

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 compete for patients by prescribing antibiotics. Using antibiotics limits antibiotic-treatable infections, but fosters the growth of antibiotic-resistant infections. The paper demonstrates a ‘Goldilocks’ effect from provider competition. A perfectly competitive market for providers over-prescribes antibiotics because providers do not bear the cost of antibiotic-resistant infections. A patient monopolist 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. Due to more moderate antibiotic use, oligopolistic competition can be the optimal decentralized market structure. The paper then demonstrates how the model can be used for policy analysis by computing the optimal

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licensing regime, prescription quota, and tax on antibiotics.

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

JEL Classification Numbers: I11, L13, Q2

1 Introduction

Antibiotics are a special economic good. Using antibiotics causes both a positive externality by limiting the spread of antibiotic-treatable infections and a negative externality by fostering the growth of antibiotic-resistant infections (Brown and Layton 1996). While antibiotic use has greatly tempered the level of treatable infections, resistant infections have become a burdensome public health problem, costing the United States an estimated twenty billion dollars and twenty three thousand lives annually (CDC 2013; Roberts et al. 2009). The Center for Disease Control and Prevention states that the rise in “antimicrobial resistance is one of our most serious health threats” (CDC 2013). Understanding how economic incentives affect antibiotic use is a problem of first-order importance.

This paper develops a model of a fee-for-service healthcare system in which healthcare providers compete for patients by prescribing antibiotics. This is a natural component of the provider-patient relationship to focus on, as patients frequently request and receive antibiotics from their providers, even when use is medically inappropriate (Bauchner et al. 1999; Stivers 2005). The extent of such misuse by providers appears to be severe: in the United States, patients with a sore throat are prescribed antibiotics about seventy percent of the time, even though antibiotics are only effective in about ten percent of cases (Barnett and Linder 2014). Further, outpatient antibiotic use, which is the focus of the model, significantly contributes to the development of resistant infections (Seppälä et al. 1995).

The model is used to study a number of questions. First, how do the dynamics of antibiotic use generated by the decentralized decisions of providers and patients compare to the socially optimal dynamics? Relatedly, what is the optimal market structure of healthcare providers? There is significant variation in the market concentration of providers across counties in the United States (Schneider et al. 2008). More competitive markets for providers use antibiotics more frequently than less competitive markets (Bennet et al. 2014; Fogelberg

2013). However, given the competing effects of antibiotic use on treatable and resistant infection, the optimal market structure of providers is an open question in the economics literature on antibiotic resistance. Finally, what role can public policy play in correcting the potential misuse of antibiotics by providers?

To study these questions, the paper embeds an economic model of the provider-patient relationship into a dynamic epidemiological model of infection and antibiotic resistance. The economic model describes how provider and patient behavior determine antibiotic use and the epidemiological model describes how infection evolves in response to antibiotic use.

The epidemiological model features an antibiotic-treatable infection and an antibiotic-resistant infection. The two infections compete for resources in the ecosystem. When a patient with the treatable infection 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 infection, the patient's use of the antibiotic allows the resistant infection to grow and spread at a faster rate in the future.

Figures 1 and 2 show the evolution of Penicillin-treatable and Penicillin-resistant invasive *Streptococcus Pneumoniae* infections in Baltimore City, Maryland and the Nashville-Davidson region of Tennessee from 2003-2009.¹

Baltimore City, featured on the top, 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 campaign to reduce antibiotic use.² The graphs highlight competition between the treatable and resistant infections for resources: one infection grows

¹I 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/>

²<http://health.state.tn.us/ceds/Antibiotics/index.htm>

Invasive *Streptococcus Pneumoniae* Dynamics

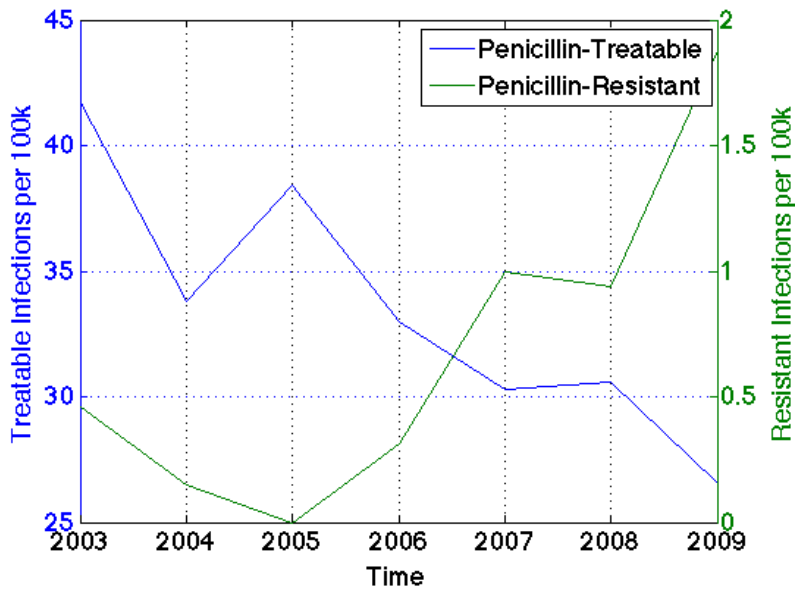


Figure 1: Baltimore City

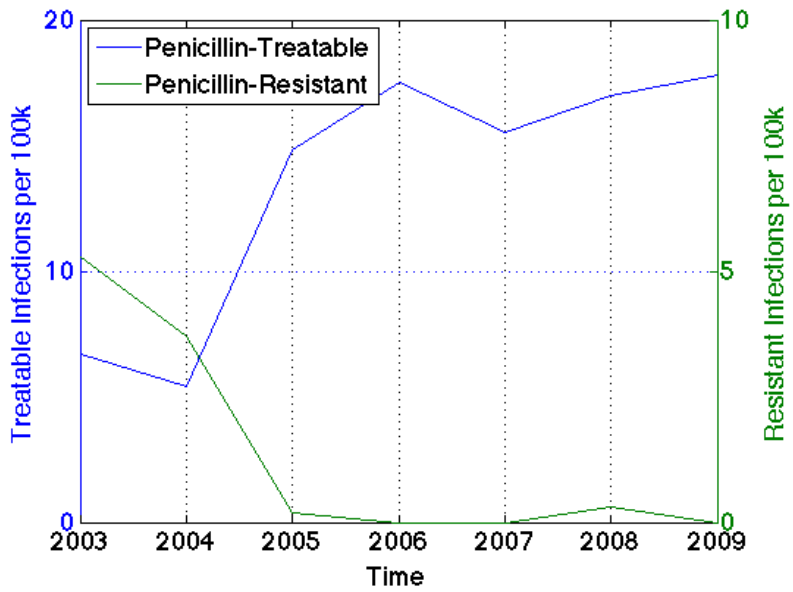


Figure 2: Nashville-Davidson

at the expense of the other. It is this competition between the two infections, the winner of which is determined by the frequency of antibiotic use, that the epidemiological model describes.

The economic model consists of a matching process between providers and patients. Providers 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 prescription behavior, the effectiveness of the antibiotic, and an 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. 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 providers extract as they treat patients with antibiotics. In the classic extraction models à la Levhari and Mirman (1980), 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.

I first look at the market allocation under perfect competition. When the market is competitive, providers have a negligible effect on the evolution of disease and therefore 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 high antibiotic use is initially a boon for society as the level of treatable infection quickly falls, the resistant infection grows at a quicker rate than is socially optimal, eventually causing a

loss that more than offsets the initial gain.

At the other end of the competitive spectrum, a patient monopolist *under-prescribes* antibiotics relative to the planner. This increases profit along two margins for the monopolist. First, the monopolist maintains 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 finds conditions for the 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 duopolists 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. However, as the market structure becomes increasingly competitive, antibiotics eventually 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 in Section 4.4). Social welfare initially increases as the market becomes more competitive from monopoly, peaks at some level of oligopolistic competition, and then decreases as the market becomes perfectly competitive. Oligopolistic competition can thus be the optimal market structure.

This “Goldilocks” effect 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 epidemiological states to increase the number of patients willing to pay the fee. This is accomplished by under-prescribing antibiotics to encourage the growth of the treatable infection and prevent the growth of the resistant infection.

At high levels of competition, the reward for patient volume incentivizes providers who lack

any market power to try to attract as many patients as possible in the current period. This leads to over-prescription of antibiotics that, while it quickly limits the treatable infection, encourages faster long-run growth of the resistant infection than is optimal.

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 does, but gives enough of a stake in the future that providers do not drive down the treatable infection as quickly as a perfectly competitive market does, slowing the growth of the resistant infection.

However, even the optimal decentralized market does not use antibiotics optimally. This raises interest in policy solutions. I demonstrate how the model can be used to evaluate the welfare effects of several different public policies aimed at mitigating provider misuse. Using numerical analysis, I compute the optimal licensing regime, quota on prescription rates, and tax on antibiotics and compare the welfare associated with each policy. Taxing antibiotic use is the superior policy for the parameter values studied.

The paper makes two primary contributions to the economics literature on antibiotic resistance (which is discussed in detail in Section 2). First, the paper develops a novel model of imperfectly competitive provision of antibiotics. The paper demonstrates new results about the optimality of oligopolistic competition and generalizes results previously discussed in the literature. Second, the paper provides a framework for 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 studies the market allocation of antibiotics. Section 5 studies the effect of public policy. Section 6 concludes. For ease of exposition, all proofs can be found in the appendix.

