 2.5. Here the first two columns are the result of a restricted version of Equation 2.2 that makes no attempt to control for unobserved heterogeneity. It contains neither the exclusion restrictions nor the unobserved error components. For CVS, the coefficients on rival probability of entry both in the same block group and within two miles are statistically significant at the 1% level and over twice as big as their counterparts from the mixed logit specification. Comparing (1) to (2), when the exclusion restrictions are included, both the magnitude and significance of the competition coefficients for CVS decrease. This is

Table 2.5: Competition Effects Across Alternative Specifications

	(1)		(2)		(3)	
	<u>No Exclusion Restrictions</u>		<u>No ξ_m or ξ_{ml}</u>		<u>Mixed Logit</u>	
	CVS	Walgreens	CVS	Walgreens	CVS	Walgreens
Competitor in Block Group	5.9378*** (2.0535)	0.3674 (0.8331)	1.9284 (1.1787)	1.1523 (0.9004)	1.7803 (1.1849)	0.9991 (0.9321)
Competitor within 2 miles	3.4611*** (0.8342)	-0.1016 (0.4117)	1.4228** (0.592)	-0.0345 (0.449)	1.3898** (0.5815)	-0.0411 (0.4547)
LL at Convergence	2908		2795		2790	
Pseudo R^2	0.5690		0.5857		0.5865	
N	5738		5738		5738	

Standard Errors Reported in Parentheses

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

due to the fact that the exclusion restrictions are removing much of the unobserved heterogeneity artificially inflating the coefficients in (1). Similarly comparing (2) to (3), the competition coefficient decreases when the error structure is allowed to account for correlated unobservable shocks.

Given that Table 2.5 evidences that unobservables are being at least partially accounted for in the specification of interest, there are two other broad channels through which positive returns to competition may be occurring, namely, demand externalities and learning. Demand externalities refer to the situation where one store may benefit its nearby rivals by drawing firm indifferent customers to an area. Learning occurs when there are uncertainties about market profitability and a firm can take another's location as a signal of market profitability. I will assume that learning is not possible in my model as firms make entry decisions simultaneously. This leaves demand externalities to be explored as the possible source of the positive competition effects.

Demand externalities have been studied by many, including Konishi (2005) and Yang (2014). The idea is that firms will locate near one another when the additional consumer demand this co-location creates offsets any potential business stealing effects. Specifically retail clinics may benefit from a demand informational spillover. As retail health clinics are a new innovation, consumers may be uninformed about the industry. This lack of knowledge may be about either the existence of retail clinics, their prices, or even their scope of practice. When a retail clinic enters

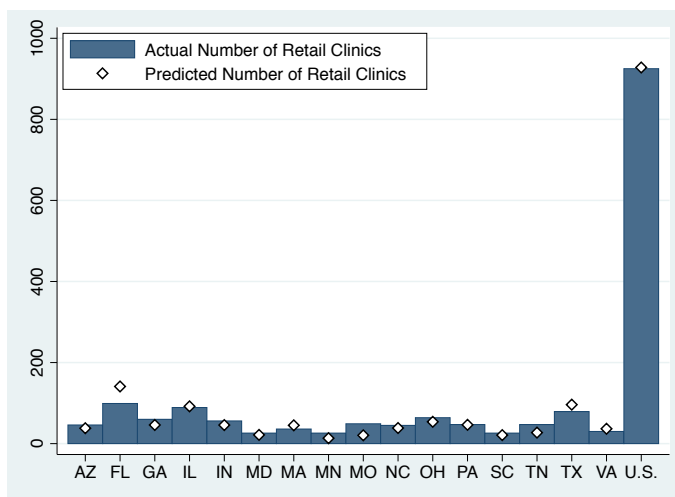
a market, it is common for them to advertise their presence. Consider a firm-indifferent consumer, who learns of a store from its advertising and then drives to that location. Once a consumer has arrived near the advertising retail clinic, their commute cost is sunk. If upon arrival she finds that there are two competing stores in the area, their indifference may lead them to equally chose either store.

An interesting and persistent feature of the results from Table 2.5 is the positive coefficient on competition is only present for CVS, while it is not statistically significant for Walgreens. This means that CVS is more likely to open a MinuteClinic in an area where they believe that Walgreens is likely to open a HealthCare Clinic. One potential explanation for this strategy is that CVS has a vertically integrated Pharmacy Benefit Manager (PBM) which has allowed the company to make more profit per claim than Walgreens in recent years. A Pharmacy Benefit Manager (PBM) is a service that administers the processing, filling, delivery, and claim payments related to prescription pharmaceuticals. CVS has a vertically integrated PBM, which is the second largest in the United States. Since 2011, Walgreens does not have its own PBM and relies on contracts with third parties for PBM related services. This vertical integration allows CVS to earn more revenue per customer than Walgreens, which translates into needing fewer daily customers than Walgreens to have a profitable retail clinic. Therefore if informational demand spillovers are occurring, CVS would benefit from them more benefit from them than Walgreens.

