

Original Contribution

Merging Economics and Epidemiology to Improve the Prediction and Management of Infectious Disease

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Abstract: Mathematical epidemiology, one of the oldest and richest areas in mathematical biology, has significantly enhanced our understanding of how pathogens emerge, evolve, and spread. Classical epidemiological models, the standard for predicting and managing the spread of infectious disease, assume that contacts between susceptible and infectious individuals depend on their relative frequency in the population. The behavioral factors that underpin contact rates are not generally addressed. There is, however, an emerging class of models that addresses the feedbacks between infectious disease dynamics and the behavioral decisions driving host contact. Referred to as “economic epidemiology” or “epidemiological economics,” the approach explores the determinants of decisions about the number and type of contacts made by individuals, using insights and methods from economics. We show how the approach has the potential both to improve predictions of the course of infectious disease, and to support development of novel approaches to infectious disease management.

Keywords: economic epidemiology, epidemiological economics, incentives, infectious disease

ECONOMIC EPIDEMIOLOGY AND EPIDEMIOLOGICAL ECONOMICS

Economic behavior is known to play a key role in disease transmission. Throughout history, new pathogens have emerged with the opening of new markets or trade routes. The Black Death in the fourteenth century, and the sixteenth century Columbian exchange—which brought smallpox and typhus to the Americas, and syphilis to Europe, are the best-known examples (McNeill 1977; Yoo et al. 2010). In the last few decades, the growth of global trade and travel have been implicated in the emergence of human infectious diseases such as plague, cholera, HIV (Tatem et al. 2006a, b), West Nile virus (Lanciotti et al. 2000), SARS (Guan et al. 2003; Hufnagel et al. 2004), as well as livestock diseases such as H9N2 Avian influenza, Bovine Spongiform Encephalopathy, Bluetongue or Foot and Mouth disease (Rweyemamu and Astudillo 2002; Karesh et al. 2005; Fevre et al. 2006; Purse et al. 2008), and diseases of wildlife—potentially white-nose syndrome in bats (Pikula et al. 2012). In the USA, many other wildlife diseases and zoonoses have been linked to live animal imports (Smith et al. 2009a). Trade and travel affect the likelihood that pathogens are spread internationally by altering the number and variety of infectious-susceptible contacts (Smith et al. 2007; Jones et al. 2008; Suhrcke et al. 2011; Daszak 2012; Kilpatrick and Randolph 2012). In the same way, the decisions people make to engage with others in their own community affect the spread of disease nationally. Since people take account of potential disease risks, it is possible to analyze the spread of disease as a function of the costs and benefits of disease risk management.

In recent years, work at the boundary between ecology, epidemiology, and economics has shed new light on the way that economic behavior affects the spread of pests and pathogens (reviewed in Perrings 2014). The approach, referred to either as economic epidemiology or as epidemiological economics (hereafter EE), initially focused on the relationship between preventive behavior and disease prevalence (Philipson 2000). More recently, it has focused on the economic causes and epidemiological consequences of the number and type of contacts people make (Gersovitz and Hammer 2003, 2004; Barrett and Hoel 2007; Funk et al. 2009; Funk et al. 2010; Springborn et al. 2010). That is, the economic factors behind contact and mixing decisions are treated as part of the disease transmission mechanism. The approach provides a deeper understanding of the dynamics of epidemics, and opens up a new set of disease manage-

ment options that target either the contact rate (Kremer 1996; Auld 2003) or the probability that contact leads to infection (Geoffard and Philipson 1996).

