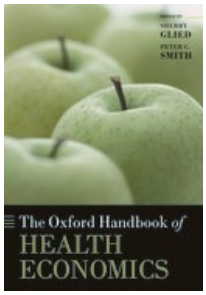

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CHAPTER

9 Economics of Infectious Diseases

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Abstract

Infectious diseases remain a central preoccupation in many countries. This article sets out the economic issues that arise in this highly complex domain. It reviews four main strands of literature on the economics of infectious diseases. It discusses the economic impact of infectious diseases on labor productivity and investment decisions. It focuses on the interplay between disease prevention and treatment and individual risk-taking behavior. It discusses vaccination as an important tool in the prevention of infectious diseases and presents a classic public goods problem. Disease reporting and eradication efforts are also global public goods. A fourth strand of literature is on the optimal design and allocation of resources for prevention and treatment programs. These programs are based on epidemiological models of disease spread that present significant mathematical challenges.

Keywords: infectious diseases, economic impact, vaccination, treatment, epidemiological

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INFECTIOUS diseases remain an important cause of poor health in developing countries (Lopez et al. 2006). Hygiene, sanitation, vaccination, and treatment access have improved and thus reduced the burden of infections in developed countries, but these continue to pose barriers to similar reductions in low- and middle-income countries. Even in developed countries, influenza and HIV/AIDS remain challenges that demand attention from public health authorities. From an economist's perspective, infectious diseases are distinguished from many other health issues by the central role played by externalities.[1] Control of infectious diseases yields both positive externalities (prevention and treatment can delay or reduce spread of infection to uninfected individuals) and negative externalities (over-use of treatment can lead to drug resistance, which has global consequences for treatment effectiveness).

In this chapter, we review four main strands of literature on the economics of infectious diseases. First is the economic impact of infectious diseases, discussed in section 9.1. Although there are direct impacts on life years, there are also important impacts on labor productivity and perhaps investment decisions. These impacts have been studied in contexts ranging from hookworm eradication in the United States and malaria control in India, Africa, and Vietnam to HIV/AIDS epidemics in Africa.

A second strand focuses on the interplay between disease prevention or treatment and individual risk-taking behavior, discussed in section 9.2. Much of this literature builds upon Peltzman's idea of risk compensation, which holds that people adjust their behavior to a regulation in ways that counteract the
p. 190 intended effect of the regulation (Peltzman 1975). For example, when the government passes a seatbelt ↳ law, some drivers may respond by driving less safely. In the case of infectious diseases, individuals respond to greater risk of disease by taking greater protective measures. Conversely, a reduction in the expected cost from disease due to the availability of treatment could disincentivize self-protection measures (Philipson and Posner 1993). Another theme in this literature is that vaccination, an important tool in the prevention of infectious diseases, presents a classic public goods problem. Society gains from individual vaccination because of herd immunity, but this value is not recognized by individuals, who have an incentive to free-ride on vaccination by other individuals.

A third strand of literature relates to incentives faced by institutions and nations to respond to the emergence and spread of infectious diseases, discussed in section 9.3. Disease reporting and eradication efforts are also global public goods. Individual countries may fail to internalize the benefits of prompt reporting on the global spread of disease. Likewise, the benefits for the last country to eliminate a disease are much less than the benefits that accrue to all countries when the disease is permanently wiped out from the planet. Policies relying on prompt reporting of disease outbreaks or domestic eradication of disease must recognize the incentives that individual countries face.

A fourth strand of literature is on the optimal design of and allocation of resources for prevention and treatment programs, discussed in section 9.4. These programs are based on epidemiological models of disease spread that present significant mathematical challenges. Section 9.5 concludes the chapter.

9.1 Economic Impact of Infectious Diseases

The primary cost of infectious disease is loss of life. According to Lopez et al. 2006, five infectious diseases (lower respiratory infections, HIV, diarrheal diseases, tuberculosis, and malaria) were among the top ten global causes of death in 2001. Because onset is earlier in life than other top causes, such as ischemic heart disease, their impact is magnified when the outcome is not merely death but loss of life years. Together, all infectious diseases account for more than 25 percent of premature death globally.

