The Economics of Infectious Diseases

Summary

Economics can make immensely valuable contributions to our understanding of infectious disease transmission and the design of effective policy responses. The one unique characteristic of infectious diseases makes it also particularly complicated to analyze: the fact that it is transmitted from person to person. It explains why individuals' behavior and externalities are a central topic for the economics of infectious diseases. Many public health interventions are built on the assumption that individuals are altruistic and consider the benefits and costs of their actions to others. This would imply that even infected individuals demand prevention, which stands in conflict with the economic theory of rational behavior. Empirical evidence is conflicting for infected individuals. For healthy individuals, evidence suggests that the demand for prevention is affected by real or perceived risk of infection. However, studies are plagued by underreporting of preventive behavior and non-random selection into testing. Some empirical studies have shown that the impact of prevention interventions could be far greater than one case prevented, resulting in significant externalities. Therefore, economic evaluations need to build on dynamic transmission models in order to correctly estimate these externalities. Future research needs are significant. Economic research needs to improve our understanding of the role of human behavior in disease transmission; support the better integration of economic and epidemiological modeling, evaluation of large-scale public health interventions with quasi-experimental methods, design of optimal subsidies for tackling the global threat of antimicrobial resistance, refocusing the research agenda toward underresearched diseases; and most importantly to assure that progress translates into saved lives on the ground by advising on effective health system strengthening.

Introduction

Infectious diseases remain a major source of morbidity and mortality in many countries, despite great advances in vaccines, diagnostics, therapeutics, and infection control measures. The Global Burden of Disease Study 2013 reports that of the around 2.5 billion disability-adjusted life-years (DALYs) caused by all diseases in 2013 worldwide, 520 million were attributable to infectious diseases. Four infectious diseases were among the top 10 causes of disease burden globally: lower respiratory tract infections (113 million DALYs), diarrheal disease (72 million), HIV/AIDS (70 million), and malaria (65 million) (Murray et al., 2015). These four diseases, plus tuberculosis (TB), comprised 5 of the top 12 causes of death worldwide in 2015 (Feigin, 2016). Death rates due to communicable diseases significantly declined from 2005 to 2015, gains largely attributable to decreases in mortality rates due to HIV/AIDS and malaria. Progress was slower for certain diseases, such as lower respiratory infections, and for some deaths even increased, such as for dengue. People living in low- and middle-income countries suffer disproportionally from the infectious disease burden. In addition, antimicrobial resistance (AMR) has made many previously effective therapies obsolete, which, together with the slow pace of clinical development of new ones, leaves fewer treatment options for many infections. The burden of AMR is difficult to estimate and predict. It may result in at least 700,000 deaths every year, a number that could increase to 10 million deaths by 2050 and a reduction in gross domestic product of 2 to 3.5% if no measures to contain the threat are undertaken (O'Neill, 2014).

Because onset of infectious diseases occurs earlier in life than most of the major non-communicable diseases, their impact is magnified when considering loss of healthy life-years. In addition, some infectious diseases, most prominently HIV/AIDS, have a large economic burden because they hit primarily young productive adults (Haacker, 2016; Laxminarayan & Malani, 2011). Adding to the predictable burden of endemic disease, the threat of epidemics and worldwide pandemics is ever-present. Recent epidemics of Ebola and Zika have shown that diseases that have slumbered for decades in Africa and Asia can become global health emergencies. Outbreaks can be associated with a sudden steep increase in morbidity and mortality if health systems are ill-prepared and with high economic costs because of a decline in economic exchange. This mainly results from self-protective behavior of consumers, e.g., avoidance of travel and consumption of certain products, and the anticipation of such demand changes by producers (Keogh-Brown, Wren-Lewis, Edmunds, Beutels, & Smith, 2010). The re-emergence of some infectious diseases is facilitated by environmental factors including the depletion of forests (Guerra, Snow, & Hay, 2006), reduction of biodiversity (Keesing et al., 2010), expansion and modernization of agricultural practices (Patz et al., 2004), and urban development that increases floods and areas with stagnant water (Ahern, Kovats, Wilkinson, Few, & Matthies, 2005). These lead to changes in ecological niches that favor certain pathogens or fuel their adaptation to humans. Sociodemographic factors such as increase in population density, poor living conditions and infrastructure, increased travel, conflicts, and social instability can also facilitate transmission.

The objective of this article is to provide an overview of the status of economic research on infectious diseases. In the 1990s, the new field of "economic epidemiology" set out to merge insights from economics and epidemiological modeling of infectious disease transmission dynamics (Philipson, 2000). This article is a selective review of the empirical

literature generated in the two decades since these early seminal contributions. It focuses on the issues generic to infectious diseases and on those where economic analysis can make a contribution. It takes stock of what has been achieved and identifies areas where further research is needed. Readers may wonder why the economics of infectious diseases requires its own article and cannot be covered more generally under the economics of public health. The one unique characteristic of infectious diseases—the fact that it is transmitted from person to person—makes it particularly complicated to analyze. It makes individuals' behavior—specifically their choices about prevention and treatment—a central topic for the economics of infectious diseases because of its impact on other individuals. In many situations there is a discrepancy between the choices that individuals would find optimal if made on their own and the choices that would be optimal if made collectively through government. Externalities are the domain of welfare economics, and it therefore has the potential for a large contribution to the understanding of how human behavior affects infectious diseases and the role of government in controlling them.

A Very Brief Epidemiology of Infectious Diseases

Pathogens or microorganisms capable of causing infectious disease usually enter our bodies through the eyes, mouth, nose, or urogenital openings or through wounds or bites that breach the skin barrier. Some diseases spread via direct contact with infected skin, mucous membranes, or body fluids. Diseases spread that way include hepatitis B (blood, saliva, semen, and vaginal fluids), hepatitis C (blood), HIV/AIDS (semen and vaginal secretions, blood, breastmilk), Ebola (urine, saliva, sweat, feces, vomit, breast milk, semen), chickenpox (saliva, skin contact, breastmilk), gonorrhea (semen and vaginal secretions), and glandular fever (saliva). Pathogens can also be spread by indirect contact when an infected person touches a surface such as a doorknob, leaving behind microbes that are then transferred to

another person who touches that surface and then touches his or her eye(s), mouth, or nose. Droplets spread by sneezes, coughs, or simply talking can transmit infection if one person comes in contact with the mucous membranes of the eye(s), mouth, or nose of another person. Diseases spread that way include the common cold, meningococcal disease, and rubella. Influenza is spread by airborne droplet transmission but more commonly by indirect contact on surfaces. Acute lower respiratory tract infections, mainly pneumonia, are spread via droplets or are complications of influenza and the leading cause of death in children under five years, with low birthweight, malnutrition, and poor living conditions being contributing factors. Pathogens can also spread when residue from evaporated droplets or dust particles containing microorganisms is suspended in air for long periods of time. Diseases spread by airborne transmission include TB, measles, chickenpox, Hantavirus pulmonary syndrome, and legionnaires' disease. Common vehicles such as contaminated water, food, or soil may spread pathogens. Rotavirus and *Escherichia coli* are the two most common organisms spread that way in low-income countries, causing moderate-to-severe diarrhea. Cholera, typhoid, and salmonella also enter the digestive system in this manner.

