Immigration Laws and Immigrant Health: Modeling the Spread of Tuberculosis in Arizona

Laura Catron [1], Ambar La Forgia[2], Dustin Padilla[3], Reynaldo Castro[3], Karen Rios-Soto[4], and Baojun Song[5]

[1]East Tennessee State University
[2]Swarthmore College
[3]Arizona State University
[4]University of Puerto Rico - Mayagüez
[5]Montclair State University

Abstract

The United States has observed a steady decline in the number of reported Tuberculosis (TB) cases in the past fifty years, but many states, such as Arizona, have had rates consistently above the US average. TB has been regarded as a disease of the disadvantaged, where poverty, overcrowding and malnourishment are responsible for much of the continued spread. Accordingly, the majority of TB cases in Arizona occur in the foreign-born population, whose households usually fall below the poverty line and have less access to adequate health care. Within this population, undocumented immigrants are the most socially and economically disadvantaged. Therefore, immigration laws, including some of the provisions in SB 1070, are likely to cause further marginalization as the increased fear of deportation will discourage undocumented individuals from seeking work and healthcare. Such laws could potentially exacerbate the spread of TB among undocumented immigrants and the low-income communities in which they reside, eventually extending to all socioeconomic classes. To observe the spread of TB in Arizona we employ a TB epidemic model that considers low and high income groups and accounts for different degrees of interaction within and between these socioeconomic classes. We also adjust the model parameters to simulate changes in behavior of undocumented immigrants before and after the implementation of an immigration law such as SB 1070.

1 Introduction

Over six million people die from Tuberculosis (TB), Malaria and HIV/AIDS each year, where about two million of these deaths are caused by TB [26]. Traditionally, TB is considered a disease of impoverished countries where overcrowding, unsanitary living conditions and malnourishment are still responsible for much of the continued spread. Therefore, it often afflicts developing nations. The United States has one of the lowest TB rates with only 4.6 reported cases per 100,000 people in 2007 [28], but this low number of TB cases masks the large differences between states and ethnicities. In fact, in 2007 the TB rate for US-born individuals was 2.3 per 100,000 people while the TB rate for foreign-born individuals was 21.9 per 100,000 people, and this ratio has been consistently increasing since cases started being recorded in 1953 [28]. Furthermore, in 2006, for the third consecutive year, more TB cases were reported among Hispanics than any other racial/ethnic population, with Mexico accounting for most cases [28]. Consequently, states with large Hispanic populations hold disproportionate TB rates. In fact, Mexico border states comprise a majority of all TB cases in the United States.

Border states are particulary most affected by foreign-born TB cases since approximately one million persons cross the U.S.-Mexico border daily [27]. With that large volume of influx there are three ways TB can be transmitted into the United States: 1) people with active TB disease move northward across the border, 2) people with latent TB infection experience active disease after arrival in the United States and 3) U.S. residents touring Mexico, including immigrants, acquire TB disease after returning to the United States [15]. After a person with TB enters the United States, further transmission might occur, which, in turn, contributes to TB morbidity in the United States directly from source patients and indirectly from their contacts. As a result of these scenarios, California, Texas, New Mexico and Arizona, and six Mexican states actively collaborate to address health issues at the border. Despite these efforts, illegal immigration prevents millions of people from being tested for TB upon entering the United States.

In 1998 the number of TB cases among Mexican-born individuals as a proportion of the total population was 43% for Arizona, 41% for New Mexico and 42% for Texas [17]. About ten years later, as seen in Figure 1, Mexican-born persons account for 57% of TB cases. This is coupled with a steady TB case rate between 4.6 and 5.5 per 100,000, which is consistently higher than the US rate for the past decade [30]. Arizona is also one of the states most afflicted by illegal immigration, and to address this issue, Governor Jan Brewer signed Senate Bill SB 1070. Which would legally allows law enforcement officials to question a suspect's immigration status if there is "reasonable suspicion" they entered the country illegally [1]. Arizona is not the only state to consider such polemic laws and immigration reform in general is continually receiving increased political attention.

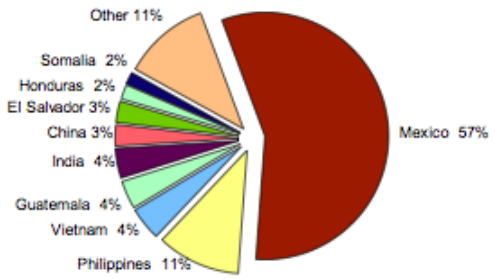

Figure 1: Country of Origin for Foreign-Born TB Cases, Arizona, 2007. Source: AZDHS Surveillance Report 2007

Some individuals in the medical community are in an uproar because of the health consequences of immigration laws such as SB 1070. Doctor Lucas Restrepo of the Barrows Neurological Institute in Phoenix wrote in the New England Journal of Medicine that "[the law] specifies that those who conceal, harbor or shield or attempt to conceal, harbor or shield a foreign person who came to the United States illicitly are guilty of a class 1 misdemeanor punishable by a fine of at least $1,000 (Sec. 5, Section 13-2929). It can be argued that health care providers who neglect to report illegal immigrants under their care will violate the law and be considered criminals." Yet the greatest fear physicians have is with the behavioral changes among undocumented immigrants. For example, after the law "Why would illegal immigrants, or legal immigrants without their papers handy, go to the emergency room or a healthcare center that can be policed?" posed Valerie Arkoosh, President of the National Physicians Alliance.

Therefore, immigration laws have the potential to completely isolate healthcare access to undocumented immigrants, who are already the least likely demographic to utilize healthcare. At least before a law, undocumented immigrants would utilize emergency health services when necessary and have access to free clinics [22, 34]. After a law, despite no definite denial of care, the fear of deportation as well as lack of clarity on free clinic access could cause a complete removal of all healthcare usage. Additionally, after a severe immigration law, undocumented immigrants will be changing their day-to-day lives to avoid conflict with police. Such measures include using more public transportation and staying near their communities, thus furthering their social isolation in the state. This combination of decreased health and increased social isolation does not bode well for the spread of communicable diseases such as TB, and will also affect those with pre-existing conditions.

TB treatment is very costly and requires up to six months of taking multiple drugs and patients often fail to follow the full regimen, only temporarily halting the disease [27]. Moreover, individuals with type 2 diabetes and HIV/AIDS are more susceptible to TB infection [13]. This is a problem for Hispanic immigrants in particular, whose change in eating patterns upon entering the United States results in an increased risk and prevalence of type 2 diabetes [9]. In fact, 33.4% of Arizona's hispanic population is obese, making it one of the six most obese states among Hispanics [10].

Consequently, immigration laws such as SB 1070 could foster the spread of TB within low-income communities where the majority of undocumented immigrants reside. Yet, other socioeconomic groups will also be at risk, since the social isolation created by the law will not completely limit contact. In fact, as undocumented immigrants lose work in restaurants, schools, and corporations, they will most likely turn to odd jobs, such as gardening and house cleaning, thus remaining in contact with individuals of all income levels though potentially at a reduced level. TB is a specific worry because the TB vaccine is rarely administered in the United States and even those vaccinated are still susceptible to multiple strains of TB especially after childhood [35]. Also, it is a highly contagious airborne disease, passed along through sneezing, coughing and breathing in contaminated droplets from the air. As a result, immigrations laws have the potential to foster the growth of TB among all socioeconomic groups.

This paper models the spread of TB in Arizona before and after the implementation of an immigration law. As outlined previously, Arizona will soon be in a unique situation because of it's large undocumented immigrant population, relatively high number of TB cases and the potential side effects of SB 1070 on immigrant health. Our prediction is that the prevalence of TB will grow among undocumented immigrants, creating a domino effect from this population to the general low-income populations in the communities in which they reside. Then, despite the reduced contact with more elite socioeconomic groups, there will still be mixing between all groups, actually increasing the probability of infection for high-income communities. To analyze this scenario we employ a TB epidemic model of susceptible, exposed and infected individuals, where infected individuals continuously return to the latent stage in which they have the disease but can not infect others. This is a modification of the TB model originally proposed by Waaler [31] and later developed by Feng, Castillo-Chavez and Capurro [11]. We then partition the population into two groups, low and high income, where parameters are adjusted to account for different levels of social contact between these two groups. Ultimately, this model captures the spread of TB within income groups and between income groups.

This paper is organized as follows: Section 2 explains the basic TB model in detail. Section 3 analyzes the basic control number and the case of a backward bifurcation. Section 4 establishes our parameters and presents before and after simulations of an immigration law on the spread of TB. Section 5 discusses a sensitivity analysis of our basic control number with respect to selected parameters as well as single-effort and multi-effort control methods to reduce the basic control number. Section 6 provides results and conclusions.

