Managing Livestock Disease Transmission as a Nonpoint Source Biological Pollutant

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INTRODUCTION

Features of disease ecology models generally drive the specification and potential results of bioeconomic models of infectious livestock disease management. Three features have particularly important implications:

Uniform compartments. Traditional disease ecology models segment a population (e.g, of animals or herds) within a region into *compartments* (e.g., susceptible, *S*, infected, *I*, recovered, *R*), which are distinct state variables. The approach implies uniform transmission risks within a region, which requires homogenous benefits and costs of disease control and results in uniform policy tools being optimal. Adding spatial or economic heterogeneity requires adding a new set of states (e.g., *S*, *I*, *R*) for each new spatial or economic category, with the number of dynamic variables growing geometrically as categories are added.

Non-random transmission. Simple disease ecology models are deterministic, involving non-random transmission. This becomes a particular concern when behavioral responses to risk are important. For instance, deterministic models result in certain disease spread in each period, which may elicit a costly policy response. But for many diseases, disease spread is a rare occurrence that only happens sporadically. Some bioeconomic models do assume random transmission. This complicates attempts to add heterogeneities.

Observable states. Traditional disease ecology models assume current disease states are known in aggregate. Within this framework, bioeconomic models require assumptions about the observability of individual herds' health states, as this influences private and public choice sets, economic decisions, and outcomes. Assuming freely observable health states limits the role of uncertainty and treats individual producers as point sources of biological pollution (disease transmission), as infected producers could be targeted to manage externalities. A richer analysis arises by assuming unobservable (without cost) herd health states, but this also implies a paradox: how is it then that aggregate health states are freely known?

OBJECTIVE

We develop a bioeconomic framework in which economic considerations drive the construction of a realistic disease model. Particular features are:

Heterogeneity. We assume heterogeneous benefits and costs of disease control, which in turn drives non-uniform behaviors and disease risks.

Random transmission. Actions and states only affect the probability of transmission.

Partially observable, probabilistic states. States are unknown due to imperfect and costly testing. This complicates matters in some respects. However, it also allows us to reduce the number of states, making it easier to incorporate heterogeneity into the model. Note that unobservability means each direct or indirect animal contact is risky, with transmission externalities in this framework more akin to a nonpoint source pollutant.

We adopt a partially observable Markov decision process (POMDP) model, which has seen recent applications in bioeconomic settings (Springborn and Sanchirico 2013; MacLachlan et al. 2017) and which utilizes Bayesian state-space modeling that has recently been introduced to disease ecology (Hobbs et al. 2015).

MODEL

Modeling choices

- A region with *N* producers indexed by $n \in \{1,\ldots,N\}$
- Stochastic transmission & uncertainty about the status of detected herds
- Only state variables are $p_{n,t}$: the probability herd *n* is infected at *t*
- Many choices: inputs = $\mathbf{x}_{n,t}$, testing effort = $s_{n,t} \in [0,1]$

Bayesian dynamics

$$p_{n,t+1} = \overbrace{B_n(\tau_{n,t}|s_{n,t},p_{n,t})}^{\substack{\text{Bayesian update}\\ \text{of } pn_t \text{ after testing}}} \overbrace{\left(1-h_n(\mathbf{x}_{n,t})\right)}^{pr(\text{no recovery at } t)} + \left(1-B_n(\tau_{n,t}|s_{n,t},p_{n,t})\right)\times \underbrace{g_n(\mathbf{x}_t,\mathbf{p}_t)}_{pr(\text{infected by others})}$$

A stochastic difference equation where $\tau_{n,t}$ = random test result

$$B_n(\tau_{n,t}|s_{n,t},p_{n,t}) = \frac{\Pr_n(\tau_{n,t}|s_{n,t},i_{n,t}=1)p_{n,t}}{\Pr_n(\tau_{n,t}|s_{n,t},i_{n,t}=1)p_{n,t}+\Pr_n(\tau_{n,t}|s_{n,t},i_{n,t}=0)(1-p_{n,t})}$$

where $i_{n,t}$ = 1 if the herd tests positive for infection and zero otherwise, and

$$g_n(\mathbf{x}_t,\mathbf{p}_t) = g_n\big(g_{1,n}(\cdot)\,B_1(\cdot),\ldots,\underbrace{g_{j,n}(\mathbf{x}_{j,t},\mathbf{x}_{n,t})\,B_j(s_{j,t},p_{j,t})}_{\substack{\text{producer } j\text{'s contribution to producer } n\text{'s infection likelihood}}},\ldots,\,g_{N,n}(\cdot)\,B_N(\cdot)\big)$$