A secondary impact of infectious disease is reduction in income and thus consumption. There is a large literature on the impact of disease, and health generally, on income. Although much of this literature studies infectious diseases, it does not identify any impacts of contagious disease that differ from impacts of
p. 191 non-contagious disease and infectivity is treated as having little economic consequence. This is surprising, ↳ as one might suspect that infectious diseases may have important consequences for the location or dispersion of economic activity.[2]

Focusing broadly on the effects of health on income and development, it is useful to distinguish partial equilibrium and general equilibrium effects. Bleakley (2010) offers a theoretical framework to think about partial equilibrium effects. Health has both a direct effect and an indirect effect on discounted lifetime income: $Y(H, E(H))$. The direct effect is increased productivity during working years and increased number

of working years. The indirect effect is changes in the level of investment in human capital *E* (*H*) and thus income.

Although a number of prominent papers (e.g. Miguel and Kremer 2004; Bobonis et al. 2006) focus on the effect of disease or disease control interventions on human capital investments, there are strong theoretical reasons to suspect that the indirect effects of disease are only of second-order importance. First, improvements in childhood health have theoretically ambiguous effects on, say, years of schooling. Health improves both the returns to schooling (the investment pays off over a longer working life) as well as the opportunity costs of schooling (the loss of wages during days in school).[3] This trade-off does not work against improvements in adult health. An increase in anticipated health after schooling increases the return to schooling without increasing the loss of wages during school. The effect of adult health on human capital investment is dampened, however, if improvements in longevity are associated with a reduction in hours worked per year (Bleakley and Lange 2009).

There is a second reason to think the indirect effect is minor: the envelope theorem. An individual chooses the level of human capital investment such that marginal benefit is equal to marginal cost. Even if health increases marginal benefit, the marginal cost is so high that the overall marginal return is likely to be small. This may explain conflicting results on the effect of disease control on education (compare Lucas 2010 with Cutler et al. 2007 on the effects of malaria eradication) and why Bleakley (2007a, 2007b), Cutler et al. (2007), and Behrman (2009) find that health seriously affects income but not human capital accumulation.[4]

p. 192 Further (indirect) support comes from ↳ papers that find a significant effect of improvements in adult health on labor productivity (e.g. Thomas et al. 2003; Larson et al. 2004; Russell 2004; Ashraf et al. 2009).

A contrary view is given by Corrigan et al. (2004, 2005) and Bell et al. (2006). These papers argue that parents transmit human capital to their children and that increases in (young) adult mortality reduce this transmission. Simulation exercises based on overlapping generation models incorporating this mechanism suggest both larger and longer duration effects from HIV epidemics. For example, Bell et al. (2006) predict that the 20 percent HIV prevalence in South Africa could shrink that economy by half in as little as four generations.

Finally, we turn to the general equilibrium effects of improvements to health (see also Chapter 5 in this volume). As Bleakley and Lange (2009) highlight, the central issue here is the fixed factor problem. Although investment in labor and physical capital can respond to health-induced improvements in labor productivity, land may be a fixed factor. If improvements in health increase population and thereby labor supply,[5] then there may be less land per worker. This will offset the positive effect of health on labor productivity and may even reduce income per capita. This effect is less significant in developed countries, where urbanization and trade have reduced the importance of the fixed factor land. But it may explain why cross-sectional comparison regressions frequently find insignificant effects of disease on per capita income (e.g. Bloom and Mahal 1997; Acemoglu and Johnson 2006). Calibrated general equilibrium models that also find modest effects of health on income include Young et al. 2005 and Ashraf et al. (2009).