2 The TB Epidemic Model

Infection with Tuberculosis leads to active TB by one of three possible routes: primary progression after a recent infection, activation of a latent infection, or exogenous reinfection of a previously infected individual. Traditional mathematical models for exogenous reinfection, such as the TB model proposed by Feng, Castillo-Chavez and Capurro, use four epidemiological classes with susceptible, exposed and infectious stages as well as a treatment

stage. We simplify this model by removing the treatment stage and instead assume that all those infected with TB in our population never fully recover from TB, thus returning to the latent or exposed stage upon treatment. Upon entering the exposed stage, individuals can continually progress from exposed to infectious and back to exposed. Furthermore, we divide our population into two groups: a low-income class and a high-income class. Then, each group is divided into three epidemiological classes described in the table below:

Symbol, $i = L, H$	Name	Definition
S_i	Susceptible	Not infected but susceptible to TB infection
E_i	Exposed	Infected but unable to infect others
I_i	Infected	Active TB infection (individual is able to infect others)

Table 1: Symbols and Definitions of Populations for TB Model

$S_i(t)$ denotes the susceptible population at time t, $E_i(t)$ is the latently infected (assumed not infectious) class at time t, and $I_i(t)$ denotes the actively infected (assumed infectious) class at time. In order to determine the population size of the income groups in Arizona, we take the total number of individuals living in poverty (earning less than \$30,000) in a year as our low-income population. By assumption, undocumented immigrants are captured by these low-income communities. The high-income class captures all people earning more than \$30,000 a year and are by assumption the part of society least susceptible to TB and more removed from undocumented immigrants though they still have some degree of mixing. The system of ordinary differential equations for the spread of TB within the low-income group is given by

$$\begin{aligned}
\frac{dS_L}{dt} &= \Lambda_L - \beta_L \lambda_L S_L - \mu S_L, && \text{(1a)}\\
\frac{dE_L}{dt} &= \beta_L \lambda_L S_L - \mu E_L - q\beta_L E_L \lambda_L - kE_L + r_L I_L, && \text{(1b)}\\
\frac{dI_L}{dt} &= q\beta_L E_L \lambda_L + kE_L - r_L I_L - dI_L - \mu I_L, && \text{(1c)}\\
N_L &= S_L + E_L + I_L.
\end{aligned}$$

While the system for the high-income group is given by

$$\begin{aligned}
\frac{dS_H}{dt} &= \Lambda_H - \beta_H \lambda_H S_H - \mu S_H, && \text{(2a)}\\
\frac{dE_H}{dt} &= \beta_H \lambda_H S_H - \mu E_H - q\beta_H E_H \lambda_H - kE_H + r_H I_H, && \text{(2b)}\\
\frac{dI_H}{dt} &= q\beta_H E_H \lambda_H + kE_H - r_H I_H - dI_H - \mu I_H, && \text{(2c)}\\
N_H &= S_H + E_H + I_H.
\end{aligned}$$

Symbol, $i = L, H$	Explanation	Units
Λ_i	Recruitment rate	$\frac{1}{year}$
β_i	Infection rate for susceptible individuals by an infectious TB individual per contact per unit time	$\frac{1}{year}$
k	Per capita natural progression rate of latent TB to active TB	$\frac{1}{year}$
μ	Per capita natural death rate	$\frac{1}{year}$
d	Per capita excess death rate due to TB	$\frac{1}{year}$
q	Reduction in susceptibility of a latently-infected individual	Unitless
r_i	Per capita TB treatment rate (returns infected to exposed)	$\frac{1}{year}$

Table 2: Symbols and Definitions of Parameters for TB Model

We set $\beta_i = pC_i$, where p is the probability that a contact is effective for TB transmission given that a susceptible has contact with an actively infected individual and C_i is the average number of contacts of group i per person per unit time. Therefore, β_i is interpreted as the average number of effective contacts a susceptible has per unit of time. An important term for our model is λ_i, the force of transmission, since it captures the probability of mixing between groups and thus connects the six equations. It is given by

$$\lambda_i = \left[P_{iL} \left(\frac{I_L}{N_L} \right) + P_{iH} \left(\frac{I_H}{N_H} \right) \right],$$

where

$$P_{ij} = f_i \delta_{ij} + (1 - f_i) \frac{(1 - f_j) C_j N_j}{(1 - f_L) C_L N_L + (1 - f_H) C_H N_H} \tag{3}$$

and

$$\delta_{ij} = \begin{array}{ll} 1, & i = j \\ 0, & i \neq j \end{array}.$$

Above, P_{ij} represents the probability that an individual in group i contacts an individual in group j given group i had a contact, where f_i is an individuals preference for their own income group and $(1 - f_i)$ is an individuals preference for the other income group [4, 16]. When δ is equal to one, there exists preferred mixing only for one's own group. This means that, for example, the probability that a low-income individual interacts with another low-income individual (P_{LL}) is equal to the preference that the low-income individual has for low-income individuals (f_i), plus the preference that a low-income individual has for the rest of the population ($1-f_i$) multiplied by the random mixing that will occur with the remainder of low-income individuals as a proportion of the entire population. Furthermore, when f_i=f_j=0

the preference term is removed from the equation and there is simply proportional mixing between income classes. Therefore, by multiplying these probabilities with those infected as a proportion of each population, we see that λ_i captures how likely an individual is to come in contact with an infectious individual given their mixing preferences. Thus, the product of λ_i with β_i captures how likely an individual is to be infected given their mixing preference. The dynamics of the interactions between groups is best seen in the compartment model in Figure 2, where the dashed lines represent inter-group contact between susceptibles and infected.

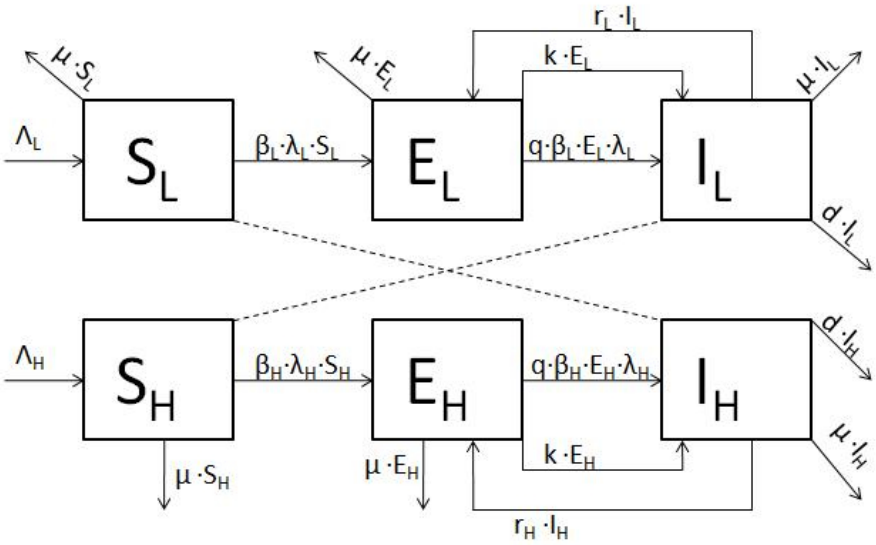

Figure 2: Flowchart of 2-Group TB Model

3 Mathematical Analysis

3.1 Disease-Free Equilibrium Point

The disease-free equilibrium is a steady-state solution by which there is no disease. Notice that in the absence of disease, $S_L = N_L, S_H = N_H, E_L = 0, E_H = 0, I_L = 0$,and $I_H = 0$. Thus, we have a disease-free equilibrium: DFE= $(S_L^*, S_H^*, E_L^*, E_H^*, I_L^*, I_H^*) = (\frac{\Lambda_L}{\mu}, \frac{\Lambda_H}{\mu}, 0, 0, 0, 0)$. The disease-free equilibrium will be used when computing the basic control number.

3.2 Basic Control Number

We use the next generation operator method to compute the basic control number where the basic control number, R_c, is a measure of the number of primary infections caused by

a "typical" infectious individual in a fully susceptible population during entire infectious period. If $R_c < 1$, the disease-free equilibrium point is locally asymptotically stable and the disease will die out in the population. While $R_c > 1$, the disease-free equilibrium is unstable and the disease will invade the population. Let $\mathcal{F}$ be a vector containing all new infections. That is, $\mathcal{F}$ will contain all rates from uninfected compartments into infected compartments. $\mathcal{V}$ will be a vector containing all other rates of transfer to all compartments. Thus,

$$\mathcal{F} = \begin{pmatrix} \beta_L S_L(P_{LL}\frac{I_L}{N_L} + P_{LH}\frac{I_H}{N_H}) \\ \beta_H S_H(P_{HL}\frac{I_L}{N_L} + P_{HH}\frac{I_H}{N_H}) \\ 0 \\ 0 \\ 0 \end{pmatrix},$$

and

$$\mathcal{V} = \begin{pmatrix} q\beta_L E_L(P_{LL}\frac{I_L}{N_L} + P_{LH}\frac{I_H}{N_H}) + E_L(\mu + k) - r_L I_L \\ q\beta_H E_H(P_{HL}\frac{I_L}{N_L} + P_{HH}\frac{I_H}{N_H}) + E_H(\mu + k) - r_H I_H \\ I_L(r_L + d + \mu) - kE_L - q\beta_L E_L(P_{LL}\frac{I_L}{N_L} + P_{LH}\frac{I_H}{N_H}) \\ I_H(r_H + d + \mu) - kE_H - q\beta_H E_H(P_{HL}\frac{I_L}{N_L} + P_{HH}\frac{I_H}{N_H}) \\ \beta_L S_L(P_{LL}\frac{I_L}{N_L} + P_{LH}\frac{I_H}{N_H}) + \mu S_L - \Lambda_L \\ \beta_H S_H(P_{HL}\frac{I_L}{N_L} + P_{HH}\frac{I_H}{N_H}) + \mu S_H - \Lambda_H \end{pmatrix}.$$

We then compute the Jacobian of each at the disease-free equilibrium. This gives us

$$D(\mathcal{F}) = \begin{pmatrix} 0 & 0 & \beta_L p_{LL} & \beta_L p_{LH}\frac{N_L^*}{N_H^*} & 0 & 0 \\ 0 & 0 & \beta_H p_{HL}\frac{N_H^*}{N_L^*} & \beta_H p_{HH} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix},$$

and

$$D(\mathcal{V}) = \begin{pmatrix} \mu + k & 0 & -r_L & 0 & 0 & 0 \\ 0 & \mu + k & 0 & -r_H & 0 & 0 \\ -k & 0 & r_L + d + \mu & 0 & 0 & 0 \\ 0 & -k & 0 & r_H + d + \mu & 0 & 0 \\ 0 & 0 & \beta_L p_{LL} & \beta_L p_{LH}\frac{N_L^*}{N_H^*} & \mu & 0 \\ 0 & 0 & \beta_H p_{HL}\frac{N_H^*}{N_L^*} & \beta_H p_{HH} & 0 & \mu \end{pmatrix}.$$

Where $N_L^* = \frac{\Lambda_L}{\mu}$ and $N_H^* = \frac{\Lambda_H}{\mu}$. Additionally, p_{ij} is P_{ij} evaluated at the disease-free equilibrium.