2.5 Model Accuracy and Counterfactual Analysis

The model predictions fit the observed distribution of retail clinics quite well. Figure 2.2 shows the actual and predicted number of clinics in those states with at least 25 retail clinics. The figure also shows the number of actual and predicted clinics in the 40 states that are considered in my entry model. In these states there are 925 actual clinics and my model predicts that there will be 928 clinics. At the state level my model's predictions for the number of retail clinics by each operator have 92.66% correlation with the actual number of MinuteClinics and a 85.36% correlation with

Figure 2.2: Actual and Predicted Retail Clinics by State



Note: For clarity of exposition only those states with at least 25 clinics are displayed. The U.S. column contains all 40 states that were included in the entry model.

the actual number of Healthcare Clinics.

A useful feature of structural analysis is the ability to perform out-of-sample predictions. I will apply my model to California as it was not included in the original analysis due to its corporate practice of medicine laws. This counter-factual analysis will be informative as it will allow for the inference as to how different California may be from the rest of the country with respect to retail clinic profitability. California is an ideal state for this type of analysis because it has a large number of pre-existing CVS and Walgreens retail locations and it has many diverse regions. Figure 2.3 shows a map of California with part (a) displaying the actual number of retail clinics per county and part (b) showing the counterfactual number of clinics in each California county. There is a significant under-entry of retail clinics in California. While I cannot say with certainty that this underdevelopment is due to CPOM laws, I can say that if California were like the 40 states used in my entry model, then there should be over 128 retail clinic in California, as opposed to the observed 43.

2.6 Conclusion

In this paper I examine retail clinics by modeling their location decisions. To do this, I employ a structural discrete choice framework where the choice is the outcome of an entry game of incomplete information with oligopolistic competition. Using a structural framework enables for the otherwise unobservable competition effect to be estimated as well as for useful counterfactual analyses to be performed. My analysis shows that clinics are more likely to locate in areas that are populous, wealthy, educated, and white, and that they are less likely to locate in traditionally under-served communities. This is not to say that retail clinics could not bolster access for these groups, but only that as currently operated, they are not finding it profitable to do so.

Figure 2.3: California Market Structure With:

