Strategic Dynamics of Antibiotic Use and the Evolution of Antibiotic-Resistant Infections

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Abstract

This paper studies a dynamic model of a fee-for-service healthcare system in which healthcare providers attract patients by prescribing antibiotics. Using antibiotics limits antibiotic-treatable infections, but causes the development of antibiotic-resistant infections. The focus of the paper is the optimal market structure of healthcare providers given the competing, dynamic externalities from antibiotic use. The paper demonstrates a 'Goldilocks' effect. A perfectly competitive market for providers over-prescribes antibiotics relative to the planner because providers do not fully bear the cost of antibiotic-resistant infections. At the other end of the industrial organization spectrum, a patient monopolist provider under-prescribes antibiotics in order to increase the level of treatable infection. This is because while infection is a 'bad' for society, infection is a 'good' for a provider of antibiotics under a fee-for-service regime. The main result of the paper is that due to more moderate antibiotic use than perfect competition or monopoly, oligopolistic competition can be the optimal market structure. The paper then turns to policy analysis and shows that a state-dependent quota/subsidy scheme can incentivize a perfectly competitive market to implement the planner's solution. Should implementing the scheme be infeasible, the paper demonstrates how the model can be used for constrained policy analysis.

Key Words: economics of antibiotic resistance, healthcare competition, renewable resources, Markov equilibria

JEL Classification Numbers: I1, I11, I18, I30, L13, Q2

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1 Introduction

Antibiotics have long been a boon to human well-being by fighting infection. However, using antibiotics also causes the development of antibiotic-resistant infections. (Levy, 1992; Seppälä et al, 1995). While antibiotic use has tempered the current level of treatable infection, antibiotic-resistant infections have grown to become a costly public health problem.[1] The Center for Disease Control and Prevention goes so far as to say "antimicrobial resistance is one of our most serious health threats."[2] Understanding how economic incentives affect antibiotic use and the infection/resistance balance is a problem of first-order importance.

This paper studies the incentives of healthcare providers who prescribe antibiotics in a fee-for-service healthcare system. I propose a model in which providers attract patients to pay a fee by prescribing antibiotics. This component of the provider-patient relationship is a natural one to focus on, as numerous studies have found that patients frequently request antibiotics from providers and that providers comply in order to keep patients satisfied (Bauchner et al 1999; Stivers 2005).

The main purpose of the model is to investigate how competition among providers affects social welfare. There is significant variation in the market concentration of healthcare providers across counties in the United States (Schneider et al 2008). More concentrated markets for providers have been linked empirically to more frequent antibiotic use than less concentrated markets (Bennet et al 2014). However, given the competing, dynamic effects of antibiotic use on treatable and resistant infection, the optimal market structure of healthcare providers is an open question. This paper develops a framework to understand the welfare implications of a fee-for-service healthcare system and provider competition taking into account the dynamic epidemiological effects of antibiotic use.

The framework has an epidemiological component and an economic component. The epidemiological component describes how infection evolves in response to antibiotic use and the economic component describes how provider and patient behavior determine antibiotic use.

The epidemiological component is an off-the-shelf dynamic model of an infection that has two strains: an antibiotic-treatable strain and an antibiotic-resistant strain. The two strains compete for resources (healthy bodies) in the ecosystem. When a patient infected

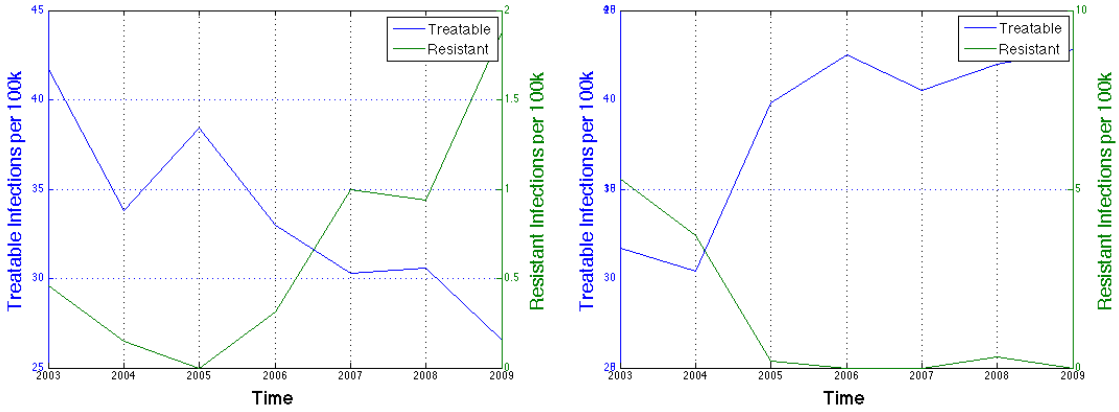
[1] See, for example, Klevens et al (2007) and Roberts et al (2009).

[2] Antibiotics Resistance Threats in the United States, 2013 http://www.cdc.gov/drugresistance/threat-report-2013/pdf/ar-threats-2013-508.pdf

with treatable bacteria takes the antibiotic, the patient raises his probability of recovery. The patient is then less likely to infect other healthy agents. However, by removing a source of competition for the resistant bacteria, the patient's use of the antibiotics allows the resistant bacteria to grow and spread at a faster rate in the future.

Below I display the evolution of Penicillin-treatable and Penicillin-resistant invasive *Streptoccocus Pneumoniae* infections in Baltimore City, Maryland and the Nashville-Davidson region of Tennessee from 2003-2009.[3]

Invasive *Streptococcus Pneumoniae* Dynamics



(a) Baltimore City

(b) Nashville-Davidson

Baltimore City, featured on the left, displays patterns typical from antibiotic use: a decrease in the rate of treatable infections and an increase in the rate of resistant infections. Nashville-Davidson, on the other hand, displays the opposite pattern: an increase in the rate of treatable infections and a decrease in the rate of resistant infections. Interestingly, in 2001, Nashville-Davidson started a social campaign to reduce antibiotic use.[4] The graphs highlight competition between the resistant and treatable infections for resources: one strain of the infection grows at the expense of the other. It is this competition between the resistant and treatable infections, crucially affected by antibiotic use, that the epidemiological component seeks to capture.

The economic component is a model of the provider-patient matching process. Providers

[3]I thank Dr. David Blythe and the Maryland Department of Health and Mental Hygiene for access to the Baltimore City data. The Tennessee data is available online at: http://health.tn.gov/Ceds/WebAim/

[4]http://health.state.tn.us/ceds/Antibiotics/index.htm

choose how frequently to prescribe antibiotics for their patients and patients choose whether to see a provider and which provider to see. Patients gain utility from a provider's antibiotic prescription behavior, the current effectiveness of the antibiotic (i.e. the proportion of treatable infection to total infection), and some idiosyncratic component related to the quality of their match with a provider. Patients choose whether or not to pay a fixed fee to see their most preferred provider, ignoring the external effects of their antibiotic use on infection and resistance. Providers choose a prescription rate to maximize profits, given the behavior of other providers and patients, internalizing their own effect on the future levels of infection and resistance. Provider and patient behavior together determine the aggregate amount of antibiotic use, which is then embedded into the epidemiological model.

The fully fleshed model is a variant of the resource extraction "fish war" model, where treatable infection is a scarce resource that gets extracted as providers treat patients with antibiotics. In the classic extraction models a la Levhari and Mirman (1980), though, the social objective is to extract the resource so as to maximize the lifetime utility from consumption. In the set-up here, the social goal is to use antibiotics to extract the treatable disease so as to minimize the lifetime disutility from total infection, taking into account the effect that extracting the treatable infection accelerates the growth of the resistant infection. A key feature of the model is that, due to the crowding out effect that the treatable infection has on the resistant infection, the socially optimal level of the treatable infection will not be zero. At the same time, due to the beneficial effects of antibiotic use, the socially optimal level of the resistant infection will not be zero either.

Due to the idiosyncratic utility shocks, providers sell differentiated products to patients. This allows for the model to study antibiotic provision under any number of providers.

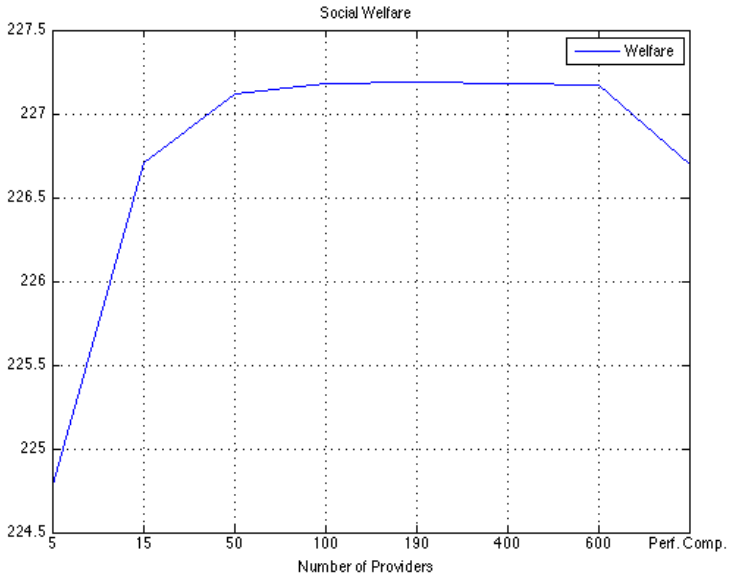
I first study the case in which the number of providers tends to infinity, a perfectly competitive market. When the market is competitive, providers have a negligible effect on the evolution of the system and no incentive to conserve the treatable infection. This incentivizes providers to perpetually over-prescribe antibiotics in order to attract as many patients in the current period as possible. While this can initially be a boon for society as treatable infection quickly falls, the resistant infection grows at a quicker rate than is socially optimal, eventually leading to a loss for society.

Interestingly, over-use by a competitive market does not always hold. This is because in order to be prescribed antibiotics, a patient must be willing to pay the fee to be seen by a healthcare provider. At low levels of antibiotic efficacy, depending on the parameter values of the model, a patient may choose to forego purchasing healthcare because the expected

private benefit is below the healthcare fee. However, purchasing healthcare would be socially beneficial due to the potentially decreased spread of treatable infection.

At the other end of the industrial organization spectrum, I show that a patient monopolist *under-prescribes* antibiotics relative to the planner. This increases profit along two margins for the monopolist. First, the monopolist maintains a higher antibiotic effectiveness, which increases the value of the monopolist's product and, in the long run, induces more patients to pay the fee. Second, the monopolist can maintain a perpetually higher total level of infection than the social planner desires, which results in a larger pool of potential fee-payers. The paper gives sufficient conditions for a monopolist to maintain a steady-state characterized by an undesirably high level of infection.

Increasing the level of competition from monopoly to duopoly generates a steady-state with a lower level of infection. Competition for patients induces the duopolist's to prescribe antibiotics more frequently than the monopolist, leading to a steady-state that yields strictly lower treatable infection and higher welfare than the monopolist's steady-state. As the market structure becomes increasingly competitive, though, antibiotics can become over-prescribed. Using numerical analysis, I show that social welfare as a function of the number of providers can take on an inverted U-shape (illustrated below). That is, social welfare can initially increase as the market becomes more competitive from monopoly, peak at some level of oligopolistic competition, and then decrease as the market moves towards perfect competition. Oligopolistic competition can thus be the optimal market structure.



This "Goldilocks" effect - the concept that an intermediate value is preferable to either of the extremes - arises because fee-for-service rewards patient volume. At low levels of competition, this aspect of fee-for-service incentivizes providers to use their market power to manipulate the states to increase the number of patients willing to pay the fee. This is accomplished by under-prescribing antibiotics to prevent the growth of the resistant infection and encourage the growth of the treatable infection.

At high levels of competition, the reward for patient volume incentives providers who lack any market power to attract as many patients as possible in the current period. This leads to over-prescribtion of antibiotics that, while it quickly limits the treatable infection, encourages quick long-run growth of the resistant infection.

An intermediate level of competition creates a better incentive structure for providers to trade off limiting the growth of the treatable infection in the current and limiting the growth of the resistant infection in the future. This is because an intermediate level of competition prevents providers from letting treatable infection grow as a monopolist would, but gives enough of a stake in the future that providers would not drive down the treatable infection as quickly as a perfectly competitive market would, thus slowing the growth of the resistant infection.

The model has several policy implications. First, I show that a state-dependent quota/subsidy scheme can incentivize a perfectly competitive market to implement the socially optimal solution. The quota is a cap on providers' prescription rates. The subsidy lowers the price of the provider service for a patient. The state-dependent quota/subsidy puts a cap on providers' prescription rate, which prevents over-use, while ensuring that patients always find it individually rational to be seen by a provider. Second, if implementing the state-dependent quota/subsidy is infeasible due to complexity or commitment problems, the model can serve as a tool to evaluate second-best policy alternatives. I show how the model can be used to compare two policies: a licensing regime and a state-invariant quota on competitive providers' prescription behavior.

The paper makes two primary contributions to the economics literature on antibiotic resistance (which is discussed in detail in Section 2). First, it provides a novel dynamic model of antibiotics provision under imperfect competition. The model illuminates new results about the optimality of oligopolistic competition and generalizes results previously discussed in the literature.

Second, the paper provides a characterization of optimal policy in a dynamic model of infection and resistance which has market provision of antibiotics. In case implementing the optimal policy is infeasible, the paper gives a framework for constrained policy analysis.

The rest of the paper will proceed as follows: Section 2 discusses the related literature. Section 3 presents the epidemiological model of infection and antibiotic resistance and the economic model of provider competition. Section 4 presents the results of the model. Section 5 analyzes public policy. Section 6 concludes. For ease of exposition, all proofs can be found in Appendix A.

2 Related Literature

There exists an economics literature on infection and antibiotic resistance. Layton and Brown (1996) first formalize the competing externalities from antibiotic use. Laxminarayan and Brown (2001) and Laxminarayan and Weitzman (2002) look at the optimal use of antibiotics in a variety of settings. Recently, attention has turned to how markets allocate antibiotics. Elbasha (2003) estimates a static model of market provision of antibiotics. Tisdell (1982) is an early contribution of a stylized two period model in which a competitive market over-uses an antibiotic-like good relative to the planner. Herrmann and Gaudet (2009) extend the analysis to an infinite-horizon model of infection and resistance. Mechoulan (2007) uses numerical simulations to show that a monopolist can settle into a steady-state with a positive level of infection whereas a planner may prefer to use antibiotics to achieve full eradication of the disease. Finally, Herrmann (2010) studies the pricing behavior of a pharmaceutical company that produces an antibiotic that is temporarily protected by a patent after which pharmaceutical companies have open-access to the antibiotic.

Herrmann and Gaudet (2009) is the closest to the present paper. They study an environment with market demand for antibiotics given by a demand curve that gets supplied by competitive pharmaceutical companies. The analysis here focuses on the patient-provider matching process, which allows for the study of antibiotic provision under imperfect market competition. This is an important gap to fill in the literature because, as the paper shows, oligopolistic competition can be the optimal market structure. The paper also generalizes results discussed in the aforementioned literature. For example, this paper finds conditions for which a sufficiently patient monopolist has an undesirably high steady-state level of infection, a broad extension of the results found in Mechoulan (2007).