2 Related Literature

This section reviews the 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) study the optimal use of antibiotics. Recently, attention has turned to how markets allocate antibiotics. Elbasha (2003) estimates a static model of market provision of antibiotics in which use only causes a negative externality. Tisdell (1982) is an early contribution of a stylized two period model in which a competitive market over-uses antibiotics 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. Herrmann et al. (2015) study a monopolist's incentive to innovate a new antibiotic that is tied to the same pool of efficacy as a generically produced antibiotic. Finally, Herrmann (2010) studies the pricing behavior of a pharmaceutical company that produces an antibiotic that is temporarily protected by a patent after which there is open-access to the antibiotic.

Mechoulan (2007) and Herrmann and Gaudet (2009) are the closest to the present paper. These papers study an environment with market demand for antibiotics given by a demand curve that gets supplied by a monopolist and competitive pharmaceutical companies respectively. The primary contribution of this paper is a model that allows for the study of antibiotic provision under imperfect market competition. This is useful because not only does imperfect competition more closely reflect the structure of healthcare markets, it turns out that some degree of imperfect competition can in fact be the *optimal* market structure.

3 The Model

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

3.1 Epidemiological Model

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), being infected with a treatable infection (I^T), or being infected with an antibiotic-resistant strain (I^R). Critically, agents can be infected with the treatable or the resistant strain, but not both. This captures the notion of 'bacterial interference' (Reid et al. 2001).³

³In a more elaborate epidemiological framework, patients could be colonized with both the treatable and resistant bacteria. The externalities from antibiotic use would operate through a similar albeit more

Agents know if they are infected, but do not know the strain of the infection. Antibiotic effectiveness, E , is defined as the ratio of treatable disease to total disease in society:

$$E = \frac{I^T}{I^T + I^R} \quad (1)$$

E is the probability that, conditional on being infected, an agent is infected with the treatable infection.

Both infections are equally contagious. Let β reflect the contagiousness of the disease. Infected agents recover naturally from the infection with some probability. Let p^T denote the probability that an agent infected with the treatable disease recovers. Let p^R denote the probability that an agent infected with the resistant disease recovers. Let $\Delta p \equiv p^R - p^T$. I assume that the difference is strictly positive. A positive Δp describes what is known in the epidemiological literature as the ‘fitness cost of resistance’. The intuition is that resistance comes at a cost to the bacteria that allows the treatable bacteria to outcompete the resistant bacteria “if the selective pressure from antibiotics is reduced” (Anderson and Hughes 2010).

If an agent 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 $p^T < p^R < p^T + p^A$. That is, agents who are infected with the treatable disease and are treated with antibiotics recover quickest while agents infected with the treatable disease and are not treated recover slowest. Agents infected with the resistant disease recover at an intermediate rate. This ordinal ranking of recovery probabilities makes antibiotic effectiveness a renewable resource in the model.

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:

complicated mechanism than the one studied here.

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

where $I_t^T \beta S_t$ describes the measure of new treatable infections, $I_t^T p^T$ describes the measure of treatable infections that recover naturally, and $I_t^T p^A F_t$ the measure that recover due to antibiotic use.

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

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

For simplicity, I impose a “no-death” condition.⁴ Because the population measure stays constant, the change in the susceptible population is simply the inverse of the change in the infected population:

$$S_{t+1} = S_t - (I_{t+1}^T - I_t^T) - (I_{t+1}^R - I_t^R) \quad (4)$$

The dynamic externalities can be understood as follows: patients who are treatable and take the antibiotic recover quicker, which means they infect less of the susceptible population with the treatable strain (the positive externality). However, the patients that recovered are now susceptible to reinfection by the resistant strain, which accelerates its growth (the negative externality). This notion that patients who take antibiotics to clear a treatable infection can quickly be reinfected with resistant bacteria is observed in Kuster et al. (2014), who find that patients who recently received a course of antibiotics were more likely to be infected with a

⁴The paper studies infinitely-lived agents. However, the model can be extended to one with finitely lived agents that die due to the infection. This would require the additional structure of a birth process. Including these features would not substantively change the intuition underlying the main results.

resistant strain of *Streptococcus Pneumoniae* than patients who had not recently received a course of antibiotics.

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

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

and

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

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 $F_t > \frac{\Delta p}{p^A}$, antibiotic effectiveness is decreasing. Effectiveness decreases at high levels of antibiotic use because the treatable infection is driven out 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 recover 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.

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 is

reached asymptotically:

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

(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 is also reached asymptotically. Note that this steady-state has a higher level of infection than the other two steady-states:

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

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

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

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 *evolutionary time-scale*, and so, particularly in the case of asymptotic steady-states, the analysis of the steady-state is intended largely as a point of reference. The ‘action’ in the paper occurs along the transition path.

Note that the epidemiological model is deterministic. A natural way to make the model stochastic would be to shock β , the transmission rate. In this case, the model would feature steady-state distributions over infection and efficacy. However, for simplicity, this paper focuses on the deterministic case.

3.2 Economic Component

I now introduce an economy with a provider-patient matching process. Infected patients search for healthcare providers, who are the gatekeepers 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 an 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. Provider financial incentives have been shown to be a major driver of antibiotic abuse (Currie et al. 2014). However, 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 and providers have common knowledge of the states I and E . Utility is comprised of a component related to a patient's health status and a component related to the medical care they may purchase. The health status portion of utility is given by:

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

Infected patients can choose to be seen by a provider who may possibly prescribe antibiotics. I assume that providers cannot diagnose whether patients have the treatable or resistant infection and that their prescription rates are public knowledge. Patients pay the provider a fixed price whether or not an antibiotic is prescribed. The price is taken to be exogenous, but can be imagined to be set by an insurance company or government. The exogeneity 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 the price to 1. Providers incur a constant marginal cost of c in administering antibiotics.

Providers pick a prescription rate, which from a patient's perspective is the probability that the provider will prescribe him antibiotics. Patients randomly draw idiosyncratic utility for each provider at time 0. This can be thought of as the degree to which a healthcare provider's non-medical features, e.g. bed-side manner, distance, etc. agree with a patient. I introduce this match-specific utility component so that providers sell differentiated products. Patient m 's idiosyncratic utility for provider i is given by:

$$\varepsilon_m^i \sim U[0, 1] \tag{11}$$

If f^i is provider i 's prescription rate, then the net utility that patient m receives from seeing provider i is:

$$U_m^i(f^i, \varepsilon_m^i, E) = f^i E p^A U + \varepsilon_m^i - 1 \tag{12}$$

The first term captures the utility gained from healthcare, the second the idiosyncratic utility from provider i , and the third the cost of the visit. I assume three additional conditions. First, that $p^A U \leq 1$. This assumption ensures that demand for provider care is elastic over the entire range of admissible values in the model. Second, that $U > (p^T + p^A)U - c + 1$. Third, that $U > p^R U + 1$. These latter two assumptions ensure that from a social welfare

perspective, being healthy is always preferred to being infected and receiving care from a provider.

3.3 Social Planner's Problem

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

$$\max_{\{F_t\}_{t=0}^{\infty}} \sum_{t=0}^{\infty} \delta^t \left[(1 - I_t)U + I_t \left([E_t p^T + (1 - E_t)p^R + F_t E_t p^A]U + 1 \right) - cF_t I_t \right] \quad (13)$$

such that (5), (6), $I_0, E_0, F_t \in [0, 1]$

The discount factor is $\delta < 1$. 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.⁵ 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 infection, taking into account the cost of antibiotic use and the effect of treatment on the growth of the treatable and the resistant infections. The planner's solution can be characterized recursively through the Bellman equation:

$$V^{SP}(I, E) = \max_F \left\{ (1 - I)U + I([E p^T + (1 - E)p^R + F E p^A]U + 1) - cFI + \delta V^{SP}(I', E') \right\} \quad (14)$$

⁵I assume that the planner is able to give the best possible idiosyncratic provider service to each infected patient.

such that (5), (6), $F \in [0, 1]$.

For a positive F , the planner has the first-order condition:

$$Ep^AUI - cI + \delta \left[\frac{\partial V^{SP}(I', E')}{\partial I'} \frac{\partial I'}{\partial F} \Big|_{F=F^*} + \frac{\partial V^{SP}(I', E')}{\partial E'} \frac{\partial E'}{\partial F} \Big|_{F=F^*} \right] \geq 0 \quad (15)$$

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. Let $F^{SP}(I, E)$ refer to the planner's prescription rate when the states are (I, E) .

I use value function iteration to numerically investigate the planner's problem. I use the parameter values $\delta = .9, U = 2, \beta = .4, p^R = .25, p^T = .1, p^A = .5$, and $c = .9$ for the numerical analysis of the paper.

The planner's value function is displayed in Figure 3.