Living organisms such as fleas, mites, and ticks—called vectors—can also transmit disease. The most common vector for human infection is the mosquito, which transmits malaria, West Nile virus, Chikungunya, dengue fever, yellow fever, and Zika. Other vector-borne diseases are plague (fleas) and Lyme disease (ticks). Helminths are parasitic worms that produce a high burden of disease in low-income countries and are most commonly transmitted through ingestion of contaminated soil or vegetables, drinking water, and raw or undercooked meat. The most serious helminth diseases are schistosomiasis, transmitted by snails living in contaminated water, and soil-transmitted *helminthiasis[https://en.wikipedia.org/wiki/Soil-transmitted_helminthiasis]*, roundworm, hookworm, and whipworm.

The Epidemiological Model of Disease Transmission

The standard approach to economic evaluations of healthcare technology and interventions rarely works for infectious diseases (Brisson & Edmunds, 2003). Economic evaluations inform policymakers on welfare-maximizing policy interventions and technologies. A randomized or quasi-randomized research design is used to compare the benefits and costs of a group of individuals treated using an intervention with a group receiving the current standard of care. Estimates of costs and benefits may be projected into the future, and this is usually done by modeling closed cohorts, i.e., groups of individuals that are followed until a specific point in time or until they have all died. In the case of infectious disease, however, it is necessary to project the benefits and costs not only for the treated group but also for those groups not directly treated but indirectly affected via positive or negative externalities, now and in future periods. The closed cohort approach needs to be replaced by a dynamic model that can model the externalities.

The starting point for evaluating policy interventions for infectious diseases is the classical dynamic transmission model from mathematical epidemiology (Anderson, May, and Anderson, 1992; Keeling & Rohani, 2008). In a basic compartmental model of person-to-person transmission, individuals in the population are assigned to different subgroups or compartments, each representing a specific stage of the epidemic. The transition rates from one class to another are mathematically expressed as derivatives, hence the model is formulated using differential equations. In each time period, a group of susceptible individuals, S, is uninfected but at risk of getting infected with a certain probability that is endogenous, the "force of infection." It depends upon the rate of contact between S and infected (and infectious) individuals, I, the transmission probability at each contact between S and I, and the prevalence of the disease in the population , I/N, at each point in time, with N denoting the total population. Infected individuals may recover from the disease or die with a

certain probability in each time period. In the case of an incurable disease, most prominently HIV, the only possible outcome is (eventual) death, giving rise to the *S-I* model. Recovered individuals, *R*, may become immune to any future infection (examples are measles or mumps), which results in the *S-I-R* model. For some diseases, for example seasonal influenza, immunity wanes over time, resulting in the *S-I-R-S* model. If persons surviving the infection do not acquire immunity, they are at risk of catching the disease again and therefore re-enter *S,* resulting in the *S-I-S* model, such as for malaria or TB. Vaccinations move *S* directly into the group of *R,* but at less than 100% if it is only partially effective.

More sophisticated models can accommodate transmission dynamics involving vectors, statistical uncertainty, changes in *N* by allowing for births, natural deaths or migration, and more complex natural histories for specific diseases, for example, incubation and latent periods, asymptomatic infection, infectious period with multiple stages or treatment that is associated with reduced transmission risk, and virus-strain specific immunity. An infectious disease is in an *endemic[https://en.wikipedia.org/wiki/Endemic_(epidemiology)]* steady state when it is sustained in a population without external inputs and when, on average, each infected person is infecting one other person. Any less, and the disease will burn out. Any more, and the number of infected increases exponentially up to a maximum incidence, when many individuals in the population have either become infected, died, or recovered and acquired immunity, and there are not many susceptibles remaining. If the proportion of *N* that is immune exceeds a critical unique threshold called "herd immunity," then the disease can no longer persist in the population. The ultimate goal of prevention interventions is to achieve herd immunity. The main objective of modeling is to predict the dynamics of the spread of the disease over time and space, specifically to project the number of people that will become infected and that will die, under alternative policy interventions. The standard transmission model assumes that the behavior of individuals with respect to prevention and treatment

choices is fixed—or exogenous—over the course of an epidemic and that individuals either behave in the same way or can be allocated into a small number of groups displaying different behavior (e.g., high- and low-risk groups). This assumption is unrealistic to economists.

Self-Protective Behavior of Uninfected Individuals

To an economist, all individuals make active choices about preventing infection. They may get vaccinated (flu, measles, yellow fever), abstain from sex, choose uninfected sexual partners or use condoms (sexually transmitted infections), avoid being in confined spaces with others (TB), install sanitation (diarrhea), wear protective clothing (Ebola), boil drinking water (cholera), use mosquito repellent (dengue), or sleep under insecticide-treated bednets (malaria). In making those preventive choices, individuals trade off the costs of prevention against the benefits of not getting the disease in future. Costs and benefits can be both monetary and non-monetary, and if they occur in the future, individuals discount them to the present day. Discount rates vary across individuals with different time preferences, and they can be time-inconsistent (e.g., a trade-off made between days 1 and 2 is different from one made between days 30 and 31). In order to predict the benefit of prevention, individuals must estimate the expected cost of disease and their personal risk of getting sick. Because the risk of getting infected is uncertain in most situations, the prevention decisions are affected by individuals' risk preferences, with risk-averse individuals more likely to demand prevention. The trade-offs underlying preventive choices are not generic to infectious diseases. What is unique in comparison to non-communicable diseases is the fact that the risk of getting a disease is directly related to how many other individuals have the disease and that the preventive choice made by one individual impacts on the risk of others getting the disease.

Prevalence-Elastic Demand for Prevention: The Theory

In its basic form, an epidemiological model assumes that the force of infection is an increasing function of prevalence. The larger is I/N (the prevalence of the disease in the population), the larger the number of susceptibles who become infected in the next period. Contact rate and transmission probability are assumed constant. Most epidemiological models assume that all individuals behave in the same way or that they belong in a few groups with the same behavior, for example, high-risk and low-risk individuals. Some started to challenge this assumption with the proposition that susceptibles display individual-specific risk-compensating behavior. This implies that an increase in the prevalence of infectious disease I/N induces an increase in self-protective behavior of individuals, defined as "prevalence-elastic demand for prevention" (Geoffard & Philipson, 1996, 1997; Philipson & Posner, 1993). As I/N increases, susceptible individuals face a greater risk of infection and hence increase their demand for prevention. Depending on the strength of this effect, the force of infection may be a decreasing function of $I/N,$ contrary to epidemiological model predictions. This would occur through reductions in contact rate and/or transmission probability or an increase in prevention efforts (e.g., vaccination rates) that would move S into R at a higher rate.