The literature has started examining public policy responses to antibiotic resistance. Elbasha (2003) describes the optimal tax in a static model of market provision of antibiotics. Herrmann et al (2014) study optimal tax schemes in a dynamic environment with market provision of multiple antibiotic-like goods, but in which use of the good only causes

a negative externality. This paper contributes to this strand of the literature by giving a characterization of optimal policy in a dynamic model of infection and resistance.

3 The Model

The model is infinite-horizon and in discrete time. There are two components: an epidemiological component describing how disease evolves in response to antibiotic use and an economic component describing how provider and patient behavior determine antibiotic use. Each are considered in turn.

3.1 Epidemiological Component

The model of infection and antibiotic resistance is based on the *Susceptible - Infected - Susceptible* model of disease transmission (Kermack and McKendrick 1927). Related models of antibiotic resistance have appeared in the epidemiological literature such as Bonhoeffer et al (1997), Débarre et al (2009), and Porco et al (2012) as well as in the economics literature such as Herrmann and Gaudet (2009) and Laxminarayan and Brown (2001).

Resistant and non-resistant bacteria compete for resources (healthy bodies) in the ecosystem. When a patient infected with non-resistant bacteria takes the antibiotic, the patient raises his probability of recovery. The patient is then less likely to infect other healthy people. However, by removing a source of competition for the resistant bacteria, the patient's use of the antibiotics allows the resistant bacteria to grow and spread at a faster rate in the future. This is referred to as the 'natural selection' effect in the literature.

There is a society of measure one. Agents in the society cycle between being susceptible to an infection (S) and being infected (I). There is one class of infection and one antibiotic available to treat the infection. Infected agents have a strain that is either treatable by antibiotics or a strain of the infection that is resistant to antibiotics. Let I_t^T denote the measure of agents infected with the treatable version of the infection and I_t^R denote the measure of agents infected with the resistant version of the infection at time t.

Antibiotic effectiveness, E, is defined as the ratio of treatable disease to total disease in society. This the probability that, conditional on being infected, an agent is infected with the treatable strain of the infection.

$$E_t = \frac{I_t^T}{I_t^T + I_t^R} \tag{1}$$

Both strains of the infection are equally contagious. The measure of new treatable infections at time $t+1$ is given by $\beta I_t^T S_t$. Similarly, the measure of new resistant infections at $t+1$ is given by $\beta I_t^R S_t$. The parameter β reflects the contagiousness of the disease.[5]

Patients recover naturally from the infection with some probability. Let p^T denote the probability that a patient infected with the treatable disease recovers. Let p^R denote the probability that a patient infected with the resistant disease recovers. If a patient is infected with the treatable disease and takes the antibiotic, then they have an increase in their recovery probability of p^A, for a total recovery probability of $p^T + p^A$.

I assume that $p^T < p^R < p^T + p^A$. This means that patients who are infected with the treatable disease and are treated with antibiotics are more likely to recover than patients who are infected with the resistant disease or the treatable disease and are untreated. Patients who are infected with the resistant version of the disease are more likely to recover than patients who are infected with the treatable version and are not treated. I define $\Delta p \equiv p^R - p^T$. Δp captures what is referred to as the 'fitness cost of resistance'. Pathogens have resistance to antibiotics at some cost to the pathogen that allows non-resistant bacteria to outcompete resistant bacteria "if the selective pressure from antibiotics is reduced" (Anderson and Hughes 2010).

I denote the fraction of the infected population that is treated with antibiotics at time t by F_t. The law of motion describing the evolution of treatable disease is given by:

$$I_{t+1}^T = I_t^T[1 + \beta S_t - p^T - p^A F_t] \tag{2}$$

The law of motion describing the evolution of resistant disease is similar, except that there is no benefit of taking antibiotics.

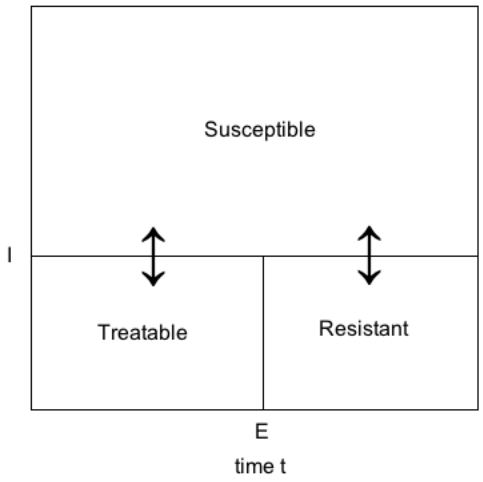
$$I_{t+1}^R = I_t^R[1 + \beta S_t - p^R] \tag{3}$$

For simplicity, I impose a no-death condition. Because the population measure stays constant, the change in the susceptible population is simply the inverse of the change in the infected population:

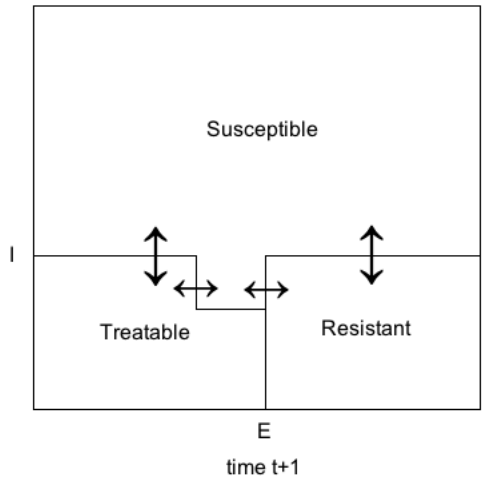
[5] β can alternatively be thought of as capturing a matching process between susceptible and infected agents.

$$S_{t+1} = S_t - (I^T_{t+1} - I^T_t) - (I^R_{t+1} - I^R_t) \tag{4}$$

The mechanism through which antibiotic use affects the evolution of the system can be understood through the following sequence of diagrams:

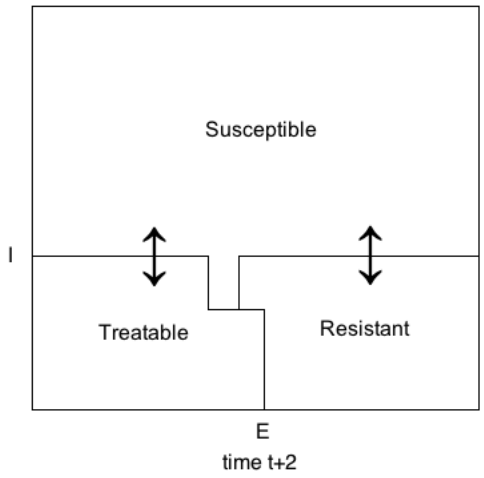


The relative size of each segment in the box represents the proportion of society in that state. There is some natural flow from the susceptible population into the infected states and vice versa. Antibiotic use at time t increases the flow of agents moving from being infected with the treatable strain to being susceptible in the following period (captured by an increase S_{t+1}).



However, because there is now a larger susceptible population at $t+1$, the resistant infection

will grow at a faster rate than if there was no antibiotic treatment. This is captured by an increase in I^R_{t+2}.[6]



The driving assumption behind this mechanism is that an agent can be infected with either the treatable or the resistant strain, but not both. This is known as 'bacterial interference' (Reid et al. 2001), a feature that has been observed in the data.[7]

Combining the definition of antibiotic effectiveness with the laws of motion governing susceptibility and infection, the system can succinctly be written recursively as:

$$I_{t+1} = I_t + I_t[\beta(1 - I_t) - p^R + E_t(\Delta p - p^A F_t)] \tag{5}$$

$$E_{t+1} = E_t + \frac{E_t(1 - E_t)(\Delta p - p^A F_t)}{1 + \beta(1 - I_t) - p^R + E_t(\Delta p - p^A F_t)} \tag{6}$$

Equation (5) describes the evolution of total infection. Equation (6) describes the evolution of antibiotic effectiveness - the proportion of treatable to total infection. Notice that for

[6]Epidemiological models frequently include some period of immunity to becoming infected again after recovery. In the set-up here, agents are immediately susceptible to the disease again after recovery. Were the immune response to be included, the mechanism through which antibiotics affects the system would fundamentally remain the same, only with a time lag.

[7]Though there is an epidemiological basis for this assumption, the assumption that patients can only be infected with the treatable or resistant strain can be relaxed. In a more elaborate epidemiological framework, patients could be colonized with both the treatable and resistant bacteria. Antibiotic use clears the patient of treatable bacteria, which allows the resistant bacteria to grow at a quicker rate in the patient. For simplicity, I focus on resistance evolving at the population level rather than the individual level.

$F_t > \frac{\Delta p}{p^A}$, antibiotic effectiveness is decreasing. Effectiveness decreases at high levels of antibiotic use because sufficiently high antibiotic use drives out the treatable infection at a quicker rate than the resistant infection (the natural selection effect). For $F_t < \frac{\Delta p}{p^A}$, effectiveness is increasing. When there is sufficiently low antibiotic use, patients with the resistant strain heal quicker on average than patients with the treatable strain (the fitness cost effect), leading to an increase in antibiotic effectiveness. At $F_t = \frac{\Delta p}{p^A}$, the two effects exactly offset and effectiveness stays constant.

I assume two further conditions. First, that $p^R < \beta < 1$. This ensures that both the treatable and resistant infections are endemic (always exist in the population) but not pandemic (spread over the entire population), a common feature of infectious disease. Second, that $\frac{\Delta p}{p^A} < 1$. This ensures that both the natural selection effect and fitness cost effect referenced above exist within the model.

The relevant epidemiological parameters and variables are summarized in the following table:

Parameter	Interpretation
β	Intensity of disease transmission
p^R	Natural recovery probability of those infected with resistant disease
p^T	Natural recovery probability of those infected with treatable disease
Δp	Fitness cost of resistance: $p^R - p^T$
p^A	Increase in recovery probability from taking antibiotics if treatable
Variable	Interpretation
I_t	Measure of those infected at time t
E_t	Antibiotic effectiveness at time t
F_t	Fraction of infected population treated with antibiotics at time t

A steady-state is a fixed point of Equations (5) and (6) - the laws of motion governing infection and antibiotic effectiveness. For a constant treatment rate F, the dynamic system tends to one of three possible steady-states described below.

(1) For $F > \frac{\Delta p}{p^A}$, the treatable strain clears the system at a faster rate than the resistant strain due to the natural selection effect. The system tends to a corner steady-state where the treatable strain goes extinct and only the resistant strain remains. This steady-state cannot be reached in finite time.

$$(\bar{I}, \bar{E}) = (\frac{\beta - p^R}{\beta}, 0) \tag{7}$$

(2) For $F < \frac{\Delta p}{p^A}$, the resistant strain clears the system faster than the treatable strain due to the fitness cost effect. The system tends to a corner steady-state where the resistant strain goes extinct and only the treatable strain remains. This steady-state also cannot be reached in finite time. Note that this steady-state has a higher level of infection than the other two steady-states.

$$(\bar{I}, \bar{E}) = (\frac{\beta - p^T - p^A F}{\beta}, 1) \tag{8}$$

(3) For $F = \frac{\Delta p}{p^A}$, the fitness cost effect and the natural selection effect exactly offset. The system tends to an interior steady-state level of antibiotic effectiveness:

$$(\bar{I}, \bar{E}) = (\frac{\beta - p^R}{\beta}, E) \text{ for } E \in (0, 1) \tag{9}$$

When applicable, the paper characterizes the steady-states of economic actors. However, the time horizon of the paper is the *human time-scale* rather than the *geological time-scale*, and so, particularly in the case of asymptotic steady-states, the analysis of the steady-state is intended only as a point of reference. The 'action' in the paper occurs along the transitional path.

3.2 Economic Component

I now introduce an economy with a provider-patient matching process and a fee-for-service healthcare system. Infected patients search for healthcare providers, who are the gate-keepers of antibiotics. Patients gain utility from a provider's prescription rate, the current effectiveness of the antibiotic (higher antibiotic quality increases the utility that agents get from providers), and some idiosyncratic component related to the quality of their match with a provider.

Patients choose whether or not to pay a fee to see their most preferred medical provider, who in turn chooses whether or not to prescribe antibiotics. Providers prescribe antibiotics to maximize their profits given the behavior of the other providers, patients, and the laws of motion governing the system.

Whether or not providers maximize profits is a debated issue in the literature. While financial incentives affect providers' treatment behavior (Clemens and Gottlieb 2014), many models of the provider-patient interaction study environments in which providers maximize a convex combination of profits and patient welfare (see Chonè and Ma 2011 or Jacobson et al 2013 for two recent examples). It would be possible to build this feature or an ethical constraint (e.g. the Hippocratic oath) into the model, but in order to both make the model more tractable and not impose structure that could assume the problem away, I refrain from doing so.

Patients are risk neutral. Each patient knows if he is infected, but does not know if he is infected with the treatable or resistant version of the disease. Patients have common knowledge of the states I and E. From a patient's perspective, E represents the conditional probability that if infected, the patient is infected with the treatable version of the disease. Utility is comprised of a component related to their health status and a component related to the medical care they may receive. The health status portion of utility is given by:

$$\begin{cases} U & \text{if healthy} \\ [E_t p^T + (1 - E_t) p^R] U & \text{if infected} \end{cases} \tag{10}$$

Infected patients can choose to be seen by a provider and may possibly be prescribed antibiotics. I assume that providers cannot diagnose whether patients have the treatable or resistant version of the disease and that their prescription rates are public knowledge. Patients pay the provider a fixed price p, regardless of whether or not an antibiotic is prescribed. The price p is taken to be exogenous, but can be imagined to be set by an insurance company or government. The exogoneity of the price reflects the fact that providers often have limited price-setting capacity and instead compete based on the quantity of services offered. I normalize p to be 1. Providers incur a constant marginal cost of c in administering antibiotics. I assume that $p^A U > c$. This assumption ensures that consumption of the antibiotic is economically efficient for an individual consumer who is infected with the treatable version of the disease.

When an infected patient takes the antibiotic, the patient has an expected increase in his utility of $E_t p^A U$. When provider i prescribes antibiotics at rate f_t^i, the patient's expected utility gain from the provider's healthcare is $f_t^i E_t p^A U$. Patients also gain idiosyncratic utility from going to the provider. Patients randomly draw a valence utility for each provider at time 0.[8] This can be thought of as the degree to which a healthcare provider's

[8] Although valence utility is fully persistent over time, this model is isomorphic to one in which valence utility is periodically redrawn.

non-medical features, e.g. bed-side manner, agree with a patient. I introduce this match-specific utility component so that providers sell differentiated products.

Patient m's valence utility for provider i is given by:

$$\varepsilon_m^i \sim U[0, \kappa] \tag{11}$$

I assume four additional conditions. First, that $p^A U \leq 1$. Second, that $1 - p^A U \leq \kappa \leq 1$. These assumptions are without loss of generality. I adopt it to ensure that patient demand for provider care is elastic over the entire range of admissible values in the model. Third, that $U > (p^T + p^A)U - c + \kappa$. Fourth, $U > p^R U + \kappa$. These latter two assumptions ensure that from a social welfare perspective, being healthy is always preferred to being infected.