9.2 Individual Incentives

The literature on the role that individual incentives play in infectious disease dynamics and control can usefully be sorted into three categories. The first includes papers that examine the impact of disease prevalence on self-protection and, in return, the effect of self-protection on prevalence. The main conclusion here is that self-protection both slows the spread of infections and reduces the return from public interventions to slow disease. The second category comprises papers that examine the demand for treatment and vaccines. These papers conclude that both treatment for the infected and vaccination to prevent infections have positive externalities on population prevalence. The challenge is how to solve this public goods problem, especially given that treatment or vaccination and other forms of self-protection are
p. 193 substitutes. The third set of papers ↳ examines the demand for information on one's infection state—that is, on testing. For diseases such as HIV/AIDS, testing is important because it is a prerequisite for treatment, and the availability of treatment may reduce the cost of risky behavior. Below we review these three subsets of the literature in order.

9.2.1 Prevalence and the Demand for Risk

Initially, models and simulations of disease dynamics in the mathematical epidemiology literature assumed that individual risk taking was exogenous (e.g. Anderson and May 1991).[6] An increase in disease prevalence accelerated the spread of disease and thus its consequences. These models also suggested that interventions to reduce prevalence—either treatment for the infected or vaccination that prevented infections—actually had significant effects on prevalence.

More sophisticated analyses introduced sensitivity analysis that examined the effects of interventions while varying the degree of risk taking by individuals. Although risk taking was still exogenous (it did not decrease *because* of prevalence), these analyses demonstrated that high levels of risk taking could swamp the efficacy of treatments and vaccines (Blower and McLean 1994; see also Hadeler and Castillo-Chavez 1995; Blower et al. 2002; Bogard and Kuntz 2002; Stover et al. 2002; Blower et al. 2003; Gray et al. 2003; Smith and Blower 2004; Anderson and Hanson 2005).

In the late 1980s and early 1990s, biologists (Liu et al. 1986, 1987; Blythe et al. 1991; Hethcote et al. 1991; Brauer et al. 1992; Velasco-Hernandez and Hsieh 1994; Gubbins and Gilligan 1997a, 1997b) and economists (e.g. Philipson and Posner 1993; Geoffard and Philipson 1996; Kremer 1996) began endogenizing risk taking in infectious disease models (mainly susceptible-infected, or SI, categorical models). The economic models posited a formal demand for risky behavior, such as sex, and assumed that the cost of risky behavior included infections. Since disease prevalence increased the chance of infection, it reduced demand for sex. The notion of prevalence-response elasticity was born. The important implication was a feedback loop that slowed the spread of disease without any public health intervention: growth in prevalence reduced risk taking, which in turn reduced growth in prevalence. Indeed, it is theoretically possible that self-protection alone could reduce steady-state levels of prevalence and even lower the basic reproductive rate of infection (R_0)—the number of persons whom one infected individual infects—below one and thus extinguish a disease. Subsequent models expanded upon this insight by considering such variations as heterogeneity in
p. 194 the levels of risk taking ↳ across individuals (Kremer 1996) and the role of individuals' expectations about the future epidemics on current levels of risk taking (Auld 2003).

9.2.2 Demand for Treatment and Prevention

For infectious disease, both vaccines and treatment have positive externalities. By getting treated or vaccinated, one enters and expands the population of uninfected or resistant individuals. The larger this population, the lower the risk of infection for other individuals. Because individuals do not internalize this external benefit, however, there is a socially insufficient demand for treatment and vaccines, though in practice it is assumed the private benefit of treatment is sufficient to induce universal demand. The natural implication is that vaccination should be subsidized (e.g. Vardavas et al. 2007).[7]

The concept of prevalence-response elasticity, however, complicates this conclusion. Because public health interventions reduce prevalence, they also reduce self-protection (e.g. Lakdawalla et al. 2006). As a result, public subsidies and self-protection are substitutes (Philipson 1996). Indeed, if vaccination is voluntary, not even subsidies will be able to achieve universal coverage. As the fraction of the population that gets vaccinated approaches one, the demand for vaccination falls to zero unless the subsidy is greater than the private cost of the vaccine (Bauch and Earn 2004). Even a mandatory vaccination program may fall short if it does not cover the entire population: the mandatory program will reduce demand among the exempt population (Geoffard and Philipson 1997; Bauch et al. 2003).