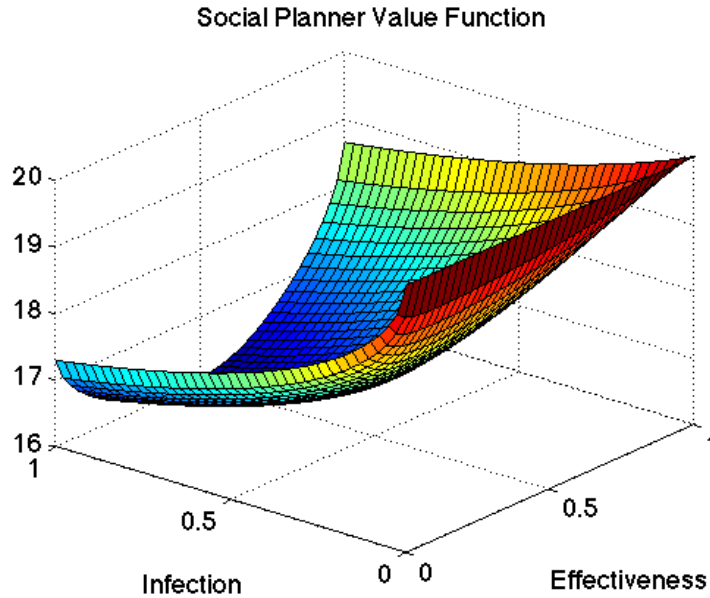


Figure 3: Planner's value function

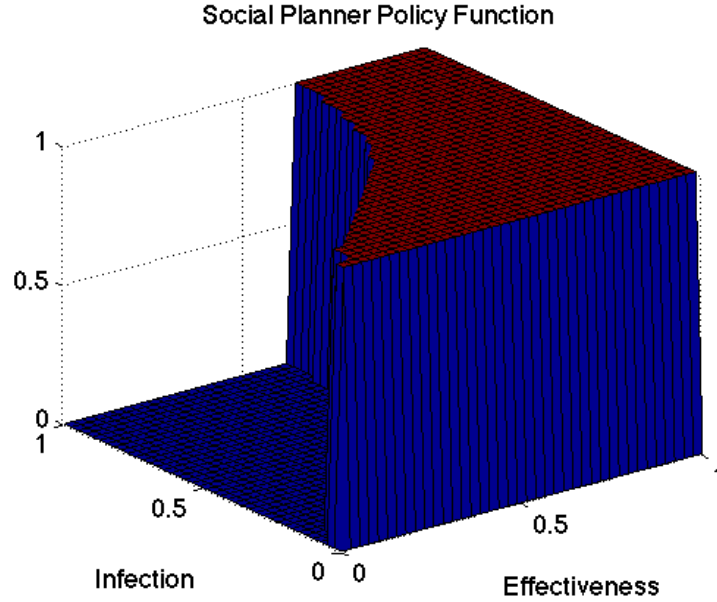


Figure 4: Optimal prescription rate

Note that the value function is convex in infection. This is because of the non-linear spread of infection. Recall that new infections are given by $I\beta(1 - I)$. Note that the rate of increase is decreasing in I . A marginal increase in 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. This is a standard result in the literature on treatment in SIS systems, although typically the literature has focused on models in which there is a single strain of infection (Goldman and Lightwood, 2002).

The value function can be used to compute the optimal prescription rate, which is displayed in Figure 4. The optimal prescription rate tends to be either zero or one - a “bang-bang” solution that is also typical in the literature on SIS systems. 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

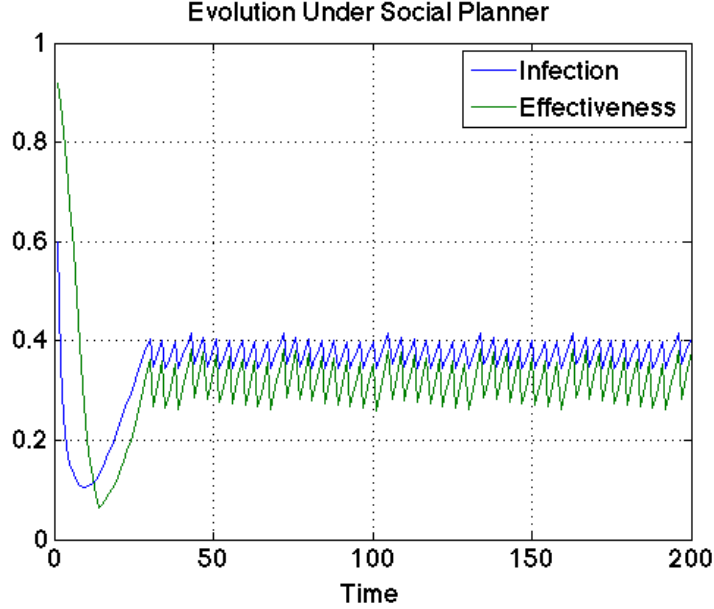


Figure 5: Planner's dynamic path

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 in Figure 5. For initial values, I use $I_0 = .6$ and $E_0 = .92$. The length of this and all other simulations is 200 periods. Due to discounting, this finite-horizon simulation well-approximates the infinite-horizon model presented in the paper.

The planner does not drive out the treatable strain entirely (because of the crowding out effect it has on the resistant strain), nor does the planner drive the resistant strain out entirely (because doing so would generate a perpetually higher level of infection). Rather than settle into a steady-state, the planner cycles antibiotic use. This is a markedly different result than has previously appeared in the literature and so it is worth elaborating on.

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. Due to competition between the two strains of the infection and the renewability of antibiotic effectiveness, the planner finds it more beneficial to cycle between periods of high treatment that drive infection and antibiotic effectiveness down and periods of low treatment in which infection and effectiveness recover than to maintain constant treatment rates.

3.4 Market Payoffs

I now describe the decentralized market decisions. If there are n total providers, then an infected agent m is willing to pay the fee to see provider i if:

$$U_m^i(f^i, \varepsilon_m^i, E) \geq U_m^j(f^j, \varepsilon_m^j, E) \quad \text{for } j = 0, \dots, i-1, i+1, \dots, n \quad (16)$$

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 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:

$$\Omega(f_t^i, f_t^{-i}, E_t) = Pr\left(U_m^i(f_t^i, \varepsilon_m^i, E_t) \geq U_m^j(f_t^j, \varepsilon_m^j, E_t) \quad j = 0, \dots, i-1, i+1, \dots, n\right) \quad (17)$$

Provider i 's per period payoff is:

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

Provider and patient behavior determines an aggregate prescription rate:

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

which can be inserted into Equations (5) and (6) to determine how infection and antibiotic effectiveness evolve. Critically, both provider prescription behavior and patient matching behavior determine the aggregate prescription rate.

Given a sequence of prescription rates, the lifetime profits of provider i are:

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

such that (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 studies symmetric Markov Perfect Equilibrium. 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 empirically observed in previous studies of patient-provider choice (Mold et al. 2004).

A Markov strategy for provider i is a mapping:

$$\sigma^i : [0, 1]^2 \rightarrow [0, 1] \quad (21)$$

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:

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

such that (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 $\sigma = (\sigma^1, \sigma^2, \dots, \sigma^n)$ 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 $i = 1, \dots, n$:

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

The equilibrium is symmetric if every provider plays the same strategy function. The Markov Perfect equilibrium can be characterized as the solution to the system of Bellman equations. Provider i 's Bellman equation is:

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

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

The first-order condition for a positive f^* in the symmetric equilibrium is:

$$\begin{aligned} [1 - cf^*] \frac{\partial \Omega(f^i, f^{-i^*}, E)}{\partial f^i} \Big|_{f^i=f^*} I - c\Omega(f^*, f^{-i^*}, E)I \\ + \delta \frac{\partial V^i(I', E'; \sigma)}{\partial I'} \frac{\partial I'}{\partial f^i} \Big|_{f^i=f^*} + \delta \frac{\partial V^i(I', E'; \sigma)}{\partial E'} \frac{\partial E'}{\partial f^i} \Big|_{f^i=f^*} \geq 0 \end{aligned} \quad (25)$$

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 equilibrium first-order condition is derived in Appendix B.

4 Market Provision

This section studies the market allocation of antibiotics. I first look at the limit case as the number of providers, n , tends towards infinity, a perfectly competitive market. I then turn to 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. Let $F^{PC}(I, E)$ denote the aggregate prescription rate under perfect competition when the states are (I, E) .

Theorem 1

1. *The symmetric equilibrium outcome under perfect competition is for every provider to set $\sigma^i(I, E) = 1$ for all (I, E) . The aggregate prescription rate is $F^{PC}(I, E) = 1$ for all (I, E) .*
2. *$F^{PC}(I, E) \geq F^{SP}(I, E)$ for all (I, E) and strictly greater for some (\tilde{I}, \tilde{E}) .*
3. *The steady-state level of effectiveness under perfect competition is $\bar{E}_{PC} = 0$.*
4. *The steady-state level of infection under perfect competition 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 possible.

This generates perpetual over-use of antibiotics by a competitive market relative to the

planner and leads to a steady-state in which the treatable strain is driven out in the limit. Contributing to this strong over-use result is the assumption that the upper bound on the support of idiosyncratic utility is the same as the provider fee (both equal to 1). This assumption means that the idiosyncratic utility patients can gain from providers is sufficiently high that patients are willing to match with their most preferred provider in a competitive setting along the entire dynamic path. While it simplifies the remaining analysis, the key takeaways from the paper do not explicitly depend on this assumption.

I display the dynamic path of infection and effectiveness under perfect competition in Figure 6. The competitive market uses the maximum amount of antibiotics. This causes total infection to initially fall, but then rebound towards the steady-state level as the resistant infection quickly grows to fill the void left from extracting the treatable infection.

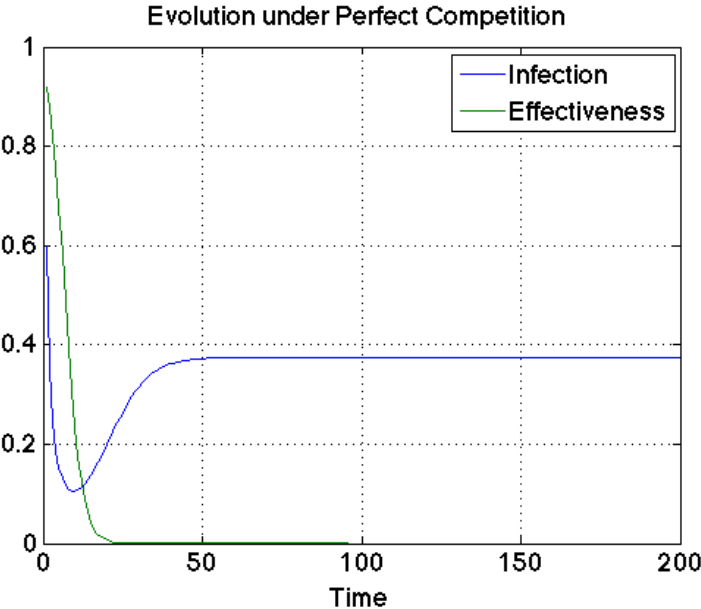


Figure 6: Perfect competition’s dynamic path

4.2 Monopolist

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 the following 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 effectiveness would tend towards one.⁶ I use this assumption to ensure that, if such behavior arises, then it is due in part to economic considerations by the monopolist.

