Optimal Antimicrobial Use
under Countervailing Externalities

Anthony R. Delmond and Haseeb Ahmed

Over- or underprovision of antimicrobials under free-riding and resistance externalities can be economically important through their impacts on animal health, human health, and food security. This paper models antimicrobial use given disease dynamics with (i) free-riding incentives and (ii) antimicrobial resistance. Our results suggest a strong potential for overprovision of antimicrobials when ignoring resistance dynamics. Numerical simulation indicates an increase in the cost of disease management with increases in resistance levels. Policy implications are discussed in the context of animal health and disease-control subsidy programs in the developing world as well as unregulated sale of antimicrobials.

Key words: antimicrobial resistance, free riding, livestock disease, smallholder production

Introduction

Livestock owners use antimicrobials (antibiotics, acaricides, and other antiparasitic agents) for disease prevention and treatment. However, the use of antimicrobials, especially for prophylaxis and subclinical disease management, can be suboptimal due to free-riding incentives (Bauch and Earn, 2004; Gramig, Horan, and Wolf, 2009; Hennessy and Wolf, 2018). Further, limited access to veterinary services, low availability of antimicrobials, and credit constraints also can hamper the demand for antimicrobials, especially in smallholder agricultural households (Marsh et al., 2016; Caudell et al., 2017; Railey et al., 2018). Given that livestock health has economic, food security, and public health implications, some governments have designed subsidy programs to promote antimicrobial use in the livestock sector to overcome access and free-rider problems (Maziku, Mruttu, and Tegegn, 2016; Mwaseba and Kigoda, 2017).

Since access, availability, and free ridership are not the only problems associated with antimicrobial use, subsidy programs that promote antimicrobial use in agriculture may end up having unintended consequences. The costs of morbidity and mortality have been increasing worldwide, largely owing to antimicrobial resistance (Laxminarayan et al., 2016), and the use of veterinary antimicrobials in livestock can be important contributors to the emergence and transmission of antimicrobial resistance (Carlet et al., 2012; Van Boeckel et al., 2015; Ahmed et al., 2018). Since private decision makers may not account for the external costs imposed on other livestock owners through resistance, they may overuse antimicrobials from a social economic perspective, exacerbating the emergence of antimicrobial resistance (Brown and Layton, 1996; Laxminarayan and Brown, 2001; Secchi and Babcock, 2002; Althouse, Bergstrom, and Bergstrom, 2010).

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This paper examines the optimal level of preventive antimicrobial use in agriculture under free-riding incentives and antimicrobial effectiveness in the context of smallholders in a developing country. We focus on a smallholder setting since commercial farms may have easy access to antimicrobials (commonly used for prophylaxis and growth promotion) and less incentive to free ride given their share of the market. Therefore, externalities associated with drug resistance may be more pronounced in that setting (Levy, 1998; Orzech and Nichter, 2008). However, the extent of free ridership and resistance is rather unclear in the smallholder setting.[1]

Antimicrobial effectiveness is modeled as a nonrenewable resource in a dynamic optimization framework, following the existing literature on the economics of antibiotic resistance (Laxminarayan and Brown, 2001; Elbasha, 2003; Wilen and Msangi, 2003). The initial model of free-riding incentives is fairly standard, and it illustrates the effects of free riding on antimicrobial use given disease dynamics. We then add antimicrobial resistance as a dynamic constraint and compare the two models, examining several cases conditional on the extent of both externalities. Our numerical simulation and phase diagrams indicate that it becomes increasingly difficult to manage a steady level of disease prevalence as levels of resistance increase. Policy implications are discussed in the context of animal health and disease control subsidy programs of the developing world as well as unregulated antimicrobial sales and use, which can be conceptualized as an implicit subsidy of sorts.

This article contributes to the literature in two ways. First, free-riding and resistance externalities are modeled separately in the literature on antimicrobial use. Modeling the two separately could lead to two types of errors in policy prescription. Only modeling access issues and the free-rider problem could prescribe subsidization policies, which in turn lead to increased antimicrobial use and resistance. Conversely, modeling only resistance could lead to policies that may limit or ban the use of antimicrobials where they are necessary for disease prevention or treatment. Second, in modeling the two externalities together in a dynamic optimization framework, the paper shows how infection burden becomes increasingly unsustainable as levels of resistance increase, suggestive of increasing costs of dynamic disease management under increasing resistance.

Related Literature

Explicitly modeling free-rider and resistance externalities jointly in the context of antimicrobials (and more specifically for smallholders in the developing world) is novel, though the two externalities have been previously modeled and discussed independently. The general free-rider problem has featured in the literature of public choice since as early as Hume (1955, originally published 1740), who described the complexity of procuring public goods. Wicksell (1967, originally published 1896) and Lindahl (1967, originally published 1919) are credited with first formalizing the problem, which has since been extended and expounded upon by Musgrave (1939), Bowen (1943), and numerous others.

Regarding models of antibiotics and vaccination, Althouse, Bergstrom, and Bergstrom (2010) built on the mathematical framework and economic theory of public choice introduced by Samuelson (1954, 1955) . As in our paper, they base their model on private decision making, and their main concern is to identify optimal allocation of public health interventions, defined as vaccinations, antibiotics, and antivirals. They use both economic and epidemiological modeling approaches, depending on the intervention method, but their analysis excludes prophylaxis or subclinical disease—the main focus of this research. This eliminates the free riding associated with preventative

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[1] Smallholders account for about 60% of global food production and may be important contributors to antimicrobial use and antimicrobial resistance (Graeub et al., 2016; Caudell et al., 2017). The World Bank defines smallholder farms as those with less than 2 hectares of land. For our purposes, we define a smallholder farmer as someone with a limited number of production animals and no market power. Indeed, we can find many countries and contexts in which such farms exist in abundance and provide an important source of food production. For example, farmers with fewer than 11 animals produce 75% of the milk in Pakistan (FAO, 2008). Similarly, 70% of the milk consumed in Kenya comes from farms that own 2–5 cattle and produce about 5 kg of milk per day (Muriuki, 2011).

action. Their multiple models, each illustrating a specific disease and treatment method, result in a specific and unambiguous relationship between private and social equilibrium. Our model, however, considers multiple equilibria based on the relative sizes of free-riding and resistance externalities, with a focus on the specific effects of disregarding resistance dynamics, especially in a smallholder, developing-world setting.

Our modeling approach diverges from most of the existing literature on antimicrobial resistance dynamics, which typically utilizes an epidemiological approach to modeling (e.g., susceptible–infected–susceptible (SIS) or susceptible–infected–recovered (SIR) models). Laxminarayan and Brown (2001) provide a standard model in the context of antimicrobial resistance, constructing a dynamic SIS model from a social planner's perspective to account for antimicrobial resistance with two drugs of differing effectiveness. The economic contribution of their paper relies on the inclusion of constant benefit of successful treatment, measured in dollars per person, scaled by fraction of infected who are treated and the effectiveness of treatment. Herrmann and Gaudet (2009) extend the epidemiological SIS framework to examine antibiotic effectiveness from the perspective of producers under open access. Similar to our model, they find that under open access and depending on the model parameters, antibiotic efficacy could be higher or lower than the social optimum. Unlike our model, they partition the population into susceptible and infected groups, each with their own subgroups of those exposed to resistant and nonresistant disease strains. Their model excludes any free-rider problem in the consumer decision-making process.