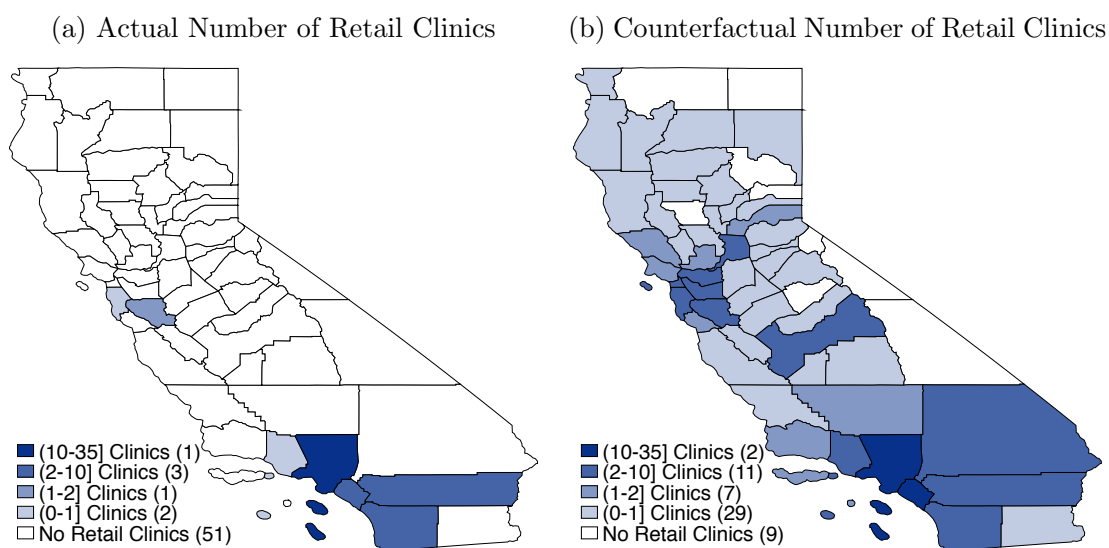


Table 2.6: Results Under Alternative Specifications

	Homogeneous Entrants as in Seim	Heterogeneous Entrants without Competition	
		CVS	Walgreens
Constant	-3.4334*** (0.3809)	-6.5097*** (0.6087)	-4.2907*** (0.7231)
Block Group Population (in 100k)	2.8804** (1.2563)	5.2085*** (1.4637)	-0.6736 (2.2662)
Population w/in 10 mi. (in 100k)	0.0356*** (0.0088)	0.0245* (0.0132)	0.0313* (0.0163)
% Black	-0.6084*** (0.2099)	-0.7742** (0.3142)	-0.4137 (0.3306)
% Hispanic	-0.65*** (0.2477)	-0.0061 (0.3595)	-0.1578 (0.4154)
% Some College	1.3276*** (0.2292)	2.4028*** (0.2996)	0.0806 (0.3906)
% No Car	-1.6637*** (0.362)	-0.8531* (0.4838)	-0.9767 (0.644)
% Insured	-0.0179 (0.4315)	2.0944*** (0.5986)	1.795** (0.796)
Household Median Income (in 10k)	0.0339*** (0.0123)	0.066*** (0.0158)	0.0297 (0.0237)
APRN per capita	0.0136 (0.0409)	0.0164 (0.0431)	0.1352* (0.0795)
PCP per 1000	-0.0084 (0.0103)	-0.0091 (0.0155)	-0.0057 (0.0198)
# Urgent Care	0.2727*** (0.0864)	0.3256*** (0.1236)	0.3542** (0.1533)
Distance to Nearest Hospital	0.0095 (0.0129)	0.0144 (0.017)	-0.0188 (0.0311)
Distance to Nearest Affiliate		-1.0323 (1.5107)	-0.3336*** (0.0438)
# of stores in Market		-0.0522 (0.0642)	0.0578 (0.061)
Drive-Through		0.946*** (0.1386)	1.0375*** (0.1209)
Competitor in Block Group	1.8207*** (0.5948)		
Competitor within 2 miles	1.1318*** (0.3173)		
LL at Convergence	3013	2795	
Pseudo R^2	0.5534	0.5857	
N	5738	5738	

Standard Errors Reported in Parentheses

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

CHAPTER 3

RETAIL HEALTH CLINICS: EMERGENCY DEPARTMENT DIVERSION

The recent increase in the number of Americans with health insurance due to the Affordable Care Act (ACA) has lowered the out of pocket cost of accessing health care services for many Americans. A lower cost of service will likely induce the newly insured to seek primary care services that were previously inaccessible to them. As there are not enough primary care physicians to adequately service the pre-ACA levels of demand, it is not clear how the U.S. health care system will accommodate the newly insured (HRSA, 2014b). There is some evidence that difficulty accessing primary care physicians will lead to an increase in unnecessary Emergency Department (ED) visits (Cowling et al., 2013). A shift of primary care provision to the ED is not a desirable outcome. Care in the ED is resource intensive and costs significantly more for an otherwise similar visit to a primary care physician or urgent care (CareFirst, 2014). In addition, over 60% of EDs in the United States are already operating at or over capacity “necessitating medical care to be provided in ED hallways and other makeshift examination areas” (Trzeciak and Rivers, 2003). Trask (2011) found that use of basic primary care services such as a free clinic was associated with reduced emergency room usage among uninsured patients. Retail clinics have made similar claims that they are able to help divert unnecessary ED visits.

This chapter evaluates the claim that retail clinics reduce unnecessary visits to nearby hospitals. To establish a causal relationship I must deal with two sources of potential endogeneity. First that the true impact of retail clinics on emergency room diversion may be muted by the endogenous location choices of profit maximizing clinic operators. In other words, retail clinics may find it more profitable to locate in areas where there are more emergency room visits to divert. To account for this first source of potential bias, the location predictions from the entry model serve

as an instrument for actual clinic locations. Predicted clinic locations are created using the structural model found in this chapter and are not a function of ED visits. Therefore by construction they are exogenous to the concern that a clinic locations might be responding to changes in ED visits. The second potential source of endogeneity is that urgent care facilities may be locating near retail clinics and may be driving any observed reductions in hospital visits. In order to address this concern I use the set of potential retail clinic locations prior to the rise of retail clinics as an instrument for observed clinic location.