Therefore,

$$p_{ij} = f_i\delta_{ij} + (1-f_i)\frac{(1-f_j)C_j\frac{\Lambda_i}{\mu}}{(1-f_L)C_L\frac{\Lambda_L}{\mu} + (1-f_H)C_H\frac{\Lambda_H}{\mu}}.$$

Our reproductive number, R_c, is the dominant eigenvalue of matrix FV^{-1} where F and V are equal to

$$F = \begin{pmatrix} 0 & 0 & \beta_L p_{LL} & \beta_L p_{LH}\frac{N_L^*}{N_H^*} \\ 0 & 0 & \beta_H p_{HL}\frac{N_H^*}{N_L^*} & \beta_H p_{HH} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

and

$$V = \begin{pmatrix} \mu+k & 0 & -r_L & 0 \\ 0 & \mu+k & 0 & -r_H \\ -k & 0 & r_L+d+\mu & 0 \\ 0 & -k & 0 & r_H+d+\mu \end{pmatrix}.$$

Thus,

$$FV^{-1} = \begin{pmatrix} \frac{\beta_L p_{LL}k}{\Theta_L} & \frac{\beta_L p_{LH}k}{\Theta_H}\left(\frac{N_L^*}{N_H^*}\right) & \frac{\beta_L p_{LL}(\mu+k)}{\Theta_L} & \frac{\beta_L p_{LH}(\mu+k)}{\Theta_H}\left(\frac{N_L^*}{N_H^*}\right) \\ \frac{\beta_H p_{HL}k}{\Theta_L}\left(\frac{N_H^*}{N_L^*}\right) & \frac{\beta_H p_{HH}k}{\Theta_H} & \frac{\beta_H p_{HL}(\mu+k)}{\Theta_L}\left(\frac{N_H^*}{N_L^*}\right) & \frac{\beta_H p_{HH}(\mu+k)}{\Theta_H} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

where $\Theta_L = (\mu+k)(\mu+d+r_L) - kr_L$ and $\Theta_H = (\mu+k)(\mu+d+r_H) - kr_H$.

Notice that two of the eigenvalues of our matrix $FV^{-1} - \lambda I$ are zero. Therefore, we need only consider the matrix GZ^{-1} when determining our largest eigenvalue

$$GZ^{-1} = \begin{pmatrix} p_{LL}\frac{\beta_L k}{\Theta_L} & p_{LH}\frac{\beta_L k}{\Theta_H}\left(\frac{N_L^*}{N_H^*}\right) \\ p_{HL}\frac{\beta_H k}{\Theta_L}\left(\frac{N_H^*}{N_L^*}\right) & p_{HH}\frac{\beta_H k}{\Theta_H} \end{pmatrix}. \tag{4}$$

The above matrix can be expressed as:

$$GZ^{-1} = \begin{pmatrix} R_{LL} & R_{LH} \\ R_{HL} & R_{HH} \end{pmatrix}$$

where each entry in our matrix is a reproductive number in and of itself. R_{ij} is the expected number of primary infections in a fully susceptible population of group i produced by an infected individual in group j.

$$\lambda_{1,2} = \frac{(R_{LL} + R_{HH}) \pm \sqrt{(R_{LL} - R_{HH})^2 + 4(R_{HL}R_{LH})}}{2}.$$

It follows that the largest eigenvalue, our basic control number, is equal to

$$R_c = \frac{(R_{LL} + R_{HH}) + \sqrt{(R_{LL} - R_{HH})^2 + 4(R_{HL}R_{LH})}}{2}.$$

We can also consider an alternate form of our model where there is only proportionate mixing between groups. That is, all individuals in the population mix randomly, without reserved contact preference for certain individuals from their own group. If we recall from equation (3), this means that $f_i = 0$ and thus, the new mixing probabilities, which we will call p^*_{ij}, are now of the form:

$$p^*_{iL} = \frac{C_L N_L}{C_L N_L + C_H N_H}$$

and

$$p^*_{iH} = \frac{C_H N_H}{C_L N_L + C_H N_H}.$$

Under this condition and using the next generation operator method (which we outlined in this section), we were able to obtain a basic control number, which we will call R^a_c. That is,

$$R^a_c = \frac{C_L N_L}{C_L N_L + C_H N_H}\left(\frac{k\beta_L}{\Theta_L}\right) + \frac{C_2 N_2}{C_1 N_1 + C_2 N_2}\left(\frac{k\beta_H}{\Theta_H}\right) \tag{5}$$

where, once again, $\Theta_L = (\mu + k)(\mu + d + r_L) - kr_L$ and $\Theta_H = (\mu + k)(\mu + d + r_H) - kr_H$.

3.3 Interpretation of Basic Control Number

Recalling matrix (4), we can now look more closely at the biological interpretation of our basic control number R_c. If we examine the individual entries of matrix (4), we see that for an expression

$$\frac{\beta_i p_{ij} k}{(\mu + k)(\mu + d + r_i) - kr_i}$$

we can rewrite the expression as

$$p_{ij}\left(\frac{\beta_i}{\mu + k}\right)\left(\frac{k}{\mu + d + r_i}\right)\left(\frac{1}{1 - \frac{kr_i}{(k+\mu)(\mu+d+r_i)}}\right).$$

The term $\frac{\beta_i}{\mu+k}$ represents the average amount of time an individual spends in the latent class multiplied by the infection rate, β_i. Likewise, the term $\frac{k}{\mu+d+r_i}$ represents the average amount

of time an individual spends in the infectious class multiplied by the natural progression rate to the infectious class, k. The remaining term, $\frac{1}{1-x}$ with $x = \frac{kr_i}{(k+\mu)(\mu+d+r_i)}$, can be rewritten in the form of a geometric series: $1 + x + x^2 + x^3 + ...$. This series, along with the prior two expressions, represents the proportion of people moving through a cycle of entering the infectious class, returning to the latent class via treatment, and then once again returning to the infectious class. When multiplied by p_{ij}, which is the conditional probability that an individual in i contacts an infectious individual in j, the result is our basic control number, R_c. Notice that if $i \neq j$, we now need to take into account the ratio of individuals in population i with respect to population j. This ratio is accounted for by the factor $\frac{N_i}{N_j}$.

We can also look more closely at R_c^a for a biological interpretation. We can rewrite equation (5) as

$$R_c^a = \frac{C_L N_L}{C_L N_L + C_H N_H}\left(\frac{\beta_L}{\mu+k}\right)\left(\frac{k}{\mu+d+r_L}\right)\left(\frac{1}{1-x_L}\right) + \frac{C_H N_H}{C_L N_L + C_H N_H}\left(\frac{\beta_H}{\mu+k}\right)\left(\frac{k}{\mu+d+r_H}\right)\left(\frac{1}{1-x_H}\right)$$

where $x_L = \frac{kr_L}{(k+\mu)(\mu+d+r_L)}$ and $x_H = \frac{kr_H}{(k+\mu)(\mu+d+r_H)}$.

The terms $\frac{\beta_i}{\mu+k}$, $\frac{k}{\mu+d+r_i}$, and $\frac{1}{1-x_i}$ can be interpreted in the same way as illustrated above. The difference lies in the p_{ij}^* terms. Notice that the p_{ij}^* terms for this alternate model are no longer dependent upon contact mixing preferences.

3.4 Existence of Endemic Equilibrium

A possible state for our model is one in which TB exists within our population in an endemic state. Through a numerical approach, we can show that such a state exists for our model. To solve for the endemic equilibrium where $R_c > 1$, $\dot{S_L}$, $\dot{S_H}$, $\dot{E_L}$, $\dot{E_H}$, $\dot{I_L}$,and $\dot{I_H}$ are set equal to zero and we solve for S_L, S_H, E_L, E_H, I_L, and I_H. Notice that

$$\frac{dS_L}{dt} + \frac{dE_L}{dt} + \frac{dI_L}{dt} = \Lambda_L - \mu N_L - dI_L$$

and

$$\frac{dS_H}{dt} + \frac{dE_H}{dt} + \frac{dI_H}{dt} = \Lambda_H - \mu N_H - dI_H.$$

If we assume that death due to TB is very uncommon, $d \approx 0$. Thus, we can assume constant populations where $N_L = \frac{\Lambda_L}{\mu}$ and $N_H = \frac{\Lambda_H}{\mu}$.

From here, using a computer algebra system, we were able to reduce the systems to two equations $F_L(I_L, I_H)$ and $F_H(I_L, I_H)$, by expressing the equilibrium values for E_L, E_H, S_L, and S_H in terms of I_L and I_H. These equations are of the form

$$F_i(I_L, I_H) = q\beta_i E_i^* p_{iL}\frac{I_L}{N_L} + q\beta_i E_i^* p_{iH}\frac{I_H}{N_H} + kE_i^* - I_i(r_i + d + \mu)$$

where

$$S_i^* = \frac{\Lambda_i}{\beta_i p_{iL}\frac{I_L}{N_L} + \beta_i p_{iH}\frac{I_H}{N_H} + \mu}$$

and

$$E_i^* = \frac{1}{\mu}\left(\beta_i S_i^* p_{iL}\frac{I_L}{N_L}\right) + \frac{I_i}{\mu}\left(\beta_i p_{iH}\frac{S_H}{N_H} - \mu - d_i\right).$$

From here, we are able to reduce the system (1,2) yet again to one biologically relevant equation, $f(I_L)$, of the form

$$f(I_L) = A_7 I_L^7 + A_6 I_L^6 + A_5 I_L^5 + A_4 I_L^4 + A_3 I_L^3 + A_2 I_L^2 + A_1 I_L + A_0.$$

To show the existence of the endemic equilibrium, we now consider $f(0)$ and $f(N_L)$. However, because of the size of these expressions, we are unable to determine the behavior. Therefore, using parameters from Table 4, we will use a numerical approach to determine the behavior of our system for our particular parameter values. From here, we were able to show that $f(0) < 0$ and $f(N_L) > 0$. Thus, by the Intermediate Value Theorem, $f(I_L)$ must have a root and in turn, the endemic equilibrium must exist for our particular model.