Among papers employing an alternative economic approach to modeling resistance dynamics, a few stand out but remain different from this paper. Horowitz and Moehring (2004) model human antibiotic use wherein a social planner maximizes social benefits—the price of antibiotics plus a constant marginal external benefit of reducing public infection—subject to the social cost of increasing antibiotic resistance. Herrmann and Gaudet's (2009) intuitive approach is similar to that of Horowitz and Moehring, but the former returns to an underlying SIS framework. In addressing their objective to decrease antibiotic resistance through reductions in open access (via patent extension or monopsony), Horowitz and Moehring ignore the private household decision-making process and the free-rider problem, instead dealing exclusively with the social planner's aggregated welfare problem. Elbasha (2003) and Brown and Layton (1996) construct economic models of antibiotic resistance aggregated from the private household decision. Following the basic structure of Phelps (1989), Elbasha employs a static model to estimate the deadweight loss associated with antibiotic overuse.

One central feature separates our paper from the models outlined above: We deal with prophylactic rather than therapeutic antimicrobials. An existing bacterial infection requires active treatment, hence the free-rider problem is absent in most of the above literature. Introducing an *ex ante* household decision on antimicrobial use creates the additional issue of providing a public good, which is key to this paper. Among free-rider models of disease prevention, many papers use the context of livestock vaccination, which by its nature does not lend itself to a discussion of resistance dynamics. Another important distinction of our model is the inclusion of a natural, external growth rate of disease prevalence. Brown and Layton's (1996) model of resistance dynamics, though fairly similar in structure, ignores this element, opting instead for a single dynamic constraint that assumes changes in bacteria's susceptibility to antibiotics as a function of antibiotic use.

Regarding the focus on smallholder households, this was a deliberate decision serving multiple purposes. First, the free-rider problem is more prominent where livestock ownership is more widely distributed—a situation more representative of (though not exclusive to) developing nations. Farm size could play a major role in individual households' decisions about antimicrobial use, but this paper develops only the framework that later can be modified to incorporate such additional complexity. Second, these smallholders in the developing nations play a critical role in food production and security and are an understudied population from the perspective of antimicrobial use and resistance (Redding et al., 2014; Caudell et al., 2017).

Model

We consider the privately and socially optimal use of prophylactic antimicrobials for livestock under dynamic societal disease prevalence. First, we examine the difference between the equilibrium of antimicrobial use between decision makers who do or do not account for the free-riding externality when antimicrobial resistance is not included in the model. We then incorporate antimicrobial resistance by adding a variable and dynamic constraint to the model. Our model illustrates that individual decision makers will underprovide antimicrobials relative to the socially optimal level when ignoring antimicrobial resistance dynamics. When the decision maker includes antimicrobial resistance in decision making, the optimal amount of antimicrobial use could be higher or lower than antimicrobial use when this externality was not considered, depending on the relative sizes of free-riding and resistance externalities.

A dynamic model is preferred to mirror the ever-changing nature of disease ecology and antimicrobial effectiveness (Herrmann and Gaudet, 2009; Viboud, Simonsen, and Chowell, 2016). Further, a dynamic model allows us to explore the changes in steady states under different levels of antimicrobial effectiveness. Additionally, phase diagrams help in discussing policy implications of the likely tipping points in the disease and antimicrobial effectiveness relationship.

Model without Antimicrobial Resistance

The decision maker maximizes the expected value of their welfare over an infinite time horizon. For livestock owners, welfare is determined largely by the health of their livestock. Suppressing other sources of uncertainty (e.g., prices, capacity constraints, etc.) and assuming homogeneity in terms of herd size/livestock holdings, the expected value of a household's welfare (W) takes the following form:

$$(1) \qquad W = \int_{t=0}^{\infty} [v(a(t), p(t)) - ca(t)] e^{-rt} dt,$$

where $v(\cdot)$ is the value derived from keeping livestock, $a(t)$ is the household's antimicrobial use at time t, $p(t)$ is the regional disease prevalence, r is the discount rate, and c is the cost of antimicrobials, which is treated here as constant. Indeed, the choice of disease management technologies is dependent on the background disease risk (Marsh et al., 2016; Ahmed et al., 2018; Railey et al., 2018), which is captured by $p(t)$. We assume the following intuitive relationships between the value of keeping livestock and its arguments:

$$(2) \qquad v_a > 0, \; v_p < 0, \; v_{aa} < 0, \; \text{and } v_{ap} = v_{pa} > 0.$$

In other words, the value of keeping livestock increases with antimicrobial use, but the magnitude of those marginal gains decreases as antimicrobial use increases (satisfying the concavity assumption for maxima to exist). Intuitively, the value of owning livestock decreases as the level of regional disease prevalence increases (i.e., $v_p < 0$). Further, we assume symmetry in cross-partial derivatives and that the marginal value of antimicrobial use increases as societal disease prevalence increases ($v_{ap} = v_{pa} > 0$). Societal disease prevalence changes according to the following disease dynamics constraint:

$$(3) \qquad \dot{p} = \theta(a(t)) \delta(p(t)),$$

which is impacted by $a(t)$, but the individual decision maker does not account for how his/her disease control efforts affect disease ecology. The social planner, however, does take this into account. In other words, the private decision maker and the social planner optimize the same welfare

function but different constraints in our model.[2] $\theta(\cdot)$ represents the way antimicrobial use induces a change in infection burden without accounting for antimicrobial resistance. We assume that $\theta_a < 0$ and $\theta_{aa} > 0$, which represents the notion that antimicrobial use reduces disease prevalence at a decreasing rate, assuming antimicrobials remain effective. The natural growth of disease prevalence is captured in $\delta(\cdot)$, and this function should follow some sort of an exponential path such that $\delta_p > 0$ and $\delta_{pp} > 0$.[3] A multiplicatively separable form of the disease dynamics constraint is used since it is highly tractable and emphasizes the different effects antimicrobial use and current levels of prevalence can have on changes in the latter. Last, we assume the initial level of disease prevalence, p_0, is finite and known.