EE models extend classic compartmental epidemiological models that divide the population into compartments defined by health and demographic status. The classic models focus on the basic reproductive ratio of the disease, R_0 —the number of secondary cases in a naïve, wholly susceptible, and disease-free population that result from the initial introduction of pathogen (Kermack and Mckendrick 1929; Anderson and May 1979, 1991). In the simplest models, R_0 is the product of three factors: the contact rate, the conditional probability of transmission per contact, and the duration of the infectious period. It is used to indicate whether or not the infection prevalence will increase or decrease. When $R_0 > 1$ the pathogen may spread, when $R_0 < 1$ it will not. The basic reproductive ratio, or variants such as the *effective reproduction number* (which measures transmission in a population that may be only partially susceptible) and the *control reproduction number* (which measures transmission in a susceptible population with control measures in place), are then used to inform disease management (Brauer and Castillo-Chavez 2013). The EE approach treats the reproduction number as a function of the decisions that underpin contact between susceptible and infected individuals. It thus opens up a different set of management options.

The EE approach is ultimately grounded in bioeconomic models of renewable resource management (Clark 1973, 1976, 1979). EE models focus on the optimal disease avoidance strategy and how that feeds back into the spread of infectious diseases of people (Geoffard and Philipson 1996; Kremer 1996; Auld 2003; Francis 2008) and animals (Horan and Wolf 2005; Horan et al. 2010, 2011). The approach also considers the consequences of disease risk management for economic development (Barrett and Hoel 2007) and growth (Grossman 1972; Boucekine and Laffargue 2010; Chakraborty et al. 2010). In what follows, we focus on two risk management strategies—contact reduction and selective mixing. However, we note that considerable attention has also been paid to vaccination (Francis 1997, 2004; Boulier et al. 2007; Cook et al. 2009).

A common feature of EE models is that behavior affects, and is affected by, the disease risks involved in both contact and mixing decisions (Fenichel et al. 2011; Aadland et al. 2013; Fenichel and Wang 2013; Morin et al. 2013). While the term risk is used in many non-economic appli-

cations to denote the probability of an undesirable or bad outcome, we use the term risk to denote the product of the probability and the value of the bad outcome. It is an expected cost. Hence, disease risk is the probability of infection multiplied by the cost of infection. There is a considerable literature on the impact of disease risk, in the expected cost sense, on behavior (Francis 1997; Auld 2003; Chen 2004; Del Valle et al. 2005; Bootsma and Ferguson 2007; Klein et al. 2007; Chen 2009; Funk et al. 2009; Reluga 2010; Chen et al. 2011; Gersovitz 2011), at least some of which is empirically based (Caley et al. 2008; Gersovitz 2011; Fenichel et al. 2013). The evidence suggests that the expected cost of disease, or at least the part of cost that is carried directly by decision-makers, is weighed amongst the benefits and costs of contact and mixing decisions. An improved understanding of how these behavioral responses feed back into infectious disease dynamics strengthens capacity to predict the course of epidemics (Bauch and Earn 2004; Reluga 2010; Perra et al. 2011; Fenichel and Wang 2013).

Beyond improved prediction, the EE approach has the potential to reduce the social cost of disease management relative to classical approaches. Specifically, it allows public health authorities to go beyond traditional control methods such as vaccination, treatment, or social distancing, and to use economic incentives that change the course of epidemics by changing private contact and mixing decisions (Francis 2004; Chowell et al. 2009a; Fenichel 2013). In this paper, we review the development of the EE approach, and show how it is creating new options for the way epidemics are evaluated and managed.

THE BASIC STRUCTURE AND RESULTS OF EE MODELS

It is useful to distinguish between the private decision problem (the decision-problem facing susceptible and infectious individuals, or those trading potentially infected animals or animal products) and the social decision-problem (the decision-problem facing public health or sanitary authorities). The main elements of both problems are an objective function describing the decision-maker's goals, a constraint set describing the dynamics of the system being managed, a control or choice set—the mechanisms by which the decision-maker is able to influence those dynamics, and the feedback loops that link these components.