We can now consider a sub-model of our model where each income group is independent of one another, having no interactions outside of their own group. That is, $f_i = 1$ and consequently, $\lambda_i = \frac{I_i}{N_i}$. From here, we can find an expression for the endemic equilibrium. To solve for the endemic equilibrium where $R_c > 1$, the equations for $\dot{S}_i$, $\dot{E}_i$, and $\dot{I}_i$ are all set equal to zero and we find the values of S_i, E_i, and I_i where $i = L$ or H. Notice that the equation for the total population is given by:

$$\frac{dS_i}{dt} + \frac{dE_i}{dt} + \frac{dI_i}{dt} = \Lambda_i - \mu N_i - dI_i.$$

Therefore,

$$N_i = \frac{\Lambda_i - dI_i}{\mu}.$$

and notice that

$$I_i \leq \frac{\Lambda_i}{d}.$$

This condition must hold since we are assuming our population is positive. Solving the system in terms of I_i, we obtain

$$S_i^* = \frac{\Lambda_i N_i}{\beta_i I_i^* + \mu N_i}$$

and

$$E_i^* = \frac{I_i^*}{\mu}\left(\beta_i \frac{S_i^*}{N_i} - \mu - d\right).$$

We can now reduce the system to one equation,

$$f(I_i) = (q\beta_i\mu I_i + k\Lambda_i - kdI_i)(\beta_i\Lambda_i - (\mu+d)(\Lambda_i + \beta_i I_i - dI_i)) - \mu(\mu+d+r_i)(\Lambda_i - dI_i)(\beta_i I_i - dI_i + \Lambda_i)$$

which can be expressed as:

$$f(I_i) = A_2 I_i^2 + A_1 I_i + A_0 I_i$$

.

The coefficients of this quadratic expression are

$$A_0 = -\Lambda_i^2((k+\mu)(\mu+d+r_i) - kr_i - k\beta_i),$$

$$A_1 = \Lambda_i(2d(k+\mu)(\mu+d+r_i) - 2kr_id - \beta_i(k+\mu)(\mu+d+r_i) - \beta_i kr_i - q\beta_i\mu d - q\beta_i\mu^2 + q\beta_i^2 + q\beta_i^2\mu - kd\beta_i),$$

and

$$A_2 = (\beta_i - d)(d(k+\mu)(\mu+d+r_i) - dkr_i - q\beta_i\mu d - q\beta_i\mu^2).$$

To show the existence of the endemic equilibrium, we now consider $f(0)$ and $f\left(\frac{\Lambda_i}{d}\right)$, respectively. That is,

$$f(0) = \Lambda_i^2(k\beta_i - k(\mu+d) - \mu(\mu+r_i+d))$$

and

$$f\left(\frac{\Lambda_i}{d}\right) = \left(q\beta_i\mu\frac{\Lambda_i}{d}\right)\left(-\mu\beta_i\frac{\Lambda_i}{d}\right).$$

Notice that since all parameters are positive, $f\left(\frac{\Lambda_i}{d}\right) < 0$. While rewritting $f(0)$ in terms of R_c^* we obtain

$$f(0) = \Lambda_i^2((k+\mu)(\mu+d+r_i) - kr_i)(R_c^* - 1)$$

where

$$R_c^* = \frac{k\beta_i}{(k+\mu)(\mu+d+r_i) - kr_i}.$$

Since the endemic equilibrium exists when $R_c^* > 1$, $f(0) > 0$. Thus, by the Intermediate Value Theorem, $f(I_i)$ has a root and, in turn, the endemic equilibrium must exist for this model.

3.5 Backward Bifurcation

In models with only two steady states, a disease free and endemic equilibrium, that exhibit a transcritical bifurcation, $R_0 \leq 1$ implies that the disease free state is stable, and thus the disease dies out, while $R_0 > 1$ implies that the endemic state is stable. Bifurcation theory has shown that TB models with exogenous reinfection, much like the one shown here, may exhibit a so-called backward bifurcation, rather than a forward transcritical one [7]. When $R_c < 1$ for a model which exhibits a backward bifurcation, there are three biologically feasible equilibria: a stable disease free equilibrium, a small unstable positive equilibrium and a larger stable endemic equilibrium. If backward bifurcation occurs, a small increase in the transmission rate β_i causes a large increase in the number of infected individuals but a subsequent small decrease in the transmission rate does not lead to the sudden disappearance of an endemic disease. Consequently, the occurrence of a backward bifurcation has important implications for the design of epidemiological control measures. Therefore, an epidemic may persist at steady state even if $R_c < 1$. Attention needs to be put toward the initial infection levels in order to guarantee being in the basin of attraction for the disease free equilibrium [7,11].

Figure 3 illustrates that a biologically relevant backward bifurcation exists for the TB model, provided that f_i is equal to 1. Under this condition, the populations have no interactions and the model for each income group has a backward bifurcation. For this, we can show that

Theorem 1. *Existence of Backward Bifurcation*

Under the condition that $f_i = 1$, it is necessary that in order for a backward bifurcation to exist,

$$\frac{q\beta_i^*\mu}{q\mu d + q\mu^2 + kd} > 1$$

and q, β_i^*, and μ must be greater than 0.

Proof. We chose β_i as the bifurcation parameter. Recall that our system of equations in terms of I_i is of the form

$$A_2 I_i^2 + A_1 I_i + A_0 = 0$$

where our coefficients A_2, A_1, and A_0 are expressions in terms of β_i. That is,

$$A_2(\beta_i) I_i^2 + A_1(\beta_i) I_i + A_0(\beta_i) = 0.$$

The characteristic which distinguishes backward bifurcations from other types of bifurcations is the behavior of the system at $R_c = 1$. The disease-free equilibrium is asymptotically stable when $R_c < 1$. However, at $R_c = 1$ the system undergoes a backward bifurcation. As opposed to a forward bifurcation, where the system would then assume a positive slope and travel along a stable endemic equilibrium, a system with a backward bifurcation assumes a negative slope and travels along an endemic equilibrium at $R_c < 1$. Therefore, we want to look at how $I_i(\beta_i)$ changes with β_i, especially at $I_i(\beta_i^*)$ where β_i^* corresponds to $R_c^* = 1$. Through implicit differentiation, we can examine this behavior.

$$A_2'(\beta_i) I_i^2 + 2 I_i I_i' A_2(\beta_i) + A_1'(\beta_i) I_i + I_i' A_1(\beta_i) + A_0'(\beta_i) = 0$$

We evaluate this function at $\beta_i = \beta_i^*$ where β_i^* is the value of β_i by which $R_c^* = 1$ and, noticing $I_i(\beta_i^*) = 0$ because we are in a disease-free state, the above equation becomes

$$I_i' A_1(\beta_i) + A_0'(\beta_i) = 0$$

or

$$I_i' = \frac{-A_0'(\beta_i^*)}{A_1(\beta_i^*)}.$$

Notice that if

$$\frac{-A_0'(\beta_i^*)}{A_1(\beta_i^*)} < 0$$

a backward bifurcation exists. In turn,

$$\frac{A_0'(\beta_i^*)}{A_1(\beta_i^*)} > 0.$$

For our model,

$$\frac{A_0'(\beta_i^*)}{A_1(\beta_i^*)} = \frac{k\Lambda_i}{2d(k+\mu)(\mu+d+r_i) - \beta_i^*(k+\mu)(\mu+d+r_i) - 2dkr_i + \beta_i^* kr_i + q(\beta_i^*)^2\mu - \beta_i^* q\mu d - q\beta_i^*\mu^2 - kd\beta_i^*}.$$

Clearly, $k\Lambda_i > 0$. Thus, we need only examine

$$2d(k+\mu)(\mu+d+r_i) - \beta_i^*(k+\mu)(\mu+d+r_i) - 2dkr_i + \beta_i^* kr_i + q(\beta_i^*)^2\mu - \beta)i^* q\mu d - q\beta_i^*\mu^2 - kd\beta_i^* > 0.$$

This can be rewritten in terms of our R_c^*. That is,

$$2d(k+\mu)(\mu+d+r_i)(1-R_c^*) + q(\beta_i^*)^2\mu > \beta_i^*(k+\mu)(\mu+d+r_i)(1-R_c^*) + \beta_i^* q\mu d + \beta_i^* q\mu^2 + \beta_i^* kd.$$

Recall that at $\beta_i = \beta_i^*, R_c^* = 1$. Thus,

$$\frac{q\beta_i^*\mu}{q\mu d + q\mu^2 + kd} > 1.$$

□

Notice that without q ($q = 0$), the parameter associated with exogenous reinfection, the condition is then

$$0 > 1$$

which is false. Consequently, this means that a backward bifurcation does not occur.