Prevalence-elastic demand or risk compensation implies that behavior is endogenous to the disease dynamics. This may have important implications for the spread of epidemics and optimal policies (Philipson, 2000): first, the growth of infectious disease is self-limiting not only because the group of susceptibles depletes but also because it induces preventive behavior among susceptibles; second, since the decline of a disease discourages prevention, initially successful public health efforts make it progressively harder to eradicate infectious diseases, with quite profound implications for optimal control of epidemics. For example, municipal efforts to reduce the amount of stagnant water for mosquito habitats decimates

vector populations and malaria prevalence, but the resulting reduced risk of infection could make individuals less inclined to sleep under insecticide-treated bednets (ITNs). The long-term net impact of the municipal intervention would be much smaller than expected.

Prevalence-elastic demand for prevention can lead to a "vaccination game" (Barrett, 2003; Bauch, Galvani, & Earn, 2003): when vaccine coverage level is sufficiently high to achieve herd immunity, a disease can be eradicated without vaccinating everyone. This positive externality of vaccination provides a completely self-interested individual with less incentive to vaccinate as coverage increases, since non-vaccinators can gain the benefits of herd immunity without the cost of vaccination. As such, indirect protection by vaccination generates discrepancies between individual and group interests. The behavioral response is stronger the higher the—real or perceived—costs of vaccination, all else being equal (Bauch & Earn, 2004). The magnitude of the elasticity predicts the extent of risk compensating behavior. If demand is elastic (i.e., the elasticity is greater than one), a percentage decline in prevalence will lead to a greater percentage decline in prevention efforts of individuals. Consequently, it will become progressively more expensive to achieve further decreases in prevalence and the disease may never be eradicated. Alternatively, an inelastic prevention demand (i.e., the elasticity is between zero and one), implies that as infection risk declines, individuals will reduce their preventive behaviors less than proportionally, allowing for the possibility that prevention may lead to the eradication of the disease. Precise estimates of the elasticity's magnitude are therefore crucial to predict the effect of individual choices and the need for government intervention.

There is growing recognition of the importance of incorporating endogenous behavior change into transmission models (Ferguson, 2007). A number of theoretical models ("econ-epi models") have been developed that attempt this integration, and this exciting multidisciplinary endeavor is an ongoing area of research (Manfredi & D'Onofrio, 2013;

Verelst, Willem, & Beutels, 2016; Weston, Hauck, & Amlôt, 2018). However, it has proven difficult to generate empirical estimates of endogenous self-protective behavior. These would be needed to apply theoretical simulations to the real world and develop valid models for predictions and informing policy. At least two problems plague empirical studies of the behavioral response to changes in prevalence: misreporting of preventive behavior and reverse causality between the demand for prevention and prevalence. Self-protective behavior is difficult to measure and often based on self-report, which usually overstates preventive behavior. There is evidence that people underreport their risky sexual behavior to conform with social norms (Minnis et al., 2009). In the area of HIV/AIDS, studies have relied on condom use (Ahituv et al., 1996; Anglewicz & Clark, 2013; Godlonton & Thornton, 2013; Oster, 2012; Young, 2007), rate of partner change (Auld, 2006; Godlonton & Thornton, 2013; Lakdawalla, Sood, & Goldman, 2006; Oster, 2012), and measures that are imperfect proxies for unprotected sex, including contraceptive use (Magadi & Agwanda, 2010), abortion rate (Medoff, 2012), and fertility (Boucekkine, Desbordes, & Latzer, 2009; Young, 2007). Recent studies attempt to correct for underreporting of risky sexual behavior by using common sexually transmitted infections (STIs) as biomarkers in combination with statistical adjustments (Norwood, Hughes, & Amico, 2016), but this research has yet to be adopted by empirical studies of risk-compensating behavior. The second problem that is affecting econometric modeling of the demand for prevention is reverse causality. It is tricky to estimate the impact of prevalence on preventive behavior if the same behavior in previous periods has contributed to spread of the disease. This is likely to bias estimates upwards unless appropriate research designs or econometric methods are employed (Oster, 2012).

Prevalence-Elastic Demand for Prevention: The Evidence

Empirical studies are dominated by HIV/AIDS. Of 10 empirical studies on HIV, 7 found evidence that behavior is reactive to prevalence or objective infection risk (Guillon & Thuilliez, 2015). However, only four studies produced estimates of prevalence elasticity that could be directly used in dynamic transmission models. The others proxied for prevalence, risk behavior, or both. A 1% increase in AIDS prevalence implies an 8.48% increase in the probability of condom use among young heterosexual men and women, clearly an elastic response (Ahituv et al., 1996). Among men who have sex with men in San Francisco, however, it results in a 0.46% decrease—an inelastic response—in the rate of partner change but almost no change in the propensity to participate in sexual activities (Auld, 2006). Elasticity is higher for groups classified as "low"-risk activity types (Auld, 2006). Among women, a 1% prevalence increase implies a 0.7% decrease in abortion rates in state-level data (Medoff, 2012). Lakdawalla et al. (2006) addressed the issue of reverse causality between demand for prevention and prevalence with instrumental variable estimation. They used the number of infected individuals under treatment as a proxy for HIV prevalence (and thus infection risk) and instrumented it by the generosity of Medicaid eligibility rules. This rests on the assumption that treatment increases the number of infected (and infectious!) individuals on treatment (at the time, the potential preventive effect of Anti-retroviral therapy [ART] was not known). Lakdawalla et al. (2006) found that only low-risk uninfected individuals seemed to react to the increase in HIV prevalence—and alleged infection risk—by reducing the number of their sex partners, but the effect was very small.

A study on couples in Malawi found that a partner's HIV test result (as an objective measure of risk) did not influence subsequent condom use for either men or women, but the results are potentially affected by non-random selection into testing (Anglewicz & Clark, 2013). A study on Kenyan women found no statistically significant association between women's fertility and local HIV prevalence (Magadi & Agwanda, 2010). Two country-level

studies confirm this finding for sub-Saharan countries (Boucekkine et al., 2009; Young, 2007). A study on men and women in Malawi proxied local HIV prevalence with the percentage of community members who learned their HIV status after a testing campaign (Godlonton & Thornton, 2013). The rationale is that people tend to overestimate the prevalence of HIV and would, following a testing campaign in their village, revise these estimates downward. This decrease in risk perception would then lead to an increase in risk behaviors. The study found evidence for this: after 10% more community members had learned their HIV results, individuals two years later were 38% points less likely to have used a condom with the current or last three partners. One study addressed the problem of reverse causality using instrumental variables regression with distance to Congo (the viral origin of HIV) as instrument (Oster, 2012). A doubling of HIV prevalence leads to a 1.8% decline in the chance of having multiple partners and an approximate 2% decline in having multiple partners without condom use for married individuals. The behavioral response is lower among unmarried individuals, a counterintuitive result.