To illustrate the EE approach, consider the private decision-problem faced by susceptible individuals seeking to manage the risks of an infectious disease of humans. Let the disease dynamics be described by a three-compartment (susceptible, infected and infectious, and recovered) discrete time S, I, R model:

$$\begin{aligned} S_{t+1} - S_t &= -C(w_{ct}, S_t, I_t, R_t)\beta(w_{ht}, H_t)S_tP(w_{mt}, S_t, I_t, M_t^{SI}) \\ I_{t+1} - I_t &= C(w_{ct}, S_t, I_t, R_t)\beta(w_{ht}, H_t)S_tP(w_{mt}, S_t, I_t, M_t^{SI}) - I_t\nu, \\ R_{t+1} - R_t &= I_t\nu \end{aligned}$$

w_{ct} , w_{ht} and w_{mt} denote the costs of contact, C_b prophylactic measures, H_t , and mixing decisions, M_t^{SI} . The function $C(\cdot)$ is the rate at which susceptible individuals make contact with others within the population, $\beta(\cdot)$ is the probability that an infectious contact results in infection, $P(\cdot)$ is the conditional probability that a susceptible person will encounter an infected person—the outcome of susceptible individuals' mixing choices, and ν is the recovery rate. In the simplest epidemiological models, the functions $C(\cdot)\beta(\cdot)$ are assumed to be the same for all individuals regardless of health status. Indeed, it is common to find $C(\cdot)\beta(\cdot)$ combined into a single parameter that assumes contacts to be proportional to the size of the population. Mixing is also commonly assumed to be homogeneous: i.e., $P(\cdot)$ is assumed to take the form, I_t/N . We make no special claims about the value of the S, I, R over other compartmental epidemiological models. We use it only to demonstrate an approach that has been applied to many different models.

In EE models, the time paths of $C(\cdot)\beta(\cdot)$ and $P(\cdot)$ are derived from the solution to an economic decision problem in which individuals seek to meet their goals by choosing, respectively, the number of contacts they make, precautionary measures that reduce the probability that a contact will lead to an infection, and/or the effort they commit to avoiding contact with infectious hosts. One example of $C(\cdot)$ allows contact choices to vary with health status, as individuals in different health classes make different choices (Fenichel et al. 2011; Fenichel 2013). The contact function between susceptible and infected individuals takes the form:

$$C_t^{SI}(\cdot) = C_t^S C_t^I N(S_t C_t^S + I_t C_t^I + R_t C_t^R),$$

where C^S , C^I , and C^R is a measure of the average number of contacts an individual in the health class j makes. Another example derives the contact rate from the individual's aversion to disease risk. The number of contacts an individual makes is assumed to depend on the exposure, and

the resulting probability of infection, they are willing to accept (Aadland et al. 2013). In both variants, the key implication is that contact choices vary over time, and by health class, in response to changes in the cost of disease and disease mitigation arising from changes in the states S , I , and R . Models that focus on mixing rather than contact decisions have their roots in the affinity-based mixing models developed in the 1990s to explore the consequences of the choice of with whom to mix with, rather than how much to mix (Dietz and Hadelers 1988; Busenberg and Castillo-Chavez 1991; Hadelers and Castillo-Chavez 1995; Morin et al. 2010; Galeotti and Rogers 2013). In such models, susceptible individuals choose $P(\cdot)$ rather than $C(\cdot)$, the value of $P(S_t, I_t, M_t^{SI})$ depends on the efforts susceptible individuals make to avoid mixing with infectious individuals. Whether people choose $P(\cdot)$ or $C(\cdot)$, their choices people depend on the costs and benefits of alternative actions. Specifically, people balance the benefits from making contacts (e.g., the benefits from buying or selling goods or services) against the costs of disease. They choose the number of contacts and/or the disease class with whom to make contact so as to maximize some index of wellbeing (utility), balancing the benefits of contact against the expected cost of disease and disease mitigation, conditional on the current states so that the choices change over time.