Notice that when in an environment where death due to tuberculosis is very uncommon($d_i \approx 0$), the condition for backward bifurcation reduces to

$$\frac{\beta_i^*}{\mu} > 1 \text{ or } \beta_i^* > \mu.$$

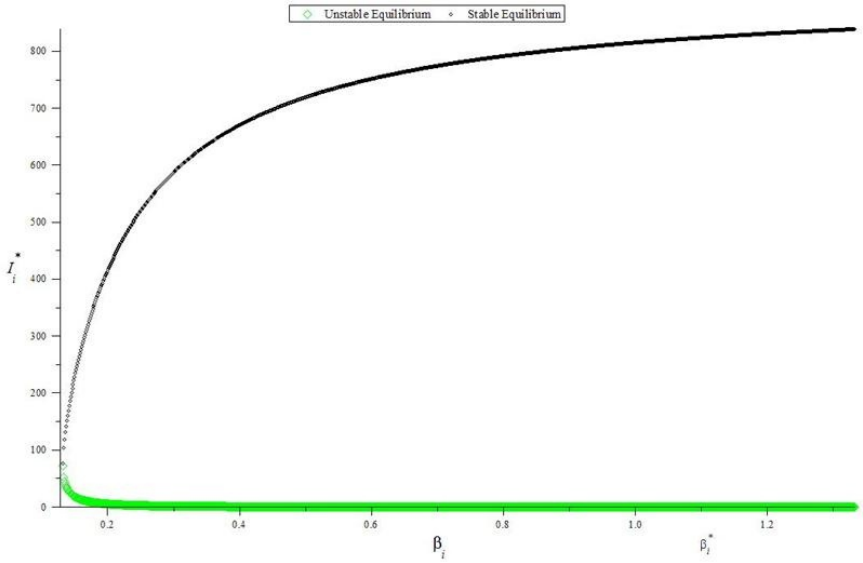

Figure 3: Backwards Bifurcation Diagram

In Figure 3, the x-axis corresponds to values for β and the y-axis are solutions to I_i, for specific parameter values (listed in Table 3) for the model. It can be shown that for the values of I_i in the diagram, there exists corresponding positive solutions for S_i and E_i. By further examining the backward bifurcation diagram, we observe that at the parameter value $\beta = 1.11$, $R_c = 1$.

Parameter	Value
Λ_i	1.0
μ	0.001
d	0.0001
k	0.00001
r_i	0.01
q	0.1

Table 3: Parameter Values Used for Backward Bifurcation

4 TB Model Parameter Estimation

Most parameter values were obtained from census data and previous TB model manuscripts as seen in Table 4.

Symbol, $i = L, H$	Explanation	Value	Reference
N_i	Total population	$N_L = 955,521$, $N_H = 5,544,964$	[2]
β_i	Infection rate for susceptible individuals	$\beta_L = 0.4$, $\beta_H = 0.2$	Estimated
C_i	Contact rate	$C_L = 5475$, $C_H = 3650$	Estimated
f_i	Reserved contacts	$f_L = 0.6$, $f_H = 0.6$	Estimated
r_i	Treatment rate	$r_L = 0.13$, $r_H = 0.7$	Estimated
k	Natural progression rate	0.005	[7]
μ	Natural death rate	0.00694	[36]
d	Death rate due to TB	$0.00000002 \approx 0$	[36]
q	Reduction in susceptibility of latently-infected	0.4	[7]

Table 4: Initial Conditions for Parameters

To establish the low-income and high-income populations, we used census data for 2007 to find the proportion of people living in poverty, or those making less than $30,000 a year. All persons under this level are in the low-income class (N_L), and all others are in the high-income class (N_H). To determine contacts made per year, we assume that the low-income class comes in contact with more people on a daily basis because they tend to live in larger households, more densely populated communities and are more likely to utilize public transportation. Therefore, we assume a low-income individual, on average, comes into close contact with 15 people per day while a high-income person makes 10 close contacts per day. Respectively, this results in 5475 and 3650 close contacts per year. In order to determine the birth rates, we plotted the population growth of our low-income group against time and fit an exponential curve, estimating a 0.041 per capita birth rate. Using the same method, the high-income group per capita birth rate was found to be 0.035. With both of these values, Figure 4 illustrates our model fit against the total population growth data for Arizona.

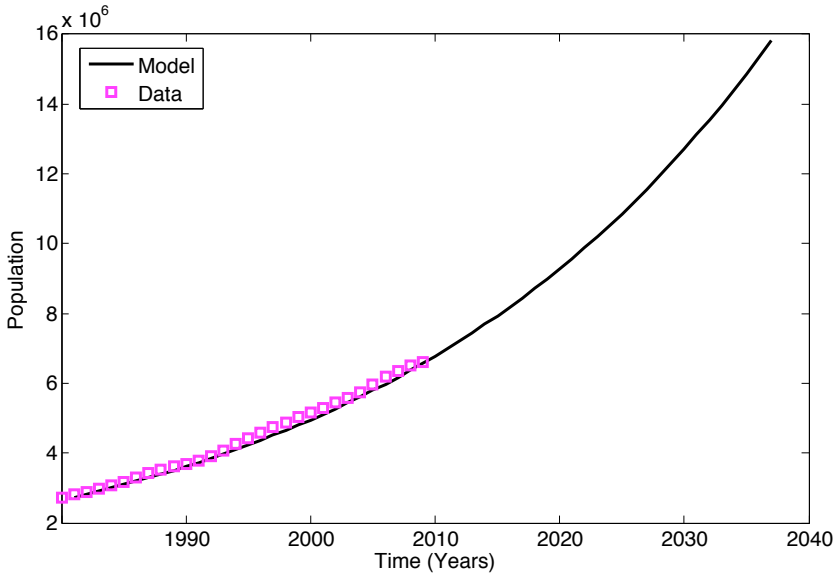

Figure 4: Exponential curve fitting total population of Arizona.

Before an immigration law such as SB 1070 passes, we assume that both low-income and high-income groups have some preference for their own groups, meaning they reserve contacts for people in their same socioeconomic class. Therefore, probabilities of 0.6 are assigned to f_i for each group. For our other varying parameters, β_i and r_i, we used TB cases for the past ten years to estimate the most appropriate values. As seen in Figure 5, the actual TB data is sporadic and difficult to fit (most likely because the data is incomplete due to unknown cases of TB), but our model does follow the general trend, suggesting plausible parameters. Furthermore, in this case, our basic control number,R_c is 1.55, confirming that TB is prevalent before a law such as SB 1070 even passes. This follows the general trend of the actual data, which has slightly increasing number of infectious TB cases.

Our parameter values are also realistic in context because we assume β_i is lower for low-income communities who are more likely to come in contact with infectious individuals. This is because this population has lower healthcare access, higher rates of diabetes and HIV/AIDS, making them more susceptible to TB. As a result, it makes sense to have a before law value of 0.4 for the low-income group and 0.2 for the high-income group. The treatment rate, r_i, also varies. Even though some counties in Arizona provide free treatment to citizens, often times low-income patients are unaware they are infected and do not know of the resources available for treatment. Therefore, we assume a low use of treatment with $r_L = 0.13$, meaning 13% of actively infected low-income individuals seek treatment in a year and $r_H = 0.7$, meaning 70% of actively infected high-income individuals seek treatment in a year. Lastly, our values for k and q mimic results of previous papers modeling TB in the United States.

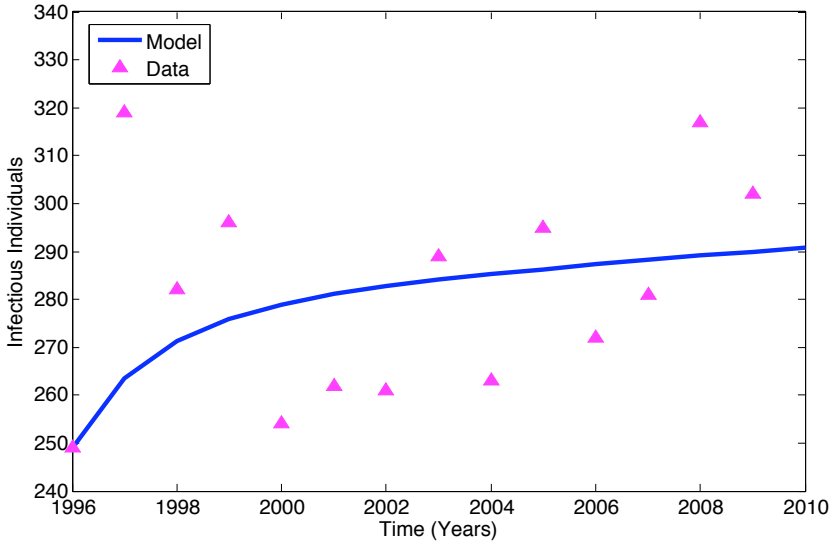

Figure 5: Fitting TB Model to Actual TB Prevalence in Arizona by Year

4.1 Simulations for Post Immigration Law Scenarios

The simulations presented below model two before and after scenarios for the effects of the spread of TB in Arizona. All the simulations measure time in years from 2010 (the year of law enactment) to 2050, to best capture the long-term effect of an immigration law on TB dynamics. The parameter values used are provided in Table 5.

Simulation	β_i		f_i		r_i		R_c	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
Scenario 1	$\beta_L = 0.4$ $\beta_H = 0.2$	$\beta_L = 0.8$ $\beta_H = 0.4$	$f_L = 0.6$ $f_H = 0.6$	$f_L = 0.8$ $f_H = 0.6$	$r_L = 0.13$ $r_H = 0.7$	$r_L = 0.08$ $r_H = 0.7$	1.55	5.75
Scenario 2	$\beta_L = 0.4$ $\beta_H = 0.2$	$\beta_L = 0.6$ $\beta_H = 0.3$	$f_L = 0.6$ $f_H = 0.6$	$f_L = 0.8$ $f_H = 0.6$	$r_L = 0.13$ $r_H = 0.7$	$r_L = 0.10$ $r_H = 0.7$	1.55	3.54

Table 5: Parameter Values for Scenario 1 and 2

Scenario 1 simulates a worst-case scenario after the implementation of an immigration law. We double β_L to represent a large increase in susceptibility for the low-income community given the decrease in health access of the undocumented immigrant population. The treatment rate r_L also decreases so that only 8% of the population receives treatment after a year. Lastly, the reserved contact rate f_L increases because undocumented immigrants will most likely become more reclusive and remain within their communities due to fear of deportation. As seen in Figure 6, immigration laws such as SB 1070 have a drastic effect in the increase of TB among the low-income population, increasing from 200 cases to 700 cases

in 40 years. Furthermore, despite the decreased contact with high-income groups, there is a slight increase in TB cases for this socioeconomic group as well. However, the cases only increase from 50 cases to 70 cases, which is not a dramatic increase given the large changes in parameter values.