Second, that:

$$\left[1 - 2c\left(\frac{\Delta p}{p^{A2}U}\right)^{\frac{1}{2}}\right] - \frac{\left[1 - c\left(\frac{\Delta p}{p^{A2}U}\right)^{\frac{1}{2}}\right]}{\beta - p^R} 2\Delta p < 0 \quad (\text{A1})$$

I refer to this assumption in later text as Assumption A1.

Theorem 2

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

The patient 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. These effects can be understood through the monopolist's first-order

⁶The monopolist's market share is $Pr(fEp^AU + \varepsilon - 1 \geq 0) = fEp^AU$ and so the aggregate prescription rate is $F = f^2Ep^AU$. If $\frac{\Delta p}{p^A} > p^AU$, then the aggregate prescription rate is always $F < \frac{\Delta p}{p^A}$ and effectiveness tends to one.

condition:

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

First, the monopolist has a positive current period marginal payoff requirement. When the monopolist increases his prescription rate, he has a marginal benefit from attracting new patients, but bears a loss from increasing costly treatment on patients already purchasing his services.⁷ As elaborated on below, the latter two terms in the monopolist's first-order condition are negative. This means that in order to satisfy the first-order condition, the monopolist requires that his steady-state current period marginal payoff is positive. This implies that:

$$\frac{1}{2c} \geq f_M^* \quad (27)$$

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).

Second, 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. Increasing his prescription

⁷This effect is analogous to the standard case in which a price-setting monopolist lowers his price to attract more consumers but takes a loss on the consumers already purchasing his product. As in the case of the standard monopolist, this effect can cause under-provision.

rate to attract more patients today causes a future loss to the monopolist by lowering the willingness of future patients to purchase his services. 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.

Third, under fee-for-service, infection is a good for a healthcare provider. Increasing his prescription rate to attract more patients today causes a future loss to the monopolist by lowering the number of infected patients and hence the size of the monopolist's future market. 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.

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 pay the fee.

I use value function iteration to study the behavior of the monopolist outside of the steady-state. The monopolist's value function is displayed in Figure 7.

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 in Figure 8.

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

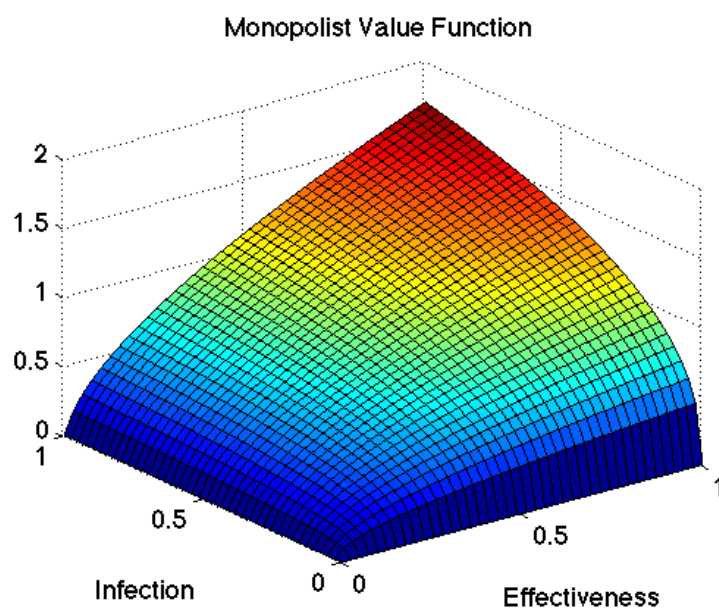


Figure 7: Monopolist's value function

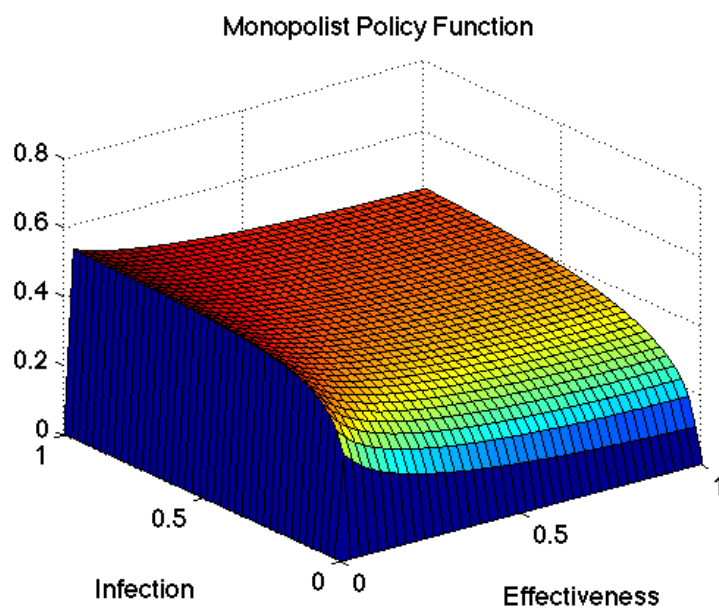


Figure 8: Monopolist's prescription rate

in Figure 9. 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.

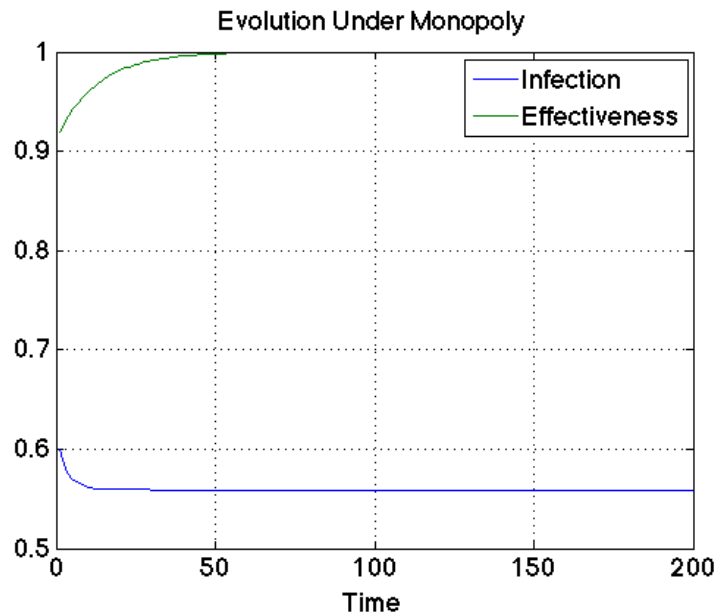


Figure 9: Monopolist's dynamic path

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 3

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 and a welfare gain for society.

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

Computational Algorithm

Step 1. Start with 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 in Figure 10.

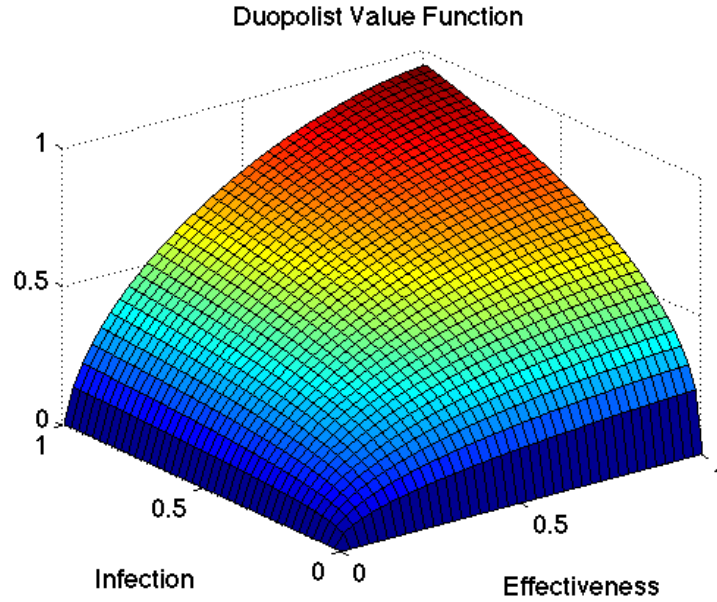


Figure 10: Duopolist's value function

The value function can be used to derive the policy function, displayed in Figure 11. In the simulation, 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, which is shown in Figure 12