The likelihood of becoming infected depends on the number of contacts made, and the riskiness of those contacts. Typically, the contact choices of a forward-looking susceptible individual are modeled as the solution to a dynamic programming problem:

$$V_t(S) = \max_{C_t^S, M_t^{SI}} \left\{ U^S(S_t, C_t^S, M_t^{SI}) + \rho \sum_j Q^{Sj}(C_t^S, M_t^{SI}, S_t, I_t, R_t)(V_{t+1}(j)) \right\},$$

where ρ is a discount factor; Q^{Sj} is the probability of transition from health state S to health state j conditional on the choice of C_t^S and/or M_t^{SI} , and on the current state of the system (the health state of others); and $V_{t+1}(j)$ is the future value of being in health state j . In solving the problem, people increase disease risk mitigation up to the point where the marginal cost of mitigation equals the state-dependent marginal benefit of reductions in disease risk.

Because the choices people make change infectious disease transmission rates, they also change epidemiological dynamics. It follows that disease dynamics are sensitive both to the cost of disease (the income forgone during

illness and the direct cost of illness) and the cost of disease avoidance. If the cost of disease is very low there is little incentive to avoid it, and disease dynamics will be those associated with proportionate mixing. If the cost of illness is very high, people will invest substantial resources in disease avoidance. In extreme cases, private decisions about selection of contacts can lead to an effective quarantine on infected individuals—an effect that would never occur in classical models. Disease dynamics are also sensitive to the benefits of contact. People trade-off disease risks against the benefits of contact. If there is much to be gained from contact they will accept much greater disease risks than if there is little to be gained (Areal et al. 2008; Fenichel et al. 2010; Gramig and Horan 2010; Horan et al. 2010).

Improved understanding of the behaviors that influence disease dynamics improves disease management. It increases both the number of control options open to public health authorities, and identifies how much public intervention is warranted. Depending on people's goals, their resources, and the opportunities open to them, the behavior of some individuals may slow epidemics, while the behavior of others can speed them up (Kremer 1996; Aadland et al. 2013). If the private and social costs of disease and disease avoidance are the same, then the decisions people make in their own self-interest coincide with the decisions they would make if they were acting with the interests of society in mind. If the private and social costs of disease and disease avoidance are different—if people make private decisions that are not in the social interest—then public health authorities can use an understanding of the private decision process to incentivize people to make different decisions. In so doing, they can minimize the expected social cost of the disease and its control.

This opens up a novel set of disease management instruments aimed at confronting individuals with the external costs of their actions or compensating them for the external benefits their actions provide. Specifically, public health managers may select instruments that change the course of disease by changing contact and mixing incentives. The same costs of disease and disease avoidance that drive private contact and mixing decisions become potential points of leverage on contact and mixing behavior. If the social decision-maker is able to alter those costs through, for example, taxes, subsidies, access fees, penalties, and so on, then the social decision-maker is also able to change private behavior and disease dynamics (Francis 1997; Auld 2003; Francis 2004). For example, where tracking mechanisms allow the sale of diseased animals to

be traced back to a specific hub in the supply chain, opening the responsible individuals up to legal penalties provides them with an incentive to exercise care. By increasing the private payoff to actions that confer benefits on others, it is possible to enhance the public good even if individuals act only in their private self-interest (Francis 1997; Sandler and Arce 2002).

EVIDENCE

There are as yet relatively few empirical studies of the relation between the costs and benefits of contact, the decisions that people make involving trade or travel contacts, and the spread of either animal or human diseases. However, those studies that do exist are informative. They test two of the main hypotheses suggested by theoretical work on private disease risk mitigation: (a) that efforts to reduce contact with infected people are likely to be increasing in the cost of disease and decreasing in the cost of avoidance, and (b) that disease risk mitigation reduces disease prevalence and lengthens disease epidemics (Figs. 1, 2) (Chowell et al. 2007, 2009b; Fenichel et al. 2011). Since the cost of disease avoidance is greater the more difficult it is to identify infected individuals, the first of these hypotheses also implies that risk mitigation is likely to be increasing in the quality of the signals about which individuals are infected (Fenichel and Horan 2007b).