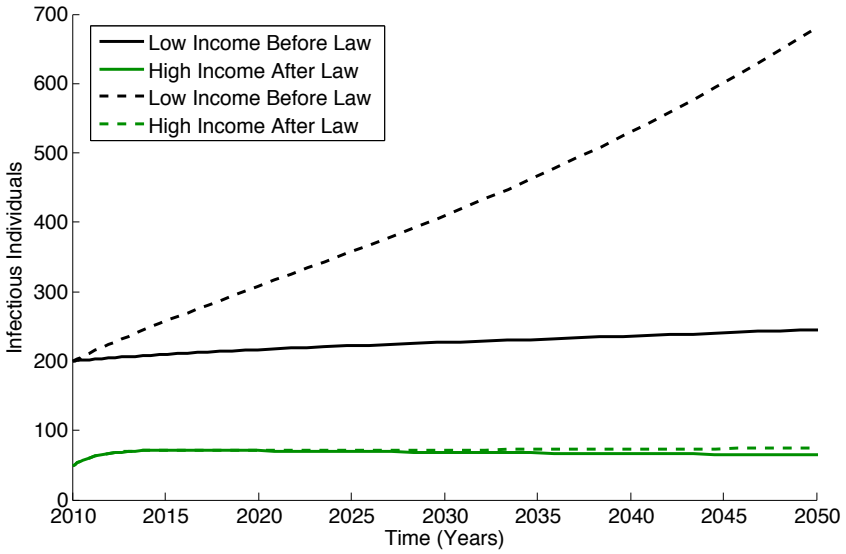

Figure 6: Scenario 1 (Extreme) for Pre and Post Immigration Law Simulation

Scenario 2 simulates a more plausible scenario after an immigration law. Both infection rates are increased modestly and r_L only decreases from 0.12 to 0.10. Despite these small changes, there is still a substantial increase in the number of infectious individuals among the low-income population, more than doubling from 200 to 450 TB cases. Though contrary to our initial prediction, there is virtually no effect on high-income individuals.

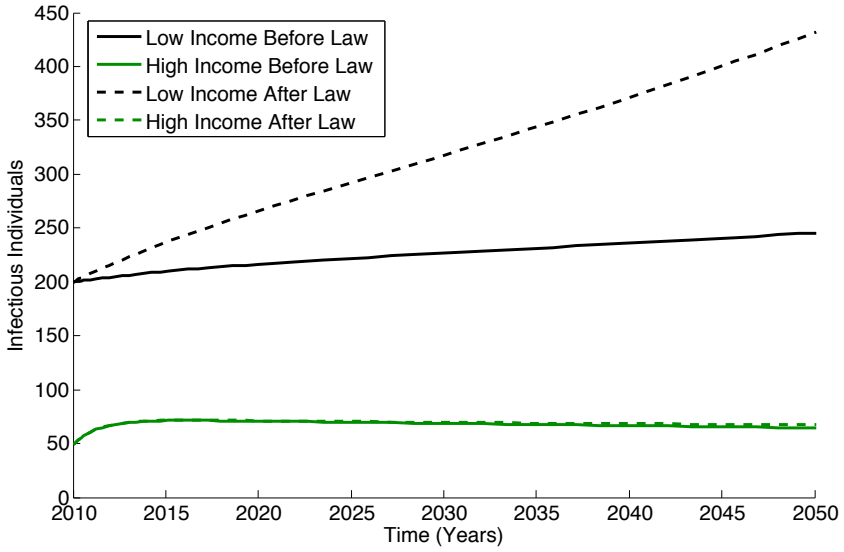

Figure 7: Scenario 2 (Moderate) for Pre and Post Immigration Law Simulation

However, a limitation to our previous scenarios is that our mixing preferences may be too strict. This is because our low-income cut off at $30,000 dollars does not necessarily capture the separation between low and high income groups and so individuals for a whole range of income levels would be mixing. Therefore, preferences may not be as pronounced as initially assumed and both classes can be treated as interacting at a more random level. To account for this, we introduce two more scenarios where the initial conditions for each f_i change from from 0.6 to 0.3. Table 6 provides the new parameter values for these new scenarios.

Simulation	β_i		f_i		r_i		R_c	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
Scenario 3	$\beta_L = 0.4$ $\beta_H = 0.2$	$\beta_L = 0.8$ $\beta_H = 0.4$	$f_L = 0.3$ $f_H = 0.3$	$f_L = 0.5$ $f_H = 0.3$	$r_L = 0.13$ $r_H = 0.7$	$r_L = 0.08$ $r_H = 0.7$	1.05	4.07
Scenario 4	$\beta_L = 0.4$ $\beta_H = 0.2$	$\beta_L = 0.6$ $\beta_H = 0.3$	$f_L = 0.3$ $f_H = 0.3$	$f_L = 0.5$ $f_H = 0.3$	$r_L = 0.13$ $r_H = 0.7$	$r_L = 0.10$ $r_H = 0.7$	1.05	2.52

Table 6: Parameter Values for Scenario 3 and 4

In Scenario 3, we model a worst case scenario under the assumption of more inter-group mixing (lower f_i). Under these assumptions, the increase in infected individuals in the high-income group is larger than Scenario 1, doubling from 50 infected individuals to 100 over 40 years. This is because our high-income group is interacting more with potentially susceptible members of the low-income group. Additionally, the number of our low-income cases triples from 200 to 600. This is less than in Scenario 1 because a disease is more likely to spread

in a contained population. Therefore, decreasing f_i will reduce the number of infections for the low-income group but increase the number of infections for the high-income group.

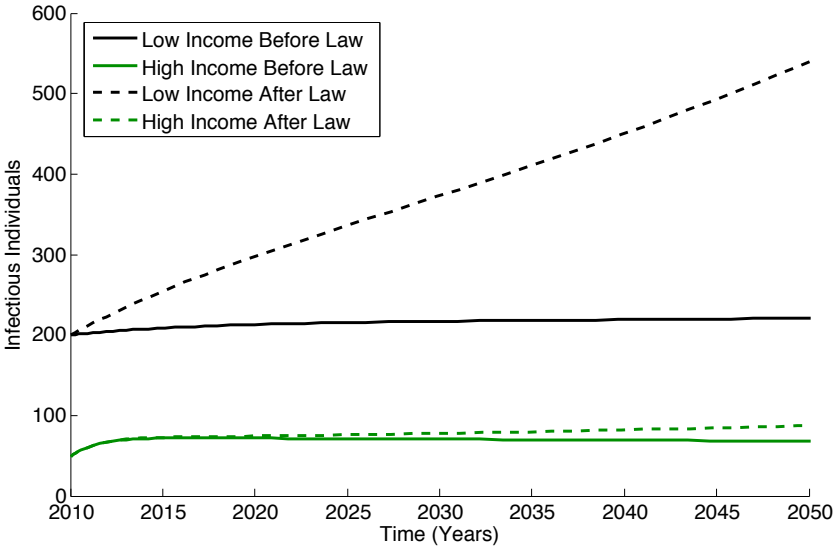

Figure 8: Scenario 3 (Extreme) for Pre and Post Immigration Law Simulation

In Scenario 4, we mimic Scenario 2 except under the assumption of a lower f_i. The results follow a similar relationship to Scenario 1 and 3, where our high-income population experiences more infections over time and our low-income population less infections. As one of our more plausible scenarios, it is important to notice the increase in the number of infectious TB cases for both income groups.

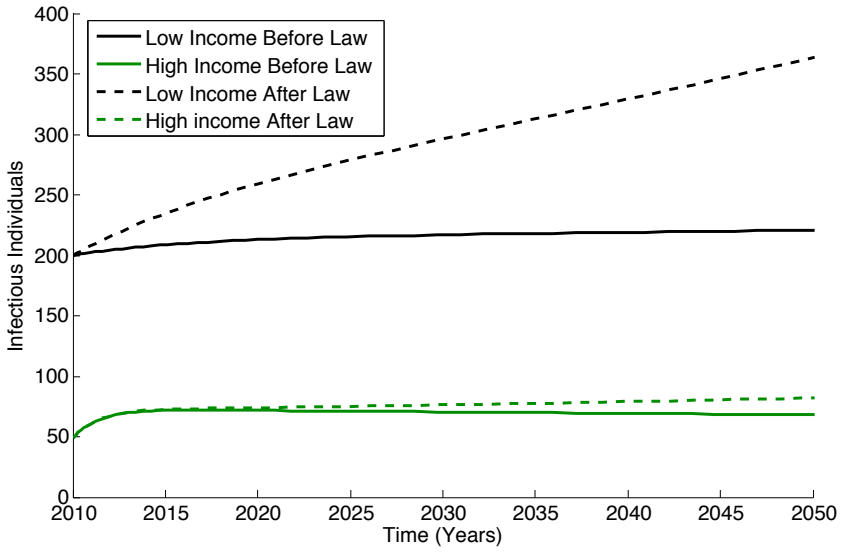

Figure 9: Scenario 4 (Moderate) for Pre and Post Immigration Law Simulation

5 Sensitivity Analysis of R_c

Sensitivity analysis provides us with important insight into what effect the parameters in our model have on our control reproductive number. By making small perturbations to certain parameters, our sensitivity analysis unveils to us which of these parameters are most sensitive. The parameters of particular interest to us are β_L, β_H, r_L, r_H, f_L, and f_H.