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

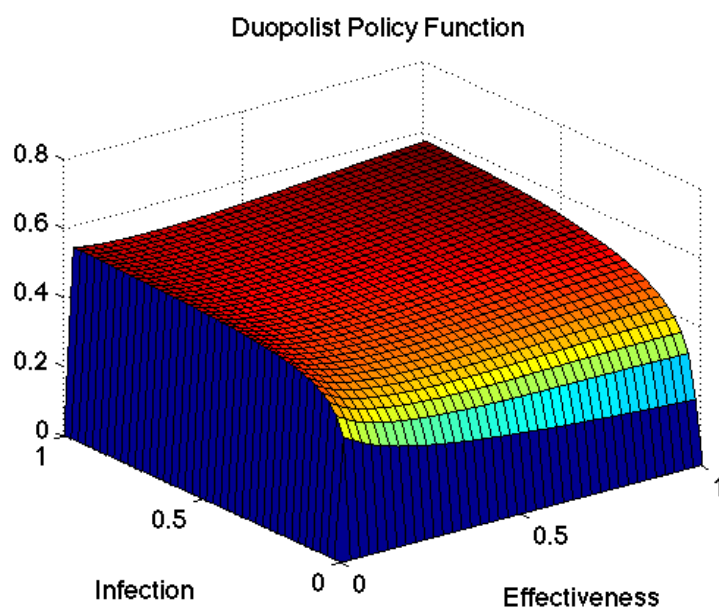


Figure 11: Duopolist's prescription rate

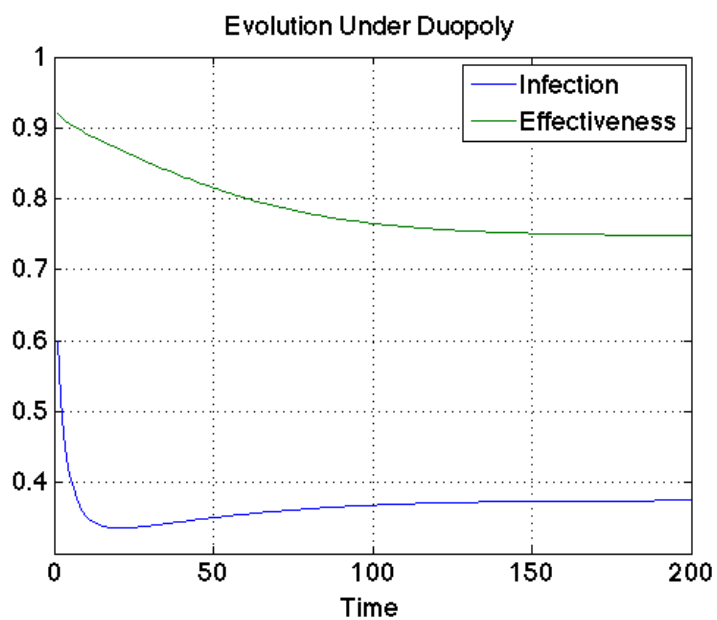


Figure 12: Duopolist's dynamic path

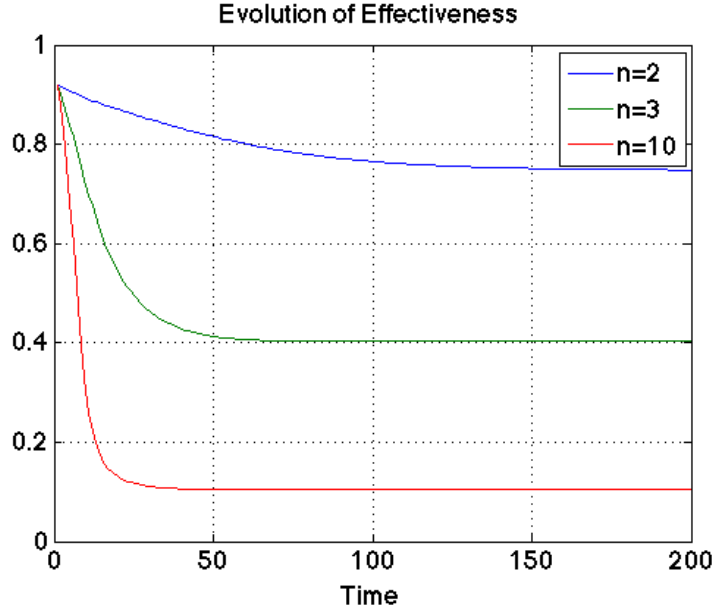


Figure 13: Effectiveness under different market structures

less on maintaining favorable states in the future and more on attracting patients in the present. Figure 13 shows 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 in Figure 14.

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

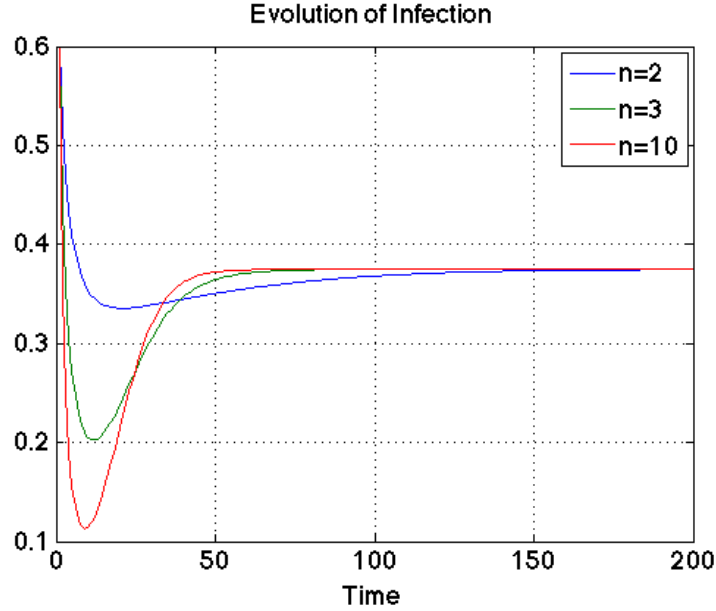


Figure 14: Infection under different market structures

infection for $n = 10$.

Note that all the market structures illustrated converge to the same steady-state level of infection, i.e. they all implement the same aggregate prescription rate in the steady-state. To see how this can occur, suppose that the market is in the steady-state and the level of competition is increased. Increased competition induces individual providers to prescribe at a higher prescription rate. However, this drives down the level of antibiotic effectiveness, which makes patients less willing to see a provider. Hence, even though individual providers are prescribing at a higher rate, enough patients become unwilling to see a provider so that the aggregate prescription rates eventually become the same.

4.4 Optimal Number of Providers

One of the central questions of the paper asks about the welfare effects of provider competition. Figure 15 gives an answer.

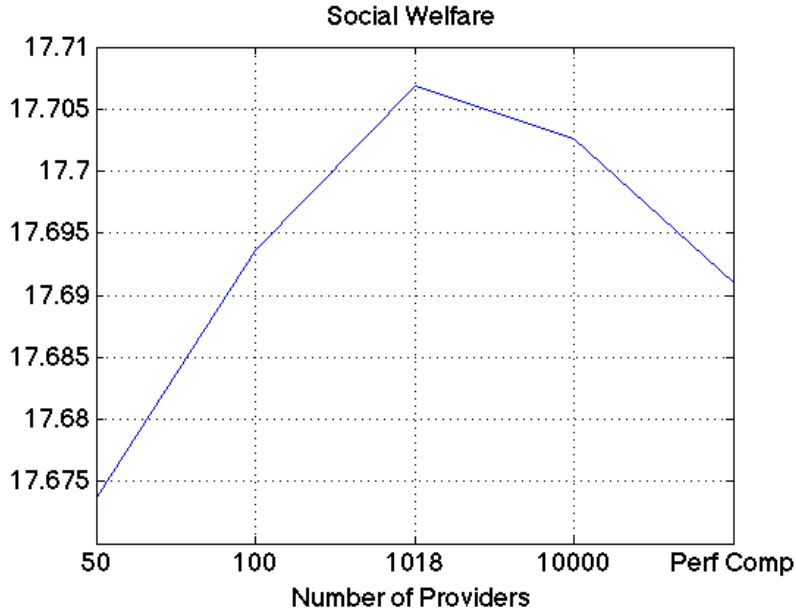


Figure 15: Oligopolistic competition is the optimal market structure

This graph plots social welfare as a function of the number of providers in a 200 period simulation. A 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 subtle. 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. Further, as competition increases, so too does the idiosyncratic utility patients gain from their providers (since they are more likely to draw a higher quality match). However, the future loss from the increase in resistant infections eventually becomes sufficiently high so as to outweigh those benefits.

The growth of the resistant infection causes two sources of welfare loss. First, it lowers the likelihood that the antibiotic will work for future users. Second, it increases the level of total infection. As illustrated in the previous section, this effect can cause the total level of infection to become higher in a more competitive market than a less competitive market.

The model thus captures a ‘Goldilocks’ effect: to a rough approximation, markets with low levels of competition use too few antibiotics and markets with high levels of competition use too much antibiotics. The optimal number of providers in the simulation is $n^* = 1018$. An interpretation is that a provider should serve about $\frac{1}{1000}$ of their market. 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

The misuse of antibiotics by a decentralized market has created interest in policy solutions. The *National Action Plan For Combating Antibiotic-Resistant Bacteria*, which was released by the White House in 2015, states: “Perhaps the single most important action to slow the development and spread of antibiotic-resistant infections is to change the way antibiotics are used.” The report instructs various governmental agencies to “review existing regulations and propose new ones, as needed... to implement robust antibiotic stewardship programs.”

The model presented here can be used to evaluate the effects of public policies aimed at mitigating the misuse of antibiotics by providers. I consider three types of policies: restricting the numbers of providers (licensing), capping how frequently a provider can prescribe antibiotics (a prescription quota) and charging an additional fee to use antibiotics (a tax).

Up to this point, the paper has studied the normative effects of market structure without

explicitly considering the number of providers as a policy variable. However, a possible public policy is restricting the number of providers through licensing. The optimal licensing regime implements the optimal decentralized market structure. The model provides a simple way to compute this: compute the equilibrium and welfare of different market structures and choose the maximum. Given the parameter values used for the simulation, the optimal number of providers is 1018 and this generates total social welfare of 17.707.