On the first hypothesis, the effort made to avoid risk, and so disease prevalence, has been found to be increasing in the cost of disease (Mummert and Weiss 2013). There is evidence that people are willing to pay more to avoid diseases they believe to be serious, and that their willingness to pay changes

as their perception of the seriousness of the disease changes. A study of the number of passengers missing previously purchased flights during the 2009 swine flu or A/H1N1 influenza epidemic used flight records, Google Trends and the World Health Organization's FluNet data to show that concern over H1N1 accounted for a small proportion (0.34%) of missed flights during the epidemic. The authors estimated that this represented around \$50 M in travel-related benefits. They noted that while this was consistent with a self-protective response to the epidemic, the timing of responses correlated poorly with FluNet data. They concluded that responses were motivated by subjective rather than objective perceptions of risk (Fenichel et al. 2013).

For animal diseases (and emerging zoonoses), it has been shown that decisions affecting the national and international movement of livestock reflect the costs and benefits of disease risk mitigation, and strongly influence the probability of spread (Keeling et al. 2001; Kilpatrick et al. 2006, 2009). Analyses of the 2001 foot and mouth disease (FMD) outbreak in the UK, and the 2004 H5N1 avian influenza outbreak in Thailand, for example, show that differences in the compensation schemes applied in each case had significant effects on the relative costs of disease and disease avoidance, and hence on the dynamics of the disease. In the UK FMD outbreak, the structure of compensation to farmers perversely reduced the cost of disease and increased the cost of disease avoidance, so discouraging disease avoidance (Davies 2002). In the Thailand H5N1 outbreak, by contrast, the government offered farmers 100% compensation for every animal killed (significantly above the compensation formally allowed under the Animal Epidemic Act), effectively reducing the private cost of disease avoidance to zero (Tiensin et al. 2005).

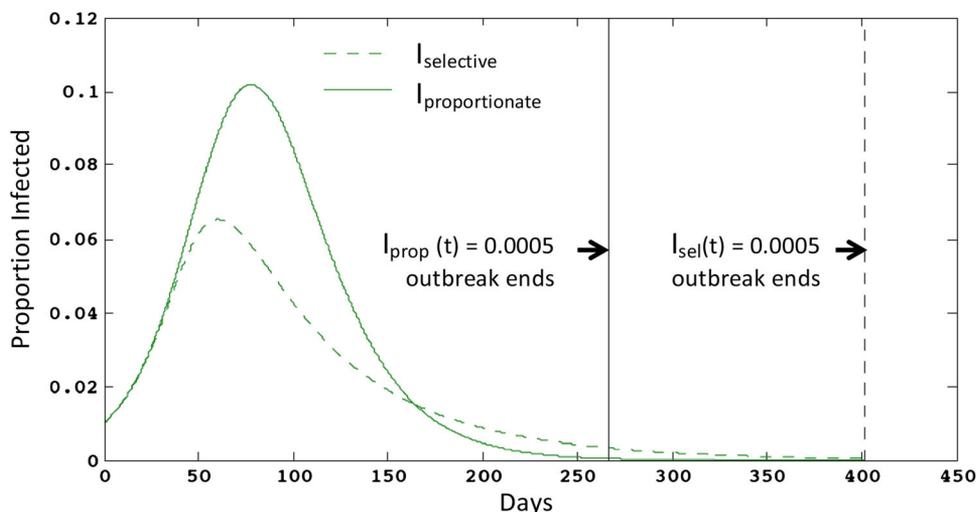


Figure 1. The effect of disease risk mitigation through selective mixing on disease prevalence and the duration of an epidemic. *Solid lines* show prevalence and duration where susceptible individuals mix with other individuals randomly (proportional mixing). *Dashed lines* show prevalence and duration where susceptible individuals avoid mixing with infected and infectious individuals (selective mixing).