It should be noted that our control reproductive number is in terms of R_{LL}, R_{LH}, R_{HL}, and R_{HH}. Therefore, when evaluating $\frac{\partial R_c}{\partial p_k}$ for some parameter p_k, we must use the chain rule. This gives us

$$\frac{\partial R_c}{\partial p_k} = \frac{\partial R_c}{\partial R_{LL}}\frac{\partial R_{LL}}{\partial p_k} + \frac{\partial R_c}{\partial R_{LH}}\frac{\partial R_{LH}}{\partial p_k} + \frac{\partial R_c}{\partial R_{HL}}\frac{\partial R_{HL}}{\partial p_k} + \frac{\partial R_c}{\partial R_{HH}}\frac{\partial R_{HH}}{\partial p_k}.$$

Thus, our normalized sensitivity index for a given parameter p_k will be defined as

$$S_{p_k} = \frac{p_k}{R_c}\frac{\partial R_c}{\partial p_k}.$$

Table 1 shows the sensitivity indeces and the associated percent needed to affect a 1% decrease in R_c. Notice that $\frac{1}{S_{p_k}}$ will provide us with the percent of change needed to change the R_c by roughly 1%.

Sensitivity Index	Value
S_{β_L}	0.9998
S_{β_H}	0.0065
S_{r_L}	-0.9118
S_{r_H}	-0.1208
S_{f_L}	0.5472
S_{f_H}	0.1414

Table 7: Sensitivity Analysis of R_c

We found that the most sensitive parameters to R_c were β_L and r_L, respectively. The sensitivity index $S_{\beta_L} = 0.9998$ means that a 1.0002% decrease in β_L results in roughly a 1% decrease in R_c. The sensitivity index $S_{\beta_H} = 0.0065$ means that a 153.846% decrease in β_H results in roughly a 1% decrease in R_c. The sensitivity index $S_{r_L} = -0.9118$ means that a 1.097% increase in r_L results in roughly a 1% decrease in R_c. The sensitivity index $S_{r_H} = -0.1208$ means that a 8.278% increase in r_H results in roughly a 1% decrease in R_c. Similarly, sensitivity index $S_{f_L} = 0.5472$ means that a 1.828% decrease in f_L results in roughly a 1% decrease in R_c. Lastly, the sensitivity index $S_{f_H} = 0.1414$ means that a 7.072% decrease in f_H results in roughly a 1% decrease in R_c.

5.1 Reducing R_c through Single-Effort and Multi-Effort Control Methods

Though single-effort control methods we can determine the minimum change that should be made to an individual parameter in order to decrease R_c to less than 1. Recall that the value of R_c prior to the enactment of a law such as SB 1070 is 1.555. This means that R_c must decrease by roughly 35.756% to fall just below 1. Through single effort control methods, the only feasible options for this to occur are to increase r_L to 0.211 or to decrease f_1 to 0.06. That translates to a 61.6% increase in treatment rates for low-income individuals or a 90% decrease in low-income individuals contact preference, the latter condition being more difficult to realistically meet. By plotting the effect of r_L and r_H on our R_c, we are able to visually witness the influence of increased treatment in each income group. Notice that even with an increase in treatment rate to 1.0 in the high-income group, our R_c still remains above 1, most likely contributed to the relatively small number of infectious individuals.

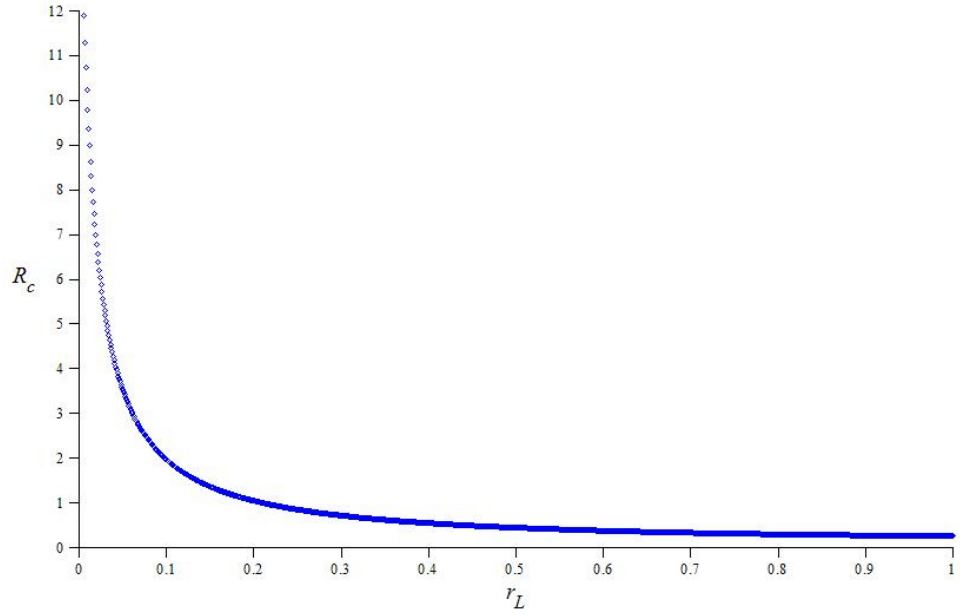

Figure 10: Low-Income Treatment Rate versus Basic Control Number

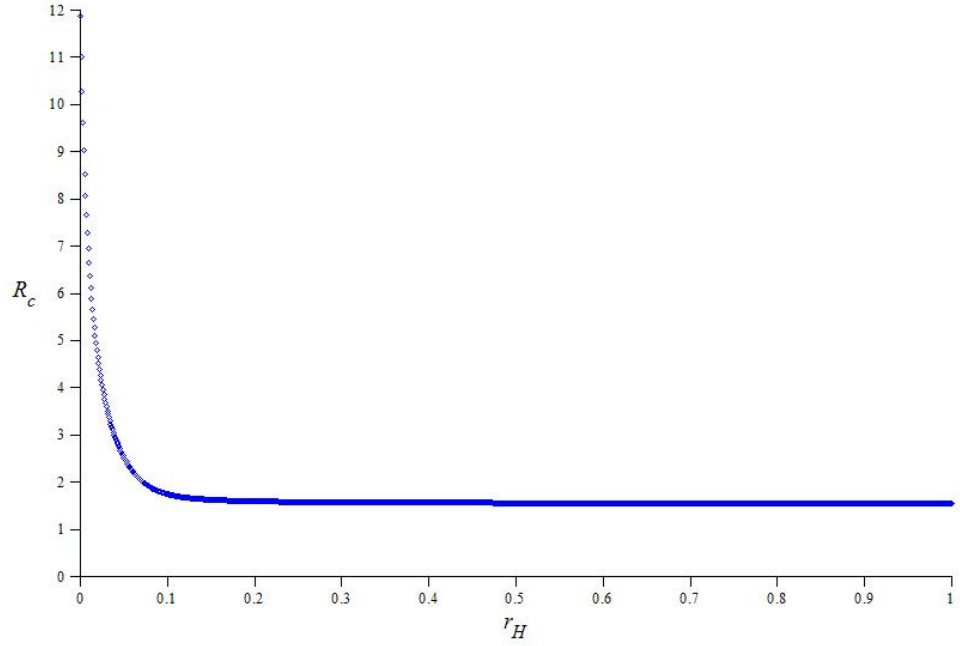

Figure 11: High-Income Treatment Rate versus Basic Control Number

If we consider single-effort control methods in Scenario 1, the only feasible option to reduce our R_c to less than 1 is by increasing our value of r_L to 0.53. This translates to a 562.5% increase in the treatment rates for low-income individuals. Likewise, if we look at Scenario 2, the only feasible option to reduce our R_c to less than 1 is by increasing our value of r_L to 0.39. That is, by increasing treatment rates for low-income individuals by 290%. This tells us that, given our model, the control of the spread of tuberculosis through single-effort control methods is easier prior to the enactment of a law.

Recall that when considering a population with less prefered mixing ($f_L = 0.30$ and $f_H = 0.30$), R_c was 1.05. In this situation, there are three feasible single-effort control methods: 1) increasing r_L to 0.14, 2) decreasing f_L to 0.25, or 3) decreasing f_H to 0.09. This translates to a 7.69% increase in treatment rates for low-income individuals, a 16.7% decrease in contact preferrence for low-income individuals, or a 70% decrease in contact prefference for high-income individuals, respectively. Interestingly, increasing r_H,the treatment rate for high-income individuals, to the maximum value of 1.0 is not sufficient to reduce our R_c to less than 1.

If we consider single-effort control methods in Scenario 3, the only feasible option to reduce our R_c to less than 1 is by increasing our value of r_L to 0.39. This translates to increasing treatment rates for low-income individuals by 387%. Looking at a less extreme scenario, Scenario 4, we see that the only feasible option to reduce our R_c to less than 1 is to increase the value of r_L to 0.28, a 180% increase in the treatment rates for low-income individuals.

Relatively minor changes of multiple parameters may be more practical than an extreme single-effort control method, resulting in what is called multi-effort control methods. Consider the model with less preferred mixing (lower f_i) before the enactment of a immigration law, where the value of R_c is 1.05. Altering the parameters β_L from 0.40 to 0.36, r_L from 0.13 to 0.143, f_L from 0.30 to 0.27, and f_H from 0.3 to 0.27 which are only 10% changes. These small adjustments result in $R_c = 0.841$ which is approximately a 20% decrease.

For Scenario 3, the parameters are adjusted as follows: β_L from 0.8 to .44, r_L from 0.08 to .124, f_L from 0.5 to 0.225, and f_H from 0.3 to 0.135 which are 55% changes. These modifications vary R_c from 4.071 to 0.96 which is approximately a 76% changes. For Scenario 4, the parameters are feasibly adjusted as follows: β_L from 0.6 to 0.36, r_L from 0.1 to 0.14, f_L from 0.5 to 0.3, and f_H from 0.3 to 0.18 which are 40% changes. These modifications vary R_c from 2.517 to 0.902 which is approximately a 64% change.