The private decision maker will solve the present-value maximization problem above for his private level of antimicrobial use, $a(t)$. That optimal level of antimicrobial use is characterized by the maximum principle derived in Appendix A, the first-order condition of which can be rearranged to show that the marginal value of an additional unit of private antimicrobial use is equal to its cost ($v_a = c$). The social planner will maximize a similar equation to get optimal antimicrobial use, but they will internalize the reduction in disease prevalence stemming from individual antimicrobial use. The optimum is characterized by the second maximum principle derived in Appendix A, the first-order condition of which indicates that the societal value of an additional unit of antimicrobial use is equal to its cost plus an additional element that depends on disease prevalence and the effectiveness of antimicrobials ($v_a = c + \lambda \theta_a \delta$). Comparing these two, the model demonstrates a basic free-rider result that optimal private antimicrobial use is below the socially optimal level. This result stems from an incentive structure under which households are not fully able to internalize the broader welfare effects of antimicrobials. As expected, free ridership tends to underprovide antimicrobials relative to the social optimum when ignoring resistance dynamics.

Model with Antimicrobial Resistance

To incorporate antimicrobial resistance, we modify the objective function to include an additional resistance variable that directly affects the value of livestock:

$$(4) \qquad W = \int_{t=0}^{\infty} [v(a(t), p(t), I(t)) - ca(t)] e^{-rt} dt,$$

where $I(t)$ is antimicrobial ineffectiveness such that $v_I < 0$ and $v_{II} > 0$, and the other partial derivatives defined previously remain unchanged. Higher levels of antimicrobial resistance diminish the value of keeping livestock; however, the value decreases at a decreasing rate. We also assume the symmetry of cross-partials holds and the intuitive relationship, $v_{aI} = v_{Ia} < 0$, which indicates that as the marginal value of antimicrobials decreases resistance increases.

Accounting for antimicrobial resistance in terms of its effects on disease prevalence, the disease dynamics constraint becomes

$$(5) \qquad \dot{p} = \theta(a(t), I(t)) \delta(p(t)).$$

Antimicrobial resistance affects the growth rate of disease prevalence both directly and indirectly through its interaction with antimicrobial use. This modification of the disease dynamics constraint illustrates the more ambiguous effect of the $\theta(\cdot)$ function on changes in disease prevalence. An increase in antimicrobial use decreases the growth rate of disease prevalence, while antimicrobial resistance has an opposite effect ($\theta_a < 0$, $\theta_I > 0$). Further, we assume that $\theta_{aa} > 0$ and $\theta_{II} > 0$ (i.e.,

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[2] It is worth noting that the social planner does not account for resistance externalities in the first model. These externalities are introduced later.

[3] Disease prevalence is generally modeled as some sort of exponential growth function (see, e.g., Anderson, May, and Anderson, 1992; Viboud, Simonsen, and Chowell, 2016).
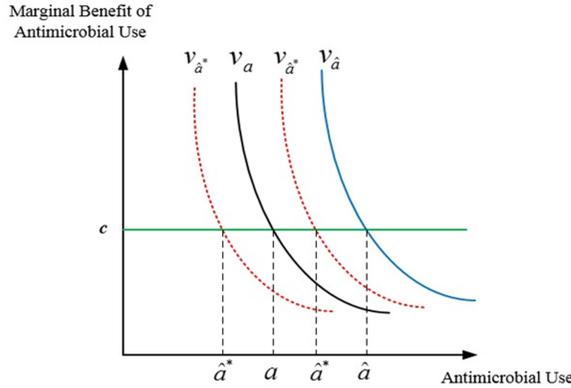
Figure 1. Illustration of the Marginal Benefit of Antimicrobial Use as a Public Good, Accounting for Antimicrobial Resistance

an increase in antimicrobial use hampers the growth of disease at a decreasing rate and an increase in antimicrobial ineffectiveness increases the growth of disease prevalence at an increasing rate).[4] To account for an interaction between antimicrobial use and antimicrobial resistance, we make the additional assumption that $\theta_{aI} = \theta_{Ia} < 0$ (i.e., the marginal ability of antimicrobials to hamper the growth of disease prevalence goes down as antimicrobial resistance goes up; Herrmann and Gaudet, 2009; Bloom and Cadarette, 2019).

    The evolution of antimicrobial resistance is characterized by the resistance dynamics constraint,

$$(6) \qquad\qquad\qquad\qquad \dot{I} = \xi\left(a\left(t\right)\right),$$

where $\xi\left(\cdot\right)$ is the rate at which antimicrobials become ineffective, and we assume this is increasing in antimicrobial use, such that $\xi_a > 0$ and $\xi_{aa} > 0$ (Van Boeckel et al., 2015, 2019). The effectiveness of antimicrobials is nonrenewable (Laxminarayan and Brown, 2001; Elbasha, 2003; Wilen and Msangi, 2003), so the level of ineffectiveness is finite, $I = [0, \bar{I}]$. The initial levels of disease prevalence and antimicrobial ineffectiveness, p_0 and I_0, are assumed to be finite and known.

    The maximum principles (derived in Appendix B) for each case define the respective optima and antimicrobial use. Comparing the first-order conditions, we see that the privately optimal antimicrobial decision ($v_a = c$) again differs from the social planner ($v_a = c + \lambda \theta_a p + \mu \xi_a$). To clarify our findings, we define the optimal levels of antimicrobial use derived in each model as follows:

- $a\left(t\right)$ represents the privately optimal level of antimicrobial use in the absence of antimicrobial resistance;

- $\hat{a}\left(t\right)$ represents the socially optimal level of antimicrobial use in the absence of antimicrobial resistance;

- $a^*\left(t\right)$ represents the privately optimal level of antimicrobial use when including antimicrobial resistance dynamics; and

- $\hat{a}^*\left(t\right)$ represents the socially optimal level of antimicrobial use when including antimicrobial resistance dynamics.

We treat $a\left(t\right) \equiv a^*\left(t\right)$ as identical terms since the private decision maker does not internalize externalities in either case.

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[4] Indeed, if the disease grows exponentially and antimicrobials become increasingly ineffective, then it is hard to change the path of disease growth (Bloom and Cadarette, 2019).

From these optimal levels derived in the models, we make three conjectures about the private and socially optimal levels of antimicrobial use under free ridership and antimicrobial resistance.

PROPOSITION 1. $a(t) < \hat{a}(t)$.

Private decision makers underprovide antimicrobials to their livestock compared with the social optimum in the absence of antimicrobial resistance. This is a basic result of free ridership. Proposition 1 follows from the comparison of first-order conditions of private and social-planner problems, where substituting out costs yields

$$(7) \qquad\qquad v_a = v_{\hat{a}} - \lambda\,\theta_{\hat{a}}\delta$$

which implies that $v_a > v_{\hat{a}}$, since $\theta_{\hat{a}} < 0$. Based on the concavity of the value function $v(\cdot)$, this implies that $\hat{a}(t) > a(t)$ (see Appendix A for more details).

PROPOSITION 2. $\hat{a}^*(t) \le \hat{a}(t)$.