Some analyses have gone further and suggested that treatment and vaccination could cause so much risk taking that the epidemic would actually grow (Blower and McLean 1994; Blower et al. 2000). Indeed, Auld 2003 speculates that this risk may have contributed to the decision not to release then-existing, semi-efficacious HIV vaccines.

The above intuition, and indeed much of the literature on demand for risk taking and vaccination, can be described with a simple model. An individual engages in greater self-protection or vaccination if the benefit outweighs the costs: $[p_0 — p_1]\, D \rangle\ c$. The benefits are calculated as the difference between the probability of infection if an individual does not self-protect or vaccinate (p_0) and the probability if he does (p_1), all times the health cost of infection (D). The cost (c) is the utility from risk-taking or the monetary price and health risk from vaccination. Obviously, the risk of infection will *inter alia* increase with the number of individuals who are infected: $p_1(I)$, $p_1'(I) \langle 0$. Since the level of infection falls with vaccination subsidies ($I'(s) \langle 0$), so does the benefit from and demand for individual vaccination.

A large strand of the literature focuses on the role of fatalism or competing risks on incentives. The probability of infection is capped at one. If there are two factors that cause infection, then we may write $p_1 =$
p. 195 $\max\{p_{11} + p_{12}, 1\}$, where p_{11} is the immediate probability ↳ of infection if under self-protection or vaccination this period, and p_{12} is another unavoidable source of risk. If p_{12} is high, then p_{11} will generate little demand for self-protection or vaccination (because p_{12} either closes the gap between p_1 and p_0 or makes the cap bind). For example, if $p_{12}$is a competing cause of death for survivors of an initial infection, then they will have less incentive to take a vaccine (Dow et al. 1999).[8] If individuals expect a serious epidemic in the next period (p_{12}), then they have less incentive to reduce risk-taking in this period (p_{11}) (Auld 2003). If life without the disease is not very pleasant (L), the demand for self-protection may be low. Oster 2007 suggests this may explain why prevalence elasticity may be lower in Africa than in the United States.

Indeed, our model captures the intuition behind an interesting paper by Heal and Kunreuther 2005 that challenges the conventional notion of individuals free-riding on public vaccination programs. Their paper relaxes the assumption that vaccines are effective against infection through contact with other humans and assumes that vaccines confer some protection against infection through contact with the environment. Our model can account for this by making $p_{12}(I\,(s))$ rather than p_{11} a function of the prevalence. Now, an increase in vaccination subsidies will lower I but will decrease a *competing* cause of death. This will increase the

marginal utility of vaccination (p_{11}). Vaccinationby others is not a substitute for self-vaccination because it is ineffective at protecting against contact with other humans.

9.2.3 Demand for Testing

The third topic that has garnered substantial attention in the literature on individual incentives is the demand for information on one's disease state. Information is important for two reasons. First, it may be a prerequisite for obtaining treatment. For example, one cannot get a script for an antiretroviral without a test result confirming that one is HIV+. Second, aggregate data on prevalence may depend on voluntary testing. In this case, the data will report prevalence only among the population that chooses to test.

Because information is costly, either because tests are costly or because there is a stigma to being sick, demand for information is not universal. People will demand information only if they expect it may materially change their prior beliefs. Low-risk individuals who are fairly certain they are not infected will not demand testing. Nor will high-risk individuals who are fairly certain they are, unless testing is necessary for treatment. It is mainly individuals who have priors around, say, a one-half probability of infection, who will test (Boozer and Philipson 2000).

The public health community thought quite differently about testing for HIV/AIDS. The conventional view was that testing was necessary for people to obtain treatment, and that treatment saved lives. Philipson and
p. 196 Posner 1993 questioned this logic. They ↳ noted that if information was a conduit for treatment and if treatment increased demand for risky behavior, then information obtained through mandatory testing could increase the demand for risky behavior after a positive test and possibly *worsen* an epidemic. Survey data collected by epidemiologists suggested that individuals were more cautious after testing positive for HIV.[9] Mechoulan 2004 uses a simulation model to show that a small number of altruists could ensure that testing does not exacerbate an epidemic.