3.3 Social Planner's Problem

To provide a benchmark for future analysis, I now describe the social planner's problem. The social planner treats a fraction of the infected population with antibiotics to solve the problem:

$$max_{\{F_t\}_{t=0}^{\infty}} \sum_{t=0}^{\infty} \delta^t [(1 - I_t)U + I_t\big([E_t p^T + (1 - E_t)p^R + F_t E_t p^A]U + \kappa\big) - cF_t I_t)] \tag{12}$$

s.t. (5), (6), I_0, E_0, $F_t \in [0, 1]$

The first term is the utility of healthy agents, the second is the utility of infected patients, and the third is the cost of treating patients with antibiotics. Note that the utility of infected patients is based on the expected recovery probability given the planner's prescription rate and the upper bound on the support of idiosyncratic utility.[9] There is no producer surplus because profits are a transfer from patients. Equations (5) and (6) are the laws of motion describing the evolution of infection and antibiotic effectiveness. I_0 and E_0 are the initial levels of infection and effectiveness.

The planner's objective is to minimize the disutility from total infection, taking into account the cost of antibiotic use and the effect of treatment on the growth resistant infection.

[9] I assume that the planner is inherently able to provide the best service possible to each infected patient and that there are no frictions that prevent the planner from providing this service to every infected patient.

The planner's solution can be characterized recursively through the Bellman equation:

$$V^{SP}(I,E) = max_F\Big\{(1-I)U + I\big([Ep^T + (1-E)p^R + FEp^A]U + \kappa\big) - cFI + \delta V^{SP}(I',E')\Big\} \tag{13}$$

st (5), (6), $F \in [0,1]$

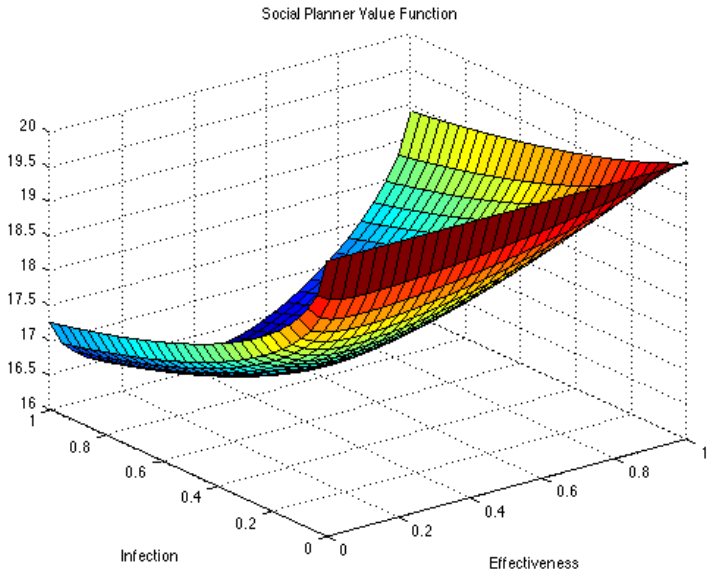
which, for a positive F, has the first-order condition:

$$Ep^A IU - cI + \delta\left[\frac{\partial V^{SP}(I',E')}{\partial I'}\frac{\partial I'}{\partial F}|_{F=F^*} + \frac{\partial V^{SP}(I',E')}{\partial E'}\frac{\partial E'}{\partial F}|_{F=F^*}\right] \geq 0 \tag{14}$$

The first-order condition reconciles the benefit of treating patients with antibiotics today and the benefit from the future decrease in infection due to treatment today with the cost of treating agents with antibiotics today and the cost of lower effectiveness in the future. I use the notation $F^{SP}(I,E)$ to refer to the planner's prescription rate when the states are are (I,E).

I use value function iteration to numerically investigate the planner's problem. I use the parameter values $\delta = .9, U = 2, \beta = .4, p^R = .25, p^T = .15, p^A = .5, c = .75$, and $\kappa = 1$. These parameter values for the remaining numerical analysis except where otherwise specified.

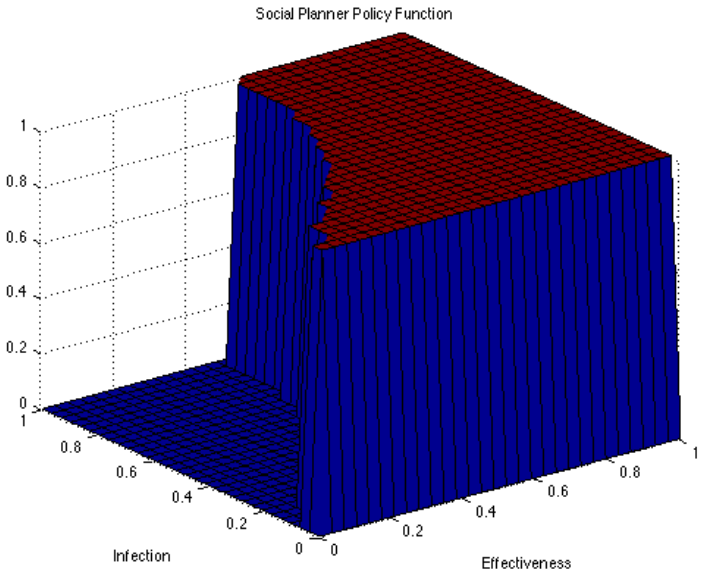
The planner's value function is displayed below:



The value function is convex in both infection and effectiveness. Convexity in infection is due to the non-linear spread of infection. Recall that new infections are given by $I\beta(1-I)$. The rate of increase is decreasing in I. Intuitively, increasing infection generates a larger negative externality when there is a large susceptible population than when there is a small susceptible population. Hence, while the planner's value function is decreasing in infection, it decreases at a less-than-linear rate.

The non-monotonicity in effectiveness stems from the cost of using antibiotics. As an extreme example, consider the case in which effectiveness is zero, i.e. every infection is resistant. Antibiotics would never be used because they are costly and have no effect. Every infected patient recovers in each period with probability p^R. Now suppose that effectiveness E is in the interior. For a low enough effectiveness, using antibiotics is not cost-justified. Initially, the average recovery probability is $(1-E)p^R+Ep^T < p^R$. Therefore, initially, patients are infected for on average a longer time than when effectiveness is zero. Due to this effect, the planner's value function initially decreases in effectiveness. However, as effectiveness increases, prescribing antibiotics eventually becomes worthwhile. Welfare begins to increase as the average recovery time decreases and the spread of infection is lessened.

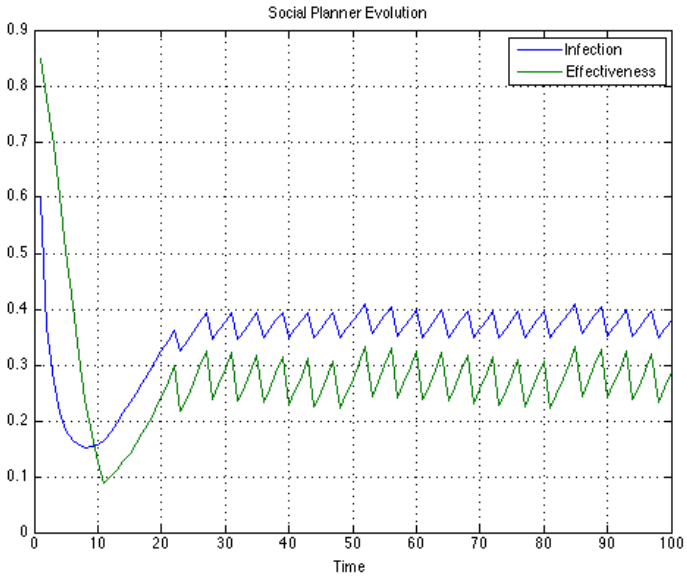
The value function can be used to compute the optimal prescription rate:



The optimal prescription rate tends to be either zero or one (however there are intermediate prescription rates along the boundary where prescription rates become positive). As elaborated on below, this generates cycling behavior along the planner's dynamic path.

Note that at low levels of antibiotic effectiveness, the optimal prescription rate is one when infection is low but zero when infection is high. When infection is lower, the planner faces a smaller marginal cost of antibiotic treatment and a higher marginal future benefit from the decrease in infection than when infection is higher. The planner is therefore willing to use antibiotics even when they are ineffective if the level of infection is low, but not when the level of infection is high.

The policy function can be used to compute the dynamic path of infection and effectiveness. For initial values, I use $I_0 = .6$ and $E_0 = .85$.



Rather than settle into a steady-state, the planner cycles antibiotic use. Given the parameter values, the level of infection in an interior steady-state is $\frac{\beta - p^R}{\beta} = .375$. By cycling antibiotic use rather than prescribing at the constant steady-state fraction $\frac{\Delta p}{p^A}$, the planner is able to induce a level of infection that is on average lower than .375 at a cost that is on average lower than the cost of maintaining the steady-state.

3.4 Market Payoffs

I now define the payoffs of healthcare providers and the solution concept, Markov Perfect equilibrium.

When provider i prescribes antibiotics at rate f_t^i, patient m's net utility from seeing provider i at time t is:

$$U_m^i(f_t^i, \varepsilon_m^i, E_t) = f_t^i E_t p^A U + \varepsilon_m^i - 1 \tag{15}$$

The first component is the utility related to a provider's prescription behavior, the second is the idiosyncratic utility gained from seeing a provider, and the third is the cost of seeing the provider. If there are n total providers, then an infected patient m will be willing to pay the fee to see provider i if:

$$U_m^i(f_t^i, \varepsilon_m^i, E_t) > U_m^j(f_t^j, \varepsilon_m^j, E_t) \quad j = 0, ..., i-1, i+1, ..., n \tag{16}$$

where $j = 0$ denotes the outside alternative of not seeing a provider and $j > 0$ denotes provider i's competitors. This condition means that m is willing to see provider i if provider i is preferred to all other providers and provider i is preferred to not seeking treatment. I assume that if patients are indifferent between their most preferred provider and the outside option then they go to the provider with probability $\frac{\Delta p}{p^A}$.

I denote provider i's market share at time t by $\Omega(f_t^i, f_t^{-i}, E_t)$, where f_t^i is provider i's prescription rate at time t and f_t^{-i} is a vector of the other providers' prescription rates at time t. Market share can be written as:

$$\Omega(f_t^i, f_t^{-i}, E_t) = Pr\Big(U_m^i(f_t^i, \varepsilon_m^i, E_t) > U_m^j(f_t^j, \varepsilon_m^j, E_t) \quad j = 0, ..., i-1, i+1, ..., n\Big) \tag{17}$$

Provider i's per period payoff is:

$$[1 - cf_t^i]\Omega(f_t^i, f_t^{-i}, E_t)I_t \tag{18}$$

Provider and patient behavior determines an aggregate prescription rate

$$F_t = \sum_{i=1}^{n} f_t^i \Omega(f_t^i, f_t^{-i}, E_t) \tag{19}$$

which can be inserted into Equations (5) and (6) to determine how infection and antibiotic effectiveness evolve. Given a discount rate $\delta < 1$ and a sequence of prescription rates, lifetime profits of provider i are:

$$\sum_{t=0}^{\infty} \delta^t \Big[1 - cf_t^i\Big] \Omega(f_t^i, f_t^{-i}, E_t) I_t \tag{20}$$

st $(5), (6), F_t = \sum_{i=1}^{n} f_t^i \Omega(f_t^i, f_t^{-i}, E_t), I_0, E_0$

The paper focuses on symmetric Markov Perfect Equilibrium. A Markov strategy for provider i is a mapping:

$$\sigma^i : [0,1]^2 \to [0,1] \tag{21}$$

where $\sigma^i(I,E) = f^i$. The strategy function takes the level of infection and effectiveness as inputs and gives a prescription rate as an output. Given a strategy function, lifetime profits for provider i can be written recursively using the value function:

$$V^i(I,E;\sigma) = [1 - c\sigma^i(I,E)]\Omega\Big(\sigma^i(I,E), \sigma^{-i}(I,E), E\Big)I + \delta V^i(I',E';\sigma) \tag{22}$$

st (5), (6), $F = \sum_{i=1}^{n} \sigma^i(I,E)\Omega(\sigma^i(I,E), \sigma^{-i}(I,E), E)$

A Markov strategy profile $\sigma = (\sigma^1, \sigma^2, ..., \sigma^n)$ constitutes a Markov Perfect Equilibrium if σ constitutes a sub-game perfect equilibrium in Markov Strategies. That is, σ is a Markov Perfect Equilibrium if for all $i = 1, ..., n$:

$$V^i(I,E;\sigma) \geq V^i(I,E;\hat{\sigma}^i, \sigma^{-i}), \forall (I,E), \forall \hat{\sigma}^i \text{ behavioral strategies} \tag{23}$$

The equilibrium is symmetric if every provider plays the same strategy function. Focusing on symmetric equilibrium makes the analysis more tractable and yields intuitive insights. In equilibrium, for example, patients will either match with the provider for whom they have drawn the highest idiosyncratic utility for or they will not pay the fee to any provider. Patient choice of provider in the model is therefore sticky, a feature that has been observed in previous studies of patient-provider choice (Mold et al 2004).

The Markov Perfect equilibrium can be characterized through the Bellman Equation:

$$V^i(I,E;\sigma) = max_f \Big\{ [1 - cf]\Omega(f^i, f^{-i}, E)I + \delta V^i(I',E';\sigma) \Big\} \tag{24}$$

s.t. (5), (6), $F = \sum_{j=1}^{n} f^j \Omega(f^j, f^{-j}, E)$, $f \in [0,1]$

When $\kappa = 1$, the first-order condition for a positive f^* in the symmetric equilibrium is:

$$[1-cf^*]Ep^AUI - c\frac{1}{n}[1-(1-Ep^AUf^*)^n]I+$$
$$\delta\frac{\partial V^i(I',E';\sigma)}{\partial I'}(-EIp^A)\big(\frac{1}{n}[1-(1-Ep^AUf^*)^n]+Ep^AUf^*(1-af^*)^{n-1}\big)+$$
$$\delta\frac{\partial V^i(I',E';\sigma)}{\partial E'}\frac{-E(1-E)p^A[1+\beta(1-I)-p^R]\big(\frac{1}{n}[1-(1-Ep^AUf^*)^n]+Ep^AUf^*(1-Ep^AUf^*)^{n-1}\big)}{\left[1+\beta(1-I)-p^R+E\Big(\Delta p-p^A\big(f^*(1-(1-Ep^AUf^*)^n)\big)\Big)\right]^2}$$
$$\geq 0 \quad (25)$$

The first-order condition reconciles the marginal benefit from increasing the prescription rate and attracting more patients today with the cost of using more antibiotics and the future effects from decreasing infection and effectiveness. The derivation of the equilibrium first-order condition is presented in Appendix B.

4 Market Provision

I now turn to the market allocation of antibiotics. I first study the limit case as the number of providers, n, tends towards infinity, a representation of a perfectly competitive market. I then study the case in which $n = 1$, monopoly, before analyzing the model under arbitrary n, oligopoly.

4.1 Perfect Competition

The equilibrium under perfect competition can be computed analytically.

Theorem 2

1. The unique symmetric equilibrium outcome under perfect competition is for every provider to set $\sigma^i(I,E) = 1$ for all (I,E).

