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**Modern Infectious Diseases:
Macroeconomic Impacts and Policy
Responses**

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Abstract

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JEL Classification: D15, D58, E10, E20, I12, I15, I18, I31, O40

Keywords: Pandemics, Epidemics, Economic Burden of Disease, Economic Growth, Macroeconomic Models, Health, Inequality, Welfare, Human Capital, Health Policy

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Modern Infectious Diseases: Macroeconomic Impacts and Policy Responses

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1. Introduction

The emergence and rapid transmission in late 2019 and early 2020 of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) and the steep increase in the prevalence of the disease it causes (coronavirus disease 2019 or COVID-19) forcefully reminded scientists, policymakers, and the wider public of the world's vulnerability to infectious disease pandemics. Within a few weeks, we moved from a few reported cases of a new pulmonary disease in the city of Wuhan in China to a state in which almost all countries reported infections and deaths, billions of people could only leave their homes for essential purposes, economic activity nosedived, and unemployment figures skyrocketed. The global economy's vulnerability to such a pandemic underscores the importance of health for economic well-being and brings to mind Arthur Schopenhauer's famous quote "health is not everything, but without health everything is nothing."

Apart from the COVID-19 crisis, the late 20th and early 21st centuries have seen the rapid transmission and difficulty of containing various diseases. Many epidemics and pandemics are ongoing, claiming millions of lives per year (such as HIV/AIDS, malaria, and tuberculosis); some are on the rise in terms of infections and deaths (such as dengue fever and various forms of hepatitis); some have nearly been eradicated but with a dangerous prospect of re-emerging should containment efforts be relaxed (such as measles and poliomyelitis); some have abated or were successfully contained (such as SARS, Ebola, H1N1 influenza, and Zika); and some have frequent outbreaks (such as cholera).

This article aims to discuss the macroeconomic effects of modern infectious disease epidemics and pandemics and the frameworks that are used to assess them. (Hereafter, we will generally use the word epidemic, except where necessary to explicitly distinguish between epidemics and pandemics.) First, we provide a brief overview of the relationship between general health improvements and macroeconomic outcomes and discuss the advantages and disadvantages of different economic frameworks to assess the macroeconomic impacts of infectious diseases. Second, we sketch a general theoretical framework for analyzing the macroeconomic impacts of infectious diseases to identify the channels by which epidemics affect the economy and the extent to which these channels matter across diseases and cross-country settings. Third, we discuss pharmaceutical and nonpharmaceutical policy interventions toward curbing and preventing epidemics and mitigating their macroeconomic repercussions. Fourth, we describe the most important modern infectious disease outbreaks and discuss their economic consequences based on the pertinent literature. In so doing, we emphasize the recent health and economic crises caused by HIV/AIDs, malaria, tuberculosis, influenza, and COVID-19.

In underscoring the economic impacts of infectious diseases, we hope to clarify the importance of (i) preventive actions against infectious diseases, such as efforts to improve hygiene and to develop vaccines and distribute them widely, and (ii) early action in the case of outbreaks such as surveillance, early and accurate reporting, cuts in unnecessary travel, transmission hotspot shutdowns, and testing and contact tracing. The advantage of these preventive policies and early responses is that their costs tend to be small compared with the costs of containment measures against full-scale outbreaks.

Important tradeoffs that policymakers must consider when enacting containment measures include the following:

- While containment measures save lives, they often impose constraints on crucial economic sectors. When constraints are especially strict, more lives could be lost due to the measures imposed than would be saved by them. For example, lifesaving medical interventions in other domains (such as surgeries, vaccination programs against other diseases, etc.) might not proceed as planned, or—particularly in poor countries—people might even starve because lockdown measures deprive them of the income they need to afford subsistence consumption.

This suggests that optimal containment measures depend on country characteristics such as general income levels and poverty rates that affect the fraction of particularly vulnerable people.