What if testing is not mandatory? Mechoulan 2007 considers the effect of treatment on demand for risky behavior when testing is voluntary (and individuals are selfish). Among inframarginal testers, treatment increases risk taking, as in prior models. However, treatment also increases the return to testing, so marginal non-testers start obtaining tests. Together, these increase prevalence, but this triggers the usual prevalence response feedback, reducing all individuals' risk-taking behavior. If prevalence—response elasticity is very high, it is possible that risk taking could even fall, on balance.

9.3 Institutional and National Incentives

Institutional incentives for control of infectious diseases operate in ways similar to those of individual incentives but with important contextual differences. Take the example of hospital incentives to control infectious disease outbreaks. Often, patients are colonized with disease-carrying bacteria in one health care institution and carry these to another institution. Investment in infection prevention at any single institution therefore benefits both that institution as well as others, which would have fewer incoming infected patients (Smith et al. 2005). Interestingly, incentives to control infections strongly depend on the number of incoming patients who are colonized with disease-causing bacteria. If the number increases, it would be in a hospital's interests to ramp up its infection control. However, if too many incoming patients are colonized, it would be hopeless for the hospital to try to control the epidemic on its own without the cooperation of other institutions, and its optimal infection control expenditure should decline.

p. 197 At the level of countries, incentives related to infectious diseases operate on similar principles. Control of malaria in South Africa, for instance, is possible if its neighbors, particularly Zimbabwe and Mozambique,

were also willing to fight malaria within their borders. Returns to investment in control are diminished if a large number of patients carry these diseases across borders. Thus, a country's incentives to control a freely moving disease like malaria are determined as much by its ability to stop the inflow of infected individuals as by the ability to control the disease within its own borders. Reducing malaria in a country could have transboundary benefits by incentivizing infection control in its neighboring countries as well.

This principle also applies more generally to the challenge of global disease eradication. The elimination of disease in all forms anywhere on the planet is a global public good, since it theoretically suggests that countries can cease all control measures and vaccination programs.[10] Eradication is a binary public good: the maximum benefits are achieved when the disease is completely gone. However, incentives for the last country to eliminate the disease may be insufficient for two reasons. First, the benefits that accrue to the entire world when the last country achieves eradication are almost certainly much larger than the costs of eradication as well as the benefits of eradication in that country alone. Second, the benefits in the last country may be small because the disease has been eliminated everywhere else and there is no risk of importation.

Diseases that have already been eliminated in high-income countries are the best candidates for eradication in the rest of the world. Indeed, it may be in the financial interests of high-income countries to finance global eradication so that they can reduce their expenditures on prevention and control. In general, disease eradication, which requires simultaneous elimination in many countries, calls for strong international institutions that can enforce a cooperative optimum even when the Nash equilibrium is for no country to eliminate the disease. Economic conditions are not sufficient. The disease must have a relatively low basic reproductive number (the number of secondary cases generated by a single infected individual in a fully susceptible population), should not have a non-human reservoir, and should be easily identifiable so that the last few cases can be eliminated. However, even with favorable epidemiological conditions and a high benefit-cost ration of eradication (believed to be about 90:1 in the case of smallpox) (Fenner et al. 1988), disease eradication is, at best, a fortuitous event and by no means inevitable, as the experience with smallpox showed.