2. The aggregate antibiotic prescription rate is:

$$F^{PC}(I,E) = \begin{cases} 1 & \text{if } Ep^AU+\kappa-1>0 \\ \frac{\Delta p}{p^A} & \text{if } Ep^AU+\kappa-1=0 \\ 0 & \text{if } Ep^AU+\kappa-1<0 \end{cases}$$

3. The steady-state level of antibiotic effectiveness is $\bar{E}_{PC} = \frac{1-\kappa}{p^A U}$

4. The steady-state level of infection is $\bar{I}_{PC} = \frac{\beta - p^R}{\beta}$

Competition with other providers for patients induces providers to prescribe antibiotics at rate one in all periods, i.e. every patient that the provider sees is prescribed antibiotics. Intuitively, when other providers prescribe at rate one, provider i has no incentive to deviate because he will not gain any patients in the current period and, due to the behavior of others, his own behavior has a negligible effect on the evolution of the system. Under perfect competition, providers have no incentive to preserve antibiotic effectiveness for future use, and so they extract the treatable infection as quickly as the market will allow.

To analyze how infection and effectiveness evolve, it is necessary to understand patient behavior as well. Recall that the upper bound on idiosyncratic utility that agents can get from a provider is κ. Under perfect competition, patients can always find a provider that gives them idiosyncratic utility arbitrarily close to κ. Given providers' behavior, the utility that a patient gets from his most preferred provider at time t is $E_t p^A U + \kappa - 1$. If this quantity is positive, then every patient will see a provider and be prescribed antibiotics.

Thus, while it is individually rational for a patient to see a provider, a perfectly competitive market will over-use antibiotics relative to the social planner (because the maximum amount of antibiotics are used under perfect-competition). This process can generate perpetual over-use of antibiotics relative to the planner. Interestingly, though, individual patients may choose to not purchase healthcare at antibiotic effectiveness levels for which the planner would find antibiotic treatment beneficial. If the healthcare fee is high relative to the idiosyncratic utility patients receive from providers, then at low levels of antibiotic effectiveness, patients may become unwilling to pay for healthcare that would be socially beneficial due to the decreased spread of infection.

I devote the remainder of the paper to studying the model when $\kappa = 1$ except where otherwise specified.

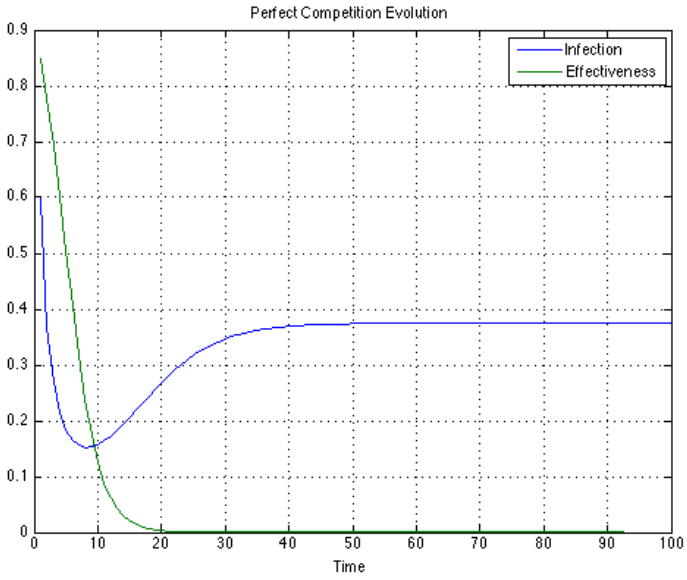
Theorem 2

If $\kappa = 1$*, then* $F^{PC}(I, E) \geq F^{SP}(I, E)$ *for all* (I, E) *and strictly greater for some* $(\tilde{I}, \tilde{E})$*.*

When $\kappa = 1$, there is perpetual over-use of antibiotics by the perfectly competitive market. The market converges to a steady-state in which the treatable infection is driven out of

the system. The assumption that the upper bound on idiosyncratic utility is the same as the provider fee (i.e. that $\kappa = 1$) is the weakest assumption needed so that the perfectly competitive market over-uses antibiotics relative to a planner along the entire dynamic path, regardless of parameter values or initial conditions. It amounts to agents in the model being willing to purchase healthcare from their most preferred provider if there is any chance of successful treatment. While it simplifies the remaining analysis, the key results from the paper do not explicitly depend on on $\kappa = 1$.

I display the dynamic path of infection and antibiotic resistance under perfect competition below.



The competitive market extracts the treatable infection as quickly as possible. Total infection initially falls, but then rebounds towards the steady-state level as the resistant infection grows to fill the void left by the treatable infection.

4.2 Monopolist

Here I turn to monopolist provision of antibiotics. In contrast to provision under perfect competition, under monopoly, the provider completely controls the flow of antibiotics to patients. The monopolist therefore has a large effect on the evolution of the system, which he fully internalizes as he chooses his prescription rate.

Before characterizing the monopolist's steady-state, I introduce two assumptions.

First, that $\frac{\Delta p}{p^A} < p^A U$. If this assumption did not hold, then regardless of the monopolist's prescription behavior, the resistant infection would clear the system at a faster rate than the treatable infection and the system would tend towards the corner steady-state in which effectiveness equals one. I use this assumption to ensure that, if such behavior arises, then it is due in part to economic considerations rather than purely biological constraints.

Second, that $(\frac{\Delta p}{p^{A2}U})^{1/2} \notin argmax_{x \in [0,(\frac{\Delta p}{p^{A2}U})^{1/2}]}[1-cx]\frac{\beta - p^T - x^2 p^A U}{\beta} x p^A U$. This expression is the monopolist's per-period payoff in the steady-state in which effectiveness equals one. $x \in [0, (\frac{\Delta p}{p^{A2}U})^{1/2}]$ are the range of prescription rates that can maintain such a steady-state - any higher prescription rate would generate a steady-state with an interior level of effectiveness. This assumption means that the monopolist's per-period payoff is not maximized on the boundary of allowable prescription rates. I refer to this assumption in later text as Assumption $\star$.

Theorem 3

1. *The monopolist's steady-state level of effectiveness* $\bar{E}_M \to 1$ *as* $\delta \to 1$.
2. *A sufficiently patient monopolist has a steady-state level of infection* $\bar{I}_M > \frac{\beta - p^R}{\beta}$.

The monopolist has a steady-state with an inefficiently high level of infection: any steady-state with infection I such that $\bar{I}_M > I > \frac{\beta - p^R}{\beta}$ generates strictly higher welfare than the monopolist's steady-state. Three reasons compel a monopolist provider to under-prescribe antibiotics in the steady-state. These effects can be understood through the monopolist's first-order condition:

$$\underbrace{[1 - 2f_M^* c] E p^A U I}_{\text{positive marginal payoff requirement}} + \delta \Bigg[\overbrace{\frac{\partial V^M(I', E'; \sigma)}{\partial I'}}^{\text{infection a good}} \frac{\partial I'}{\partial f}|_{f=f_M^*} + \underbrace{\frac{\partial V^M(I', E', \sigma)}{\partial E'}}_{\text{demand-inducement effect}} \frac{\partial E'}{\partial f}|_{f=f_M^*} \Bigg] \geq 0 \tag{26}$$

First, the monopolist's steady-state payoff is strictly increasing in antibiotic effectiveness. This is because of a demand-inducement effect: at higher levels of antibiotic effectiveness, more patients are willing to pay the fee to see the monopolist. As the monopolist becomes patient, his steady-state level of antibiotic effectiveness tends towards one because of this effect. Interestingly, although more patients are willing to pay the monopolist when the

steady-state level of antibiotic efficacy is one, patients are not, on net, better off because of the higher antibiotic efficacy. This is because the monopolist's under-use of antibiotics causes patients to be sick in expectation for a longer time.

Second, under fee-for-service, infection is a good for a healthcare provider. A monopolist can under-prescribe antibiotics so that there is a higher level of infection - resulting in a greater amount of potential fee-payers - than is socially optimal. In the steady-state in which antibiotic effectiveness is one, lowering the prescription rate has three effects on the monopolist's profits. First, by treating a smaller proportion of his patients with antibiotics, the monopolist raises his marginal profit per patient. Second, by using fewer antibiotics, the monopolist generates a steady-state with a strictly higher level of infection. Third, due to the decrease in antibiotic use, patients at the margin are less willing to pay the fee to the monopolist. Assumption $\star$ gives conditions in which the first two factors can trump the third factor.

The third reason that can cause a monopolist to under-use antibiotics is a positive current period marginal payoff requirement. Since increases in infection and effectiveness are a benefit for the monopolist, and since increasing the prescription rate decreases both, the latter two terms in Equation (26) - the monopolist's first-order condition - are negative in the steady-state. This means that in order to satisfy the first-order for a positive $\bar{f}_M$, the monopolist requires that his steady-state current period marginal payoff is positive. This implies that:

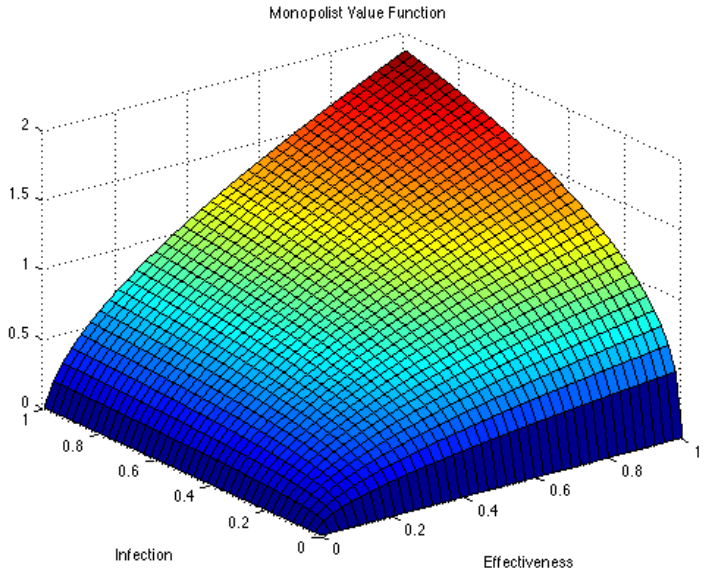
$$\frac{1}{2c} \geq \bar{f}_M \tag{27}$$

This positive current period marginal payoff requirement puts an upper bound on the monopolist's prescription rate. If the upper bound is low enough, then the aggregate steady-state prescription rate will be less than the critical threshold $\frac{\Delta p}{p^A}$, which implies a steady-state antibiotic effectiveness of one (note this also implies a steady-state with a strictly higher level of infection than is desirable). The positive current period marginal payoff requirement is another sufficient cause for under-prescription of antibiotics by a monopolist.

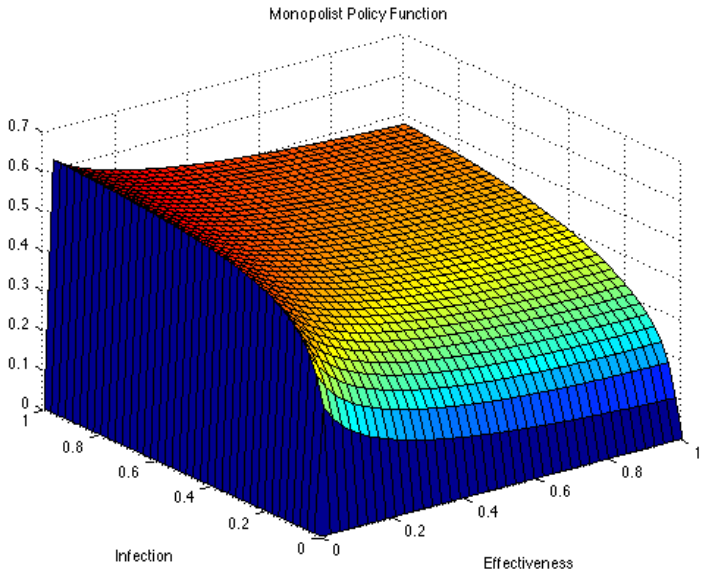
These distortions arise because of the linear payment structure of fee-for-service. By tying revenue to the volume of patients, fee-for-service incentivizes the monopolist to use his market power to manipulate the epidemiological states so as to increase the total number of patients willing to see the monopolist. The monopolist does this by under-prescribing antibiotics to increase antibiotic efficacy, which makes patients at the margin more willing

to pay the fee. Under-prescription of antibiotics also increases the total stock of infection, which increases the size of potential fee-payers.

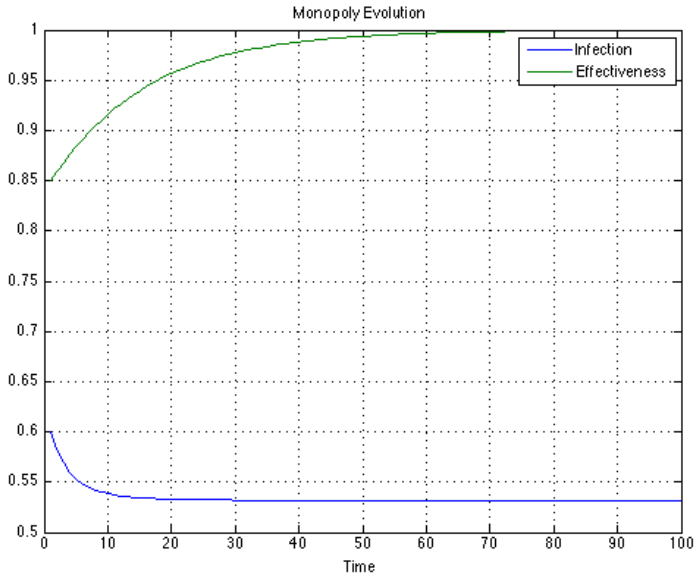
I use numerical analysis to study the behavior of the monopolist outside of the steady-state. Below I display the monopolist's value function given the assumed parameter values.



Note that the value function is strictly increasing in both infection and antibiotic effectiveness. This reflects the fact that fee-for-service makes infection and antibiotic effectiveness 'goods' for the monopolist. The value function can be used to derive the monopolist's optimal policy rule, which is displayed below.



Note that this is not the aggregate prescription rate, but rather the rate that the monopolist prescribes antibiotics to patients who pay the fee. The policy rule can be used to simulate the evolution of infection and antibiotic effectiveness under the monopolist, which is displayed below.



The monopolist uses his market power to let infection and effectiveness grow towards a corner steady-state with a higher level of infection than is socially optimal.

4.3 Oligopoly

I now turn to imperfectly competitive provision of antibiotics. Critically, unlike in the case of monopoly or perfectly competitive provision, providers now act strategically. The strategic interaction affects providers in several ways. First, whereas a patient would pay the fee to the monopolist if his individual rationality constraint was met, a patient will only pay the fee to the oligopolist if his individual rationality constraint is met and the oligopolist is preferred to all other providers. The oligopolist's prescription behavior thus affects his competitors' market share (and similarly, the oligopolist's market share is affected by his competitors' prescription behavior). Second, whereas the monopolist fully internalizes the effects of his prescription behavior, the oligopolist only partially internalizes the effects of his prescription behavior. The oligopolist's prescription behavior affects his competitors by changing the levels of infection and effectiveness in the future (and similarly, the oligopolist is affected by his competitors in this fashion).