I find that access to retail clinics causes a substantial decrease in the number of emergency department visits for bronchitis and upper respiratory infections but has no effect for visits related to other illnesses. In 2012, for the 40 states and territories with minimal barriers to retail clinic operation, the most conservative savings associated with retail clinic induced ED diversion are conservatively estimated to be over \$88 million. In California, counterfactual analysis suggests that relaxing the barriers to clinic entry would result in \$10.5 million in health care savings. While these are large and significant efficiency spillovers from retail clinics into the health care system, these efficiency gains will only be provided for hospitals in relatively more affluent areas, where the clinics are most likely to locate.

3.1 Data

Evaluation of retail clinics on nearby ED visits will be made possible by using the State Emergency Department Database (SEDD) (HCUP, 2012). SEDD is the ideal database to answer this question because it contains visits at hospital-affiliated EDs that do not result in admission to a hospital. Data on all 198 participating hospitals in Florida for 2006 and 2012 will be used in this analysis. This encounter level data is time coded in a manner that allows comparisons of the same hospital across time, facilitating a difference-in-differences estimation strategy. SEDD contains information on the primary reason for each visit, diagnoses, visit related charges, and time of visit. The primary reason or chief complaint driving the visit, as well as any

diagnoses, are recorded using the International Classification of Diseases version 9, or ICD-9. The ICD-9 code for the primary reason for the visit is the variable of interest in this analysis. The reason for this is that the choice of where to seek treatment is made by that patient based upon the disease that they believe they have. Consider a patient who has bronchitis and who visits an Emergency Department for treatment. If the patient sought treatment for bronchitis then this visit could have been diverted to a retail clinic. However if the patient had sought treatment for chest pains, but was diagnosed only with bronchitis, then this visit could not have been diverted as the patient would not have gone to or been seen by a retail clinic due to his chief complaint. In addition if a patient sought treatment for bronchitis and was not diagnosed with bronchitis, but with lung cancer, they are not included in the analysis. Stated differently, the analysis will be restricted to visits that could have been ex ante diverted rather than just visits that should have been ex post diverted. Table 3.1 shows the number of visits to each hospital in each year received by the primary reason for the visit. Reasons include the seven most common acute, episodic ailments treated at retail clinics, strep throat, bronchiolitis, and broken noses.

Table 3.1: Florida SEDD Summary Statistics, 2006 and 2012

Variable	Mean	Std. Dev.	Min.	Max.	N
Acute Upper Respiratory Infection	257.881	412.476	0	3379	396
Acute Bronchitis	79.861	156.505	0	1048	396
Acute Pharyngitis	540.073	475.736	0	3371	396
Otitis Externa	32.795	50.081	0	321	396
Otitis Media	3.722	11.062	0	165	396
Urinary Tract Infection	138.53	223.535	0	2135	396
Conjunctivitis	13.354	20.312	0	150	396
Streptococcal Sore Throat	27.457	75.614	0	752	396
Bronchiolitis	12.626	43.451	0	535	396
Broken Nose, (open and closed)	10.033	14.874	0	109	396

Both a difference-in-differences (DD) estimation strategy and a triple-difference strategy will be employed to test if the number of retail clinics within 5 miles of an ED causally reduces the number of ED visits for acute illnesses that are commonly

seen in retail clinics. In both frameworks the number of retail clinics will be the treatment. The DD estimator will compare ED visits by specific hospitals across treatment and across time. The triple difference will further restrict the analysis to the time of day in which a retail clinic is open. Retail clinics are said to be open between 9:00 AM and 7:00 PM on weekdays and between the hours of 9:00 AM and 5:00 PM on weekends. For a given ED i let the change in visits for a disease, d , from 2006 to 2012 be equal to:

$$\Delta Visits_{id} = Visits_{id}^{2012} - Visits_{id}^{2006} \quad (3.1)$$

For the same ED, let the change in visits for a disease across time and hours of operation be equal to:

$$\begin{aligned} \Delta\Delta Visits_{id} &= (Visits_{id}^{2012,RC=Open} - Visits_{id}^{2012,RC=Closed}) \\ &\quad - (Visits_{id}^{2006,RC=Open} - Visits_{id}^{2006,RC=Closed}) \\ &= \Delta Visits_{id}^{Open} - \Delta Visits_{id}^{Closed} \end{aligned} \quad (3.2)$$

Tables 3.2 and 3.3, display the relevant differences corresponding respectively to Equations 3.1 and 3.2.