- Short-run and long-run effects trade off in the sense that shutdowns can be imposed decisively and quickly, which might reduce how long they would have to be applied. In addition, lockdown measures reduce economic activity sharply at impact, while a continuing, unchecked epidemic leads to more infections and deaths over time. Thus, in the latter case, the adverse economic effects spread out over time and into the longer term. Another intertemporal tradeoff arises regarding efforts aimed at reducing the risk of pandemics and building up medical and nonmedical capacity to deal with them. Such investments may carry high and certain costs upfront while leading to delayed and uncertain, but potentially very high, returns in the future.
- Tradeoffs also arise in the context of targeting policies to specific population subgroups. Resource limitations may force the policymaker to target treatments or vaccines according to their highest effectiveness. Alternatively, if nonmedical interventions, such as lockdowns, are burdensome to those affected, issues emerge about whether the stringency of these measures should vary across different parts of the population. For example, if a disease predominantly affects specific risk groups such as older adults, discussions naturally emerge regarding the extent to which other parts of the population need to be subjected to strict lockdown measures to protect the group(s) at risk. Finally, choices must be made regarding the extent to which protective measures and economic relief should be targeted according to medical and socioeconomic vulnerability.

Some of these tradeoffs change in a crucial way when considering different diseases. For example, children seem to be particularly contagious when it comes to the seasonal flu such that closing schools is an important policy lever to stop or slow its spread (Litvinova et al., 2019). In the case of COVID-19, closing bars; canceling large indoor events; and addressing issues with air conditioning, climate control, and hygiene within meat production facilities, warehouses, and public transportation seem to be most effective.

Despite the economic focus on the tradeoffs faced when enacting policies against infectious diseases, we would like to issue a caveat up front. Narrow economic considerations take inadequate account of the ethical, normative, and political dimensions of decisions that relate to saving lives, particularly the lives of vulnerable members of the population who depend on the actions of others and who might not contribute economically by participating in the labor market (e.g., because of age or illness). While welfare economics has long gone beyond a focus on gross domestic product (GDP) and while practical well-being indicators have recently been developed that emphasize life expectancy and health (e.g., Jones, 2016; Jones and Klenow, 2016; Kuhn and Prettner, 2016; Fan et al., 2018; Frankovic and Kuhn, 2018; Bloom et al., 2020a), they might still not fully capture all aspects of living a healthy life without pain and suffering.

In addition, the value of a statistical life (VSL) metric that is typically employed when considering the tradeoff between lifesaving measures and their economic repercussions has problematic properties (cf. Viscusi and Aldy, 2003; Adler, 2020). In particular, the VSL declines with age and increases with income (cf. Murphy and Topel, 2006; Aldy and Viscusi, 2008). Thus, a disease that kills mainly older persons or poor persons would be associated with comparatively low losses in terms of the VSL. This indicates the limits of economic analysis when it comes to the implicit social contracts and ethical responsibilities that we all have in providing appropriate protections for the most vulnerable parts of the population.

As the economic literature on infectious diseases is large to begin with and the literature on COVID-19 is following its own epidemic growth at the time of writing (cf. Brodeur et al., 2020; Wyplosz, 2020, for some bibliographical analysis), we must restrict our attention to selected prominent studies. Philipson (2000), Laxminarayan and Malani (2006), Klein et al. (2007), Gersovitz (2011), Hauck (2018), and Rasul (2020) survey the economics of infectious diseases with more focused treatments of the earlier literature. Brodeur et al. (2020) provide a survey of the emerging economic literature on COVID-19, and a wealth of research has been published as part of the Centre for Economic Policy Research's *Covid Economics* series of vetted and real-time papers (cf. Wyplosz, 2020, for a summary of the editorial experience).