The paper's next result contrasts the steady-state conditions of the monopolist and duopolist.

Theorem 4

A sufficiently patient duopolist has a strictly lower steady-state level of infection than the equally patient monopolist, i.e. $\bar{I}_D < \bar{I}_M$.

When there is competitive pressure in the market, the higher levels of infection that a monopolist can maintain in the steady-state are unsustainable. The presence of a competitor lowers an individual provider's benefit from withholding antibiotic treatment to generate more favorable states in the future. This forces more weight on attracting patients in the current period, which leads to higher antibiotic use.

I use numerical analysis to compute the Markov Perfect Equilibrium. The numerical algorithm used is described below.

Computational Algorithm

Step 1. Start an initial value function $v^0(I, E) = 0$.

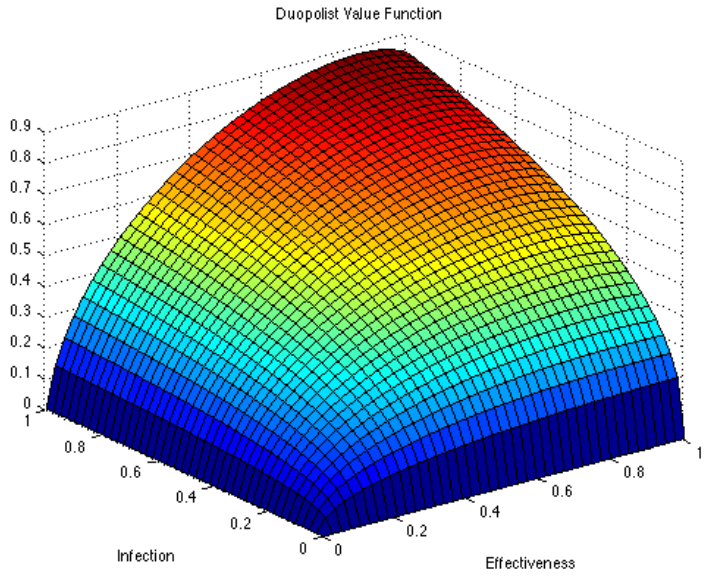
Step 2. Plug the proposed valued function into the oligopolist's first-order condition. Solve for the optimal symmetric prescription rate f^*.

Step 3. Use f^* to update the value function. Specifically, $v^{k+1}(I,E) = [1-cf^*]\Omega(f^*, f^{-i*}, E)+ \delta v^k(I', E')$.

Step 4. Iterate until the difference between $v^{k+1}(I,E)$ and $v^k(I,E)$ becomes small.

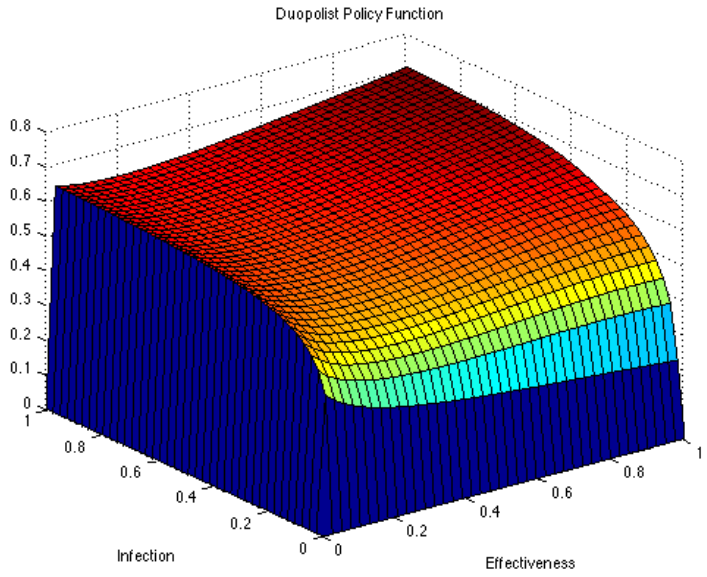
The algorithm computes the Markov Perfect Equilibrium of a finite horizon game and takes the limit as the time horizon tends to infinity.

I display the duopolist's symmetric equilibrium value function below:



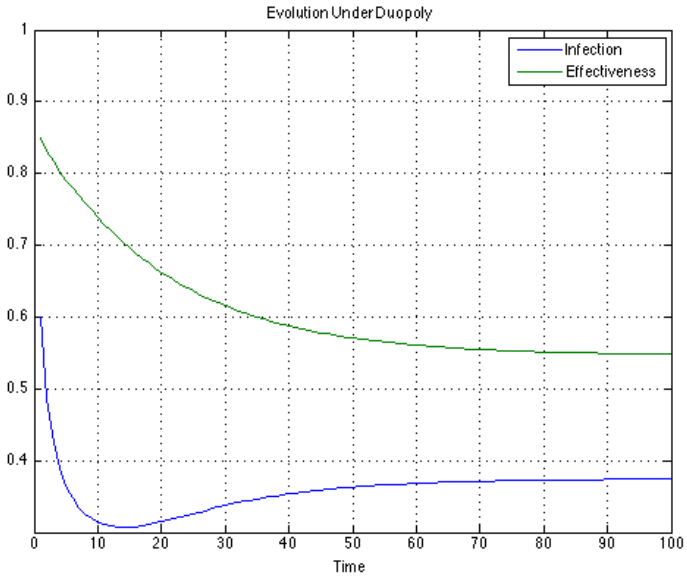
As is the case with the monopolist, the duopolist's value function is strictly increasing in infection. Interestingly, the value function is not strictly increasing in effectiveness. This is because when one duopolist prescribes antibiotics, he imposes an externality on the other duopolist by lowering the future level of infection. The more effective the antibiotic, the more strongly this externality effect is felt. While high efficacy is initially a boon for the duopolist because it draws in more patients in the current period, the future loss to the duopolist from the decrease in infection negates the initial benefit.

The value function can be used to derive the policy function, displayed below:



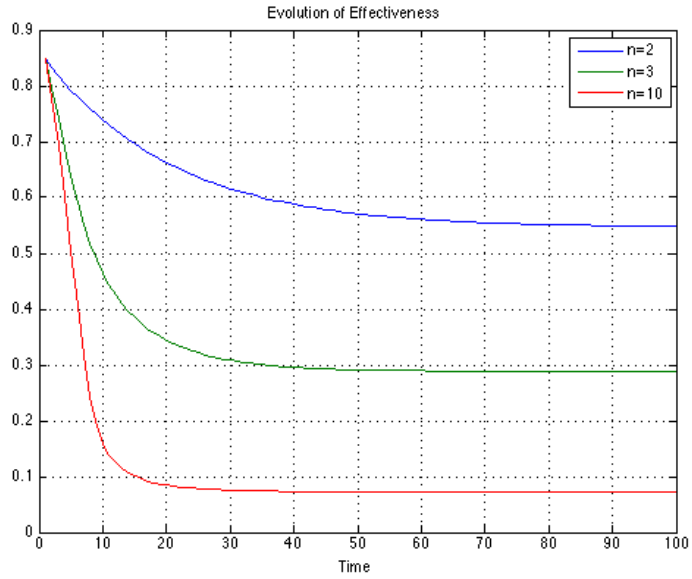
The duopolist prescribes at a strictly higher rate than the monopolist for all values of infection and antibiotic effectiveness. The presence of another provider cuts into a provider's market share, as well as diminishes the marginal effect of one's own prescription behavior on the future levels of infection and resistance relative to monopoly provision. These factors encourage greater antibiotic use under duopoly than monopoly.

The policy rule can be used to simulate the dynamic path of infection and effectiveness under the duopolist:

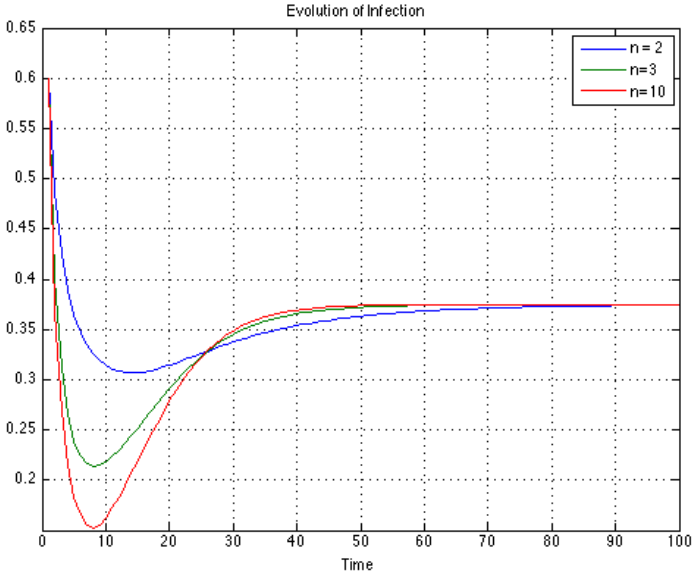


Notice that the duopolist has a strictly lower level of infection and effectiveness than the monopolist along the entire dynamic path. While fee-for-service still incentives infection as a good for the duopolist, competition for patients with the other provider prevents a provider from letting infection grow as a monopolist would prefer.

Intuitively, increasing the level of competition increases antibiotic use as providers focus less on maintaining favorable states in the future and more on attracting patients in the present. Below, I show the evolution of antibiotic effectiveness under increasing levels of competition.



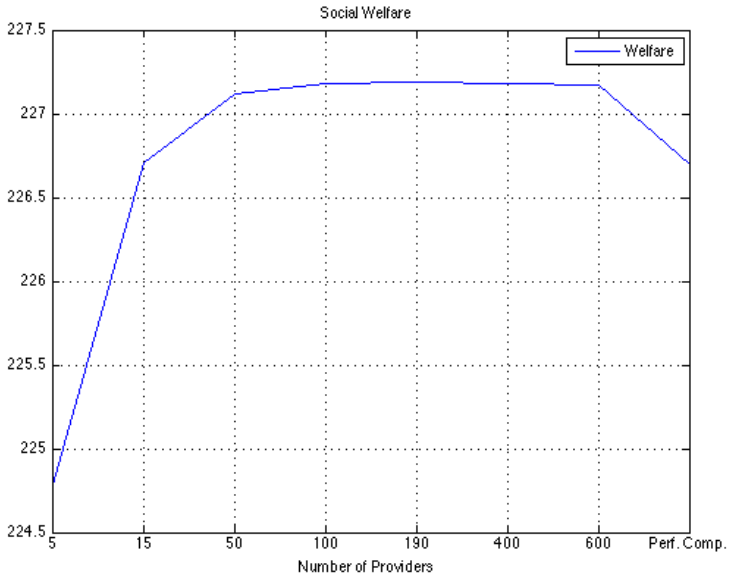
When there is more competition, antibiotic effectiveness decreases at a quicker rate and reaches a lower steady-state level than when there is less competition. This is because higher antibiotic use in more competitive markets results in quicker and more prolonged extraction of the treatable infection. The effect of competition on total infection is shown below.



Due to the increased antibiotic use, more competitive market structures generate a quicker initial decrease in total infection than less competitive markets as the treatable strain is cleared from the system. However, this also allows the resistant strain to grow at a faster rate. In fact, the resistant strain can grow sufficiently faster that the total level of infection in more competitive markets can eventually exceed the total level of infection in less competitive markets. This effect can be seen on the graph above by following the dynamic path of infection for $n = 10$. In period 10, $n = 10$ generates a lower total level of infection than the other market structures, but in period 35, a higher total level of infection than the other market structures.

4.4 Optimal Number of Providers

The central question of the paper asks about the welfare effects of provider competition. The picture below gives an answer for the parameter values $\delta = 0.99,\ V = 3,\ \beta = 0.4,\ p^R = 0.3,\ p^T = 0.1,\ p^A = 0.3,\ c = 0.89,\ I_0 = 0.05$, and $E_0 = 0.5$.



This graph plots social welfare as a function of the number of providers in a 200 period model. The key takeaway from this paper is that the graph can take on an inverted U-shape. That is, social welfare can initially increase as the market structure becomes more competitive from monopoly, peak at some level of oligopolistic competition, and then decrease as the market structure becomes perfectly competitive.

Intuitively, welfare initially increases as competition increases from monopoly because this generates lower steady-state level of infection. The reason that welfare can decrease at high levels of competition is more nuanced. As the market becomes more competitive, treatable infection decreases at a quicker rate, which initially generates higher welfare for society since the level of total infection becomes lower. However, the future loss from the increase in resistant infections becomes sufficiently high so as to negate that benefit.

The paper thus captures a 'Goldilocks' effect: to a rough approximation, low levels of competition use too few antibiotics and high levels of competition use too much antibiotics. The optimal number of providers in the simulation is $n^* = 190$. While n^* does not implement the planner's solution, n^* better treads the balance between limiting the current growth of the treatable infection and the future growth of the resistant infection given the economic constraints in the model than other market structures.

5 Public Policy

A natural question to ask is whether public policy can incentivize a decentralized market structure to optimally supply antibiotics in the model. The next result answers that question in the affirmative.

Theorem 5

For all κ, a state-dependent quota on providers' prescription rate and subsidy to patients can induce a perfectly competitive market to implement the first-best solution.

The intuition behind the result can be described as follows. In a competitive market, providers will always prescribe with the highest allowable rate. By capping the allowable prescription rate in each state by what the planner would choose, the quota prevents over-use of antibiotics.[10] The subsidy to patients ensures that it is always individually rational to purchase healthcare, which prevents under-use of antibiotics. By combining the quota/subsidy, the policy-maker can precisely pin down the aggregate prescription rate, ensuring that it equals the planner's.

The key takeaway from Theorem 5 is that in general, policy instruments (e.g. quotas or taxes) that only target providers' prescription rates will be unable to implement the first-best solution. The first-best policy must take into account the fact that patients can

[10] This can equivalently be achieved through a graduated tax on prescription rates.

become unwilling to purchase socially beneficial healthcare and therefore subsidize care accordingly.

Implementing this scheme may be infeasible. The policy is complex and requires commitment on the part of the policy-maker. In case a state-dependent tax is infeasible, the model can serve as a tool for second-best policy analysis. As two examples, I show how the model can be used to compare licensing restrictions and a time-invariant prescription quota with a competitive market. I use the parameter values from Section 4.5.

Licensing restricts the number of providers. The optimal licensing regime implements the optimal decentralized market structure. Given the parameter values used for the simulation, the optimal number of providers is 190 and this generates total social welfare of 227.192. The optimal prescription quota with competitive provision is the optimal cap on providers' prescription rates. Since competitive providers will always prescribe with the highest frequency allowable and, because $\kappa = 1$, all infected patients will see a provider, picking the optimal prescription quota is equivalent to picking the optimal time-invariant aggregate prescription rate.

The optimal time-invariant prescription quota is the solution to the following problem:

$$max_F \sum_{t=0}^{\infty} \delta^t [(1 - I_t)U + I_t[E_t p^T + (1 - E_t)p^R + F E_t p^A]U - cF I_t + I_t] \tag{28}$$

s.t. (1), (2), $I_0, E_0, F \in [0, 1]$

Given the parameter values, the optimal prescription quota sets $F = 0.7181$ and generates social welfare of 230.093. The model thus predicts that for the given parameter values, the optimal prescription quota with a competitive market is a better policy than the optimal licensing regime.