Table 3.2: Summary Statistics for Dif-in-Dif

Variable	Mean	Std. Dev.	Min.	Max.	N
Δ Acute Upper Respiratory Infection	52.288	463.485	-3077	1717	198
Δ Acute Bronchitis	26.894	213.721	-828	968	198
Δ Acute Pharyngitis	35.076	370.699	-1644	1830	198
Δ Otitis Externa	-1.48	63.756	-278	289	198
Δ Otitis Media	-0.929	15.395	-165	41	198
Δ Urinary Tract Infection	37.949	283.304	-997	2012	198
Δ Conjunctivitis	-4.283	25.591	-132	112	198
Δ Streptococcal Sore Throat	5.076	100.83	-621	752	198
Δ Bronchiolitis	-0.152	60.363	-447	535	198
Δ Broken Nose, (open and closed)	-3.985	18.309	-109	71	198

Table 3.3: Summary Statistics for Triple Difference

Variable	Mean	Std. Dev.	Min.	Max.	N
$\Delta\Delta$ Acute Upper Respiratory Infection	22.944	86.647	-218	503	198
$\Delta\Delta$ Acute Bronchitis	9.419	50.238	-140	297	198
$\Delta\Delta$ Acute Pharyngitis	12.783	132.153	-1118	422	198
$\Delta\Delta$ Otitis Externa	0.439	15.688	-54	63	198
$\Delta\Delta$ Otitis Media	0.232	3.965	-18	25	198
$\Delta\Delta$ Urinary Tract Infection	12.263	53.579	-121	263	198
$\Delta\Delta$ Conjunctivitis	-0.848	7.91	-52	26	198
$\Delta\Delta$ Streptococcal Sore Throat	0.975	23.289	-129	192	198
$\Delta\Delta$ Bronchiolitis	0.505	12.011	-51	137	198
$\Delta\Delta$ Broken Nose, (open and closed)	-0.429	5.041	-23	17	198

3.2 Empirical Model

For both estimation frameworks, the specification of interest is set up as:

$$\text{Outcome}_{id} = \Delta \text{Retail Clinics within 5 mi.} \beta_{id} + \varepsilon_{id} \quad (3.3)$$

For the difference-in-differences estimation, $\text{Outcome}_{id} = \Delta \text{Visits}_{id}$, while for the triple differences framework, $\text{Outcome}_{id} = \Delta\Delta \text{Visits}_{id}$. In both cases Δ (Retail Clinics within 5 miles) is equivalent to the number of retail clinics in 2012 as the industry did not exist in Florida in 2006. A finding of a significant reduction in visits using only the difference-in-difference estimates cannot be interpreted as the causal impact of retail clinics on emergency room diversion to the extent that it does not account for underlying trends that are correlated with both the entry of retail clinics and with health improvements. Consider the case of a hospital in which its surrounding neighborhood is becomes wealthier from 2006 to 2012. As outlined in the previous chapter, retail clinics are more likely to locate in wealthier areas. Similarly, it is expected that as wealth increases, both health and access to primary care services increases, both of which would lead to a reduction in ED visits. If wealth is not included in the model, then a significant visits reduction could be incorrectly attributed to retail clinics as the true driver of the effect was

an unobserved increase in wealth.

Thus, in addition to the difference-in-difference framework, I also include a triple-difference framework. The triple-difference further compares the visits across the time of day because it is likely that retail clinics have the greatest potential to divert visits during their hours of operation. In order for an unobservable to bias a significant negative finding in the triple difference framework, the unobservable must be more negatively correlated with ED visits during retail clinic hours of operation than when retail clinics are closed. This approach dismisses threats of bias from unobservables like wealth, which should not differentially impact emergency department visits during the day vs the evening. The triple difference reduces the bias at the cost of throwing away useful data. For example it is possible that an individual who is ill early in the morning may wait to visit a retail clinic when it opens rather than go to the ED, when she would have simply gone to the ED if the retail clinic did not exist. This further biases the triple-difference away from finding the full impact of retail clinics. The results from both the difference-in-difference and triple difference specifications where the outcome of interest is actual retail clinic locations can be found in Table 3.4.