The remainder of this article is organized as follows. Section 2 briefly presents core insights into the general relationship between health and economic growth that serve as a backbone for this survey. Section 3 introduces the main approaches used to assess the economic burden of diseases and the literature on economic epidemiology. Section 4 sketches a general theoretical framework to evaluate the interrelations between disease dynamics and behavioral responses, while Section 5 analyzes the associated macroeconomic repercussions. Section 6 discusses the effects of economic policies toward containing or eradicating infectious diseases, issues of international coordination, and macroeconomic interventions toward mitigating disease-induced recessions. Section 7 summarizes insights into the most important epidemics since World War II, while Section 8 concludes and discusses the reverse channel from economic activity to changes in the likelihood of epidemics.

2. General health and economic growth

2.1 Theoretical considerations

To better understand the effects of epidemics on economic prosperity, knowing the channels by which health affects economic growth in general and the extent to which these channels differ between rich and poor countries is important (for surveys on the health–growth nexus see Weil, 2014 and Bloom et al., 2019a). Generally, better health implies (i) a greater supply of productive labor and thus higher incomes (e.g., Bloom et al., 2020c); (ii) higher life expectancy and lower morbidity, which tends to increase schooling and thus human capital accumulation (e.g., Bleakley, 2007, 2010; Boucekine et al., 2002; Cervellati and Sunde, 2005); (iii) higher saving and investment because of the longer individual planning horizons and potentially more foreign direct investment (e.g., Alsan et al., 2006; Bloom et al., 2007); and (iv) lower fertility due to a decline in precautionary fertility and higher opportunity costs of children, which can then initiate a takeoff in education investments and the emergence of a demographic dividend (e.g., Bloom et al., 2003, 2017, 2020c; Cervellati and Sunde, 2005, 2011; Klasing and Milionis, 2020).

In low-income countries, a lack of general access to quality health care, widespread undernourishment, and the prevalence of competing infectious diseases exacerbate the impact of epidemics. Generally, low-income countries are at considerable risk of entering or remaining in a vicious cycle that links low income, a lack of investment in schooling and health, and high fertility (e.g., Galor and Weil, 2000; Lagerlöf, 2003; Galor, 2005, 2011). Low incomes prevent investments in health that through the previously described channels would help promote sustained economic development. Overall, countries with poor population health are then trapped in a Malthusian stagnation equilibrium. In such a setting, negative health shocks from an epidemic can have strongly adverse effects on economic

growth by either perpetuating stagnation, or by pushing the economy back into a poverty trap (e.g., Lagerlöf, 2003; Momota et al., 2005; Chakraborty et al., 2010).¹

In high-income countries, by contrast, access to effective treatments and a well-functioning health care sector can often mitigate negative health shocks.² The main economic effects then relate to rising health care costs, which curb the scope for investments and might lead to private or public budget deficits. Recent analysis tends to agree, however, that in rich countries the willingness to pay for health and survival is typically so high that rising health care costs are justified in social welfare terms even if they impose a drag on economic growth (Hall and Jones, 2007; Jones, 2016; Kuhn and Pretzner, 2016; Frankovic and Kuhn, 2018; Fonseca et al., 2020; Frankovic et al., 2020). In high-income countries, concerns rather relate to inequality in survival and other health outcomes as documented empirically by, e.g., Chetty et al. (2016) and theoretically by Frankovic and Kuhn (2019). In summary, high-income countries likely exhibit a large willingness to pay to contain the mortality and morbidity from large-scale epidemics even at high economic cost, but this willingness to pay may vary considerably across subgroups of the population. Inequality with respect to risks of infection and access to prevention and treatment technologies and their effective usage may thus be the prime concern.