The findings may not be applicable to other diseases or to subgroups of the population with systematically different risk behavior. In many regions of sub-Saharan Africa, the HIV epidemic is accelerated by commercial sex work, but the private market may dampen the self-limiting effect of prevalence-elastic demand. Consistent with the theory, there is evidence that in areas with a low prevalence of STIs, the premium for unprotected sex is small (Arunachalam & Shah, 2013). However, the premium for non-condom sex increases with STI prevalence. The private market compensates sex workers for the increase in risk, and the number of risky sex acts do not decrease as strongly as economic theory would predict. This would imply that commercial sex work, a major driver of STIs in many countries, would work against the dampening effect of prevalence-elastic demand. For other diseases, an early U.S. study found that prevalence of measles in the respondent's state of

residence reduces the age in months at which first measles vaccination occurs (Philipson, 1996). Malaria prevalence has a positive effect on bednet usage across nine countries in sub-Saharan Africa, but it is inelastic with values ranging from 0.42 for adult women to 0.59 for older children (Picone, Kibler, & Apouey, 2017). Adherence to therapy for asymptomatic patients with latent TB infection in the United States was found to be prevalence-elastic (Fluegge, 2015). Vaccination against human papilloma virus did not respond to changes in cervical cancer prevalence, possibly because of lack of awareness about the disease and its causal link to cervical cancer (Staben, 2016).

Other Determinants of Self-Protective Behavior

Many other factors affect the demand for prevention, most of which are not generic to infectious disease. In a review, Dupas (2011) lists financial constraints, poor delivery of preventive services, lack of information, or more subtly wrong information provided to the wrong target groups, lack of education, social learning or peer effects, and present bias or competing mortality risks whereby people who expect to die young from other conditions have weak incentives for prevention. Related to this point, the availability of effective treatment reduces the demand for prevention because it lowers the expected costs of being sick. An example is the introduction of highly active antiretroviral therapy for the treatment of HIV/AIDS, which reduced the implicit price of risky sex (Chan, Hamilton, & Papageorge, 2016; Mechoulan, 2007). A new technology or intervention that reduces transmission risk may decrease prevention efforts among susceptibles. This risk compensation is problematic if transmission risk is not reduced to zero or if the behavioral adjustment overcompensates. There is evidence that pessimistic expectations about the future of the epidemic could induce more risky behavior in the present (Auld, 2003). If future infection risk increases, then there is less "reward" for preventive behavior today. Fatalism may even result in counterintuitive

behavior change. Increased HIV infection risk creates incentives for people with low sexual activity to reduce their activity but may make high-activity people fatalistic, leading them to reduce their activity only slightly, or actually increase it (Kremer, 1996). This is because highly sexually active people are so likely to be infected by their inframarginal partners that their marginal probability of infection from an additional partner changes little. If high-activity people reduce their activity by a smaller proportion than low-activity people, the composition of the pool of available partners will worsen, creating positive feedbacks and even increasing transmission. Lastly, beliefs about one's disease status may determine preventive choices. For example, people with higher beliefs of being HIV-negative might be more likely to have multiple partners, although that has been contested (for a review, see Gong, 2015).

In summary, theoretical economic models show that demand for prevention is prevalence-elastic. Relatively few empirical studies have studied this phenomenon. Investigations would find it difficult to convincingly overcome the methodological and practical difficulties that plague this research question. If studies find an association as predicted, most estimate an inelastic response. However, some studies, particularly for low-income countries, find no association. We now move from individuals' preventive choices about their own infection risk to preventive choices affecting others' infection risk.

Preventing Infection in Others: Are Individuals Altruistic?

Many public health interventions are based on the assumption that individuals are altruistic. For example, interventions that aim to improve coverage and uptake of HIV testing are at least in part motivated by the assumption that upon learning one's status, infective individuals

will make efforts to prevent transmission of the disease to susceptible individuals. The infective individuals bear the costs of their behavior, but the benefits are enjoyed by somebody else. Classical economic utility theory considers such altruistic behavior as irrational. The standard assumption is that individuals do not consider in their utility function the benefits of their preventive choices to others, giving rise to negative externalities (Stiglitz, 1988). This explains why prevention is low if its provision is left to private markets.

Although susceptible individuals will demand prevention even if they are not altruistic, they only consider their private benefits and not the social benefit, which is higher. It is difficult to estimate the difference between private and social benefit because it is highly dependent on the characteristics of the disease and the intervention (Boulier, Datta, & Goldfarb, 2007). This is probably why the issue of altruism among susceptibles is rarely analyzed in economic studies (an exception is Shim, Chapman, Townsend, & Galvani, 2012). Most empirical studies on altruism among susceptibles have analyzed vaccination decisions against seasonal and pandemic influenza among healthcare professionals (Schmid, Rauber, Betsch, Lidolt, & Denker, 2017). When healthcare professionals lacked the belief that getting vaccinated protects patients or relatives, the perceived risk for others due to the disease was low, or when the perceived risk of transmission was low, vaccine uptake was low. Pregnant mothers were found to be altruistic toward their unborn child. However, there was no evidence of altruism among the elderly, chronically ill patients, or parents of children under 5 years of age.

The question whether individuals have a desire to prevent infection in others comes into stark view when analyzing the preventive choices of already infected (and infective) individuals. Their decisions solely concern others. To reduce the risk of transmitting the disease, infective individuals may decide to stay at home to reduce contact with others, choose sexual partners who are also HIV positive, use a condom, or sleep under a bednet.

These actions will reduce the force of infection by reducing transmission risk, contact rate, or both, and therefore lower the rate at which susceptibles become infected in each period. The following section reviews empirical evidence on altruism among infective individuals.

Prevention Decisions of Infected Individuals: The Power of a Test

Altruism in prevention decisions among infected individuals requires that individuals be able to make decisions and that they be aware of their infectious status. Often this is not the case, as certain infectious diseases are asymptomatic for some individuals or at certain stages of the disease (e.g., dengue and HIV/AIDS). Empirical studies on altruism among infected individuals usually focus on behavior change after learning about one's infectious status via a diagnostic test. Many have found that people who learn they are HIV-positive reduce behavior that puts others at risk, especially in longstanding partnerships, confirming they behave altruistically (Gersovitz, 2011).

This finding is put into question by Boozer and Philipson (2000), who established the importance of prior belief about one's disease status when investigating the impact of test results on behaviors. Only individuals who believed they were HIV negative reduced their risky behavior upon learning about their HIV-positive status, whereas individuals who believed they were infected with HIV—and received confirmation with their test result—did not change their behavior. However, empirical analyses of the impact of testing on preventive behavior are complicated by selection bias; unobserved characteristics are associated with an individual's propensity both to get tested and to change behavior once learning about his or her status. Studies have attempted to overcome this using randomized study designs or econometric modeling. HIV-positive individuals who had just learned about their status were three times more likely to purchase condoms 2 months later than HIV-positive individuals who had not learned about their status, but the effect was small as they purchased only two

additional condoms on average (Thornton, 2008). A decrease in the perceived probability of being HIV positive from 10% to 0% increased the probability of engaging in extramarital affairs from 8.3% to 14.1% (Paula, Shapira, & Todd, 2014). These studies relied on self-report, which may have biased their results. Two randomized studies that used STIs as markers of risky sex confirmed that individuals who learned they were HIV positive *increased* their risky sexual behavior, an effect that was even stronger among individuals surprised by a positive test result (Baird, Gong, McIntosh, & Özler, 2014; Gong, 2015). This seems to confirm the findings by Boozer and Philipson (2000) and suggests that the benefits of risky sex are high and altruism toward sexual partners low for some infected individuals in the countries where the studies were conducted (Malawi, Kenya, and Tanzania).