Results from Equation 3.3 and Table 3.4 cannot be taken as the causal impact of retail clinics on visits as there are two potential endogeneity problems. The first is endogeneity between retail clinic location choice and ED visits in an area. This endogeneity arises because retail clinics would profit more by seeing patients, thus they are likely to select into areas that have higher visits as this reflects a larger customer base. In order to account for this endogeneity, I will use an instrumental variables approach. I use the predicted number of retail clinics from my entry model as an instrument for actual retail clinic locations. This predicted value is constructed by selecting all block groups that are within 5 miles of each hospital. I then sum the conditional choice probabilities as calculated in Equation 2.7 for across the selected block groups and across both CVS and Walgreens. This procedure provides an expectation of the number of retail clinics that is driven by expected profit as outlined in Equation 2.2 and is not related to the number of ED visits as they do

Table 3.4: Results from All Difference-in-Differences and Triple Difference Estimations with no IV

Disease	$\Delta Visits$	$\Delta\Delta Visits$
Acute Upper Respiratory Infection	-21.55 (29.21)	-10.43* (6.28)
Acute Bronchitis	-32.54** (15.08)	-5.87 (3.60)
Acute Pharyngitis	40.92 (46.09)	12.94 (11.87)
Otitis Externa	-3.60 (4.54)	-1.13 (1.27)
Otitis Media	.59 (.85)	.22 (.25)
Urinary Tract Infection	-16.67 (18.84)	-4.41 (3.81)
Conjunctivitis	.67 (1.89)	-.01 (.68)
Streptococcal Sore Throat	-6.28 (4.48)	-1.42 (1.06)
Bronchiolitis	-2.11 (3.70)	.53 (.52)
Broken Nose (Open and Closed)	-1.44 (1.70)	-.08 (.43)
Observations	198	

Robust Standard Errors Reported in Parentheses

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Note: Each coefficient is from a separate regression

not enter into Equation 2.2. Thus, the instrument is exogenous to this source of endogeneity by construction. The first stage results of the regression of actual clinics on predicted clinics can be found in Table 3.5. The instrument performs quite well in predicting the existence of a retail clinic within 5 miles of a hospital. The coefficient implies that when our model predicts a retail clinic should exist, this is the case 70% of the time. The F-Statistic from this regression is 151.21, dismissing any concerns of a weak or irrelevant instrument. The results from running a separate 2SLS for each outcome in Tables 3.2 and 3.3 are displayed in Table 3.6. The specification of interest is the column, $\Delta\Delta$ *Visits*. An additional retail clinic within 5 miles of an ED reduces the number of yearly visits to that ED from Acute Bronchitis by 10.68 and from Acute Upper Respiratory Infections by nearly 15. Access to a retail clinic has no measurable effect on other outcomes of interest. When looking at the level of the individual hospital, these results may not initially seem to be of great economic importance, but once they are aggregated, their importance becomes clear. They indicate that in Florida in 2012 there were 1,399.08 fewer ED visits for bronchitis and 1,961.07 fewer ED visits for upper respiratory infections due to retail clinics.

Broken noses has been included as a placebo test. Broken noses are a good placebo test because retail clinics do not treat patients with broken noses and urgent cares do see patients with this ailment. A non-finding here is suggestive that the triple-difference framework is likely capturing a retail clinic effect rather than an improvement in overall health or an effect driven by urgent cares. In addition, it is possible that retail clinics could have the adverse effect of diverting some ED visits that would ideally not be seen in a retail clinic but in an ED. In order to capture this concept, Bronchiolitis has been included.

If clinics are locating in areas where there are more ED cases to divert, than this instrument would correct for that fact and the coefficients from the 2SLS specifications would be closer to zero than the specifications without the endogeneity correction. However, the coefficients found in Table 3.6 are both more negative and more precise than those coefficients in Table 3.4. This implies that endogeneity concerns related to clinics choosing locations with more ED visits are likely not an

issue. In addition, the fact that the correction yields more precise and more negative estimates indicates that the areas assigned a higher probability of receiving a retail clinic by the predictive model enjoy more diversions than those areas deemed less likely to have a retail clinic. In other words, retail clinics in affluent, populous, and white areas divert more ED visits than clinics in traditionally undeserved markets.

The triple-difference framework may yield biased results if there exists an unobservable that is more negatively correlated with ED visits during retail clinic hours of operation than when retail clinics are closed. Using the predicted number of clinics sweeps away many unobservables in this category, but it might not eliminate all sources of bias. It is possible that urgent care facilities, which have been rapidly growing in the past ten years, may be locating near retail clinics because both could be responding to similar demand shocks. While the predicted location instrument incorporates the locations of urgent cares in 2014, it does account for the opening of urgent cares from 2006 to 2012. Thus, the predicted location instrument may be prone to bias in that it is possible that urgent cares are driving some or all of the ED diversions rather than retail clinics. In order to correct for this bias a different instrumental variable will be used.