Another possible public policy takes the market structure as given and instead restricts antibiotic prescriptions. In the context of the model, a prescription quota sets a ceiling on a provider's prescription rate. The process of computing equilibrium is essentially unchanged, except now rather than being bounded by one, a provider's prescription rate is bounded by the prescription quota. Again, the model provides a simple tool to determine the optimal quota: compute the equilibrium and welfare induced by different quotas and choose the maximum. I set the number of providers $n = 10,000$. The optimal quota caps the prescription rate at $\hat{f} = .93$ and generates social welfare of 17.703.

The final policy considered here is a tax on antibiotics. I impose the tax, τ , on providers. The tax increases the provider's marginal cost of prescribing antibiotics from c to $c + \tau$. Given $n = 10,000$, the optimal tax on antibiotics sets $\tau = 0.17$ and generates social welfare of 17.722. Given the parameter values, the model predicts that a tax on antibiotics is a better policy than licensing or a prescription quota.

6 Conclusion

This paper studies a model of provider competition embedded within a dynamic epidemiological model of infection and antibiotic resistance. A 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 antibi-

otic 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 demonstrates how the model can be used for public policy analysis.

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

7 Appendix A

Throughout, I use the notation f^* to denote a provider's optimal prescription rate and \bar{f} to denote the steady-state prescription rate.

Proof of Theorem 1

Suppose that all providers prescribe at rate one for all states. Without loss of generality, suppose that provider 1 defects and prescribes at a rate less than one. Let N denote the set of providers and n the number of providers. Note that:

$$\lim_{n \rightarrow \infty} Pr\left(f^1 Ep^AU + \varepsilon^1 \geq Ep^AU + \max_{j \in N \setminus i} \varepsilon^j\right) = Pr\left(f^1 Ep^AU + \varepsilon^1 \geq Ep^AU + 1\right) = 0 \quad (28)$$

Under perfect competition, patients can always find a provider who is preferred to provider 1. Hence, by deviating, provider 1 attracts no patients and has a current period payoff of zero. Since provider 1 attracts a zero-measure set of patients whether he adheres to the equilibrium strategy or not, deviating has no effect on his continuation payoff. Therefore there is no incentive to defect from prescribing at rate one.

Since the price to see a provider is 1 and the upper bound on the support of idiosyncratic utility is also 1, given the prescription behavior of providers, the utility a patient gains from their most preferred provider is $Ep^AU > 0$. Thus all patients are seen by a provider and prescribed antibiotics. This generates an aggregate prescription rate of $F^{PC}(I, E) = 1$ for all (I, E) . The prescription behavior generates the asymptotic steady-state in which $\bar{E}_{PC} = 0$ and $\bar{I}_{PC} = \frac{\beta - p^R}{\beta}$.

To show perpetual overuse by the competitive market, it suffices to show that 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:

$$Ep^AUI - cI + \delta \left[\frac{\partial V^{SP}(I', E')}{\partial I'} \frac{\partial I'}{\partial F} \Big|_{F=F^{SP*}} + \frac{\partial V^{SP}(I', E')}{\partial E'} \frac{\partial E'}{\partial F} \Big|_{F=F^{SP*}} \right] \geq 0 \quad (29)$$

By (5) and (6):

$$\frac{\partial I'}{\partial F} = -EI p^A \quad (30)$$

and

$$\frac{\partial E'}{\partial F} = - \frac{E(1-E)p^A[1 + \beta(1-I) - p^R]}{\left[1 + \beta(1-I) - p^R + E[\Delta p - p^A F] \right]^2} \quad (31)$$

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

$$\begin{aligned} \frac{\partial V^{SP}(I, E)}{\partial I} = & [-1 + Ep^T + (1-E)p^R + F^{SP*}Ep^A]U + 1 - cF^{SP*} + \\ & \delta \frac{\partial V^{SP}(I', E')}{\partial I'} \frac{\partial I'}{\partial I} + \delta \frac{\partial V^{SP}(I', E')}{\partial E'} \frac{\partial E'}{\partial I} \end{aligned} \quad (32)$$

and

$$\frac{\partial V^{SP}(I, E)}{\partial E} = I[p^T - p^R + F^{SP*}p^A]U + \delta \frac{\partial V^{SP}(I', E')}{\partial E'} \frac{\partial E'}{\partial E} + \delta \frac{\partial V^{SP}(I', E')}{\partial I'} \frac{\partial I'}{\partial E} \quad (33)$$

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

$$-c \frac{\beta - p^R}{\beta} < 0 \quad (34)$$

which is incompatible with a positive F^* , a contradiction.

□

Proof of Theorem 2

The monopolist's market share is:

$$Pr\left(fEp^AU + \varepsilon - 1 \geq 0\right) = fEp^AU \quad (35)$$

Hence the Bellman equation is:

$$V^M(I, E; \sigma) = \max_f \left\{ [1 - cf]fEp^AUI + \delta V^M(I', E'; \sigma) \right\} \quad (36)$$

such that (5), (6), $f \in [0, 1]$, $F = f^2Ep^AU$

The first-order condition for a positive f_M^* is:

$$[1 - 2f_M^*c]Ep^AU I + \delta \left[\frac{\partial V^M(I', E'; \sigma)}{\partial I'} \frac{\partial I'}{\partial f} \Big|_{f=f_M^*} + \frac{\partial V^M(I', E', \sigma)}{\partial E'} \frac{\partial E'}{\partial f} \Big|_{f=f_M^*} \right] \geq 0 \quad (37)$$

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

$$\frac{\partial I'}{\partial f} = -EI p^A (2fEp^AU) \quad (38)$$

and

$$\frac{\partial E'}{\partial f} = - \frac{E(1-E)p^A[1 + \beta(1-I) - p^R]}{\left[1 + \beta(1-I) - p^R + E[\Delta p - p^A f(fEp^AU)] \right]^2} (2fEp^AU) \quad (39)$$

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

$$\frac{\partial V^M(I, E; \sigma)}{\partial I} = [1 - cf_M^*]f_M^*Ep^AU + \delta \frac{\partial V^M(I', E'; \sigma)}{\partial I'} \frac{\partial I'}{\partial I} + \delta \frac{\partial V^M(I', E'; \sigma)}{\partial E'} \frac{\partial E'}{\partial I} \quad (40)$$

and

$$\frac{\partial V^M(I, E; \sigma)}{\partial E} = [1 - cf_M^*]f_M^*p^AU I + \delta \frac{\partial V^M(I', E'; \sigma)}{\partial E'} \frac{\partial E'}{\partial E} + \delta \frac{\partial V^M(I', E'; \sigma)}{\partial I'} \frac{\partial I'}{\partial E} \quad (41)$$

In the steady-state, $I = I'$ and $E = E'$. The monopolist cannot have a steady-state in which $E = 0$ because $F = f^2Ep^AU$ which for low enough E is below the threshold $\frac{\Delta p}{p^A}$. Enough patients stop going to see the monopolist that he cannot maintain an aggregate prescription rate high enough to drive effectiveness to 0. There are two possible steady-state configurations: one with an interior level of effectiveness and one in which effectiveness equals one.

In an interior steady-state, the envelope conditions are:

$$\frac{\partial V^M(\bar{I}_M, \bar{E}_M; \sigma)}{\partial I} = \frac{[1 - c\bar{f}_M]\bar{f}_M\bar{E}_Mp^AU}{1 - \delta[1 - (\beta - p^r)]} \quad (42)$$

and

$$\frac{\partial V^M(\bar{I}_M, \bar{E}_M)}{\partial E} = \frac{[1 - c\bar{f}_M]^{\frac{\beta - p^r}{\beta}}\bar{f}_Mp^AU}{1 - \delta} \quad (43)$$

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

$$\frac{\Delta p}{p^A\bar{f}_M^2} \frac{\beta - p^r}{\beta} \left([1 - 2c\bar{f}_M] - \frac{\delta[1 - c\bar{f}_M]}{1 - \delta[1 - (\beta - p^r)]} 2\left(\frac{\Delta p^2}{p^{A2}U\bar{f}_M^2}\right) - \frac{\delta[1 - c\bar{f}_M]}{1 - \delta} 2\Delta p\left(1 - \left(\frac{\Delta p}{p^{A2}U\bar{f}_M^2}\right)\right) \right) \geq 0 \quad (44)$$

such that $\bar{f}_M \in \left(\frac{\Delta p}{p^{A2}U}\right]^{\frac{1}{2}}, 1]$

In the steady-state in which effectiveness equals one, the steady-state envelope condition governing infection is:

$$\frac{\partial V^M(\bar{I}_M, \bar{E}_M; \sigma)}{\partial I} = \frac{[1 - c\bar{f}_M]\bar{f}_Mp^AU}{1 - \delta(1 - (\beta - p^T - \bar{f}_M^2p^{A2}U))} \quad (45)$$

and since $\frac{\partial E}{\partial f} = 0$ in this steady-state, the monopolist's the steady-state first-order condition when effectiveness is one can be written as:

$$(\bar{I}_Mp^AU) \left([1 - 2c\bar{f}_M] - \delta \frac{[1 - c\bar{f}_M]2p^{A2}U\bar{f}_M^2}{1 - \delta(1 - (\beta - p^T - \bar{f}_M^2p^{A2}U))} \right) = 0 \quad (46)$$

such that $\bar{f}_M \in [0, \frac{\Delta p}{p^{A^2 U}}]^{\frac{1}{2}}$, $\bar{I}_M = \frac{\beta - p^T - \bar{f}_M^2 p^{A^2 U}}{\beta}$

Notice that either Equation 44 or Equation 46 admits a solution.

I now argue that the monopolist's steady-state level of effectiveness $\bar{E}_M \rightarrow 1$ as $\delta \rightarrow 1$. Suppose by contradiction that \bar{E}_M is bounded away from 1 for all δ . This means that \bar{f}_M is bounded above $[\frac{\Delta p}{p^{A^2 U}}]^{\frac{1}{2}}$ for all δ .