2.2 Empirical considerations

There are two main approaches to estimating the direct effects of health on economic growth from an empirical perspective. The micro-based approach utilizes well-identified wage regressions of the Mincerian type to isolate the effects of health on individual earnings. The results are then aggregated over the whole population to arrive at the effects of health on macroeconomic performance (cf. Shastry and Weil, 2003; Weil, 2007).³ The macro-based approach, by contrast, utilizes conditional convergence regressions along the lines of Barro (1991) and Islam (1995) to estimate how health improvements affect the growth rate of the economy. In general, the latter finds greater effects of health on economic growth than the micro-based approach (cf. Barro, 1997; Bloom et al., 2004; Sala-i-Martin et al., 2004; Aghion et al., 2011; Lorentzen et al., 2008, Bloom et al. 2020d).⁴ The explanation is that while macro-based estimates tend to include indirect effects of health on schooling and saving that foster economic growth, these effects are disregarded in the micro-based approaches by construction. Bloom et al. (2019b) seek to reconcile the two approaches by (i) carefully specifying the estimation equations, (ii) controlling extensively for indirect effects of health, and (iii) instrumenting for health and education, which allows (at least in principle) identification of the causal direct effect of health on economic growth. Their macro-based estimates imply that a 10-percentage-point increase in adult survival rates (the probability of surviving to age 60, conditional on having survived to age 15) leads to a 9.1% increase in aggregate labor productivity.⁵ This estimate is well within the range of

¹ By contrast, the Black Death is often assumed to have affected so many workers that labor became scarce and wages rose substantially above the subsistence level, at least for an extended period. The extent to which this could have helped in escaping a Malthusian trap is discussed in Jedwab et al. (2020a).

² This is not always true, of course. For HIV/AIDS, expensive treatment can now contain mortality and morbidity, but this was not the case in the 1980s. By contrast, COVID-19 lacks effective treatment options (at this writing), although health care facilities are important because in many cases intensive care and mechanical ventilation are necessary for survival. The availability of these tends to be greater in richer countries.

³ Micro data on the impact of health and health-related policy interventions on earnings is also obtained from randomized (e.g., Baird et al., 2016) or natural experiments (e.g., Bleakley, 2007, 2010). These data, too, would need to be aggregated.

⁴ Acemoglu and Johnson (2007) is an exception in that the authors find a negative effect of increasing life expectancy on economic growth. Their results change, however, when controlling for initial health and when splitting the sample into pre-demographic transition countries in which increasing life expectancy raises population growth and post-demographic transition countries in which increasing life expectancy reduces population growth (see Aghion et al., 2011; Cervellati and Sunde, 2011; Bloom et al., 2014; Hansen and Lønstrup, 2015; Klasing and Milionis, 2020).

⁵ A 10-percentage-point improvement in adult survival rates is approximately equal to going from the adult survival rate of India (82.2% in 2016; World Health Organization, 2020) to that of China (92%).

credible estimates from the micro-based approach as calculated by Shastri and Weil (2003) and Weil (2007).

2.3 Role of complementarities

Many theoretical and empirical contributions center on the evidence that strong indirect effects complement the direct effects of health on economic performance. Complementarities exist along at least four margins: (i) Complementarity between health/longevity and education. Here, Boucekine et al. (2002), Cervellati and Sunde (2013), and Sanchez-Romero et al. (2016), among others, study the role of the Ben-Porath effect, whereby extensions in life expectancy trigger greater investments in education, with Jayachandran and Lleras-Muney (2009), Fortson (2011), and Oster et al. (2013) providing empirical evidence. Bleakley (2007, 2010), by contrast, emphasizes the complementarity between childhood health and the productivity of education. Finally, a reverse effect from education to health and health behavior is also likely, although the evidence is somewhat mixed (cf. Galama et al., 2018). (ii) Complementarity among investments into improving different types of health, e.g., relating to competing risks of mortality or to morbidity (quality of life) in parallel to mortality (quantity of life) (cf. Murphy and Topel, 2006; Oster, 2012a; Oster et al., 2013). (iii) Complementarity between health/longevity and savings, as individuals who live longer tend to save more, which has repercussions for capital accumulation and economic activity (cf. Bloom et al., 2007). (iv) Complementarity between improvements in women's health and reductions in fertility, which in turn can help the escape from poverty traps (Bloom et al., 2020c; Klasing and Milionis, 2020). In the context of endemic infectious diseases, such as HIV or malaria, complementarities imply serious economic consequences, including the negative impact of HIV on human capital investments (Fortson, 2011), the negative impact of malaria during childhood on educational attainment (Bleakley, 2010), and the detrimental impact of low levels of life expectancy on risky sexual behavior in the presence of HIV (Oster, 2012a).