In their most basic form, epidemiological models of STIs assume that infected and uninfected individuals match at random. The economic theory of matching markets, however, predicts that low-risk individuals are more likely to match with other low-risk individuals, and high-risk individuals with other high-risk individuals. The complementarity in health status that generates assortative matching stems from the fact that low-risk individuals have more to gain by the choice of low-risk partners than do high-risk individuals. Assortative matching has the important implication that disease growth is slower than in the random matching case considered by epidemiological analysis. Dow and Philipson (1996) analyzed data from San Francisco and found that, on average, HIV-positive individuals are more than twice as likely as HIV-negative individuals to have positive partners, and the estimated incidence reduction implied by such matching is about one-third.

Treatment Decisions of Infected Individuals

Infected individuals may impact the infection risk of others by choosing to get treatment, with resulting positive or negative externalities. The direction and size of the externalities are

highly dependent on the clinical features of the disease and the treatment. Treatment may confer positive externalities if it decreases the force of infection. This can occur because of an increase in recovery rate, i.e., a decrease in the pool of infected *I/N,* or a decrease in transmission risk if it renders infected individuals less infectious. Treatment may not lead to recovery but increase the longevity of infected individuals. It then confers a negative externality by increasing *I/N* without a reduction in the transmission risk. Treatment creates positive benefits for the infected individual but generates potential costs for susceptibles who might be infected by treated individuals that remain infectious. A few studies speculated whether improved access and efficacy of ART was linked to the increase in HIV incidence at the same time (Lakdawalla et al., 2006; Skåtun, 2003). These findings may be superseded by the discovery that ART reduces transmission risk. However, it is unclear as yet whether this actually applies under real-world conditions where treatment adherence is imperfect. If it does, it would make HIV treatment a means of prevention.

Antimicrobial resistance is a negative treatment externality associated with the use of antimicrobials in treating or preventing infections in humans and animals. The current stock of antimicrobials can be interpreted as a depletable natural resource. The effects of antimicrobial use in terms of resistance are unlikely to be felt directly by either the consumer or the supplier of treatment (and hence they have no incentive to reduce the use of antimicrobials) but will affect the overall welfare of the community. Patients infected with a resistant microorganism are less likely to recover from infection with the first antimicrobial used in treatment and have a greater likelihood of premature death. Patients may require both extra investigations and extra treatment (usually more expensive), and for some patients a cascade of antimicrobial drugs will be tried before one is successful in eradicating the infection. This may result both in longer hospital stays and longer periods of time away from work. The costs of AMR in the United States have been estimated at $55 billion ($20 billion

in health service costs and $35 billion in lost productivity) per year overall (Smith & Coast, 2012). However, the costs of resistance could be much higher than these estimates suggest (Smith & Coast, 2013). Many antibiotics are given as prophylaxis, and infection after standard procedures (with associated morbidity and mortality) will be much more common in a world without effective antibiotics, a cost rarely considered in current estimates. Moreover, anticipation of untreatable infections could change treatment decisions; for example, clinicians may be less inclined to choose surgical treatment, with associated adverse outcomes for patients. Such adaptive behavior was found in German intensive care units; demand for reserve antibiotics grew in expectation of increasing resistance against the first-line therapy (Heister, Hagist, & Kaier, 2017).

In summary, economic theory predicts that individuals' choices regarding prevention generate externalities. This would mean that individuals are not altruistic because they do not—or do not sufficiently—take into account the effects of their actions on the infection risk or treatment outcomes of others, either negative or positive. In the case of flu vaccination, there is evidence that some individuals are altruistic and do take others into account, but this depends on circumstances. Evidence on whether infected individuals are altruistic is conflicting, and there is even reason to believe that they may reduce preventive efforts on learning about their disease status. Treatment may generate positive externalities if it reduces transmission risks or the length of time individuals are infected but negative externalities if it leaves infected individuals infectious. Antimicrobial resistance is a negative externality imposed by infected individuals or food producers of the present onto infected individuals of the future.

How Big Are the Externalities?

The indirect costs of infectious diseases—and hence the indirect benefits of preventing them—can be substantial. For example, vaccinating children against influenza reduces serious influenza-related complications in older people; use of ITNs in a village reduces malaria morbidity and mortality in villages nearby that do not use them; improving sanitation in a households leads to reduction in diarrheal disease in children in neighboring households without proper sanitation. Externalities provide justification for government intervention to align the private benefits with the social benefits of decisions. To evaluate the societal value of those policy interventions, the costs and benefits of interventions for directly and indirectly treated population groups need to be predicted. This implies estimating the extent of the externality. Otherwise, the benefit of a policy could be understated, which may tip the balance toward not implementing it. Theoretical and simulation studies predict that the marginal externality of a prevention intervention can be greater than one case of illness prevented among the non-targeted, therefore omission from policy analyses may imply serious biases (Boulier et al., 2007; Brisson & Edmunds, 2003). However, the externalities are difficult to quantify because they vary with the epidemiological characteristics of the disease, age-related patterns of transmission, prevention efficacy, characteristics of the affected populations, and the number already reached by prevention. They often need to be projected into the future with a dynamic transmission model. Randomized study designs are costly, because the presence of externalities means that evaluating treatment effects by randomizing at the individual level is flawed. For example, when evaluating the impact of deworming programs against intestinal helminths in school children, it would be erroneous to randomize children within one school into treatment and control groups. This would potentially doubly underestimate the benefits of a treatment compared against standard of care: first, by missing externality benefits to the control group from reduced disease transmission, and second, by overstating outcomes for the control group. Instead, whole

schools, or even schools within one community, must be randomized into either treatment or control group in cluster-randomized controlled trials.

Most evidence on the presence and extent of externalities comes from public health studies, which often do not use the term externalities but spillovers or community-wide effects (for a systematic review, see Benjamin-Chung et al., 2015). The contribution of economic studies is the focus on externalities with respect to schooling, economic outcomes or saved healthcare costs, the explicit integration of externalities into cost-effectiveness analyses, or evaluation of externalities with quasi-randomized research designs such as difference-in-difference analyses. Miguel and Kremer (2004) used a cluster-randomized design to evaluate the impact of a deworming program and found that deworming substantially improved health and school participation among untreated children in both treatment schools and neighboring schools and that these externalities are large enough to justify fully subsidizing mass deworming programs. However, this finding was contested by a subsequent study (Aiken, Davey, Hargreaves, & Hayes, 2015; Davey, Aiken, Hayes, & Hargreaves, 2015). Sanitation infrastructure improvements on U.S. Indian reservations were quite cost-effective because they reduced infectious respiratory disease among Native American infants and white infants living nearby (Watson, 2006). A study from rural India estimated that three-fourth of benefits of improved sanitation are due to externalities (Andrés, Briceño, Chase, & Echenique, 2017). Coverage expansion of seasonal influenza vaccination led to substantial external benefits to older adults in Ontario, Canada, and was cost saving when considering hospitalization costs and productivity losses (Ward, 2014). Kaier and Frank (2010) and Kaier (2012) quantified the negative externality associated with hospital-acquired infections caused by methicillin-resistant *Staphylococcus aureus* resulting from the use of second- and third-generation antibiotics in monetary terms and estimated the positive externality from hand disinfection. Cohen estimates the cost-effectiveness of subsidizing

ITNs and takes into account the benefits to non-users that live in the vicinity of users of ITNs (Cohen & Dupas, 2010). Bhattacharya, Dupas, and Kanaya (2013) found that when the indirect benefits of ITN subsidies to neighbors of users are ignored in cost-benefit analyses, ITN use is overestimated at lower and underestimated at higher subsidy rates. Cook et al. (2009) show that if the optimal Pigouvian subsidy for Cholera vaccine is unknown, selling them at full marginal cost may, under some circumstances, be a preferable second-best option to providing them for free.