Disease eradication represents one end of the spectrum of dealing with an infectious pathogen. The emergence and spread of pathogens from animals to humans represent the other. The global spread of zoonoses (as these diseases are known) like swine flu and SARS highlights the difficult decision governments face when presented with evidence of a local outbreak. Reporting the outbreak may bring
p. 198 medical assistance but is also likely to ↳ trigger trade sanctions by countries hoping to contain the disease. Suppressing the information may avoid trade sanctions but increases the likelihood of widespread epidemics. Malani and Laxminarayan (2010) model the government's decision as a signaling game in which a country has private but imperfect evidence of an outbreak. The first important conclusion is that not all sanctions discourage reporting. Sanctions based on fears of an undetected outbreak (false negatives) encourage disclosure by reducing the relative cost of sanctions that follow a reported outbreak. Second, improving the quality of detection technology may not promote the disclosure of an outbreak because the forgone trade from reporting truthfully is that much greater. Third, informal surveillance is an important channel for publicizing outbreaks and functions as an exogenous yet imperfect signal that is less likely to discourage disclosure. In sum, obtaining accurate information about potential epidemics is as much about reporting incentives as it is about detection technology.

A final example of a global public good in the context of infectious disease is drug effectiveness. Take the case of drugs to treat malaria. The use of antimalarials places selection pressure on parasites to evolve resistance to these drugs. Moreover, resistance is bound to arise when these drugs are misused and could have adverse consequences for all malaria-endemic countries. Efforts to manage resistance across national borders would have to rely on international agreements and regulations (Walker et al. 2009) or on tax or subsidy instruments (Arrow et al. 2004). In the absence of such agreements and regulation, countries are

unable to commit themselves to an optimal use of antibiotics, which would be in all countries' interest. At the macroeconomic level, a too-intensive use of antibiotics as an input in a country's production results (Cornes et al. 2001). A supranational authority would have to consider both the externality benefits of antibiotic use, in terms of reducing infections, and the costs, in terms of resistance (Rudholm 2002). Whether antibiotic consumption should be taxed or subsidized to reach the first-best outcome then depends on the relative magnitude of the externalities.

A relatively new class of antimalarial drugs, called artemisinins, requires a different way of thinking about optimal subsidies to manage resistance. When chloroquine, a once-powerful antimalarial drug, became obsolete, the public health world was left with the challenge of using the last remaining effective drug class, artemisinins, in an effective manner. The World Health Organization (2001) has recommended that artemisinins be used in combination with a partner drug that is unrelated in its mechanism of action and genetic bases of resistance, so that a single mutation cannot encode resistance to both components. Artemisinin combination treatments (ACTs), if used instead of monotherapies of either artemisinin or the partner drug, should slow the emergence of antimalarial resistance. However, the WHO guidelines are routinely flouted because monotherapies are much less expensive than ACTs. In response to this problem, an Institute of Medicine report (Arrow et al. 2004) recommended establishing an international fund to buy ACTs at producer cost and resell them at a small fraction of that cost.

On economic efficiency grounds there is a second-best case for subsidizing ACTs, because the ideal policy—taxing monotherapies and other antimalarials according to the marginal external cost from the elevated risk of resistance evolution—is infeasible, given their widespread use in the informal sector. The efficiency
p. 199 argument is further strengthened ↳ by the positive externality to the extent that effective treatment of one individual reduces the risk of infection transmission to other individuals. Laxminarayan et al. (2010) show that it is possible to determine the optimal subsidy in a dynamic disease modeling framework. Bioeconomic analysis has been helpful for determining whether the social benefit from the subsidy, in terms of delayed resistance and saved lives, exceeds the social cost of resistance because of increased use of ACTs (Laxminarayan et al. 2006). It was also instrumental in turning an idea into the Affordable Medicines Facility for malaria, a global financing system launched in early 2009.

9.4 Optimal Allocation of Resources to Fight Infectious Diseases

Recent papers have examined the optimal allocation of resources to fight infectious disease, especially in the context of disease treatment. Given the inevitable constraint of limited treatment resources, there is not enough money to treat all infected individuals, even for a disease like HIV with unprecedented resources made available for treatment. For instance, in Zambia, a country with one of best-funded malaria control programs in Sub-Saharan Africa, only 13 percent of children with malaria receive effective treatment. Questions of optimal allocation of resources across different populations are not just of academic interest. International policy makers at the World Health Organization and the Global Fund for AIDS, TB, and Malaria, as well as national ministries of health, are charged with allocating limited treatment resources across different populations that have different prevalence levels of infection. Although the stated objective of these agencies is to reduce the burden of disease, the focus is on populations with the highest burden of disease.