6 Conclusion

This paper studies the welfare effects of a fee-for-service healthcare system and provider competition embedded within a dynamic epidemiological model of infection and antibiotic resistance. The key finding is that oligopolistic competition can be the optimal decentralized market structure. This is because a perfectly competitive market over-uses antibiotics because providers do not bear the cost of antibiotic resistance and a monopolist under-uses antibiotics to increase infection and antibiotic efficacy. An interior level of competition has

more moderate antibiotic use which generates higher welfare. The paper also develops a framework for policy analysis.

A precise determination of the optimal market structure or public policy depends on the parameter values of the model. This is an important empirical exercise for future work. Other extensions of the model include incorporating innovation of new antibiotics by pharmaceutical companies, incorporating diagnostics procedures, and extending the analysis to a global setting.

7 Appendix A

Proof of Theorem 1

First I show that prescribing at rate one for all states is a symmetric Markov Perfect equilibrium and then I show it is unique. Suppose that all providers prescribe at rate one for all states. I show that defecting and prescribing at a rate less than one can never increase a provider's payoff.

Let N denote the set of providers. Without loss of generality, I will show that when provider 1 prescribes at rate $f^1 < 1$ and all $j \in \{2, ..., N\}$ prescribe at rate one, then in the limit as N gets large, no agent prefers going to provider 1. An agent prefers provider 1 to provider j if:

$$\varepsilon^1 - \varepsilon^j \geq E_t p^A U(1 - f^1) \tag{29}$$

Since the right-hand side is positive, I can show that agent m prefers provider j over provider 1 if there is a j for which $\varepsilon^j > \varepsilon^1$. Since

$$\lim_{N \to \infty} Pr[\max \varepsilon^j > \varepsilon^1 \text{ for } j \in \{2, ..., N\}] = 1 \tag{30}$$

there is always another provider that m prefers to 1. Hence, provider 1 gets zero patients and has a current period payoff of zero.

Next I show that the future states are the same when provider 1 prescribes at rate one or a rate less than one. This means that the continuation payoffs for provider 1 are the same regardless of his behavior. Specifically, this entails showing that every patient that would have been treated by provider 1 when 1 prescribes at rate one is treated by another provider when 1 prescribes at a rate less than one.

If a patient m is willing to see provider 1 when he prescribes at rate one, then $\varepsilon^1 \geq p - E_t p^A U$. Since all providers prescribe at probability one, m would be willing to see another provider j if $\varepsilon^j > \varepsilon^1$. By (30), the existence of such a j is ensured. Hence, provider 1's behavior has no impact on the evolution of the state variables and therefore his continuation payoff. Therefore, when every provider prescribes at rate one, defecting and prescribing at a rate less than one gives the provider a current period payoff of zero and does not increase his continuation payoff. Therefore there is no incentive to defect from prescribing at rate one.

To show uniqueness, suppose by contradiction that there existed another symmetric equilibrium in which providers prescribed at a rate less than one. Profits from adhering to the equilibrium strategy are zero, but deviating and prescribing with a higher rate would generate positive profits (because the provider would now attract a measurable set of patients). Hence the equilibrium in which providers prescribe at rate one is unique.

Recall that the price to see a provider is 1 and that the upper bound on the idiosyncratic utility that a patient can receive from the provider is κ. Under perfect competition, a patient can find a provider that gives idiosyncratic utility arbitrarily close to κ. If infected then, the patient will be willing to be seen by their most preferred provider $Ep^A U + \kappa - 1 > 0$, which would generate an aggregate prescription rate of 1. If $Ep^A U + \kappa - 1 < 0$, then no patient is willing to see their provider in which case the aggregate prescription rate is 0. If $Ep^A U + \kappa - 1 = 0$, then patients see their provider with probability $\frac{\Delta p}{p^A}$, which generates an aggregate prescription rate of $\frac{\Delta p}{p^A}$.

The only steady-state level of antibiotic effectiveness consistent with these aggregate prescription rates is $\bar{E}_{PC} = \frac{1-\kappa}{p^A U}$. Given this level of effectiveness, the steady-state level of infection is $\bar{I}_{PC} = \frac{\beta - p^R}{\beta}$.

□

Proof of Theorem 2

By Theorem 1, when $\kappa = 1$, $F^{PC}(I, E) = 1$ for all (I, E). It suffices to show that the the social planner does not prescribe at rate one for all state variables.

The planner's first-order condition for a positive F^{SP*} is:

$$Ep^A UI - cI + \delta \left[\frac{\partial V^{SP}(I', E')}{\partial I'} \frac{\partial I'}{\partial F}|_{F=F^{SP*}} + \frac{\partial V^{SP}(I', E')}{\partial E'} \frac{\partial E'}{\partial F}|_{F=F^{SP*}} \right] \geq 0 \qquad (31)$$

By (5) and (6), we have that:

$$\frac{\partial I'}{\partial F} = -EIp^A \tag{32}$$

and

$$\frac{\partial E'}{\partial F} = -\frac{E(1-E)p^A[1+\beta(1-I)-p^R]}{\left[1+\beta(1-I)-p^R+E[\Delta p - p^A F]\right]^2} \tag{33}$$

Assuming differentiability of the value function, the envelope conditions are:

$$\frac{\partial V^{SP}(I,E)}{\partial I} = [-1+Ep^T+(1-E)p^R+F^{SP*}Ep^A]U+\kappa-cF^{SP*}+\delta\frac{\partial V^{SP}(I',E')}{\partial I'}\frac{\partial I'}{\partial I}+\delta\frac{\partial V^{SP}(I',E')}{\partial E'}\frac{\partial E'}{\partial I} \tag{34}$$

and

$$\frac{\partial V^{SP}(I,E)}{\partial E} = I[p^T - p^R + F^{SP*}p^A]U + \delta\frac{\partial V^{SP}(I',E')}{\partial E'}\frac{\partial E'}{\partial E} + \delta\frac{\partial V^{SP}(I',E')}{\partial I'}\frac{\partial I'}{\partial E} \tag{35}$$

where

$$\frac{\partial I'}{\partial I} = 1+\beta(1-I)-p^R+E(\Delta p - p^A F)-\beta I \tag{36}$$

$$\frac{\partial E'}{\partial I} = \frac{E(1-E)(\Delta p - p^A F)\beta}{[1+\beta(1-I)-p^R+E(\Delta p - p^A F)]^2} \tag{37}$$

$$\frac{\partial I'}{\partial E} = (\Delta p - p^A F)I \tag{38}$$

$$\frac{\partial E'}{\partial E} = 1+\frac{(\Delta p - p^A F)[(1-2E)(1+\beta(1-I)-p^R)-(\Delta p - p^A F)E^2]}{[1+\beta(1-I)-p^R+E(\Delta p - p^A F)]^2} \tag{39}$$

Suppose by contradiction that the planner prescribes at rate one for all state variables. Then, $I \to \frac{\beta-p^R}{\beta}$ and $E \to 0$. Notice that $\frac{\partial I'}{\partial F} \to 0$ and $\frac{\partial E'}{\partial F} \to 0$ as well, and that the derivatives of the value function are obviously finite in the limit.

The planner's first-order condition converges to:

$$(31) \rightarrow -c\frac{\beta - p^R}{\beta} < 0 \tag{40}$$

which is incompatible with a positive F, hence a contradiction.

□

Proof of Theorem 3

The monopolist's Bellman equation is:

$$V^M(I, E; \sigma) = max_f \Big\{ [1 - cf] f E p^A U I + \delta V^M(I', E'; \sigma) \Big\} \tag{41}$$

s.t. (5), (6), $f \in [0, 1], F = f^2 E p^A U$

The monopolist will never have a steady-state in which he sets $f = 0$, since that is strictly dominated by prescribing any positive quantity. The first-order condition for a positive f is:

$$[1 - 2f_M^* c] E p^A U I + \delta \left[\frac{\partial V^M(I', E'; \sigma)}{\partial I'} \frac{\partial I'}{\partial f} |_{f=f_M^*} + \frac{\partial V^M(I', E', \sigma)}{\partial E'} \frac{\partial E'}{\partial f} |_{f=f_M^*} \right] \geq 0 \tag{42}$$

The effect of the monopolist's prescription rate on the states is:

$$\frac{\partial I'}{\partial f} = -EIp^A(2fEp^AU) \tag{43}$$

$$\frac{\partial E'}{\partial f} = -\frac{E(1-E)p^A[1 + \beta(1-I) - p^R]}{\Big[1 + \beta(1-I) - p^R + E[\Delta p - p^A f(fEp^AU)]\Big]^2}(2fEp^AU) \tag{44}$$

Assuming differentiability of the value function, the envelope conditions are:

$$\frac{\partial V^M(I,E;\sigma)}{\partial I} = [1 - cf_M^*]f_M^* E p^A U + \delta \frac{\partial V^M(I',E';\sigma)}{\partial I'}\frac{\partial I'}{\partial I} + \delta \frac{\partial V^M(I',E';\sigma)}{\partial E'}\frac{\partial E'}{\partial I} \quad (45)$$

and

$$\frac{\partial V^M(I,E;\sigma)}{\partial E} = [1 - cf_M^*]f_M^* p^A U I + \delta \frac{\partial V^M(I',E';\sigma)}{\partial E'}\frac{\partial E'}{\partial E} + \delta \frac{\partial V^M(I',E';\sigma)}{\partial I'}\frac{\partial I'}{\partial E} \quad (46)$$

In an interior steady-state, $\frac{\partial I'}{\partial I} = 1 - (\beta - p^r)$, $\frac{\partial E'}{\partial I} = 0$, $\frac{\partial I'}{\partial E} = 0$, and $\frac{\partial E'}{\partial E} = 1$. The steady-state envelope conditions are:

$$\frac{\partial V^M(\bar{I}_M,\bar{E}_M;\sigma)}{\partial I} = \frac{[1 - c\bar{f}_M]\bar{f}_M \bar{E}_M p^A U}{1 - \delta[1 - (\beta - p^r)]} \quad (47)$$

$$\frac{\partial V^M(\bar{I}_M,\bar{E}_M)}{\partial E} = \frac{[1 - c\bar{f}_M]\frac{\beta - p^r}{\beta}\bar{f}_M p^A U}{1 - \delta} \quad (48)$$

The steady-state first-order condition can be written as:

$$[1 - 2c\bar{f}_M]\bar{E}_M p^A U \frac{\beta - p^r}{\beta} + \frac{\delta[1 - c\bar{f}_M]\bar{f}_M \bar{E}_M p^A U}{1 - \delta[1 - (\beta - p^r)]}(-\bar{E}_M \frac{\beta - p^r}{\beta} p^A)(2\bar{f}_M \bar{E}_M p^A U) + \frac{\delta[1 - c\bar{f}_M]\frac{\beta - p^r}{\beta}\bar{f}_M p^A U}{1 - \delta}(-\bar{E}_M(1 - \bar{E}_M)p^A)(2\bar{f}_M \bar{E}_M p^A U) \geq 0 \quad (49)$$

Using the fact that in an interior steady-state, $\bar{f}_M^2 \bar{E}_M p^A U = \frac{\Delta p}{p^A}$, the steady-state first-order condition can be further simplified to:

$$\bar{E}_M p^A U \frac{\beta - p^r}{\beta} \left([1 - 2c[\frac{\Delta p}{\bar{E}_M p^{A2} U}]^{\frac{1}{2}}] - \frac{\delta[1 - c[\frac{\Delta p}{\bar{E}_M p^{A2} U}]^{\frac{1}{2}}]}{1 - \delta[1 - (\beta - p^r)]} 2\frac{\Delta p}{p^A}\bar{E}_M p^A - \frac{\delta[1 - c[\frac{\Delta p}{\bar{E}_M p^{A2} U}]^{\frac{1}{2}}]}{1 - \delta} 2\frac{\Delta p}{p^A}(1 - \bar{E}_M)p^A \right) \geq 0 \quad (50)$$

s.t. $\bar{f}_M \in ([\frac{\Delta p}{p^{A2}U}]^{\frac{1}{2}}, 1], \bar{f}_M^2 \bar{E}_M p^A U = \frac{\Delta p}{p^A}$

Note that Equation (50) may not admit an interior $\bar{E}_M$ as a feasible solution. In this case, the monopolist will have a steady-state level antibiotic effectiveness of one. When $\bar{E}_M = 1, \frac{\partial E'}{\partial f} = 0$. Note also that in this steady-state, $\frac{\partial E'}{\partial I} = 0$ and so the steady-state envelope condition governing infection is:

$$\frac{\partial V^M(\bar{I}_M, \bar{E}_M; \sigma)}{\partial I} = \frac{[1 - c\bar{f}_M]\bar{f}_M p^A U}{1 - \delta(1 - (\beta - p^T - \bar{f}_M^2 p^{A2} U))} \tag{51}$$

The monopolist's steady-state first-order condition when effectiveness is one can be written as:

$$[1 - 2c\bar{f}_M]\bar{I}_M p^A U - \delta \frac{[1 - c\bar{f}_M][\bar{f}_M p^A U][\bar{I}_M p^{A2} U 2\bar{f}_M]}{1 - \delta(1 - (\beta - p^T - \bar{f}_M^2 p^{A2} U))} = 0 \tag{52}$$

s.t. $\bar{f}_M \in [0, \frac{\Delta p}{p^{A2}U}]^{\frac{1}{2}}], \bar{I}_M = \frac{\beta - p^T - \bar{f}_M^2 p^{A2} U}{\beta}$

Note that either Equation (50) or Equation (52) admits a solution, ensuring the existence of a steady-state.

I now argue that the monopolist's steady-state level of effectiveness $\bar{E}_M(\delta) \to 1$ as $\delta \to 1$. Suppose by contradiction that $\bar{E}_M$ is bounded away from 1 for all δ. Let $\hat{E}_M$ denote the maximum value of effectiveness. Note that if the monopolist prescribed with probability one, then the steady-state value of effectiveness would be $\frac{\Delta p}{p^{A2}U}$. This is the smallest possible steady-state value of effectiveness.

Notice that for all values of effectiveness between the bounds and all values of δ, the first two terms in the monopolist's first-order condition (50) are finite. However, because effectiveness is bounded from 1, the third term of the monopolist's first-order condition becomes arbitrarily small as $\delta \to 1$. Hence for high enough δ, the monopolist's first-order condition becomes negative, a contradiction.

Therefore, the monopolist's steady-state level of antibiotic effectiveness tends towards one as the monopolist becomes patient.

In order for steady-state level of effectiveness to equal one and for the steady-state level of infection to be greater than $\frac{\beta - p^R}{\beta}$, an extra condition is needed. That condition is assumption $\star$:

$$(\frac{\Delta p}{p^{A2}U})^{1/2} \notin argmax_{x \in [0, (\frac{\Delta p}{p^{A2}U})^{1/2}]}[1 - cx]\frac{\beta - p^T - x^2 p^A U}{\beta} x p^A U$$

This assumption is necessary because the steady-state in which effectiveness equals one is never reached in finite time. This assumption ensures that there exists a path to the steady-state that in finite time yields a higher payoff than any interior steady-state.