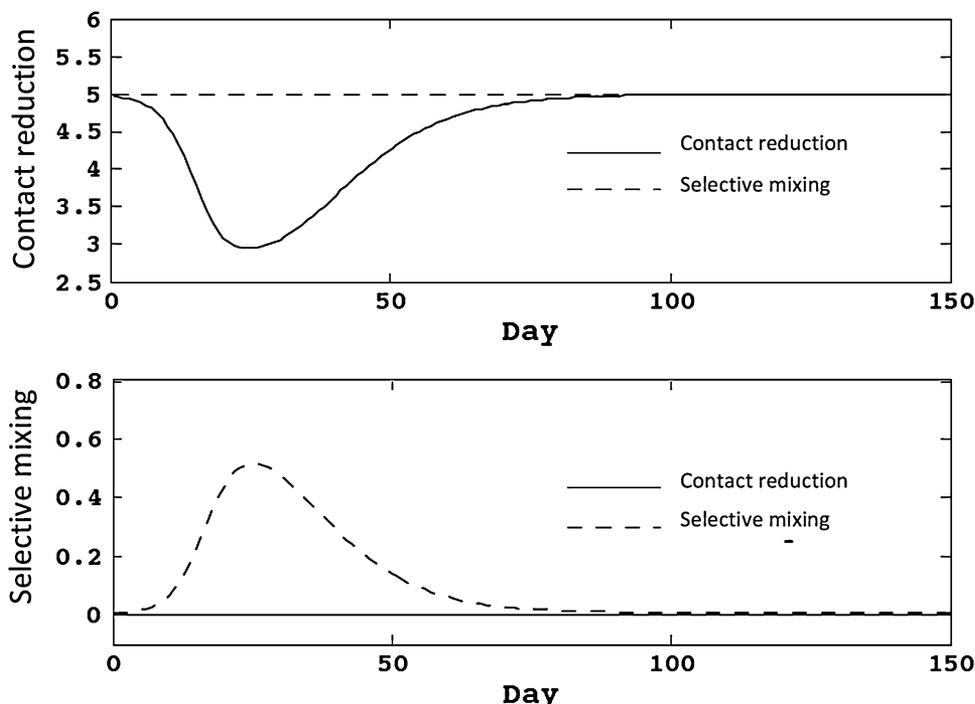


Figure 2. The symmetry between disease risk mitigation through contact reduction and disease risk mitigation through selective mixing. The *upper panel* indicates the timing and level of optimal contact reduction by susceptible individuals under each strategy (contact reduction is zero under a selective mixing strategy). The *lower panel* indicates the timing and level of effort committed by susceptible individuals to avoiding infected/infectious individuals under each strategy (avoidance effort is zero under a contact reduction strategy).

Because disease risk reflects both the probability of infection and the cost of infection, trade growth that reduces cost more than proportionately to the increase in the probability of infection can, paradoxically, reduce risk (Fenichel and Horan 2007a, b; Fenichel et al. 2010; Horan et al. 2011, 2013). While there have been no formal tests of this hypothesis, there is considerable empirical evidence that people trade-off the price of goods and services against the risks they pose (Lusk and Coble 2005), just as they trade-off the rate of return and risk on asset holdings (Ghysels et al. 2005).

There is less evidence that the public management of infectious human disease is sensitive to the incentive effects of changes in the private cost of disease and disease avoidance. Although the World Health Organization recognizes the cost effectiveness of economic instruments (World Health Organization 2004), applications to the control of infectious human diseases are limited. The most obvious and long standing examples are the use of subsidies to lower the private cost of vaccination (Brito et al. 1991; Geoffard and Philipson 1996, 1997; Cook et al. 2009) or vaccination and treatment (Gersovitz and Hammer 2004; Gersovitz 2011). By contrast, standard control measures such as travel interdictions or enforced quarantine are classic, and often poorly targeted, examples of command and control instruments. Measures of this sort have, in particular cases, proved to be extremely costly (Thompson

et al. 2002; Webby and Webster 2003; Smith et al. 2009b; Keogh-Brown et al. 2010). In some cases, for example, mandatory controls have increased the flow of infected emigrants from the epicenter of infectious disease outbreaks, so spreading the disease to uninfected sub-populations (Mesnard and Seabright 2009; Maharaj and Kleczkowski 2012).