There is a long literature of applying optimal control theory in the context of epidemiological models. ReVelle et al. (1967) analyzed how best to allocate treatment resources to contain tuberculosis. Sanders 1971 and Sethi 1974 evaluated the socially optimal level of treatment under the assumption of linear treatment costs in an optimal control framework but disagreed on whether the optimal treatment level followed a bang-bang path or was singular.

Choosing the best policy calls for a combination of epidemiological and economic insights, an approach that has been taken in recent papers in both economics and epidemiology (Goldman and Lightwood 1997; Rowthorn and Brown 2003; Smith et al. 2005). Other papers have examined the optimal allocation of resources in a dynamic setting. Gersovitz and Hammer (2004, 2005) evaluate the optimal allocation of resources between prevention and treatment by a social planner and compare this decision with that made by representative individuals who ignore disease externalities.

The allocation of scarce financial resources for disease treatment between geographical regions is usually
p. 200 guided by disease burden, but this basis does not recognize the ↳ dynamic nature of infections. Treating a single infected individual not only cures that individual but also prevents other healthy individuals who are in close proximity from getting infected. Rowthorn et al. (2009) address the question of optimal allocation of treatment resources across two connected populations when there is a period-by-period constraint on the number of treatments available to the social planner. Such a constraint is realistic and relevant from the perspective of most health authorities, which are given annual budgets and cannot transfer funds intertemporally. Rowthorn et al. find that the optimal solution is to preferentially treat the population with low prevalence of infection before allocating remaining resources to the higher-prevalence population. Whereas the usual policy is to provide a larger treatment budget for the more highly afflicted district, they find that, from an economic perspective, disease burden may be a poor criterion to use for allocating treatment resources. However, the paper by Rowthorn et al. relies on a simulation model and cannot conclusively prove that a corner solution is always optimal. This result is shown analytically in a paper by Anderson et al. (2010). The intuition underlying this finding is that the economic value of treatment is greater in this population because of the lower probability of re-infection. From a methodological perspective, the proof depends on the concavity of the cost function. We show that with just a single group, the minimized cost function in every period is weakly concave in the overall wealth allocation. Given this concavity, it is then straightforward to show that with two groups, the health authority will allocate all wealth to a single group.

Optimal allocation of resources has also been examined for the effect of de-worming treatments on school performance in Kenya. Miguel et al. (2001) find that school-based mass de-worming drug treatment for children significantly reduced school absenteeism, a finding that escaped earlier studies in which treatment was randomized among some children in a school. De-worming only some students had few lasting benefits, since treated students were likely to be quickly re-infected by untreated students. However, when treatment was administered to all students in a single school, the externality benefits from treatment (reducing infections of other students) were found to be large.

9.5 Conclusions

The emergence and spread of infectious diseases is strongly influenced by the behavior of individuals, institutions, nations, and international organizations. At the individual level, beliefs about the likelihood of infection and the consequences of being infected drive both the decision to prevent disease in the first place, whether through vaccination or self-protection, and the decision to test for infection after exposure. Greater levels of vaccination in the community are likely to reduce incentives for vaccination for any single individual because of the ability to free-ride. Indeed, because community-level protection and individual protection are substitutes, vaccination campaigns may increase the level of individual risk taking, subverting those very campaigns. These basic principles also operate at the level of institutions and
p. 201 countries, and the problems remain the ↳ same: insufficient incentives to prevent and control disease (including through vaccination), and insufficient incentives to provide information on disease outbreaks. Disease eradication and the effectiveness of drugs to treat infections are global public goods that require international cooperation and strong international institutions to ensure that they are provided at a socially appropriate level; the challenges remain.