In an interior steady-state, the monopolist's per-period payoff is:

$$\bar{\Pi}_M(\bar{E}_M) = [1 - c\bar{f}_M]\frac{\beta - p^r}{\beta}\bar{f}_M\bar{E}_M p^A U \tag{53}$$

From earlier work we know that in an interior steady-state, $\bar{f}_M = [\frac{\Delta p}{\bar{E}_M p^{A2} U}]^{\frac{1}{2}}$, so the monopolist's per-period payoff can be re-written as:

$$[1 - c[\frac{\Delta p}{\bar{E}_M p^{A2} U}]^{\frac{1}{2}}]\frac{\beta - p^r}{\beta}[\frac{\Delta p}{\bar{E}_M p^{A2} U}]^{\frac{1}{2}}\bar{E}_M p^A U \tag{54}$$

Note that this is strictly increasing in antibiotic effectiveness (this is the demand-inducement effect). Let $\hat{f}$ be defined as:

$$\hat{f} = [\frac{\Delta p}{p^{A2} U}]^{\frac{1}{2}} \tag{55}$$

That is, $\hat{f}$ is the monopolist prescription rate that implements an aggregate prescription rate of $\frac{\Delta p}{p^A}$ when antibiotic efficacy is one. By assumption $\star$, there exists an $\epsilon > 0$ such that:

$$[1-c(\hat{f}-\epsilon)]\frac{\beta - p^T - (\hat{f}-\epsilon)^2 p^{A2} U}{\beta}(\hat{f}-\epsilon)p^A U > [1-c[\frac{\Delta p}{\bar{E}_M p^{A2} U}]^{\frac{1}{2}}]\frac{\beta - p^r}{\beta}[\frac{\Delta p}{\bar{E}_M p^{A2} U}]^{\frac{1}{2}}\bar{E}_M p^A U \tag{56}$$

for all interior $\bar{E}_M$. That is, if the monopolist prescribes at rate $\hat{f} - \epsilon$, then that leads to a steady-state that yields a strictly higher payoff than the payoff in any interior steady-state. While this steady-state payoff is never reached in finite time, I show that implementing a constant prescription rate of $\hat{f} - \epsilon$ generates a strictly higher payoff than any interior steady-state in finite time. By (56), there exists an $\eta > 0$ such that:

$$[1-c(\hat{f}-\epsilon)]\frac{\beta - p^T - (\hat{f}-\epsilon)^2 p^{A2} U}{\beta}(\hat{f}-\epsilon)p^A U - \eta > [1-c[\frac{\Delta p}{\bar{E}_M p^{A2} U}]^{\frac{1}{2}}]\frac{\beta - p^r}{\beta}[\frac{\Delta p}{\bar{E}_M p^{A2} U}]^{\frac{1}{2}}\bar{E}_M p^A U \tag{57}$$

for all interior $\bar{E}_M$. Suppose that the monopolist is currently at a steady-state with an interior level of antibiotic effectiveness. Let $\pi^t(I_t, E_t, \hat{f} - \epsilon)$ denote the monopolist's time t payoff when he prescribes at constant rate $\hat{f} - \epsilon$. Given the constant prescription rate of $\hat{f} - \epsilon$, $I_t \to \frac{\beta - p^T - (\hat{f} - \epsilon)^2 p^{A2} U}{\beta}$ and $E_t \to 1$. Because of that, $\pi^t(I_t, E_t, \hat{f} - \epsilon) \to [1 - c(\hat{f} - \epsilon)] \frac{\beta - p^T - (\hat{f} - \epsilon)^2 p^{A2} U}{\beta} (\hat{f} - \epsilon) p^A U$.

This implies that for t large enough,

$$\pi^t(I_t, E_t, \hat{f} - \epsilon) > [1 - c(\hat{f} - \epsilon)] \frac{\beta - p^T - (\hat{f} - \epsilon)^2 p^{A2} U}{\beta} (\hat{f} - \epsilon) p^A U - \eta \tag{58}$$

which means that in finite time, this path generates a per period payoff that is strictly than the payoff in any interior steady-state. Hence, a sufficiently patient monopolist would prefer the path realized by prescribing at rate $\hat{f} - \epsilon$ to remaining at an internal steady-state. Hence, the only feasible steady-state is one in which antibiotic effectiveness equals one. This steady-state necessarily has a higher level of infection than $\frac{\beta - p^R}{\beta}$.

□

Lemma 1

I now state and prove a lemma that will be used in proving Theorem 4.

Lemma 2: Suppose that the equation characterizing the monopolist's steady-state first-order condition is negative for some $f \in [0, 1]$. Then, the the equation is negative for all $\hat{f} \geq f$.

Proof:

Re-write (52) as:

$$I(f) p^A U \left([1 - 2cf] - \delta \frac{[1 - cf][2f^2 p^{A2} U]}{1 - \delta(1 - \beta I(f))} \right) \tag{59}$$

where $I(f)$ denotes the steady-state level of infection when effectiveness is one and the monopolist prescribes at rate f. Suppose that the expression is negative at f. I will show that the entire expression either decreases as f increases or that the term $[1 - 2cf]$ becomes negative, thus proving the claim.

Clearly, $I(f)$ is decreasing in f. Focusing on the interior term and differentiating with respect to f gives:

$$-2c - \frac{4fp^{A2}U[1-\frac{6}{4}cf](1-\delta(1-\beta I(f))) - (1-cf)2fp^{A2}U\delta\beta\frac{\partial I}{\partial f}}{(1-\delta(1-\beta I(f)))^2} \tag{60}$$

which is negative unless $[1-\frac{6}{4}cf](1-\delta(1-\beta I(f)))$ is sufficiently negative. However, if $[1-\frac{6}{4}cf]<0$, then $[1-2cf]<0$, which means that Equation (59) is negative anyway.

□

Proof of Theorem 4

By the analysis in Appendix B, the duopolist's first-order condition is:

$$[1-2cf_D^*]Ep^AUI+\frac{1}{2}c(Ep^AUf_D^*)^2I+\delta\frac{\partial V^D(I',E';\sigma)}{\partial I'}(-EIp^A)(2Ep^AUf_D^*-\frac{3}{2}(Ep^AUf_D^*)^2)+$$
$$\delta\frac{\partial V^D(I',E';\sigma)}{\partial E'}\frac{-E(1-E)p^A[1+\beta(1-I)-p^R]}{\left[1+\beta(1-I)-p^R+E\Big(\Delta p-p^A\big(f_D^*(1-(1-Ep^AUf_D^*)^2)\big)\Big)\right]^2}(2Ep^AUf_D^*-\frac{3}{2}(Ep^AUf_D^*)^2)$$
$$\geq 0 \tag{61}$$

The envelope condition governing infection is:

$$\frac{\partial V^D(I,E;\sigma)}{\partial I} = [1-cf_D^*]\frac{1}{2}[1-(1-Ep^AUf_D^*)^2]+\delta\frac{\partial V^D(I',E';\sigma)}{\partial I'}\frac{\partial I'}{\partial I}+\delta\frac{\partial V^D(I',E';\sigma)}{\partial E'}\frac{\partial E'}{\partial I} \tag{62}$$

The envelope condition governing effectiveness is:

$$\frac{\partial V(I,E;\sigma)}{\partial E} = [1-cf_D^*]I[1-Ep^AUf_D^*]p^AUf_D^*+\delta\frac{\partial V^D(I',E';\sigma)}{\partial I'}\frac{\partial I'}{\partial E}+\delta\frac{\partial V^D(I',E';\sigma)}{\partial E'}\frac{\partial E'}{\partial E} \tag{63}$$

In an interior steady-state, (62) and (63) can be re-written as:

$$\frac{\partial V^D(\bar{I}_D,\bar{E}_D;\sigma)}{\partial I} = \frac{[1-c\bar{f}_D]\frac{1}{2}[1-(1-\bar{E}_Dp^AU\bar{f}_D)^2]}{1-\delta(1-(\beta-p^R))} \tag{64}$$

and

$$\frac{\partial V^D(\bar{I}_D, \bar{E}_D; \sigma)}{\partial E} = \frac{[1 - c\bar{f}_D]\frac{\beta - p^R}{\beta}[1 - \bar{E}_D p^A U \bar{f}_D] p^A U \bar{f}_D}{1 - \delta} \tag{65}$$

The first-order condition governing an interior steady-state level of antibiotic efficacy is:

$$\begin{aligned}
&[1 - 2c\bar{f}_D]\bar{E}_D p^A U \frac{\beta - p^R}{\beta} + \frac{1}{2} c(\bar{E}_D p^A U \bar{f}_D)^2 \frac{\beta - p^R}{\beta} + \\
&\delta \frac{[1 - c\bar{f}_D]\frac{1}{2}[1 - (1 - \bar{E}_D p^A U \bar{f}_D)^2]}{1 - \delta(1 - (\beta - p^R))}(-\bar{E}_D \frac{\beta - p^R}{\beta} p^A)(2\bar{E}_D p^A U \bar{f}_D - \frac{3}{2}(\bar{E}_D p^A U \bar{f}_D)^2) + \\
&\delta \frac{[1 - c\bar{f}_D]\frac{\beta - p^R}{\beta}[1 - \bar{E}_D p^A U \bar{f}_D] p^A U \bar{f}_D}{1 - \delta}(-\bar{E}_D(1 - \bar{E}_D) p^A)(2\bar{E}_D p^A U \bar{f}_D - \frac{3}{2}(\bar{E}_D p^A U \bar{f}_D)^2) \\
&\geq 0
\end{aligned} \tag{66}$$

s.t. $\left(\bar{f}_D(2\bar{E}_D p^A U \bar{f}_D - (\bar{E}_D p^A U \bar{f}_D)^2)\right) = \frac{\Delta p}{p^A}, \bar{f}_D \in [0, 1]$

For notational convenience, let $a = p^A U$. The steady-state first-order condition in which antibiotic effectiveness is one can be written as:

$$[1 - 2c\bar{f}_D] a \bar{I}_D + \frac{1}{2} c(a\bar{f}_D)^2 \bar{I}_D + \delta \frac{[1 - c\bar{f}_D][a\bar{f}_D - \frac{(a\bar{f}_D)^2}{2}]}{1 - \delta(1 - \beta \bar{I}_D)}(-I_D^* p^A)(2a\bar{f}_D - \frac{3}{2}(a\bar{f}_D)^2) \geq 0 \tag{67}$$

s.t. $\left(\bar{f}_D(2a\bar{f}_D - (a\bar{f}_D)^2)\right) < \frac{\Delta p}{p^A}, \bar{f}_D \in [0, 1]$

Note that a solution exists to either (66) or (67), ensuring the existence of a steady-state. Given assumption $\star$, for a sufficiently high δ, the monopolist has a steady-state level of infection $\bar{I}_M > \frac{\beta - p^R}{\beta}$.

Let δ be sufficiently high. Clearly, if the duopolist has an interior steady-state level of effectiveness, the duopolist's steady-state level of infection will be lower than the monopolist's. I will show that if the duopolist has a steady-state level effectiveness of one, then the duopolist has a lower steady-state level of infection than the monopolist. I will show this by assuming that an equally patient duopolist has a higher steady-state level of infection than the monopolist and showing that this implies that the monopolist's first-order condition is negative, a contradiction.

I will show that if the monopolist were to implement the duopolist's steady-state level of infection, then his first-order condition would be negative. The aggregate prescription rate

in the duopolist's steady-state is $F = \bar{f}_D(2a\bar{f}_D - (a\bar{f}_D)^2)$. For the monopolist to implement the duopolist's steady-state level of infection, the aggregate prescription rate must be the same.

In order for the aggregate prescription rate to be the same, the monopolist must set $f_M^2 a = \bar{f}_D(2a\bar{f}_D - (a\bar{f}_D)^2)$, or $f^M = \bar{f}_D(2 - a\bar{f}_D)^{1/2}$. The monopolist's steady-state first-order condition evaluated at the duopolist's steady-state level of infection is:

$$[1 - 2c\bar{f}_D(2 - a\bar{f}_D)^{1/2}]a\bar{I}_D - \delta\frac{(1 - c\bar{f}_D(2 - a\bar{f}_D)^{1/2})2a^2\bar{f}_D^2(2 - a\bar{f}_D)\bar{I}_D p^A}{1 - \delta(1 - \beta\bar{I}_D)} \tag{68}$$

Recall the positive current period marginal payoff requirement. In order for the monopolist's first-order condition to be non-negative, it is required that:

$$[1 - 2c\bar{f}_D(2 - a\bar{f}_D)^{1/2}] > 0 \tag{69}$$

In order for the duopolist to have a higher steady-state level of infection than the monopolist, the duopolist must prescribe at an interior rate (if the duopolist prescribed at rate one, then that necessarily would cause a lower steady-state level of infection than under the monopolist). This implies that the duopolist's first-order condition holds with equality, and therefore:

$$[1 - 2c\bar{f}_D]a\bar{I}_D + \frac{1}{2}c(a\bar{f}_D)^2\bar{I}_D = \delta\frac{[1 - c\bar{f}_D][a\bar{f}_D - \frac{(a\bar{f}_D)^2}{2}]}{1 - \delta[1 - \beta\bar{I}_D]}(\bar{I}_D p^A)(2a\bar{f}_D - \frac{3}{2}(a\bar{f}_D)^2) \tag{70}$$

Because $[1 - 2c\bar{f}_D]a\bar{I}_D + \frac{1}{2}c(a\bar{f}_D)^2\bar{I}_D > [1 - 2c\bar{f}_D(2 - a\bar{f}_D)^{1/2}]a\bar{I}_D$, the monopolist's first-order condition (Equation (68)) is less than:

$$(68) < \delta\frac{[1 - c\bar{f}_D][a\bar{f}_D - \frac{(a\bar{f}_D)^2}{2}]}{1 - \delta[1 - \beta\bar{I}_D]}(\bar{I}_D p^A)(2a\bar{f}_D - \frac{3}{2}(a\bar{f}_D)^2) - \delta\frac{(1 - c\bar{f}_D(2 - a\bar{f}_D)^{1/2})2a^2\bar{f}_D^2(2 - a\bar{f}_D)\bar{I}_D p^A}{1 - \delta(1 - \beta\bar{I}_D)} \tag{71}$$

It suffices to show that:

$$(1 - c\bar{f}_D(2 - a\bar{f}_D)^{1/2})2a^2\bar{f}_D^2(2 - a\bar{f}_D) > [1 - c\bar{f}_D][a\bar{f}_D - \frac{(a\bar{f}_D)^2}{2}](2a\bar{f}_D - \frac{3}{2}(a\bar{f}_D)^2) \tag{72}$$

which, if true, would mean that the monopolist's first-order condition evaluated at the duopolist's steady-state level of infection is negative.