Notice that for all values of δ , the first two terms in the monopolist's interior-steady state first-order condition (Equation 44) are finite. However, because \bar{f}_M is bounded above $[\frac{\Delta p}{p^{A^2 U}}]^{\frac{1}{2}}$, the third term of the monopolist's first-order condition becomes arbitrarily small as $\delta \rightarrow 1$. 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.

I now argue that for sufficiently high δ , the monopolist's steady-state level of infection is $\bar{I}_M > \frac{\beta - p^r}{\beta}$. Suppose this was not the case. Since $\bar{E}_M \rightarrow 1$ and $\bar{I}_M = \frac{\beta - p^r}{\beta}$, as $\delta \rightarrow 1$, the monopolist's first-order condition tends to:

$$\left(\frac{\beta - p^R}{\beta} p^{A^2 U}\right) \left(\left[1 - 2c \left(\frac{\Delta p}{p^{A^2 U}}\right)^{\frac{1}{2}}\right] - \frac{\left[1 - c \left(\frac{\Delta p}{p^{A^2 U}}\right)^{\frac{1}{2}}\right] 2\Delta p}{\beta - p^R} \right) < 0 \quad (47)$$

which is negative by assumption A1. Hence, a sufficiently patient monopolist has an inefficiently high steady-state level of infection.

□

Lemma 1

I now state and prove a lemma that will be used in proving Theorem 3.

Lemma 1: Suppose that

$$(I(f)p^{A^2 U}) \left(\left[1 - 2cf\right] - \delta \frac{\left[1 - cf\right] 2p^{A^2 U} f^2}{1 - \delta(1 - \beta I(f))} \right) \quad (48)$$

where $I(f) = \frac{\beta - p^T - f^2 p^{A2} U}{\beta}$ is negative for some $f \in [0, 1]$. Then, the expression is negative for all $\hat{f} \geq f$.

Proof:

The term $I(f)p^A U$ is always positive, so it suffices to show that if the second term is negative, then it is negative for all higher values of f . Suppose that the second term is negative at f . Differentiating the second term with respect to f gives:

$$-2c - \frac{4fp^{A2}U[1 - \frac{6}{4}cf]\left(1 - \delta(1 - \beta I(f))\right) - (1 - cf)2fp^{A2}U\delta\beta\frac{\partial I}{\partial f}}{\left(1 - \delta(1 - \beta I(f))\right)^2} \quad (49)$$

which, because $\frac{\partial I}{\partial f} < 0$, is negative unless $[1 - \frac{6}{4}cf]$ is sufficiently negative. However, if $[1 - \frac{6}{4}cf] < 0$, then $[1 - 2cf] < 0$, which means that Expression 48 is negative. Hence, if 48 evaluated at f is negative, then 48 is negative for all $\hat{f} \geq f$.

□

Proof of Theorem 3

Let δ be sufficiently high that the monopolist's steady-state level of infection is $\bar{I}_M > \frac{\beta - p^r}{\beta}$. By the analysis in Appendix B, the duopolist's first-order condition is:

$$\begin{aligned} & [1 - 2cf_D^*]Ep^AUI + \frac{1}{2}c(Ep^A U f_D^*)^2 I + \delta \frac{\partial V^D(I', E'; \sigma)}{\partial I'} (-EI p^A) (2Ep^A U f_D^* - \frac{3}{2}(Ep^A U f_D^*)^2) + \\ & \delta \frac{\partial V^D(I', E'; \sigma)}{\partial E'} \frac{-E(1 - E)p^A [1 + \beta(1 - I) - p^R] (2Ep^A U f_D^* - \frac{3}{2}(Ep^A U f_D^*)^2)}{\left[1 + \beta(1 - I) - p^R + E \left(\Delta p - p^A (f_D^* (1 - (1 - Ep^A U f_D^*)^2)) \right) \right]^2} \\ & \geq 0 \quad (50) \end{aligned}$$

The envelope condition governing infection is:

$$\frac{\partial V^D(I, E; \sigma)}{\partial I} = [1 - cf_D^*] \frac{1}{2} [1 - (1 - Ep^A U f_D^*)^2] + \delta \frac{\partial V^D(I', E'; \sigma)}{\partial I'} \frac{\partial I'}{\partial I} + \delta \frac{\partial V^D(I', E'; \sigma)}{\partial E'} \frac{\partial E'}{\partial I} \quad (51)$$

The envelope condition governing effectiveness is:

$$\frac{\partial V(I, E; \sigma)}{\partial E} = [1 - cf_D^*] I [1 - Ep^A U f_D^*] p^A U f_D^* + \delta \frac{\partial V^D(I', E'; \sigma)}{\partial I'} \frac{\partial I'}{\partial E} + \delta \frac{\partial V^D(I', E'; \sigma)}{\partial E'} \frac{\partial E'}{\partial E} \quad (52)$$

If the duopolist has an interior steady-state level of effectiveness, then the monopolist automatically has a higher level of infection. Suppose the duopolist has a steady-state in which effectiveness is one. For notational convenience, let $a = p^A U$. The steady-state first-order condition in which antibiotic effectiveness is one can be written as:

$$[1 - 2c\bar{f}_D] a \bar{I}_D + \frac{1}{2} c (a\bar{f}_D)^2 \bar{I}_D + \delta \frac{[1 - c\bar{f}_D] [a\bar{f}_D - \frac{(a\bar{f}_D)^2}{2}]}{1 - \delta(1 - \beta\bar{I}_D)} (-I_D^* p^A) (2a\bar{f}_D - \frac{3}{2} (a\bar{f}_D)^2) = 0 \quad (53)$$

such that $(\bar{f}_D(2a\bar{f}_D - (a\bar{f}_D)^2)) < \frac{\Delta p}{p^A}$, $\bar{I}_D = \frac{\beta - p^T - p^A(\bar{f}_D(2a\bar{f}_D - (a\bar{f}_D)^2))}{\beta}$, $\bar{f}_D \in [0, \tilde{f}]$

where \tilde{f} is defined implicitly by the equality:

$$2a\tilde{f}^2 - a^2\tilde{f}^3 = \frac{\Delta p}{p^A} \quad (54)$$

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. By Lemma 1, this means that the monopolist must pick a lower prescription rate and therefore a higher level of infection, proving the claim.

The aggregate prescription rate in the duopolist's steady-state is:

$$F = \bar{f}_D(2a\bar{f}_D - (a\bar{f}_D)^2) \quad (55)$$

For the monopolist to implement the duopolist's steady-state level of infection, the aggregate prescription rate must be the same, so the monopolist must set:

$$f_M^2 a = \bar{f}_D(2a\bar{f}_D - (a\bar{f}_D)^2) \quad (56)$$

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:

$$[1 - 2c\bar{f}_D(2 - a\bar{f}_D)^{1/2}]a\bar{I}_D - \delta \frac{(1 - c\bar{f}_D(2 - a\bar{f}_D)^{1/2})2a^2\bar{f}_D^2(2 - a\bar{f}_D)\bar{I}_D p^A}{1 - \delta(1 - \beta\bar{I}_D)} \quad (57)$$

The objective is to show that this expression is negative. Recall the positive current period marginal payoff requirement. In order for this expression to be non-negative, it is required that:

$$[1 - 2c\bar{f}_D(2 - a\bar{f}_D)^{1/2}] > 0 \quad (58)$$

Because $[1 - 2c\bar{f}_D]a\bar{I}_D + \frac{1}{2}c(a\bar{f}_D)^2\bar{I}_D > [1 - 2c\bar{f}_D(2 - a\bar{f}_D)^{1/2}]a\bar{I}_D$, Expression 57 is less than:

$$\delta \frac{[1 - c\bar{f}_D][a\bar{f}_D - \frac{(a\bar{f}_D)^2}{2}]}{1 - \delta[1 - \beta\bar{I}_D]} (\bar{I}_D p^A) (2a\bar{f}_D - \frac{3}{2}(a\bar{f}_D)^2) - \delta \frac{(1 - c\bar{f}_D(2 - a\bar{f}_D)^{1/2})2a^2\bar{f}_D^2(2 - a\bar{f}_D)\bar{I}_D p^A}{1 - \delta(1 - \beta\bar{I}_D)} \quad (59)$$

It suffices to show that 59 is either negative (which would imply that 57 is negative) or that the monopolist's current period positive marginal payoff requirement is violated (which would also imply that 57 is negative). 59 is negative if:

$$(1 - c\bar{f}_D(2 - a\bar{f}_D)^{1/2})2a^2\bar{f}_D^2(2 - a\bar{f}_D) > [1 - c\bar{f}_D][a\bar{f}_D - \frac{(a\bar{f}_D)^2}{2}](2a\bar{f}_D - \frac{3}{2}(a\bar{f}_D)^2) \quad (60)$$

Dividing both sides by $2a^2\bar{f}_D^2(1 - \frac{a\bar{f}_D}{2})$ and subtracting the right-hand side from the left-hand side yields:

$$1 - 2c\bar{f}_D(2 - a\bar{f}_D)^{\frac{1}{2}} + c\bar{f}_D + \frac{3}{4}a\bar{f}_D(1 - \bar{f}_Dc) \quad (61)$$

This expression is positive unless the monopolist's positive current period marginal payoff requirement is violated. Either way, the implication is that Equation 1.57, the monopolist's steady-state first-order condition evaluated at the duopolist's solution, is 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 higher steady-state level of infection than the duopolist.