It should be noted that if a backward bifurcation exists within these specific models, reducing R_c to just below 1 is not sufficient to control the spread of tuberculosis in our population. More extreme control methods would then be needed in order to return the population to a disease-free state.

6 Results and Conclusion

The premise of our paper was to model to what extent an immigration law such as SB 1070 would effect the spread of TB in the low-income population, and its subsequent spread to other socioeconomic groups. Under the assumption that the undocumented immigrant population resides primarily in low-income communities, we altered parameters for our low-income group to reflect behavioral changes among undocumented immigrants after the law.

Through simulations, we notice that these changes, whether modest or extreme, reflected a substantial increase in the number of TB cases among our low-income group forty years after the law. However, we do not account for changes in immigration, where our immigrant population could either substantially reduce as a result of the law, or illegal immigration could continue to occur at a constant rate. Despite this limitation, our scenarios show a TB public health concern for low-income individuals in Arizona. Additionally, under extreme parameter changes, as well as with less strict mixing preferences, there is a notable effect on the increase of infectious TB cases among our high-income group. In reality, our high-income group is the rest of our population, or those not living below the poverty line, and so realistically we expect more mixing between those at the lower end of the high-income spectrum with the low-income population. Therefore, we believe that the actual prediction of the spread of TB lies somewhere between scenario 3 and scenario 4, where both socioeconomic groups experience an increase in TB infections.

Using sensitivity analysis, we were able to show that the most feasible and effective single-effort control method in all scenarios was through increased treatment rate in the low-income community. A surprising result from examining these single-effort control methods was that even an increase in the treatment rate to 1.0 (meaning 100% of individuals in the high-income community receive treatment) would not reduce our basic control number to below 1. On the other hand, even a small increase in the treatment rate in the low-income community largely reduces our basic control number. We also found that the easiest scenarios to reduce the basic control number to below 1 through single-effort control methods were the before immigration law scenarios. Similar results follow from multi-effort control methods. Additionally, we found that our basic control number for a scenario in which individuals hold less preference for their own income group was much lower (32.26% lower) than a scenario with more preferential mixing. Despite this decrease, the amount of TB cases in our high-income group increases as seen in Scenario 3 and 4, because TB is not strictly contained in one income group.

Ultimately, our paper investigates just one of the many repercussions of an immigration law such as SB 1070. Though TB is of interest because of the direct increase of foreign-born TB cases, TB is not the necessarily the largest health threat. The logic behind our model applies to other communicable diseases from measles to the common flu.

8 Acknowledgments

This project has been supported by grants from the National Science Foundation (NSF - Grant DMPS-0838705), the National Security Agency (NSA - Grant H98230-09-1-0104), the Alfred P. Sloan Foundation; and the President and Provost Offices at Arizona State University. The Mathematical and Theoretical Biology Institute now hosted at the Mathematical, Computational and Modeling Science Center at ASU would like to give thanks to everybody involve with the program for the past 15 years. We would like to give special thanks to Leon Arriola PhD, Angela Ortiz PhD, and Benjamin Morin, for their insight and support. We also would like to thank MTBI and Carlos Castillo-Chavez PhD for the wonderful opportunity and academic experience.

References

[1] R.C. Archibold *Arizona Enacts Stringent Law on Immigration*, New York Times. April 23, 2010.

[2] *Arizona* Quickfacts from the US Census Bureau. July 15, 2010.

[3] S. Asch, B. Leake, L. Gelberg, *Does Fear of Immigration Authorities Deter Tuberculosis Patients From Seeking Care?*, West J Med. 161 (1994) 373-376.

[4] Brauer, Fred. *Heterogeneity in Disease Transmission Style* Bulletin of Mathematical Biology. (2008) 70:1869-1885.

[5] *Border County Health Workforce Profiles: Arizona*, US Department of Health and Human Services (2010 July).

[6] C. Castillo-Chavez, Z. Feng, *Global Stability of an Age-Structured Model for Tuberculosis and its Applications to Optimal Vaccination Strategies*, Mathematical Biosciences. 151 (1998), 135-154.

[7] C. Castillo-Chavez, B. Song, *Dynamical Models of Tuberculosis and Applications*,Journal of Mathematical Biosciences and Engineering. 1 (2004) 361-404.

[8] C.Y. Chiang, L.W. Riley, *Exogenous Reinfection in Tuberculosis*, Lancet Infect Dis. 5 (2005) 629-636.

[9] *Diabetes in Arizona: Status Report*, Arizona Department of Health Services, Diabetes Prevention and Control Program (2005 November).

[10] *F as in Fat: How Obesity Threatens America's Future 2010.* Robert Wood Johnson Foundation. June 2010.

[11] Z. Feng, C. Castillo-Chavez, A.F. Capurro, *A Model for Tuberculosis with Exogenous Reinfection*, Theor. Pop. Biol. 57 (2000) 235-247.

[12] Z. Feng, W. Huang, C. Castillo-Chavez *On the Role of Variable Latent Periods in Mathematical Models for Tuberculosis*,Journal of Dynamics and Differential Equations. 13 (2001) 425-452.

[13] S.P. Fisher-Hoch, E. Whitney, J.B. McCormick, G. Crespo, B. Smith, M.H. Rahbar, B.I. Restrepo, *Type 2 Diabetes and Multi-Drug Resistant Tuberculosis*, Scand J Infect Dis.40 (2008), 888-893.

[14] H.W. Hethcote, *Qualitative Analysis for Communicable Disease Models*, Mathematical Biosciences. 28 (1976) 335-356.

[15] D. Jackson, *US/Mexico Border and Tuberculosis*, Institute of Medicine.

[16] Jacquez, John, Carl P. Simon, James Koopman, Lisa Sattenspiel and Timothy Perry. *Modeling and Analyzing HIV Transmission: The Effect of Contact Patterns*. Mathematical Biosciences. 92 (1988) 119-199.

[17] M.N. Lobato, J.P. Cegielski, *Preventing and Controlling Tuberculosis Along the U.S.-Mexico Border*, MMWR. 50 (2001) 1-27.

[18] M.M. McEwen, *Mexican Immigrants' Explanatory Model of Latent Tuberculosis Infection*, J Transcult Nurs. 16 (2005) 347-355.

[19] M.T. McKenna, E. McCray, I. Onorato, *The Epidemiology of Tuberculosis among Foreign-Born Persons in the United States, 1986 to 1993*. N Engl J Med. 332 (1995) 1071-1076.

[20] A. Pablos-Mndez, J. Blustein, C.A. Knirsch, *The Role of Diabetes Mellitus in the Higher Prevalence of Tuberculosis Among Hispanics*, Am J Public Health. 87 (1997) 574-579.

[21] A. Ponce-De-Leon, M.L. Garcia-Garcia, M.C. Garcia-Sancho, F.J. Gomez-Perez, J.L. Valdespino-Gomez, G. Olaiz-Fernandez, R. Rojas, L. Ferreyra-Reyes, B. Cano-Arellano, M. Bobadilla, P.M. Small, J. Sifuentes-Osornio, *Tuberculosis and Diabetes in Southern Mexico*, Diabetes Care. 27 (2004) 1584-1590.

[22] R. Rector, *Providing Health Care for Illegal Immigrants: Understanding the House Health Care Bill*, The Heritage Foundation. (2009).

[23] Reluga, Timothy, Jan Medlock and Alan Perelson. *Backward Bifurcations and Multiple Equilibria in Epidemic Models with Structured Immunity*. Journal of Theoretical Biology. 252 (2009) 155-165.

[24] B.I. Restrepo, S.P. Fisher-Hoch, J.G. Crespo, E. Whitney, A. Perez, B. Smith, J.B. McCormick, *Type 2 Diabetes and Tuberculosis in a Dynamic Bi-national Border Population*, Epidemiol. Infect. (2007) 483-491.

[25] B. Song, C. Castillo-Chavez, J.P. Aparicio, *Tuberculosis Models with Fast and Slow Dynamics: The Role of Close and Casual Contacts*, Mathematical Biosciences. 180 (2002) 187-205.

[26] *The Global Tuberculosis Epidemic: Fact Sheet*, US Global Health Policy. June 2010.

[27] *Treatment of TB: Guidelines for National Programmes*, World Health Organization. (2003).

[28] *Trends in Tuberculosis Incidence: United States, 2006*, JAMA. April 25, 2007.

[29] *Tuberculosis Surveillance Report Arizona, 2005*, Arizona Department of Health Services, Bureau of Epidemiology and Disease Control, Office of Infectious Disease Services (2006 July).

[30] *Tuberculosis Surveillance Report Arizona, 2008*, Arizona Department of Health Services, Bureau of Epidemiology and Disease Control, Office of Infectious Disease Services (2010 July).

[31] H. Waaler, A. Geser, S. Andersen, *The Use of Mathematical Models in the Study of the Epidemiology of Tuberculosis*, AM. J. Publ. Health Nations Health 52 (1962) 1002-1013.

[32] X. Wang, Z. Feng, J.P. Aparicio, C. Castillo-Chavez, X. Wang, Z., *On the Dynamics of Reinfection: the Case of Tuberculosis*, BIOMAT. (2009) 304-330.

[33] C.D. Wells, M.Ocaña, K. Moser, D. Bergmire-Sweat, J.C. Mohle-Boetani, N.J. Binkin, *A Study of Tuberculosis Among Foreign-Born Hispanic Persons in the U.S. States Bordering Mexico*, Am. J. Respir. Crit. Care Med. 159 (1999) 834-837.

[34] R. Wolf, *Rising Healthcare Costs Put Focus on Illegal Immigrants*, USA Today. January 22, 2008.

[35] *Vaccine Education Center*, Children's Hospital of Philadelphia. July 15, 2010.

[36] *Vital Statistics*, Arizona Department of Health Services. July 15, 2010.