The instrument is the potential retail clinic locations in 2005, the year prior to the opening of the first CVS and Walgreens operated clinics. These data were collected from loopnet.com. A histogram of this dataset can be seen in Figure 3.1. In order for the triple difference results using this instrument to be biased due to urgent cares it would have to be the case that urgent cares located near CVS and Walgreens that were there before 2006 affect day time ED visits relative to night visits differently than those urgent cares near to newer Walgreens and CVS locations. The first stage of the instrument can be seen in Table 3.7. The coefficient is statistically significant and implies that areas with more CVS and Walgreens locations in 2006 are more likely to have retail clinics than locations with fewer drug-stores. Specifically an area with at least 15.7 CVS or Walgreens locations in 2006 is likely to have one retail clinic. The F-Statistic of the first stage is 136.1, which dismisses concerns of a weak instrument.

The results from running a separate 2SLS for each outcome are displayed in Table 3.8. The specification of interest is the column $\Delta\Delta$ *Visits*. If urgent care facilities are the actual drivers of the results found in in Table 3.6 than the use of available building stock as an instrument would result in coefficients that would be closer to zero. However, the coefficients found in Table 3.8 are statistically different than zero, and they are not statistically different than their analogous results in Table 3.6. Thus, the potential endogeneity concern that the diversion effect is being driven by the urgent cares can be set aside because these results indicate that the diversion is being caused by retail clinics not urgent cares. Specifically, the results show that an additional retail clinic within 5 miles of an ED reduces the number of yearly visits to that ED from Acute Bronchitis by 11.34 and from Acute Upper Respiratory Infections by nearly 18.63. As in the other specification, access to a retail clinic has no measurable effect on other outcomes of interest, including the placebo test of broken noses and bronchiolitis. Once aggregated, the results using this instrument indicate that in Florida in 2012 there were 1,485.54 (14%) fewer ED visits for bronchitis and 2,440.53 (7%) fewer ED visits for upper respiratory infections due to the introduction of retail clinics by CVS and Walgreens.

The fact that the coefficients in Table 3.8 are more negative and more precise than those coefficients in Table 3.4 implies that those areas with more CVS and Walgreens drugstores in 2006 see more diversions from retail clinics than those areas with fewer CVS and Walgreens drugstore in 2006. This could be driven by the fact that those areas with more drugstores would be able to refer or recommend to their customers that they visit the nearby location which has a retail clinic. In other words there appear to be complementarities between the number of drugstores in a location and the effectiveness of retail clinics in diverting ED visits.

3.3 Results and Welfare Analysis

Using the charge information contained for 2012 in the SEDD dataset for Florida, it is calculated that the average ED charges for an acute bronchitis visit were \$2,391.39

Figure 3.1: Year of Construction for Each CVS and Walgreens Location in Florida

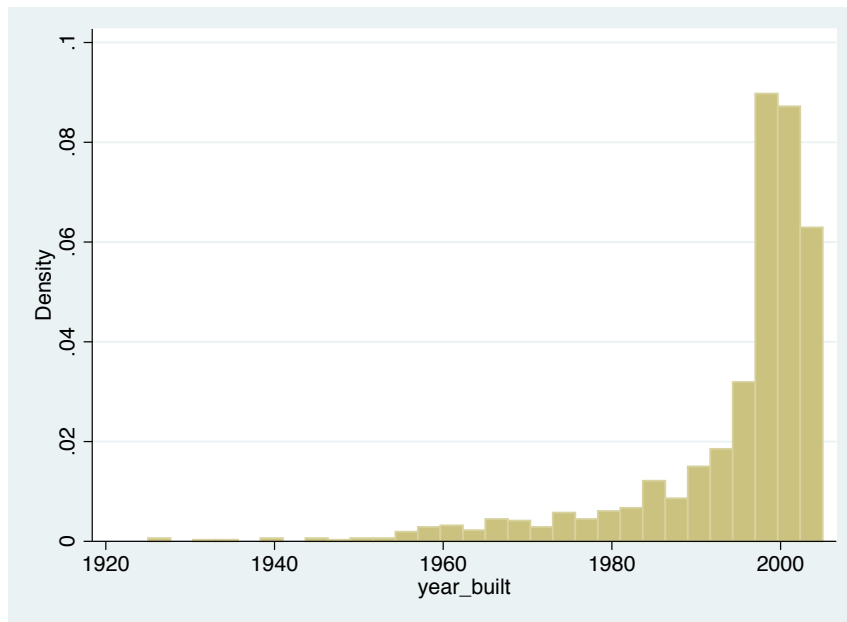


Table 3.5: First Stage Regression of Actual Number of Retail Clinics on Predicted Number of Retail Clinics within 5 miles of an ED