Proposition 2 follows from a similar comparison of the two social planner's problems, where substituting out costs yields

$$(8) \qquad\qquad v_{\hat{a}} - \lambda\,\theta_{\hat{a}}\delta = v_{\hat{a}^*} - \lambda\,\theta_{\hat{a}^*}\delta - \mu\,\xi_{\hat{a}^*}$$

Given that $\mu\,\xi_{\hat{a}^*} \ge 0$, we obtain $v_{\hat{a}} - \lambda\,\theta_{\hat{a}}\delta \le v_{\hat{a}^*} - \lambda\,\theta_{\hat{a}^*}\delta$. Given the assumptions on $v(\cdot)$ and $\theta(\cdot)$ outlined in the model, this inequality implies that $v_{\hat{a}} \le v_{\hat{a}^*}$ and $\lambda\,\theta_{\hat{a}}\delta \le \lambda\,\theta_{\hat{a}^*}\delta$, which in turn implies that $\hat{a} \ge \hat{a}^*$. That is, the socially optimal level of antimicrobial use when excluding antimicrobial resistance from the model is at least as high as (and possibly higher than) the socially optimal amount of antimicrobial use when including resistance dynamics.

PROPOSITION 3. *Based on specific cases, one of the following is true:*

1. *$a \le \hat{a}^* \le \hat{a}$; or*

2. *$\hat{a}^* < a < \hat{a}$.*

One may observe that Proposition 3 follows directly from Appendix B and Propositions 1 and 2. Following is a brief examination of each of the two cases in Proposition 3.
    Case 1: This is the case when the relative size of the free-riding externality is greater than the resistance externality; therefore, private antimicrobial use is less than or equal to the socially optimal level with either externality.
    Case 2: This is the case when the relative size of the free-riding externality is smaller than the resistance externality; therefore, socially optimal antimicrobial use with both externalities is less than private use, which is less than the socially optimal level with only the free-riding externality.
    Broadly, Proposition 3 indicates that when antimicrobial resistance dynamics are incorporated into the model, the socially optimal amount of antimicrobial use is (i) never greater than that in the no-resistance model, (ii) may be lower than the privately optimal amount in the no-resistance model, and (iii) may still be underprovided at the private level in the resistance model.
    Figure 1 presents general graphical illustrations of the three propositions. The marginal private and social benefits of antimicrobial use without accounting for antimicrobial resistance dynamics are given by v_a and $v_{\hat{a}}$, respectively. The marginal social benefit accounting for resistance dynamics, $v_{\hat{a}^*}$, is uncertain. It will be less than the marginal social benefit without resistance dynamics, but it is unclear where it will fall in relation to marginal private benefits without resistance dynamics.
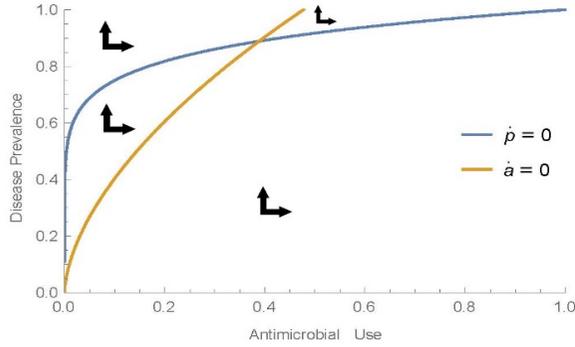
Figure 2. Phase Diagram for Initial Model with Free Riding But No Antimicrobial Resistance

Simulation

Given the model assumptions for the social planner problems, we construct phase diagrams in (a, p) space examining the steady-state levels of antimicrobial use and (regional) disease prevalence in both the no-resistance and the resistance cases. First, we must adopt some explicit functional forms and add parameters that satisfy the assumptions of the model. This exercise allows us to compare steady-state levels of antibiotic use and disease prevalence under the two alternative model specifications.

Parameterized Illustration

The slope and curvature of the $\dot{p} = 0$ and $\dot{a} = 0$ isoclines determine the steady-state equilibrium and transition dynamics of regional disease prevalence and antimicrobial use (Figure 2). An upward sloping $\dot{p} = 0$ isocline indicates that when disease prevalence is low (high), a small (large) amount of antimicrobials will keep the change in disease prevalence steady at 0. Conversely, the $\dot{a} = 0$ isocline slopes upwards, indicating that to maintain a steady state of no change in antimicrobial use over time, higher levels of antimicrobial use correspond to higher levels of disease prevalence.

Taking the present-value Hamiltonian for the dynamic problem without antimicrobial resistance, we use the maximum principle to solve for the steady-state equilibrium. We begin by isolating the $\dot{a}$ equation:

$$(9) \qquad \dot{a} = \frac{-\theta_a \delta v_p + 2(v_a - c)\theta \delta_p - v_{ap}\theta}{v_{aa} - (v_a - c)\frac{\theta_{aa}}{\theta_a}}.$$

Equation (3) provides us the $\dot{p}$ equation. Next, we select functional forms that satisfy our prior assumptions to demonstrate the transition dynamics as given in Table 1. The value function, $v(a, p)$, and associated parameters are chosen such that the optimality conditions are satisfied (i.e., $v_a > 0$, $v_p < 0$, $v_{aa} < 0$ and $v_{ap} > 0$). Therefore, the chosen functional form is $v(a, p) = a^{\kappa_1} p^{\kappa_2} - p^{\kappa_3}$, with $\kappa_1 = 0.5$, $\kappa_2 = 1$ and $\kappa_3 = 2.$[5] Similarly, $\theta(a) = a^{\kappa_5}$, with $\kappa_5 = -0.25$, is chosen such that it satisfied the conditions set in the model (i.e., $\theta_a < 0$ and $\theta_{aa} > 0$), representing the notion that antimicrobial use reduces disease prevalence at a decreasing rate. Additionally, $\delta(p) = p^{\kappa_7}$, with $\kappa_7 = 2$, such that $\delta_p > 0$ and $\delta_{pp} > 0$, representing the exponential path of growth of disease prevalence (Anderson, May, and Anderson, 1992; Viboud, Simonsen, and Chowell, 2016). A multiplicatively separable form of the disease dynamics constraint is used since it is highly tractable and emphasizes the different effects that antimicrobial use and current levels of prevalence can have on changes in the latter.

Using the functional forms in Table 1 and parameter values discussed previously, the initial model (without resistance dynamics) can be solved numerically for given levels of a and p. Figure 2

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[5] A brief examination of the model's sensitivity to small changes in the parameters follows.

Table 1. Functional Form Selections for Parameterized Illustrations

Function	No Resistance Dynamics	Resistance Dynamics
Value of keeping livestock	$v(a,p) = a^{\kappa_1} p^{\kappa_2} - p^{\kappa_3}$	$v(a,p,I) = a^{\kappa_1} p^{\kappa_2} I^{\kappa_4} - p^{\kappa_3}$
Antimicrobial effects on disease prevalence	$\theta(a) = a^{\kappa_5}$	$\theta(a,I) = a^{\kappa_5} I^{\kappa_6}$
Independent rate of change in disease prevalence	$\delta(p) = p^{\kappa_7}$	$\delta(p) = p^{\kappa_7}$
Antimicrobial effects on antimicrobial resistance		$\xi(a) = a^{\kappa_8}$

plots the isoclines for $\dot{a} = 0$ and $\dot{p} = 0$. As long as the parameter values satisfy the assumptions of the model, the qualitative results should be quite similar regardless of the parameters' magnitudes.