$g_{j,n}$ = a probabilistic biological externality imposed by producer *j* onto producer *n*

Note that externalities are filterable (e.g., Shogren and Crocker 1991): self-protection by producer *n* in period *t* reduces risks to others in future periods

Social planner's Bellman's equation:

$$V(\mathbf{p}_t) = \min_{\mathbf{x}_t,\mathbf{s}_t}\left\{\sum_n \big[\overbrace{C_n(\mathbf{x}_{n,t},p_{n,t})}^{\substack{\text{Expected control}\\ \text{and damage costs}}} + \overbrace{w_n(s_{n,t})}^{\substack{\text{Expected}\\ \text{testing costs}}}\big] + \beta E_\tau\{V(\mathbf{p}_{t+1})\}\right\}$$

Individual producer's Bellman's equation:

$$V_n(\mathbf{p}_t) = \min_{\mathbf{x}_{n,t},\mathbf{s}_{n,t}}\left\{C_n(\mathbf{x}_{n,t},p_{n,t}) + w_n(s_{n,t}) + \beta E_\tau\{V_n(\mathbf{p}_{t+1})\}\right\}$$

RESULTS

Necessary conditions

Social planner's first-order conditions (FOCs) for input $x_{nm,t}$:

$$\frac{\partial C_n(\mathbf{x}_{n,t},p_{n,t})}{\partial x_{nm,t}} = -\beta\sum_j \underbrace{E_\tau\left\{\frac{\partial V(\mathbf{p}_{t+1})}{\partial p_{j,t+1}}\right\}}_{+}\underbrace{E_\tau\left\{\frac{\partial p_{j,t+1}}{\partial x_{nm,t}}\right\}}_{+/-} - \beta\sum_j \underbrace{cov_\tau\left\{\frac{\partial V(\mathbf{p}_{t+1})}{\partial p_{j,t+1}},\frac{\partial p_{j,t+1}}{\partial x_{nm,t}}\right\}}_{+/-}$$

- RHS = producer-specific marginal expected benefits of the input
- First RHS term: mean marginal effect is positive (negative) for inputs that increase (reduce) recovery chances and reduce (increase) infection likelihoods
- Second RHS term: inputs that increase the variation of $p_{j,t+1}$ increase social risks and reduce expected net benefits of disease control
- Externalities arise because producers do not consider effect of input use on risks to others, and will not account for others' probability updates

Similar results arise for the FOC for testing, $s_{n,t}$, except that:

- The mean effect of testing is positive (a benefit), producing benefits
- An additional term arises: more testing is likely to increase positive test results (a cost; sometimes ignorance is bliss!)

Similar externalities arise in the adjoint conditions

Policy design implications

- Each producer is optimally regulated as a potential polluter
- Not efficient to tax the probabilistic externality if more than one choice
- Efficiency requires producer-specific and time-varying incentive rates applied to
  - Each input affecting transmission risks to oneself or others
  - Testing efforts
  - The producer's probability of transmission

Special case:

- A producer generates no externalities if s/he has no way of reducing risks to others, including no self-protection. In that case producer is only a victim.
- If risks only due to trade, with no other risk-filtering opportunities, then can target exporter only; otherwise importers are potential polluters

CONCLUSIONS

- We should not let features of conventional disease ecology models drive the construction of economic models
- Economic features such as heterogeneity and uncertainty can significantly affect producer decision-making and policy recommendations
- Like NPS pollution problems, separately targeting each input affecting risks is efficient – it is not enough to target the probability of infection (which is the closest thing to the externality in this model)
- Unlike NPS pollution problems, need to also target the probability of pollution (due to filterable nature of externalities)

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