Subtracting the right-hand side of (72) from the left-hand side yields:

$$2a^2\bar{f}_D^2 - 2a^3\bar{f}_D^3 - 2a^2\bar{f}_D^3 c(2-a\bar{f}_D)^{3/2} + 2a^2\bar{f}_D^2 - \frac{5}{2}a^3\bar{f}_D^3 + \frac{3}{4}a^4\bar{f}_D^4 c \tag{73}$$

Dividing by $2a^2\bar{f}_D^2$ yields:

$$1 + \frac{1}{4}a\bar{f}_D - c\bar{f}_D(2-a\bar{f}_D)^{3/2} + \frac{3}{8}a^2\bar{f}_D^2 c + \frac{c}{2}(2-a\bar{f}_D) \tag{74}$$

Recall the current period positive marginal payoff requirement (i.e. $[1-2c\bar{f}_D(2-a\bar{f}_D)^{1/2}] > 0$. This implies that:

$$(74) > 2c\bar{f}_D(2-a\bar{f}_D)^{1/2} + \frac{1}{4}a\bar{f}_D - c\bar{f}_D(2-a\bar{f}_D)^{3/2} + \frac{3}{8}a^2\bar{f}_D^2 c + \frac{c}{2}(2-a\bar{f}_D) \tag{75}$$

Since the right-hand side is strictly positive (divide through by $\bar{f}_D(2-a\bar{f}_D)^{1/2}$ and it becomes immediately clear), that implies that Equation (75) is positive and that the inequality in Equation (72) is true.

Therefore, if the monopolist chose the prescription rate that implemented the duopolist's steady-state level of infection, then the monopolist's first-order condition would be negative. By Lemma 1, the monopolist's first-order condition is negative for all higher prescription rates, and so to reconcile the first-order condition, the monopolist must have a strictly lower steady-state level of infection than the duopolist.

□

Proof of Theorem 5

Let $F^{SP}(I,E)$ denote the planner's prescription rate when the states are (I,E). Let this value be the highest allowable prescription rate for a provider under the state-dependent quota when the states are (I,E). By the same argument as presented in Theorem 2, all perfectly competitive providers will prescribe at the highest allowable prescription rate. A subsidy to patients of $1-\kappa$ ensures that all infected patients will be seen by a provider. Therefore the aggregate prescription rate will be $F^{SP}(I,E)$.

□

8 Appendix B

In this appendix, I derive the oligopolist's equilibrium first-order condition. The oligopolist's Bellman equation when there are n total providers is:

$$V(I,E;\sigma) = max_{f^i}\Big\{[1-cf^i]\Omega(f^i,f^{-i},E)I + \delta V(I',E';\sigma)\Big\} \tag{76}$$

st (5), (6), $F = \sum_{i=1}^{n} f^i\Omega(f^i,f^{-i},E)$, $f^i \in [0,1]$

The first-order condition for a positive f^{i*} is:

$$[1-cf^{i*}]\frac{\partial\Omega(f^i,f^{-i*},E)}{\partial f^i}|_{f^i=f^{i*}}I - c\Omega(f^{i*},f^{-i*},E)I + \\ \delta\frac{\partial V(I',E';\sigma)}{\partial I'}\frac{\partial I'}{\partial F}\frac{\partial F}{\partial f^i}|_{f^i=f^{i*}} + \delta\frac{\partial V(I',E';\sigma)}{\partial E'}\frac{\partial E'}{\partial F}\frac{\partial F}{\partial f^i}|_{f^i=f^{i*}} \geq 0 \tag{77}$$

To calculate the first-order condition, I need the equations that describe the marginal effect of the oligopolist increasing his prescription rate on his market share, the equilibrium market share, the aggregate prescription rate, and the marginal effect of the oligopolist increasing his prescription rate on the aggregate prescription rate.

Let N denote the set of doctors and suppose there are n total doctors. Consider doctor i who prescribes antibiotics with probability f^i. Let the remaining $n-1$ doctors prescribe antibiotics with the symmetric probability f. Then the probability that a consumer is willing to pay the fee to see provider i is:

$$\Omega(f^i,f^{-i},E) = Pr\Big(U^i(f^i,\varepsilon^i,E) \geq U^j(f,\varepsilon^j,E) \text{ for } \ j=0,...,i-1,i+1,...,n\Big) \tag{78}$$

This is the probability that a patient prefers provider i to all other providers and is preferred to the outside option. Since the other $n-1$ providers prescribe at the symmetric rate f, this can be written as:

$$\Omega(f^i, f^{-i}, E) = Pr\Bigg(f^i Ep^A U + \varepsilon^i - 1 \geq 0 \bigcap f^i Ep^A U + \varepsilon^i \geq f Ep^A U + max_{j \in N \setminus i} \varepsilon^j \Bigg) \quad (79)$$

To ease notation, let $a = Ep^A U$. Using the Law of Conditional Probability,

$$\Omega(f^i, f^{-i}, E) = Pr(\varepsilon^i > 1 - f^i a) Pr\big(\varepsilon^i - max_{j \in N \setminus i} \varepsilon^j > (f - f^i)a \big| \varepsilon^i > 1 - f^i a\big) \quad (80)$$

since $\varepsilon^i \sim U[0,1]$ Equation (80) can be re-written as:

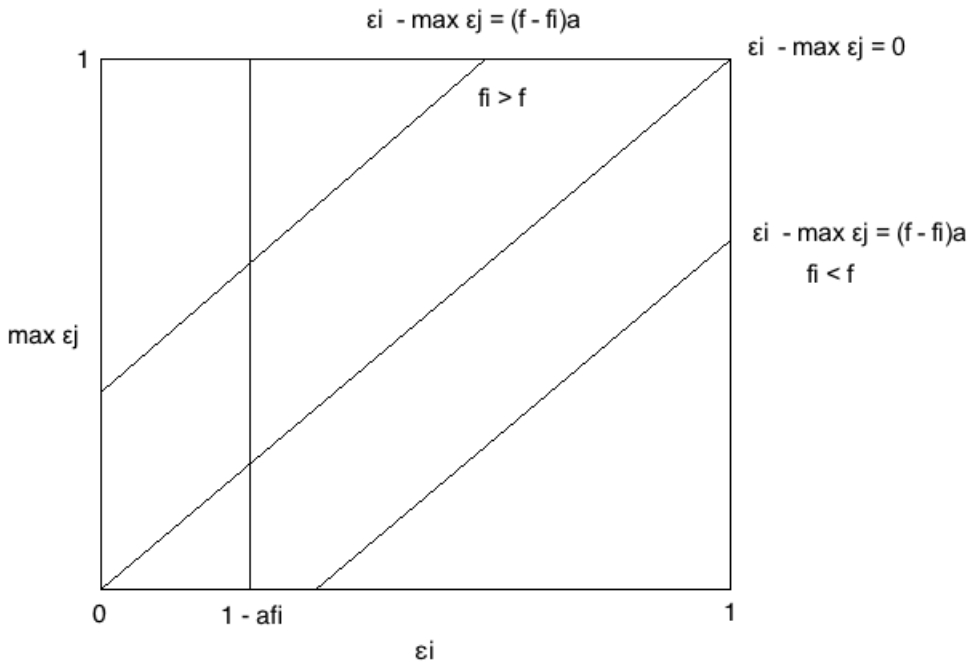
$$\Omega(f^i, f^{-i}, E) = f^i a Pr\big(\varepsilon^i - max_{j \in N \setminus i} \varepsilon^j > (f - f^i)a \big| \varepsilon^i > 1 - f^i a\big) \quad (81)$$

I now focus on the latter term of Equation (81). This is the probability that provider i is preferred to all other providers conditional on i being preferred to the outside option. Note that since there are n total providers including i, the distribution governing the maximum idiosyncratic utility a patient receives from the other $(n-1)$ providers is

$$max_{j \in N \setminus i} \varepsilon^j \sim \beta(n-1, 1) \quad (82)$$

which has pdf $f(x) = (n-1)x^{n-2}$.

I illustrate graphically the probability that provider i is preferred to all other providers conditional on i being preferred to the outside option below.



ε^i runs along the x-axis and $max_{j \in N \setminus i} \varepsilon^j$ runs along the y-axis. We are conditioning on the fact that $\varepsilon^i > 1 - f^i a$, which restricts the range of admissible values for ε^i. The iso-difference line $\varepsilon^i - max_{j \in N \setminus i} \varepsilon^j = (f - f^i)a$ gives the value of ε^i that makes the patient indifferent between seeing provider i and the next best provider, given prescription rates and a value for $max_{j \in N \setminus i} \varepsilon^j$. The region below (above) the iso-difference line are the parameter values for which provider i is preferred (not preferred) to the best of the other providers.

The probability that provider i is preferred to all other providers conditional on being preferred to the outside option is the weighted region of admissible values under the iso-difference line divided by the weighted region of all admissible values. The region of admissible values has weight af^i.

The probability - and hence Equation (81) - is a piecewise function. I derive this expression for $f^i \geq f$. In equilibrium, all providers will prescribe antibiotics at the same rate, but this more general derivation is necessary to describe the marginal effect of the oligopolist increasing his prescription rate on his market share. It can easily be verified by performing the equivalent exercise for $f^i \leq f$ that the derivative at $f^i = f$ exists.

For simplicity, I calculate the weighted region of values for which i is not preferred, divide

by the weight of admissible values af^i, and then subtract from one. The region for which i is not preferred is the triangle demarcated by the vertical line at $\varepsilon^i = 1 - af^i$, the corresponding the iso-difference line, and the top of the graph.

$$Pr\left(\varepsilon^i - max_{j\in N\setminus i}\varepsilon^j \geq a(f-f^i)\middle|\varepsilon^i \geq 1-af^i\right) = 1 - \frac{\int_{1-af^i}^{1+a(f-f^i)}\int_{x-a(f-f^i)}^{1}(n-1)y^{(n-2)}dydx}{af^i} \tag{83}$$

$$= 1 - \frac{f}{f^i} - \frac{(1-af)^n-1}{naf^i}$$

For $f^i \geq f$ provider i's market share is:

$$Pr\left(\varepsilon^i - max_{j\in N\setminus i}\varepsilon^j \geq a(f-f^i)\bigcap\varepsilon^i \geq 1-af^i\right) = af^i\left(1 - \frac{f}{f^i} - \frac{(1-af)^n-1}{naf^i}\right) \tag{84}$$

To calculate the marginal effect of the oligopolist increasing his prescription rate on his market share, differentiate (84) with respect to f^i to get:

$$\frac{\partial\Omega(f^i, f^{-i}, E)}{\partial f^i}|_{f^i=f} = a \tag{85}$$

To calculate i's equilibrium market share, set $f^i = f$ to get

$$\Omega(f, f^{-i}, E) = \frac{1}{n}\left(1 - (1-af)^n\right) \tag{86}$$

The equilibrium aggregate prescription rate is:

$$F = fPr(max_{j\in N}\varepsilon^j \geq 1-af) = f\big(1-(1-af)^n\big) \tag{87}$$

To calculate an individual oligopolist's equilibrium marginal effect on the aggregate prescription rate, consider the more general formulation,

$$F = f^i\Omega(f^i, f^{-i}, E) + fPr\Bigg(max_{j\in N\setminus i}\varepsilon^j - \varepsilon^i \geq a(f^i - f)\bigcap max_{j\in N\setminus i}\varepsilon^j \geq 1 - af\Bigg) \quad (88)$$

This expression is to be differentiated with respect to f^i and evaluated at $f^i = f$. The first part of this expression is derived using Equations (85) and (86). Focusing on the second part of the expression and using the Law of Conditional Probability,

$$fPr\Bigg(max_{j\in N\setminus i}\varepsilon^j - \varepsilon^i \geq a(f^i - f)\bigcap max_{j\in N\setminus i}\varepsilon^j \geq 1 - af\Bigg) =$$
$$fPr\Bigg(max_{j\in N\setminus i}\varepsilon^j \geq 1 - af\Bigg)Pr\Bigg(max_{j\in N\setminus i}\varepsilon^j - \varepsilon^i \geq a(f^i - f)\Bigg|max_{j\in N\setminus i}\varepsilon^j \geq 1 - af\Bigg) \quad (89)$$

Note that

$$Pr\Bigg(max_{j\in N\setminus i}\varepsilon^j \geq 1 - af\Bigg) = \int_{1-af}^{1}(n-1)x^{n-2}dx = 1 - [1 - af]^{n-1} \quad (90)$$

The last component then is a closed-form expression for

$$Pr\Bigg(max_{j\in N\setminus i}\varepsilon^j - \varepsilon^i \geq a(f^i - f)\Bigg|max_{j\in N\setminus i}\varepsilon^j \geq 1 - af\Bigg) \quad (91)$$

I derive this for $f \geq f^i$ using a similar process as before, which can then be differentiated and evaluated at the symmetric equilibrium in which $f = f^i$.

For $f \geq f^i$,

$$Pr\Bigg(max_{j\in N\setminus i}\varepsilon^j - \varepsilon^i \geq a(f^i - f)\Bigg|max_{j\in N\setminus i}\varepsilon^j \geq 1 - af\Bigg) = 1 - \frac{\int_{1-af}^{1+a(f^i-f)}\int_{x-a(f^i-f)}^{1}(n-1)x^{(n-2)}dydx}{1 - [1 - af]^{n-1}} \quad (92)$$

$$= 1 - \frac{\frac{1}{n}[1+a(f^i-f)]^n + \frac{n-1}{n}[1-af]^n - [1-af]^{n-1}[1+a(f^i-f)]}{1-[1-af]^{n-1}}$$

For $f \geq f^i$, Equation (89) can be re-written as:

$$fPr\Big(max_{j\in N\setminus i}\varepsilon^j - \varepsilon^i \geq a(f^i - f)\bigcap max_{j\in N\setminus i}\varepsilon^j \geq 1 - af\Big) =$$
$$f\Bigg(\big(1-[1-af]^{n-1}\big)\big(1-\frac{\frac{1}{n}[1+a(f^i-f)]^n + \frac{n-1}{n}[1-af]^n - [1-af]^{n-1}[1+a(f^i-f)]}{1-[1-af]^{n-1}}\big)\Bigg) \tag{93}$$

Using Equations (85), (86), and (93), we can calculate the marginal effect that increasing an oligopolist's prescription rate has on the aggregate prescription rate in the symmetric equilibrium.

$$\left.\frac{\partial F}{\partial f^i}\right|_{f^i=f} = \Omega(f, f^{-i}, E) + fa - fa + af(1-af)^{n-1} = \frac{1}{n}[1-(1-af)^n] + af(1-af)^{n-1} \tag{94}$$

We can now write the first-order condition that governs the oligopolist's symmetric Markov Perfect equilibrium. Incorporating Equations (85), (86), and (94) into the first-order condition for a positive f^* gives:

$$[1-cf^*]aI - c\frac{1}{n}[1-(1-af^*)^n]I + \delta\frac{\partial V(I', E';\sigma)}{\partial I'}(-EIp^A)\big(\frac{1}{n}[1-(1-af^*)^n] + af^*(1-af^*)^{n-1}\big) +$$
$$\delta\frac{\partial V(I', E';\sigma)}{\partial E'}\frac{-E(1-E)p^A[1+\beta(1-I)-p^R]}{\Big[1+\beta(1-I)-p^R+E\Big(\Delta p - p^A\big(f^*(1-(1-af^*)^n)\big)\Big)\Big]^2}\big(\frac{1}{n}[1-(1-af^*)^n] + af^*(1-af^*)^{n-1}\big)$$
$$\geq 0 \tag{95}$$

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