The use of command-and-control instruments is particularly common at the national level, where governments have the authority to implement emergency controls on subject populations (World Health Organization 2006; Stern and Markel 2009; Steelfisher et al. 2012). Interestingly, it is also the preferred approach at the international level where the control options are prescribed by two multilateral agreements, the International Health Regulations and the Sanitary and Phytosanitary Agreement, even though there is no supranational body with sovereign authority over nation states (Perrings et al. 2010a, b). While measures of this sort do not directly target the incentives facing susceptible individuals they do have incentive effects. A study of the 2009 H1N1 epidemic in Mexico, for example, concluded that the prolongation of the epidemic through a second wave was induced by the private response to social distancing measures implemented by the health authorities (Herrera-Valdez et al. 2011). Similar effects were observed in the 2007 Dengue outbreak in Taiwan (Hsieh and Chen 2009), and the 2002–2003 SARS epidemic (Chowell et al. 2004).

The question to consider is whether mandatory measures are cost effective, once the incentive effects of those measures are taken into account. The optimal control program in all cases depends on a number of factors, including the nature of disease, the size of each population, the length of the time horizon, or the discount rate applied, as well as the characteristics of the controls (Brandeau et al. 2003). This makes it difficult to generalize. We are unaware of empirical studies of the relative cost effectiveness of mandatory and incentive-based measures for the *ex post* control of outbreaks. It seems clear, however, that incentive-based measures are able to reduce the *ex ante* risk of disease more cost effectively than direct controls over the mobility of people or the movement of goods. A study of the cost effectiveness of a number of different classes of primary disease prevention (controls aimed at preventing new cases of disease) found that that measures aimed at changing the environment within which people make decisions are significantly more cost-effective than measures aimed at clinical or nonclinical interventions on individuals (Chokshi and Farley 2012). Measures aimed at changing the environment within which people make decisions include, for example, taxes designed to increase the private cost of risky behaviors. Measures aimed at individuals include, for example, quarantine or screening programs. The study showed that in terms of costs per quality-adjusted life-year the proportion of preventive measures that are cost saving is higher among environmental interventions (46%) than among clinical interventions (16%) or nonclinical, person-directed interventions (13%). Given that individual restrictions or obligations also pose more legal and ethical challenges (National Research Council 2007), this indicates that incentive-based measures may offer a significant advantage.

For plant diseases, a recent example of the use of incentives, in the form of conditional market access, concerns management of disease risk associated with international plant trade. With a 2011 amendment to the Plant Protection Act, the USDA established a new “gray list” designation available for plants known as “Not Approved Pending a Pest Risk Analysis” (NAPPRA) for species that might be pests, or serve as hosts of pests or pathogens (US Department of Agriculture-Animal and Plant Health Inspection Service 2011). This rule change made it simpler to restrict access to US markets for particular taxa of plants which pose a biological risk (Liebhold et al. 2012). Currently, the only mechanism for approving NAPPRA listings for importation is a detailed pest risk assessment (PRA)

assessing the threat of pest infestation, transit, colonization, spread, and damage. In April 2013, the USDA formally proposed a further amendment that would allow US import market access for NAPPRA listings conditional on exporters’ adoption of Integrated Pest Risk Management Measures (IPRMM) (US Department of Agriculture-Animal and Plant Health Inspection Service 2013). IPRMM involves certification that sufficient phytosanitary measures are being applied from the beginning of production to the end of distribution. Market access in an IPRMM program would be particularly flexible and dynamic. Access for approved producers could be revoked if the producer failed to meet the conditions at any time (US Department of Agriculture-Animal and Plant Health Inspection Service 2013). While the US is at the forefront of the IPRMM approach, interest is global. In 2012 parties to the International Plant Protection Convention adopted a standard known as ISPM-36 which recommended and outlined the use of integrated measures to manage pest and pathogen host risk for international plant trade (International Plant Protection Convention 2012). Attempts to bring pathogen introduction risks into the Fish and Wildlife Service injurious species regulations are an effort to follow this, but so far have not been successful.