Negative externalities may arise if interventions are substitutes. As discussed above, risk compensation may render preventive interventions ineffective. This occurs when individuals' demand for a prevention intervention decreases when it is crowded out by another intervention, or when individuals adapt their preventive behavior to new information. For example, individuals may risk compensate when learning about changes in disease prevalence, public health interventions going on in their community that may impact on prevalence, or by getting new information on ones' own risk of infection. This is problematic if the new prevention method is only partially effective, a scenario analyzed for the association between a partially effective vaccine against HIV and risky sexual behavior in the 1990s (Anderson & Hanson, 2005; Blower & MCLean, 1994). Public interventions may crowd out private prevention efforts. For example, the substitutability of clean water and sanitation may cause water supply improvements to actually worsen sanitary conditions. Households find it costly to build and maintain latrines, handle waste properly, and remove the waste left by children and livestock. Clean water may allow recipients to shirk in terms of sanitary behavior without adverse health impacts for themselves but result in adverse effects on other households in the community that do not have access to clean water. With greater municipal provision of piped water, public defecation became a severe problem in the Philippines (Bennett, 2012). In the case of malaria, on the other hand, individuals exposed to

prophylaxis via indoor residual spraying continued sleeping under a bednet (Picone et al., 2017). In the case of STIs, circumcised men (who have a reduced infection risk for STIs) were not more likely to engage in risky sex (Godlonton, Munthali, & Thornton, 2016; Wilson, Xiong, & Mattson, 2014). There may be another negative externality associated with prevention if supply of the prevention product (e.g., a vaccine) is limited and individuals who need prevention most (i.e., individuals with high costs of infection) do not get it because supply is depleted by individuals with a low cost of infection, but there is no empirical evidence (Arifoğlu, Deo, & Iravani, 2012).

Policy Interventions

Policymakers need to strike the optimal balance between prevention and treatment for different types of infectious disease. Under constrained budgets, there is a trade-off between prevention and treatment, with prevention increasing in attractiveness as the costs of treatment increases (Berthélemy, Gaudart, & Thuilliez, 2015). For some diseases, treatment is also prevention if it increases the likelihood of recovery (malaria, TB) or reduces transmission risk (HIV). A third consideration is the allocation of resources to diagnostic testing. If diagnostic tests are expensive but treatment simple, cheap, and associated with few side effects, then treating everybody in a high-prevalence community may be the most cost-effective strategy—for example, mass deworming in schools. In the following section I provide a brief and somewhat selective review of the empirical evidence on the impact of government interventions, focusing specifically on economic contributions. There are a number of detailed reviews on policy interventions (Bishai & Adam, 2006; Hall, 2011; Kremer & Glennerster, 2011) and on the trade-off between prevention and treatment (Canning, 2006).

Control of Infectious Disease

Subsidies

The classical intervention to align private and marginal demand for prevention are Pigouvian subsidies; they can be targeted toward the supply or the demand side. Both will result in an increase of preventive products on the market in comparison to a situation without government intervention. Prevention is often not a binary choice, but there are variations in the degree of preventive effort (e.g., proportion of sex acts for which condoms are used, how often hands washed or water boiled, how often individuals are protected with insect spray, or sleep under bednets). The subsidy needs to be adapted to the type of prevention method. Many countries fully subsidize vaccinations against childhood diseases because of the high indirect benefits to the unvaccinated. HIV and malaria prevention and treatment interventions are often at least partially subsidized, with international donors covering a major share of the costs in the poorest countries.

This can create problems for governments when donors reduce support; empirical studies have demonstrated that reducing subsidies even by relatively small amounts can lead to dramatic declines in demand for health-protective technologies among the poor (Ashraf, Berry, & Shapiro, 2010; Cohen & Dupas, 2010; Kremer & Miguel, 2007). However, for experience goods (products which's characteristics cannot be ascertained in advance but only upon consumption) this may actually not apply; Dupas et al. (2014) found that one-off subsidies did boost long-run adoption of an improved antimalarial bednet. Subsidies for artemisinin-based combination therapies for malaria increased purchases from private sector outlets and treatment coverage of children reporting a fever in some but not all of 166 malaria-endemic countries (Fiore, 2017).

However, broad-brush subsidies may generate little benefit if they fail to support the right mix of interventions. Very high subsidies for over-the-counter antimalarials dramatically increased access in Kenya, but nearly half of subsidized pills went to patients without malaria (Cohen, Dupas, & Schaner, 2015). It would be preferable to reduce the subsidy level and introduce rapid over-the-counter malaria tests, in particular if their use is already well established. In situations where prevention interventions involve beneficiaries' cost-sharing, uptake may be enhanced by the introduction of contracts that require prepayment of uptake costs, as demonstrated for the cost of periodic retreatment of bednets with insecticide (Tarozzi, Mahajan, Yoong, & Balckburn, 2009).

Conditional Cash Transfers

Conditional cash transfers (CCTs) are monetary payments that reward individuals for certain behaviors and have the objective of increasing demand for prevention or treatment (for a review, see Lagarde, Haines, & Palmer, 2007). Many of these programs target young women and reward them for staying in or returning to school, with the expectation that this reduces their likelihood of getting married early or engaging in sex work. Empirical evidence on their efficacy is conflicting and seems dependent on context and disease. Education subsidies did reduce adolescent girls' rates of dropout, pregnancy, and marriage but not STIs (Duflo, Dupas, & Kremer, 2015). In contrast, Baird, Garfein, McIntosh, and Özler (2012) found that cash transfers reduced the incidence of HIV and herpes simplex virus 2 among young women, but there was no difference between unconditional transfers and those that were conditional on school attendance. Teenage pregnancy and marriage rates were lower with unconditional than conditional transfers, which was due to the impact on girls who had dropped out of school (Baird, McIntosh, & Özler, 2011). CCTs for staying HIV negative for one year had no effect in Malawi, although some rewards were as high as 4 months of wages

(Kohler & Thornton, 2012). Smaller payments made more frequently and closer to the behavior being observed are more effective than larger payments in the future. Relatively small CCTs induced substantial increases in uptake of routine health checkups for children among non-farming households and households living farther away from clinics (Fink & Rockers, 2017). CCTs led to large increases in coverage from below 90% to over 95% for some vaccines in Nicaragua (Barham & Maluccio, 2009).