The extent to which infectious disease control, whether at the individual level or at the international level, involves incentives and behavior accounts for the growing literature on the economics of infectious disease. When done well, papers in this field have paid close attention to the specific dynamics of diseases, consistent with scientific understanding, to ensure the validity of conclusions drawn by economic models. We have learned much from modeling disease dynamics and control by scaling up from individual infections to epidemics in populations; these models include such factors as the spatial structure and heterogeneity of host populations and the topology of contacts between infected and susceptible individuals. Incorporating this epidemiological complexity into economic models both enriches the quality and robustness of conclusions drawn and informs the work of the vast community of global health professionals, many of whom are not economists.

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Notes

1 To be sure, externalities also play a role in designing interventions against tobacco, where second-hand smoke is an issue, and alcohol, where drunk driving imposes costs on others.

2 Another area ripe for research is the impact of development on infectious disease. Again according to Lopez et al. 2006, infectious diseases account for five of the top ten causes of death in low- and middle-income countries but only one (lower respiratory infections) of the top ten in high-income countries. Two papers that examine this disparity in the context of HIV are Oster (2005) and (2007). The latter asks the interesting question: Why is the elasticity of risk taking to HIV prevalence so high in the United States but low in Africa? The answer is that the utility of a disease-free life is greater in the United States, and thus the benefit of infection control is greater there. This is a theme that we will revisit in section 9.3.

3 It is possible, however, that improvements in health increase the productivity of inframarginal investment in human capital. This might explain why Bleakley (2007a, 2007b) finds that US hookworm eradication programs and malaria control in the United States and certain Latin American countries substantially improved adult literacy among children born after those diseases were controlled.

4 The envelope theorem argument may extend to macro investments in physical capital. Improvements in complementary labor productivity may have only second-order effects on investment in physical capital, given that investment, if set optimally, recently had zero net marginal return.

5 The effect on population is a combination of the direct reduction in mortality and the indirect response of fertility rates. The direction of the fertility effect may depend on whether health improvement is seen as a reduction in the price of quantity of children (Acemoglu and Johnson 2006; Jayachandran and Lleras-Muney 2009) or in the price of quality of children (Bleakley and Lange 2009).

6 Risk taking is the opposite of self-protection. For HIV, for example, risk taking is measured by condom use or number of sexual partners. For influenza, self-protection is largely the avoidance of public spaces. Following the literature, we will examine the demand for medical treatment and vaccinations separately from other self-protection measures, though there is no strong theoretical reason for doing so.

7 The mirror image of this conclusion is that risk taking has a negative externality and should be subject to a Pigouvian tax.

8 The flip side, of course, is that if the government lowers the risk from a competing cause (p_{12}), then the individual may take greater efforts to self-protect or vaccinate (p_{11}).

9 Testing is frequently accompanied by counseling. Individuals who received both testing and counseling also report greater condom use (Kamenga et al. 1991; Bhave et al. 1995; Deschamps et al. 1996; Jackson et al. 1997; Bentley et al. 1998; Levine et al. 1998; Voluntary HIV-1 Counseling and Testing Efficacy Study Group 2000), lower rates of unprotected intercourse (Deschamps et al. 1996; Voluntary HIV-1 Counseling and Testing Efficacy Study Group 2000). Surveys also suggest that testing and counseling are associated with lower HIV incidence (Bhave et al. 1995; Celentano et al. 2000) and rates of sexually transmitted disease (Jackson et al. 1997; Levine et al. 1998; Celentano et al. 2000). There are important caveats to these studies. First, intervention involves testing and counseling, not just testing. Second, the studies do not control for selection and thus do not demonstrate causation (Mechoulan 2004).

10 In practice, this has been true for smallpox, but here too, vaccinations have resumed for military and emergency personnel because of the threat of bioterrorist attacks using the smallpox virus. For a highly infectious disease like measles, it may never be feasible to stop vaccinations because the stock of immunity to measles is itself a global public good that is relatively expensive to replace in a short time, should the disease ever be reintroduced either accidentally or by malfeasance.