	(1)
Predicted Retail Clinics	.7230*** (.0588)
Constant	-.0345 (.0405)
F-stat	151.21
Observations	198

Robust standard errors in parentheses

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table 3.6: Results from All Difference-in-Differences and Triple Difference Estimations with Prediction as IV

Disease	$\Delta Visits$	$\Delta\Delta Visits$
Acute Upper Respiratory Infection	-24.16 (55.72)	-14.97* (8.39)
Acute Bronchitis	-40.77** (19.69)	-10.68** (4.94)
Acute Pharyngitis	46.62 (40.38)	18.43 (13.55)
Otitis Externa	-.40 (7.49)	-.63 (1.79)
Otitis Media	.50 (1.16)	.22 (.36)
Urinary Tract Infection	9.64 (35.81)	-1.49 (5.98)
Conjunctivitis	3.29 (2.52)	-.05 (.77)
Streptococcal Sore Throat	-7.41 (13.08)	-1.40 (3.07)
Bronchiolitis	1.04 (8.54)	1.73 (1.81)
Broken Nose (Open and Closed)	-.39 (2.32)	-.12 (.54)
Observations	198	
F-Stat of First Stage	151.21	

Robust Standard Errors Reported in Parentheses
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$
Note: Each coefficient is from a separate 2SLS regression

Table 3.7: First Stage Regression of Actual Number on Potential Clinic Locations before 2006 within 5 miles of an ED

	(1)
Potential Clinic Locations _{$t < 2006$}	.0673*** (.0058)
Constant	.0267 (.0449)
F-stat	136.1
Observations	198

Robust standard errors in parentheses
* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table 3.8: Results from All Difference-in-Differences and Triple Difference Estimations with Building Stock as IV

Disease	$\Delta Visits$	$\Delta\Delta Visits$
Acute Upper Respiratory Infection	-31.16 (63.16)	-18.63** (9.27)
Acute Bronchitis	-38.79** (23.22)	-11.34** (5.50)
Acute Pharyngitis	47.26 (40.36)	17.35 (13.75)
Otitis Externa	.40 (8.55)	-.36 (1.99)
Otitis Media	1.53 (1.66)	.18 (.41)
Urinary Tract Infection	16.75 (42.86)	-.30 (7.07)
Conjunctivitis	1.73 (2.86)	-.71 (.82)
Streptococcal Sore Throat	-6.13 (15.34)	-.92 (3.63)
Bronchiolitis	1.64 (10.25)	2.03 (2.23)
Broken Nose (Open and Closed)	.61 (2.58)	-.19 (.61)
Observations	198	
F-Stat of First Stage	136.1	

Robust Standard Errors Reported in Parentheses

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Note: Each coefficient is from a separate 2SLS regression

and the average ED charges for an acute upper respiratory visit were \$1,672. Thus when compared to the estimated cost of a retail clinic visit of \$75, each bronchitis visit that goes to a retail clinic rather than an ED, results in a system-wide savings of \$1,597. Similarly, each diverted upper respiratory visit saves \$2,316.39. Thus, using the most conservative estimates, retail clinics in Florida in 2012 were responsible for \$6,372,643.71 in system-wide savings due to emergency room diversion. Applying these results to the other 38 states and D.C. which were included in the entry model, it is estimated that retail clinics diverted at least 16,885 visits of bronchitis and 23,668 upper respiratory infection visits in 2012. Indicating that for the states included in the entry model, in 2012 through effective ED diversion retail clinics were responsible for saving over \$88 million dollars in health care costs. Extending this finding to the counterfactual predictions in California leads to the conclusion that the under-entry of clinics in California is leaving \$10.5 million in savings on the table.

3.4 Conclusion

In this chapter, I determine the impact of clinics on nearby emergency department visits using two different instrumental variable specifications. Both strategies provide similar estimates and thus lead to the conclusion that clinics divert a significant number of emergency department visits for both upper respiratory infections and for bronchitis. These results are robust to multiple specifications and are strengthened by the use of a placebo test. In 2012, for the states and territories with minimal barriers to retail clinic entry, the most conservative savings due to emergency room diversion caused by retail clinic was over \$88 million. In the same year in California, counterfactual analysis suggests that relaxing the barriers to clinic entry would have saved the state \$10.5 million in health care savings. While these spillovers are large, these gains will only be provided for hospitals in the areas where the clinics are most likely to locate.

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