If antimicrobial use and prevalence are both initially below their steady-state levels, it is possible to converge on the steady-state equilibrium. However, if either exceeds its steady-state level, the system will diverge from the steady state and both antimicrobial use and prevalence will increase *ad infinitum*. The rationale behind this result is simple: If disease prevalence is too high at the beginning, then no amount of antimicrobial use will allow the level of prevalence to stop changing over time. Conversely, when antimicrobial use is too high, there is no way to reduce it without increasing the level of disease prevalence.

Next, we add the antimicrobial resistance variable to the model along with its dynamic constraint and derive the associated $\dot{a}$ as

$$(10) \qquad \dot{a} = \frac{-v_{ap}\theta\delta - v_{aI}\xi + [v_p - \lambda\delta_p\theta]\theta_a\delta + \lambda\theta_{aI}\xi\delta + \lambda\theta_a\delta_p\theta\delta + [v_I - \lambda\theta_I\delta]\xi_a}{v_{aa} - \mu\xi_{aa} - \lambda\theta_{aa}\delta}.$$

Equations (5) and (6) provide us with $\dot{p}$ and $\dot{I}$ equations, respectively. In this case, there are three variables—a, p, and I—so drawing a proper phase diagram requires a three-dimensional plot of isoplanes. However, since we are only interested in differences from the initial steady state, we have opted to fix antimicrobial resistance at different levels to illustrate changes in the same (a,p) space as that used in the initial diagram. When antimicrobial resistance is fixed at different levels, we find that the steady-state levels of antimicrobials and disease prevalence begin to shift.

Given that antimicrobial resistance is added in the model, we introduce some changes to the functional forms. The value function in column 2 of Table 1, $v(a,p,I) = a^{\kappa_1} p^{\kappa_2} I^{\kappa_4} - p^{\kappa_3}$, is chosen such that it follows the optimality conditions laid out above (i.e., $v_a > 0$, $v_p < 0$, $v_{aa} < 0$ and $v_{ap} > 0$). Additionally, the value function must also satisfy $v_I < 0$ and $v_{II} > 0$. That is, increases in levels of antimicrobial resistance diminish the values from livestock keeping, and the value decreases at a decreasing rate. Further, $v_{aI} < 0$, which indicates that the marginal value of antimicrobials decreases as resistance increases. To satisfy these assumptions, the value of κ_1, κ_2, and κ_3 remain the same as above, and the value of κ_4 is set at -0.5.

Similarly, $\theta(a,I) = a^{\kappa_5} I^{\kappa_6}$ is chosen such that the assumptions $\theta_a < 0$ and $\theta_{aa} > 0$ are satisfied. Additionally, to account for an interaction between antimicrobial use and antimicrobial resistance, we make the additional assumption that $\theta_{aI} < 0$ (i.e., the marginal ability of antimicrobials to hamper the growth of disease prevalence goes down as antimicrobial resistance goes up). We choose $\kappa_6 = 2$ so that all the above-mentioned assumptions are satisfied; as before, $\kappa_5 = -0.25$ and $\delta(p)$ remains the same. Effects of antimicrobial use on antimicrobial resistance are modeled as $\xi(a) = a^{\kappa_8}$, with $\kappa_8 = 1.5$, such that $\xi_a > 0$ and $\xi_{aa} > 0$, indicating that the ineffectiveness of antimicrobials is increasing in antimicrobial use at an increasing rate. This ensures that the new functions and constraints in the resistance model satisfy the stated assumptions.

Figure 3 illustrates that as the level of antimicrobial ineffectiveness increases (at fixed levels, as shown across the three panels), the corresponding steady-state levels of antimicrobial use and
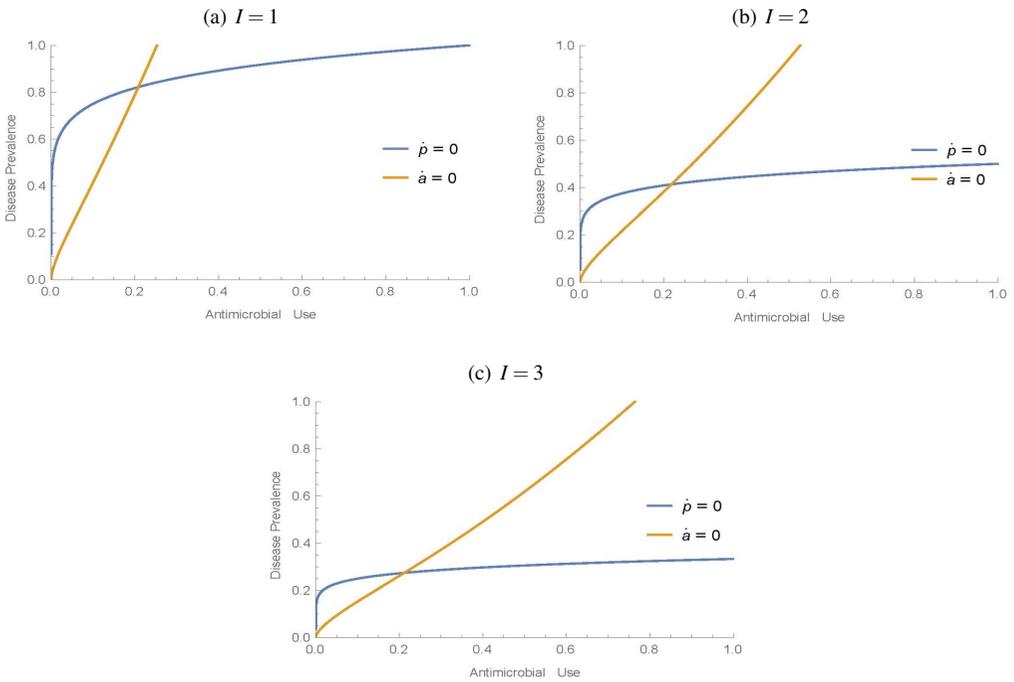
(a) $I = 1$

(b) $I = 2$

(c) $I = 3$

Figure 3. Phase Diagrams for Model with Free Riding and Resistance Externalities

disease prevalence decline. Figure 3 generates important economic insights. First, the higher the antimicrobial ineffectiveness, the lower the disease prevalence level that will be sustainable in steady state. In other words, it becomes increasingly difficult to curtail or manage a sustained level of disease as resistance increases. This suggests that disease outbreak, spread, and severity could be high under a scenario with high resistance.