DISCUSSION

In some spheres of environmental management, command-and-control instruments are being replaced, or at least supplemented, by economic instruments designed to penalize those whose actions harm others (Stavins 2003) or to incentivize those whose actions benefit others (Kinzig et al. 2011). There are many such instruments already in use for managing invasive pests and pathogens. They include charges covering the cost of inspection and interception, excise taxes, environmental bonds, damage bonds, import deposits, restoration deposits, ballast water fees, and tradable risk permits (Eisworth and Johnson 2002; Horan et al. 2002; Olson 2006; Emerton and Howard 2008; Gren 2008). A number of these instruments also reverse the burden of proof, in that they require those whose actions are a source of risk to insure society against the consequences of their actions (Perrings et al. 2002; Keller and Perrings 2011; Barbier et al. 2013).

The potential for the use of market-based mechanisms (taxes) to correct the external costs that infected individuals

impose on society in the course of an epidemic has already been demonstrated in simulation models (Goldman and Lightwood 2002; Gersovitz and Hammer 2004, 2005). Similar results have been found for the use of subsidies on the cost of vaccines (Francis 2004; Chen 2006). There is, however, scope for reducing the cost of disease avoidance in other ways. Measures that reduce the income loss from private disease avoidance, for example, can be particularly effective. Just as regulations governing physical safety in the workplace have reduced the incidence of work-related accidents, so rights to paid sick leave can reduce infectious disease risks (Aronsson et al. 2000; Skåtun 2003).

Given the pressure on public health authorities to develop more targeted and cost effective disease management strategies (Glass et al. 2006; Fenichel 2013), incentive-based disease prevention programs are increasingly attractive options. The CDC's current HIV prevention program, for example, is focused on risk targeting, bringing a geographic specificity to prevention policies, and developing a rank ordering of policies by cost effectiveness (Centers for Disease Control and Prevention 2009). The plan explicitly aims to "Identify, develop and evaluate effective behavioral interventions and strategies" (Centers for Disease Control and Prevention 2011). This requires measuring variables at scales that allow prioritization of funding across locations and risk categories. It is recognized that where the prevalence of disease is low, people will not take as much care to limit their exposure as they do where prevalence is high, making disease eradication problematic (Aadland et al. 2013). By encouraging private individuals to make decisions that are in the social interest, incentive-based measures can counteract effects of this kind.

One other implication of the EE approach is that the measures used to monitor and predict disease risk can be broadened. In addition to prevalence measures, it becomes possible to use measures of disease risk mitigation or the drivers of disease risk mitigation. Aside from the travel data used in the H1N1 study, for example, it is possible to employ time use surveys (Zagheni et al. 2008) and home media consumption measurement by audience research firms. These have the appealing feature that a representative sample of residents is monitored continuously over time and in a consistent way across a large set of countries. Coincident with an outbreak, deviations in television viewership, for example, can provide a proxy for assessing changes in time spent at home and thus in social contacts. It is also possible to exploit the much larger data base on

avoidance behavior to other sources of human health risk such as air pollution and drinking water contamination (Zivin and Neidell 2013). Beyond such measures, data on prices, sales, employment, output, exports, and imports may be as valuable for predicting epidemics as data on current disease status (Suhrcke et al. 2011).

In summary, the EE approach is opening up new options for both the prediction and management of epidemics. By improving our understanding of contact behavior the approach is strengthening capacity to project the future course of disease. By identifying the gap between the private and social cost of private disease risk mitigation, the EE approach makes it possible to induce people to behave in ways that are consistent with the public good. That is, it helps to identify both the private choices that best serve the public interest, and the incentives needed to lead people to make those choices. This opens up the prospect of more cost-effective disease control. Many governments are already committed to subsidizing vaccines. Many also use penalties to discourage importation of infected animals or plants. There is, however, scope for making more and better-informed use of instruments of this kind in the future.

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