Testing

Testing serves two purposes: advancing access to treatment and compelling infectious individuals to prevent onward transmission, i.e., demand prevention. This means that in the absence of (affordable) treatment, testing has only indirect benefits and people have little private incentive to test unless they are altruistic and concerned about infecting susceptible individuals. As discussed above, the empirical evidence is inconclusive, although the assumption of altruism in infected individuals is an important motivation behind public testing campaigns. The situation could be complicated, because aggregate effects of a testing program may be a misleading indicator of the behavioral responsive of the average individual to the information intervention. Infected individuals who learned about their HIV-positive status only reduced risky sexual behavior if they had believed before that they were HIV negative (Boozer & Philipson, 2000). The same applied to susceptibles who learned they were HIV negative—the ones surprised by the result increased risky sexual behavior. This means the behavioral responses to testing may be asymmetric. The study found that low-risk HIV-positive respondents (i.e., those who believed they were at low risk for HIV before testing but learned they were HIV positive) decreased their number of sexual contacts by 50%, whereas high-risk HIV-negative respondents increased their sexual contacts by 20%. Depending on the distribution of HIV-positive respondents across the two subgroups, the

behavioral responses may be offset with the result that the publicly subsidized testing program has very little impact on disease transmission. No studies have shown population-level reductions in the incidence of HIV or other STIs as a result of testing.

Other Interventions

Information and education campaigns are used to educate individuals about personal and local risk factors and the benefits of prevention and treatment of infectious diseases (Kremer & Glennerster, 2011). For example, online sexual health education courses in Colombia led to a reduction in STI incidence in teenagers, but there were no external benefits to untreated individuals (Chong, Gonzalez-Navarro, Karlan, & Valdivia, 2013). Community-based provision of prevention and treatment is meant to improve access by reducing travel and waiting times, with many examples ranging from TB dispensaries in the 1940s (Hansen, Jensen, & Madsen, 2017) to community health workers nowadays. Varying the framing of the perceived benefits of malaria prevention did not affect their take-up (Dupas, 2009). Education messages may have counterintuitive effects. For example, corrective information significantly reduced belief in the myth that the flu vaccine can give you the flu as well as concerns about its safety. However, the correction also significantly reduced intent to vaccinate among respondents with high levels of concern about vaccine side effects—a response that was not observed among those with low levels of concern (Nyhan & Reifler, 2015; Tarozzi et al., 2009)

For vector-borne diseases, prevention can be undertaken not only at the individual or household level but also on the community and even regional level. Environmental interventions are targeted at the mosquito larvae or adult mosquitoes, with the aim of decimating vector populations. This may entail small- or large-scale environmental management that reduces the availability of vector habitat by the removal of stagnant water

or improving the design of water storage vessels. Another option is biological control that uses natural predators to decimate vector populations. Space spray application of insecticide is used in emergency situations when a massive rapid destruction of the adult vector population is the objective, but its long-term efficacy is disputed. Careful targeting of environmental interventions is important. Castillo-Riquelme et al. (2008) investigated the geographic variation in control policies for Chagas' disease to identify the communities where implementation is cost-effective.

Many water-borne diseases are spread via groundwater contaminated with fecal pathogens from pit latrines. Helminth infections are most often transmitted via ingestion of contaminated feces through water or food, directly through the soles of the feet, or via swimming or wading in contaminated water. Long-term public health solutions are improving the quality of the water supply, sanitation, and hygiene (WASH). Walker et al. (2011) investigated the cost-effectiveness of a package of interventions to reduce diarrhea. With universal coverage, nearly 5 million diarrheal deaths could be averted for an additional cost of $12.5 billion invested across 68 priority countries for individual-level prevention and treatment interventions and an additional $84.8 billion would be required for the addition of water and sanitation interventions.

Legislative measures, and in a weaker form guidelines, are also used as public health interventions and can be temporary policies that aim to contain major epidemics. This includes legislation that aims at social distancing and restrictions on the free movement of people or goods such as quarantines, curfews, school closures, or bans on travel and the import or export of certain products such as meat from animals suspected of carrying diseases. The efficacy of some of these measures has been questioned. Knowingly infecting others with HIV is punishable by law in most countries, but there have not been many prosecutions, and in some high-endemic countries laws for the criminalization of intentional

transmission have been either weak or non-existent. Some countries require proof of yellow fever vaccination before they will issue a visa.

Policies Addressing Anti-Microbial Resistance

WHO (2017a) considers the current clinical pipeline of novel antibiotics that are in development as insufficient to mitigate the threat of antimicrobial resistance. The optimal policy response is a combination of stewardship that promotes the responsible use of existing antibiotics in humans and animals and subsidies to incentivize the pharmaceutical industry to invest in research and development (R&D) of new antimicrobials (for a review, see Renwick, Brogan, & Mossialos, 2016). Unfortunately, these two policies work against each other. Pharmaceutical companies base their R&D investment decisions on potential sales volume within the product's life cycle, but effective stewardship aims to dampen sales volume. The health system, for good public health reasons, will reserve novel antibiotics for the still relatively rare cases in which existing antibiotics are ineffective and oppose widespread distribution in primary care. Current R&D does not consider the benefits to future generations of having effective antimicrobials, which calls for government interventions that increase the returns to the pharmaceutical companies (Outterson et al., 2016). Subsidies can also be used to promote responsible use of antibiotics; for example, monetary incentives for healthcare providers may help to reduce antibiotic prescribing (Ellegård, Dietrichson, & Anell, 2017).

Global Eradication: Is It Worth It?

The discussed policies have achieved, at best, elimination of an infectious diseases in a particular country or region and as a second-best control, whereby incidence of disease is reduced below a critical level but not to zero. The global eradication of an infectious disease is an extreme—indeed, a singularly ambitious—policy goal. Eight attempts have been made

to date to eradicate infectious diseases: two successful programs targeting smallpox and rinderpest (an animal disease); four ongoing programs targeting poliomyelitis, yaws, dracunculiasis, and malaria; and two former programs targeting hookworm and yellow fever. The vector control campaigns of the 1940s and 1950s virtually eliminated yellow fever everywhere except Africa. When the disease subsided in the Americas, funding for mosquito control was reduced and resulted in a recovery of the mosquito populations. This may have contributed to recent localized outbreaks of yellow fever, the increase in the incidence of dengue over the past decades, and localized outbreaks of Chikungunya and Zika, all diseases transmitted by the same mosquito.

An important question is whether a country should push for and support international efforts to achieve global eradication, aim for elimination within its borders, or attempt optimal control, which involves moving into and sustaining a steady state with a positive level of infection. It is very difficult to identify the welfare-maximizing policy, and recommendations need to rely on projections of uncertain benefits far into the future. If a disease is already controlled at very high level, for example by vaccination, then a steady state with positive level of infections is maintained at comparably high costs, and just a slight increase in the vaccination rate would cause the disease to be eliminated or eradicated. Eradication would increase costs in the short run, and the marginal costs of the last prevented case are probably very high. If public vaccination policy is (partially) crowded out by market behavior under the assumption that the private demand for vaccination is prevalence-elastic, then economic theory suggests that eradication may only be achievable as time goes to infinity (Geoffard & Philipson, 1997).