Second, with higher levels of resistance, the $\dot{a} = 0$ isocline becomes more horizontal and lower. This indicates that the rate of change in antimicrobial use in equilibrium must be much higher to maintain a low level of disease prevalence in the presence of higher antimicrobial resistance. This change in slope of the $\dot{a}$ isocline again represents increasing costs of disease management and therapy in the face of increasing resistance. More importantly, the equilibrium level of antimicrobial use that is sustainable under the antimicrobial resistance framework is lower than the level sustainable in the free-rider model. This parameterized illustration further demonstrates the theoretical result in Proposition 2.

Sensitivity Analysis

Regarding the model's sensitivity to changes in the parameters, we conduct a rudimentary analysis to determine how the isoclines and the steady state shift with changes in individual parameter values. Due to the localized nature of our equilibria—a necessary consequence of the intuitive assumptions of our model—we find a sensitivity analysis of the parameterized model to be the most appropriate method to determine the model's robustness to minor changes. We examine parameter changes ranging from 10% to 50% in either direction, subject to the functional form assumptions of the model.

The initial model (without antimicrobial resistance) is not highly sensitive to changes in the antimicrobial term's exponent (κ_1) in the value function. Shifts of 50% around the initial parameter value leaves the isocline $\dot{p} = 0$ wholly unchanged. The isocline $\dot{a} = 0$ is altered only slightly, shifting the steady state along the isocline $\dot{p} = 0$ either to the right when κ_1 increases—indicating a higher
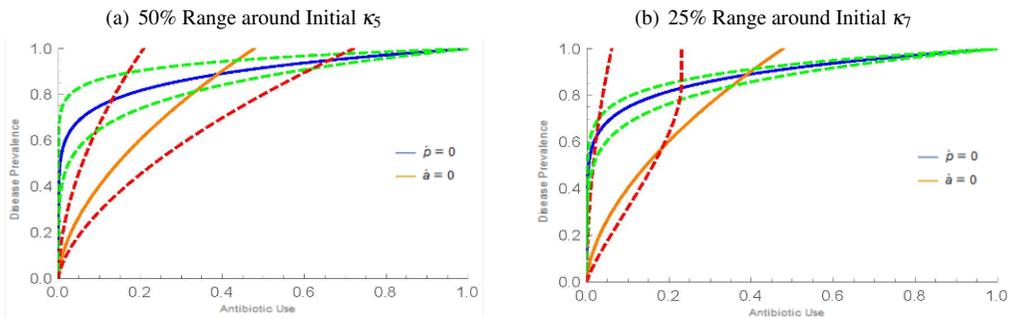
Figure 4. Phase Diagrams for Initial Model with Free Riding but No Antimicrobial Resistance with Parameter Value Ranges

steady-state level of antimicrobial use corresponding with a relatively small increase in disease prevalence—or to the left when κ_1 decreases—indicating a lower steady-state level of antimicrobial use corresponding with a relatively small decrease in disease prevalence.

Large increases in the κ_2 parameter—the exponent on the multiplicative prevalence component of the value function—leaves the isocline $\dot{p} = 0$ unchanged, stretches the isocline $\dot{a} = 0$ vertically, and reduces the steady-state level of antimicrobial use. Complete removal of that multiplicative prevalence term (setting $\kappa_2 = 0$) creates multiple stable equilibria, which are, while interesting from a graphical standpoint, too unrealistic in practice to warrant further elaboration. The exponent on the additive prevalence component of the value function (κ_3) likewise leaves the isocline $\dot{p} = 0$ unaltered, and it shifts the isocline $\dot{a} = 0$ slightly to inversely raise or lower the steady-state level of antimicrobial use.

Moving beyond the value function, we consider parameters in the dynamic constraints. While a 10% increase in the value of κ_5 (the exponent describing antimicrobials' effect on disease prevalence) has little effect on either isocline or the steady state, even the slightest decrease stretches the isocline $\dot{a} = 0$ vertically and reduces the steady-state level of antimicrobial use. Figure 3(a) illustrates a 50% change in this parameter value in either direction (by the dashed lines around the isoclines). It is representative of how the isoclines tend to shift under parameter changes; however, shifts in this parameter have more pronounced effects than shifts in most others. The isocline $\dot{p} = 0$ is not highly sensitive to small changes in the exponent describing the independent growth of disease prevalence (κ_7); however, the isocline $\dot{a} = 0$ becomes steeper, with a 10% shift in the parameter value (either direction), causing the steady-state level of disease prevalence and antimicrobial use to be lower. Figure 3(b) illustrates a 25% range around the initial κ_7 value, representing the reduction in steady-state levels of disease prevalence and antimicrobial use stemming from any upward or downward movement in this parameter value. The qualitative results remain consistent under a 25% shift in this parameter value.

We conduct a similar exercise for our resistance model. Rather than provide a pedantic explanation of each parameter, we focus on those that are specific to the resistance model. A shift of up to 50% in either direction in κ_4, the exponent on the ineffectiveness term in the value function, shifts the phase diagram only minimally. On the other hand, a shift in the exponent on the resistance dynamics constraint, κ_8, has a more pronounced effect on the phase diagram. Though the qualitative results and interpretation remain unchanged, we illustrate the effects in Figure 5. We can see that the parameter change affects the steady-state equilibrium solely through the isocline $\dot{a} = 0$.

On the whole, the resistance model seems less sensitive to changes in the parameter values than the model examining only free riding, though that sensitivity often increases as we modify the resistance terms to higher static values (i.e., as I increases in the phase diagram). As long as parameter values satisfy the functional form assumptions of the model, we find that parameter shifts of at least 50% do not affect the qualitative results and interpretations of the model. Upon further
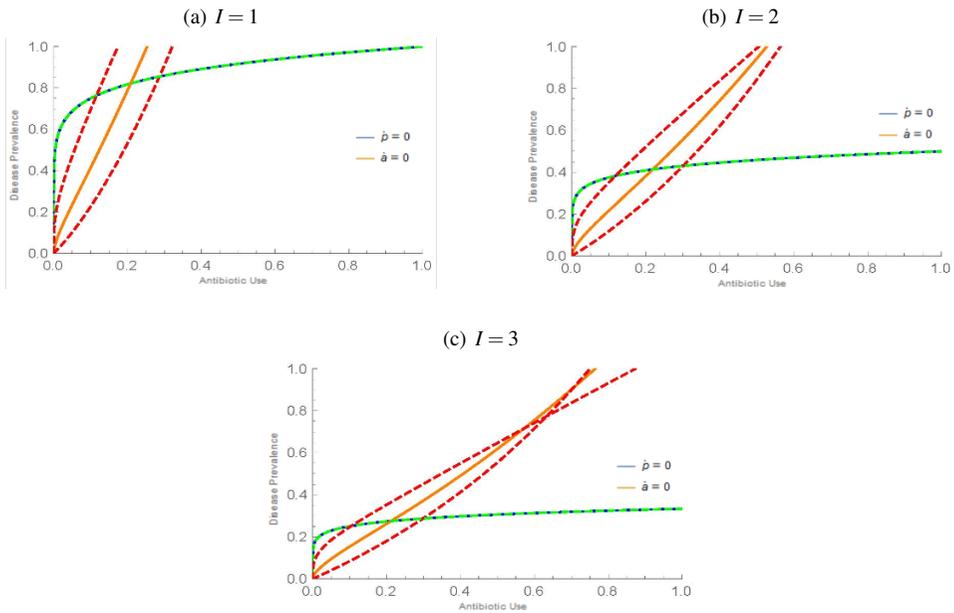
Figure 5. Phase Diagrams for Model with Free Riding and Resistance Externalities with κ_8 Ranging 50% Above and Below Initial Value

examination, we find that some parameter values in violation of the assumptions of the model lead to discontinuities, multiple equilibria, and other model inconsistencies.