□

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:

$$V(I, E; \sigma) = \max_{f^i} \left\{ [1 - cf^i]\Omega(f^i, f^{-i}, E)I + \delta V(I', E'; \sigma) \right\} \quad (62)$$

such that (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:

$$[1 - cf^{i*}] \frac{\partial \Omega(f^i, f^{-i*}, E)}{\partial f^i} \Big|_{f^i=f^{i*}} I - c\Omega(f^{i*}, f^{-i*}, E) I + \delta \frac{\partial V(I', E'; \sigma)}{\partial I'} \frac{\partial I'}{\partial F} \frac{\partial F}{\partial f^i} \Big|_{f^i=f^{i*}} + \delta \frac{\partial V(I', E'; \sigma)}{\partial E'} \frac{\partial E'}{\partial F} \frac{\partial F}{\partial f^i} \Big|_{f^i=f^{i*}} \geq 0 \quad (63)$$

To calculate the first-order condition, I need the functions that govern 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 at rate f^i . Let the remaining $n - 1$ doctors prescribe antibiotics at the symmetric rate f . Then the probability that a consumer is willing to pay the fee to see provider i is:

$$\Omega(f^i, f^{-i}, E) = Pr\left(U^i(f^i, \varepsilon^i, E) \geq U^j(f, \varepsilon^j, E) \text{ for } j = 0, \dots, i - 1, i + 1, \dots, n\right) \quad (64)$$

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:

$$\Omega(f^i, f^{-i}, E) = Pr\left(f^i Ep^AU + \varepsilon^i - 1 \geq 0 \cap f^i Ep^AU + \varepsilon^i \geq f Ep^AU + \max_{j \in N \setminus i} \varepsilon^j\right) \quad (65)$$

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

$$\Omega(f^i, f^{-i}, E) = Pr(\varepsilon^i > 1 - f^i a) Pr(\varepsilon^i - \max_{j \in N \setminus i} \varepsilon^j > (f - f^i)a | \varepsilon^i > 1 - f^i a) \quad (66)$$

since $\varepsilon^i \sim U[0, 1]$ Equation 66 can be re-written as:

$$\Omega(f^i, f^{-i}, E) = f^i a Pr(\varepsilon^i - \max_{j \in N \setminus i} \varepsilon^j > (f - f^i)a | \varepsilon^i > 1 - f^i a) \quad (67)$$

I now focus on the latter term of Equation 67. 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

$$\max_{j \in N \setminus i} \varepsilon^j \sim \beta(n - 1, 1) \quad (68)$$

which has the probability density function $f(x) = (n - 1)x^{n-2}$.

I illustrate graphically the probability that provider i is preferred to all other providers conditional on i being preferred to the outside option in Figure 16.

ε^i runs along the x-axis and $\max_{j \in N \setminus i} \varepsilon^j$ runs along the y-axis. Conditioning on the fact that $\varepsilon^i > 1 - f^i a$, 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 $a f^i$.

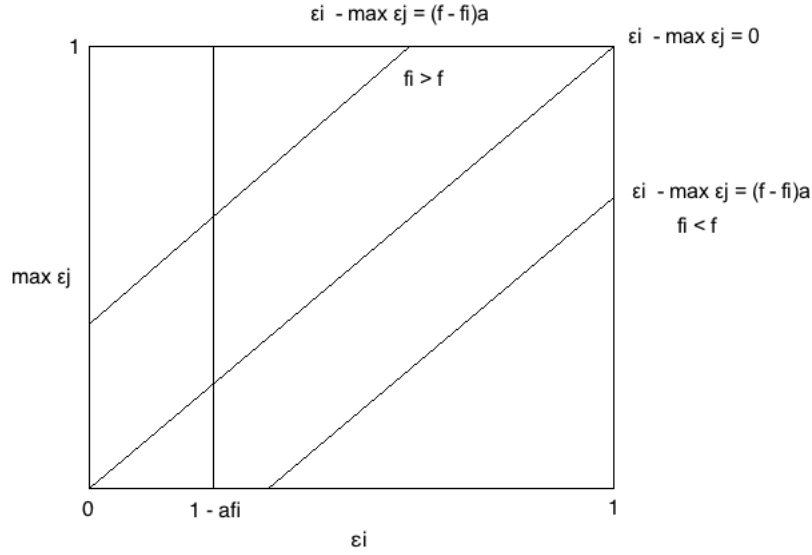


Figure 16: Illustration of parameter values for which i is preferred

The probability - and hence Equation 67 - 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.

$$Pr\left(\varepsilon^i - \max_{j \in N \setminus i} \varepsilon^j \geq a(f - f^i) \mid \varepsilon^i \geq 1 - af^i\right) = 1 - \frac{\int_{1-af^i}^{1+af-f^i} \int_{x-af^i}^1 (n-1)y^{(n-2)} dy dx}{af^i} \quad (69)$$

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

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

$$Pr\left(\varepsilon^i - \max_{j \in N \setminus i} \varepsilon^j \geq a(f - f^i) \cap \varepsilon^i \geq 1 - af^i\right) = af^i \left(1 - \frac{f}{f^i} - \frac{(1 - af)^n - 1}{naf^i}\right) \quad (70)$$

To calculate the marginal effect of the oligopolist increasing his prescription rate on his market share, differentiate 70 with respect to f^i to get:

$$\frac{\partial \Omega(f^i, f^{-i}, E)}{\partial f^i} \Big|_{f^i=f} = a \quad (71)$$

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

$$\Omega(f, f^{-i}, E) = \frac{1}{n} \left(1 - (1 - af)^n\right) \quad (72)$$

The equilibrium aggregate prescription rate is:

$$F = fPr(\max_{j \in N} \varepsilon^j \geq 1 - af) = f(1 - (1 - af)^n) \quad (73)$$

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

$$F = f^i \Omega(f^i, f^{-i}, E) + fPr\left(\max_{j \in N \setminus i} \varepsilon^j - \varepsilon^i \geq a(f^i - f) \cap \max_{j \in N \setminus i} \varepsilon^j \geq 1 - af\right) \quad (74)$$

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 71 and 72. Focusing on the second part of the expression and using the Law of Conditional Probability,

$$fPr\left(\max_{j \in N \setminus i} \varepsilon^j - \varepsilon^i \geq a(f^i - f) \cap \max_{j \in N \setminus i} \varepsilon^j \geq 1 - af\right) = fPr\left(\max_{j \in N \setminus i} \varepsilon^j \geq 1 - af\right)Pr\left(\max_{j \in N \setminus i} \varepsilon^j - \varepsilon^i \geq a(f^i - f) \middle| \max_{j \in N \setminus i} \varepsilon^j \geq 1 - af\right) \quad (75)$$

Note that

$$Pr\left(\max_{j \in N \setminus i} \varepsilon^j \geq 1 - af\right) = \int_{1-af}^1 (n-1)x^{n-2}dx = 1 - [1 - af]^{n-1} \quad (76)$$

The last component then is a closed-form expression for

$$Pr\left(\max_{j \in N \setminus i} \varepsilon^j - \varepsilon^i \geq a(f^i - f) \middle| \max_{j \in N \setminus i} \varepsilon^j \geq 1 - af\right) \quad (77)$$

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 $f \geq f^i$,

$$Pr\left(\max_{j \in N \setminus i} \varepsilon^j - \varepsilon^i \geq a(f^i - f) \middle| \max_{j \in N \setminus i} \varepsilon^j \geq 1 - af\right) = 1 - \frac{\int_{1-af}^{1+a(f^i-f)} \int_{x-a(f^i-f)}^1 (n-1)x^{(n-2)}dydx}{1 - [1 - af]^{n-1}} \quad (78)$$

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

For $f \geq f^i$, Equation 75 can be re-written as:

$$fPr\left(\max_{j \in N \setminus i} \varepsilon^j - \varepsilon^i \geq a(f^i - f) \cap \max_{j \in N \setminus i} \varepsilon^j \geq 1 - af\right) = f\left((1 - [1 - af]^{n-1})\left(1 - \frac{\frac{1}{n}[1 + a(f^i - f)]^n + \frac{n-1}{n}[1 - af]^n - [1 - af]^{n-1}[1 + a(f^i - f)]}{1 - [1 - af]^{n-1}}\right)\right) \quad (79)$$

Using Equations 71, 72, and 79, I can calculate the marginal effect that increasing an oligopolist's prescription rate has on the aggregate prescription rate in the symmetric equilibrium.

$$\left. \frac{\partial F}{\partial f^i} \right|_{f^i=f} = \Omega(f, f^{-i}, E) + fa - fa + af(1 - af)^{n-1} = \frac{1}{n}[1 - (1 - af)^n] + af(1 - af)^{n-1} \quad (80)$$

I can now write the first-order condition that governs the oligopolist's symmetric Markov Perfect equilibrium. Incorporating Equations 71, 72, and 80 into the first-order condition for a positive f^* gives:

$$\begin{aligned} & [1 - cf^*]aI - c\frac{1}{n}[1 - (1 - af^*)^n]I + \delta \frac{\partial V(I', E'; \sigma)}{\partial I'} (-EI p^A) \left(\frac{1}{n}[1 - (1 - af^*)^n] + af^*(1 - af^*)^{n-1} \right) + \\ & \delta \frac{\partial V(I', E'; \sigma)}{\partial E'} \frac{-E(1 - E)p^A[1 + \beta(1 - I) - p^R] \left(\frac{1}{n}[1 - (1 - af^*)^n] + af^*(1 - af^*)^{n-1} \right)}{\left[1 + \beta(1 - I) - p^R + E \left(\Delta p - p^A(f^*(1 - (1 - af^*)^n)) \right) \right]^2} \\ & \geq 0 \quad (81) \end{aligned}$$

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