The main benefit of eradication does not lie in the few additional infections averted but in making the pathogen disappear. Eradication would avoid the need ever to invest in prevention and surveillance of the disease in the future—the "eradication dividend," an

enormous economic benefit. A very high level of control is therefore unlikely to be optimal. The optimal policy will require either a low level of control or eradication (Barrett, 2007). In the case of smallpox, there is evidence that eradication was cost-saving and led to large economic benefits due to avoided vaccination costs, at least when considering only the incremental costs needed to eliminate smallpox from the remaining endemic countries at the time the decision was made to eradicate (Fenner, Henderson, Arita, Jezek, & Ladnyi, 1988). Smallpox was the ideal candidate for eradication due to its clinical characteristics. Unfortunately, the eradication of other diseases is likely to be more difficult and less attractive in benefit–cost terms. The probability that the elimination of malaria would be cost-saving over 50 years was estimated at a range from 0% to 42%, based on data from five sites (Sabot et al., 2010). The threat of bioterrorism weakens the economic case for eradication. Countries may feel the need to continue to vaccinate, even if at a relatively low level or to stockpile vaccine, and prepare for emergency distribution.

Eradication requires strong international cooperation, and it is a "game," because some countries may be willing to eliminate the disease within their borders only if assured that all others will eliminate the disease within their borders. International financing is also a game, because each country would rather free-ride than contribute (Barrett, 2013). If eradication fails, much of the money spent will have been wasted. If it succeeds, the world will reap the dividend.

Conclusions

This article reviewed the current status of empirical economic research on infectious diseases. Several important topics were beyond the scope here, for example, the impact of infectious diseases on economic development (for an overview, see Laxminarayan & Malani, 2011), the

long-term economic consequences of responding to infectious diseases that cannot be eradicated (e.g., HIV; see Haacker, 2016), infectious diseases of animals, global health security and international epidemic preparedness (Sands, Mundaca-Shah, & Dzau, 2016), and the association between infectious disease and migration, increased travel and globalization. The article also did not review the sociological and psychological literature on emotional responses, stigma, habits, group identity, peer effects, and networks, which attempts to explain individuals acting seemingly irrationally and in the interests of a group rather than their own.

The article is written with the growing number of economists—and epidemiologists— in mind who have the passion to tackle the considerable methodological and practical challenges that arise when integrating economic and epidemiological modeling of infectious diseases. A multidisciplinary approach is crucial because evaluating interventions without quantifying externalities—positive and negative—can lead to seriously biased findings. This requires modeling of disease transmission that takes account of secondary infections. The Modelling Good Research Practices Task Force-5 of the International Society for Pharmacoeconomics and Outcomes Research found that until 2011, only 11% of cost-effectiveness studies of vaccination programs had used dynamic modeling (Pitman et al., 2012). The Task Force set out best practices for designing and building cost-effectiveness analyses that use dynamic modeling. However, many issues are unresolved, for example the question how to jointly model uncertainty that arises from two sources: the parameters of the epidemiological model and the costs and benefit estimates from the economic model.

Epidemiology is not a social science and does not model the behavioral responses of individuals. Behavior creates feedback effects that can have substantial impact on the spread of epidemics. The article has shown that both theoretical and empirical economic research on human behavior in infectious diseases is underdeveloped, although it may be the single most

important factor impacting on the spread of disease and the efficacy of public health interventions (Pisani, 2010). So far, it is mainly epidemiologists who work on the integration of behavioral response into infectious disease models (Manfredi & D'Onofrio, 2013). Economists are needed to develop the underlying utility theory that would allow to incorporate individual preferences into infectious disease transmission models. Behavioral economists have a potentially rich field for applications; the presence of externalities implies that individual behavior is amplified because of the consequences it can have for others, as are the research efforts of a keen behavioral economist analyzing it!

Econometrics has at its disposal a powerful arsenal of quasi-experimental methods that can evaluate the impact of interventions that defy analysis in a randomized controlled trial because it would be too expensive, unfeasible, or unethical. For example, large-scale community-based interventions against vector-borne diseases are difficult to evaluate in a randomized setting. There is an increasing interest within the public health community in studies using observational data and methods developed by labor or education economics such as difference-in-difference analysis, instrumental variable methods, or regression discontinuity design.

There is considerable research effort invested into HIV/AIDS, malaria, and TB and some evidence that they have displaced research into the biggest infectious killers—respiratory tract infections and diarrhea. Diarrheal disease had a much higher priority in the 1980s but has dropped measurably on the global health agenda (Bump, Reich, & Johnson, 2013). Head et al. (2013) compared funding from the United Kingdom with disease burden (DALYs and mortality) to show low levels of investment relative to burden for gastrointestinal infections; although burden is similar to HIV/AIDS, it received only about half of the funding. Similarly, some neglected tropical diseases and antimicrobial resistance received low funding compared to their burden. WHO identified the top eight emerging infectious diseases likely to cause

severe outbreaks in the near future and for which there is insufficient R&D, and few or no medical countermeasures, with the potential to harm millions (World Economic Forum [WEF], 2017). These are Crimean-Congo hemorrhagic fever, Ebola, Marburg, Lassa fever, MERS, SARS, Nipah, Rift Valley fever: included also were three serious diseases, *Chikungunya[http://www.who.int/mediacentre/factsheets/fs327/en/]*, *severe fever with thrombocytopenia syndrome[http://www.thelancet.com/journals/laninf/article/PIIS1473-3099(14)70718-2/abstract]*, and Zika. Economists can make their contributions to avert these threats to global health and development. The increasing threat of AMR calls for insights from industrial economics to be applied to pharmaceutical markets. We need research into how incentives for R&D investments into the development of new antimicrobials can be increased. WHO's recently published global priority list of antibiotic-resistant bacteria provides guidance as to where to focus research efforts (World Health Organization [WHO], 2017b).

Increasingly, infectious disease research will need to be linked to the development of the health system infrastructure in low-income countries to translate scientific advances into operational reality. Although the WHO strategy for TB treatment has been adopted by every country, implementation has been compromised by the reach of public health systems and by the poor quality of care in private practice. The 19.5 million HIV-positive people receiving ART in 2016 represented only just over half of the 36.7 million living with HIV. Drugs to treat helminth infections have been donated in large quantities by pharmaceutical companies, and yet the proportion of eligible children receiving treatment is still far below target (Dye, 2014). Stenberg et al. (2014) estimated that around $274 billion spending on health is needed per year by 2030 to reach the ambitious Sustainable Development Goals 3 targets, of which around 75% of costs are for health system strengthening, with health workforce and infrastructure (including medical equipment) as the main cost drivers. Economics can make

valuable contributions and advise policymakers on optimal investments into health system strengthening for effective delivery of healthcare and public health interventions. The economics of infectious diseases is an exciting field of research that requires a multidisciplinary approach integrating insights from health economics, behavioral economics, econometrics, sociology, psychology, implementation science, and infectious disease modeling. If we manage this integration well, we can tackle some of the major challenges the world will face over the next decades.

Acknowledgment

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Katharina Hauck