Policy Discussion

In our model, the optimal use of antimicrobials is determined by the relative size of the free-riding and resistance externalities. Here, we discuss the implications of these results in light of subsidy and voucher programs like the Tanzanian Acaricide Subsidy Program[6] and Turkey's Animal Health Subsidy Program.[7] These programs are structured to incentivize farmers to protect their animals from disease risk, to promote growth and limit food insecurity, and to overcome free-riding behaviors. We acknowledge that such programs may have been developed to enhance food security by limiting livestock disease and increasing production, which may take precedence over judicious use of antimicrobials, especially in low- and lower-middle income countries. However, evidence suggests that ceasing growth promoting antimicrobials in animals may have negligible impacts on production (Laxminarayan, Van Boeckel, and Teillant, 2015; Van Boeckel et al., 2015). Therefore, such programs may allow for overuse of antimicrobials without many economic or food security gains. Further, these policies could be detrimental to farmers' ability to control disease losses in the future through resistance development, leading to multiple crises in terms of animal health, human health, and food insecurity. Our model emphasizes the need to recalibrate these policies by incorporating antimicrobial resistance externalities in decision calculus.[8]

   If $a > \hat{a}^*$ (i.e., private antimicrobial use is greater than the socially optimal use with free-riding and resistance externalities under disease dynamics) arises, then free riding may not be such a

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[6] http://www.snv.org/public/cms/sites/explore/download/brief_1_public_accountability_in_tanzania_pata_initiative.pdf

[7] https://gain.fas.usda.gov/Recent GAIN Publications/Turkish Livestock Support and Subsidies_Ankara_Turkey_8-12-2015.pdf

[8] This model also can motivate the problem of pest resistance in target species. However, policies to mitigate resistance in bacteria and pests may differ.

detrimental behavior, and it may in fact lead the economy closer to the socially optimal level of antimicrobial use. We cite the two subsidy programs above specifically to motivate the presence of such direct interventions; however, there are other policies that may be regarded as indirect subsidies and can incentivize overuse much like direct subsidies.

Over-the-counter availability of antimicrobials without prescription can be regarded as a subsidy on transaction costs and is a major issue in developing countries (Caudell et al., 2017). For example, China's pig production sector uses roughly 10 times more antibiotics than the United States and yet the regulatory structure for antibiotics is not implemented effectively in all regions of China (Elliott, 2015; Larson, 2015). Similarly, antimicrobial use and resistance is rapidly on the rise in important emerging economies like India, Pakistan, and Mexico (Van Boeckel et al., 2015, 2019), yet no regulatory or antimicrobial monitoring programs are in place for many of these countries. In other important livestock producing countries like Brazil, action plans are only in their infancy and not fully implemented (Cardoso, 2019). On the other hand, countries like Sweden have been successful in dealing with the resistance externality through effective regulation of antimicrobials and by consistently reducing their use in food animals without compromising growth or productivity (Elliott, 2015).[9] Lessons from these success stories could be applied to the livestock sectors of emerging countries to deal with the burden of antimicrobial resistance while simultaneously ensuring food security in low- and lower-middle income countries.

Over-the-counter availability and self-prescription of antimicrobials also leads to consumption of more broad-spectrum antibiotics (Caudell et al., 2017; Ahmed et al., 2018) and may pose the threat of more strains of bacteria becoming resistant more rapidly. These policies and the development of resistant bacteria in the emerging economies may affect global food security and health through trade and travel in an interconnected world (Frost et al., 2019). Therefore, it is important that these resistance externalities are addressed locally as well as globally (Kirchhelle et al., 2020). Indeed, considering the global dimension of antimicrobial resistance, multiple international organizations like the World Health Organization (WHO), the World Organisation for Animal Health (OIE), and the Food and Agriculture Organization of the United Nations (FAO), among others, have made this topic a priority (Rushton, Ferreira, and Stärk, 2014).

The case $a > \hat{a}^*$ (i.e., private antimicrobial use being greater than the socially optimal use with free-riding and resistance externalities) also mirrors the case in most developed countries with intensive production systems (like the United States) and little incentive to free ride (Levy, 1998; Orzech and Nichter, 2008). In developed countries, while regulatory structures and monitoring programs are in place, they are generally in their infancy, and antimicrobial consumption in food animals far exceeds their direct consumption by humans (Elliott, 2015).

Now we turn our attention to the other case, where the relative size of the free-riding externality is greater than the resistance externality (i.e., when $\hat{a}^* < a$). In such a case, incentives may be required to deal with underprovision of livestock health inputs. The free-riding externality is likely to be greater for farmers who experience credit constraints or reside in areas with limited access to veterinary services and low availability of antimicrobials (Marsh et al., 2016; Railey et al., 2018). Even in such a scenario, policy makers should account for the antimicrobial resistance externality; otherwise, they may end up overincentivizing the use of antimicrobials, which could be harmful in the long run. Further data collection is required to measure these externalities and prescribe incentives to increase or decrease antimicrobial use according to the relative sizes of these externalities.

Phase diagrams generated by our simulations also point toward important economic aspects of increasing resistance levels. Figure 3 illustrates how increases in antimicrobial resistance reduce

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[9] We acknowledge that the increase in antimicrobial use and resistance may also be related to intensification of production systems in the developing world as a response to increase in demand for animal-sourced proteins (among other factors). However, given that there is empirical evidence of increased resistance and emerging regional hotspots in low and lower-middle income countries (Van Boeckel et al., 2019), it is important that the issue of antimicrobial resistance is addressed through regulatory frameworks.

the steady state p^* but increase the steady state a^*, suggesting that disease prevalence becomes increasingly difficult to keep in check with an increasing burden of antimicrobial resistance, and more antibiotic use is required to keep a lower level of disease sustainable. This result is in line with the existing literature on antimicrobial resistance and has implications for lengths, severity, and costs of disease outbreaks and ultimately food security and global health (Rushton, Ferreira, and Stärk, 2014; Friedman, Temkin, and Carmeli, 2016; Bloom and Cadarette, 2019).

Concluding Remarks

The countervailing effects of free-riding and resistance externalities in antimicrobial use in a smallholder setting may result in suboptimal levels of disease control, which in turn may lead to high disease prevalence due either to a lack of livestock health inputs or to reductions in the inputs' effectiveness. This paper examines the optimal level of preventive antimicrobial use under free-riding incentives given antimicrobial effectiveness, which we have modeled as nonrenewable resource in a dynamic optimization framework. The first model is a standard model with free-riding incentives that elucidates the effects of free riding on antimicrobial use given disease dynamics. We then add antimicrobial resistance as a dynamic constraint and compare the two models, examining several cases conditional on the extent of both externalities. Policy implications were discussed in light of the animal health and disease control subsidy programs.

   This paper contributes to the literature of antimicrobial use in the smallholder setting by modeling the free-riding and resistance externalities simultaneously. If policy makers fail to account for both of these externalities and instead account only for free ridership, the resulting policies could aim to overprovide antimicrobials relative to the true socially optimal levels including resistance dynamics. If pathogens become resistant to antimicrobials, future costs of disease will rise in terms of disease management as shown via our phase diagrams but also likely through increased mortality and increased duration of illness. Further, increased antimicrobial resistance would undermine both public health and future food security. Therefore, the paper emphasizes the need to align the private benefits of antimicrobial use with the social benefits of these inputs, accounting fully for free riders and increasing levels of disease resistance associated with antimicrobial use.

<h1 style="text-align:center">References</h1>

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Appendix A: No Antimicrobial Resistance

The private decision maker's problem is

$$W = \int_{t=0}^{\infty} \left[v\left(a\left(t\right), p\left(t\right)\right) - ca\left(t\right) \right] e^{-rt} dt;$$

$$\text{s.t. } \dot{p} = \theta\left(a\left(t\right)\right) \delta\left(p\left(t\right)\right);$$

$$p\left(0\right) = p_0.$$

Private households maximize present value Hamiltonian:

$$H = v\left(a, p\right) - ca - \lambda\,\theta\left(a\right)\delta\left(p\right).$$

The maximum principle for private decision maker (not internalizing the constraint) gives the following:

$$H_a = v_a - c = 0 \qquad\qquad \text{(first-order condition)}$$

$$\dot{\lambda} = v_p - \lambda\,\theta\delta_p \qquad\qquad \text{(portfolio balance condition)}$$

$$\dot{p} = \theta\delta \qquad\qquad \text{(dynamic constraint)}$$

The social planner's problem in which the benevolent planner internalizes the reduction in disease persistence that comes from antimicrobial use gives use the following:

$$H_a = v_a - c - \lambda\,\theta_a\delta = 0 \qquad\qquad \text{(first-order condition)}$$

$$\dot{\lambda} = v_p - \lambda\,\theta\delta_p \qquad\qquad \text{(portfolio balance condition)}$$

$$\dot{p} = \theta\delta \qquad\qquad \text{(dynamic constraint)}$$

With the aggregate private antimicrobial use denoted as a and the socially optimal as $\hat{a}$, we can compare the private and social first-order conditions:

$$v_a - c = v_{\hat{a}} - c - \lambda\,\theta_{\hat{a}}\delta$$

$$v_a = v_{\hat{a}} - \lambda\,\theta_{\hat{a}}\delta$$

which implies that $v_a > v_{\hat{a}}$, since $\theta_{\hat{a}} < 0$. Based on the concavity of the value function $v\left(\cdot\right)$, this implies $\hat{a} > a$. In other words, private antimicrobial use will be lower than the socially optimal level, so the market is inefficient in providing antimicrobials.

Appendix B: Antimicrobial Resistance

We modify the problem now to include disease prevalence and a disease resistance dynamic constraint:

$$W = \int_{t=0}^{\infty} [v(a(t), p(t), I(t)) - ca(t)] e^{-rt} dt$$

$$s.t. \ \dot{p} = \theta(a(t), I(t)) \delta(p(t))$$

$$\dot{I} = \xi(a(t))$$

$$I(0) = I_0, \ p(0) = p_0 \text{ and } I \in [0, \bar{I}]$$

Private household with antimicrobial resistance

$$H = v(a, p, I) - ca - \lambda [\theta(a, I) \delta(p)] - \mu [\xi(a)]$$

The maximum principle gives the following:

$$H_a = v_a - c = 0 \qquad \text{(first-order condition)}$$

$$\dot{\lambda} = v_p - \lambda \theta(a, I) \delta_p \qquad \text{(portfolio balance condition 1)}$$

$$\dot{\mu} = v_I - \lambda \theta_I \delta \qquad \text{(portfolio balance condition 2)}$$

$$\dot{p} = \theta(a, I) \delta \qquad \text{(dynamic constraint 1)}$$

$$\dot{I} = \xi(a) \qquad \text{(dynamic constraint 2)}$$

The social planner internalizes the effects of antimicrobial use on disease prevalence and antimicrobial ineffectiveness. Solving the social planner's problem gives the following:

$$H_a = v_a - c - \lambda \theta_a p - \mu \xi_a = 0 \qquad \text{(first-order condition)}$$

$$\dot{\lambda} = v_p - \lambda \theta(a, I) \delta_p \qquad \text{(portfolio balance condition 1)}$$

$$\dot{\mu} = v_I - \lambda \theta_I p \qquad \text{(portfolio balance condition 2)}$$

$$\dot{p} = \theta(a, I) \delta \qquad \text{(dynamic constraint 1)}$$

$$\dot{I} = \xi(a) \qquad \text{(dynamic constraint 2)}$$

We cannot directly compare the two socially optimal choices of antimicrobials since there are multiple unknowns in each, but we can compare them indirectly using the private problem. Considering antimicrobial use in the resistance case as $\hat{a}^*$ and the private case as a^*, we have

$$v_{a^*} - c = v_{\hat{a}^*} - c - \lambda \theta_{\hat{a}^*} \delta - \mu \xi_{\hat{a}^*}$$

$$v_{a^*} = v_{\hat{a}^*} - \lambda \theta_{\hat{a}^*} \delta - \mu \xi_{\hat{a}^*}$$

Recalling that $\theta_{\hat{a}^*} < 0$ and $\xi_{\hat{a}^*} > 0$, we have the following cases:

1. $v_{a^*} \geq v_{\hat{a}^*}$ if $|\lambda \theta_{\hat{a}^*} \delta| \geq |\mu \xi_{\hat{a}^*}|$, which implies that $a^* \leq \hat{a}^*$; and

2. $v_{a^*} < v_{\hat{a}^*}$ if $|\lambda \theta_{\hat{a}^*} \delta| < |\mu \xi_{\hat{a}^*}|$, which implies that $a^* > \hat{a}^*$.

This allows for the construction of the conditional propositions (especially Proposition 3).