To summarize, this section shows important pathways by which health affects economic outcomes. The direct effects of health on economic growth as derived from micro-based and macro-based approaches are sizeable, but many indirect effects via health-induced education, savings, and fertility reductions strongly reinforce the direct effects at the macroeconomic level.

3. The economic burden of diseases: different assessment methods, their weaknesses, and potential solutions

Various approaches are used to assess the economic burden of diseases. A broad categorization would distinguish the following approaches, which have different properties, advantages, and disadvantages.

- (i) The cost-of-illness approach (COI) summarizes the economic burden of a disease by summing up all direct and indirect costs caused by the disease over a specified time period. The direct costs include personal medical costs (e.g., for inpatient and outpatient treatment), nonmedical care costs (e.g., for transportation), and personal nonmedical costs (e.g., for research). The indirect costs include the loss of income of individuals afflicted with the disease. Adding up the costs results in a monetary value that can be interpreted in a straightforward manner and compared with other figures such as the cost of eliminating the disease. Therefore, the approach lends itself to an easy-to-interpret cost-benefit analysis. However, this approach is mechanical because costs are summed up without considering general equilibrium adjustment mechanisms at the macroeconomic level or individual behavioral changes and policy interventions in response to the spread of infections. For example, the job of a worker who dies from a disease is often filled by someone else after a certain amount of time. The associated loss of income to the person

who died may therefore lead to a gain of income of another underemployed or unemployed individual if there is slack in the economy. For a more detailed description of the approach and debates on its usefulness see, for example, Currie et al. (2000), Rice (2000), and World Health Organization (2009).

- (ii) The VSL approach first infers the value that individuals attach to their lives by estimating their willingness to accept risks in wage regressions (where risk premia are paid for more dangerous occupations), or, alternatively, their willingness to pay to reduce risks based on consumption data for lifesaving or health-preserving items. From the corresponding parameter estimates inferring the value a person attaches to his or her life is then possible. Aggregating the resulting values over the whole population and weighting by the life years lost due to a disease yields again a single monetary value that represents the disease burden (Viscusi and Aldy, 2003; Murphy and Topel, 2006). In typical empirical applications, this approach also does not consider general equilibrium repercussions and behavioral changes and is thus of limited use from a macroeconomic perspective. Note, however, that recent work has included the value of life in numerical general equilibrium frameworks (Hall and Jones, 2007; Frankovic and Kuhn, 2019; Frankovic et al., 2020).
- (iii) A conceptually different approach that considers macroeconomic adjustment mechanisms is based on convergence regressions following Barro (1991) and Islam (1995). In this approach, economic growth is regressed on the prevalence of the disease of interest and a set of relevant control variables (cf. Durlauf et al., 2005; Eberhardt and Teal, 2011 for general discussions of the method). From the coefficient estimate of prevalence, the disease-specific impact on economic growth can be inferred directly. The drawbacks of this approach are that (a) the data requirements are rather high, (b) misspecifications of the regression equation and missing or inaccurately measured data strongly influence the final results, (c) the approach only works for severe diseases (to have a sufficiently high signal-to-noise ratio), and (d) it only works if the disease affects many countries over a substantial time span so as to have a sufficiently large number of observations for precise estimates in either cross-country or panel settings. The necessary conditions for the approach to work tend to be fulfilled for severe pandemics or for widespread noncommunicable diseases with a discernible impact on economic growth, such as cardiovascular diseases (see Suhrcke and Urban, 2010 for an application to cardiovascular diseases, and see Barro et al., 2020 for an application to the Spanish flu).
- (iv) As another conceptually different approach, Bloom et al. (2020b) propose calibrating a human capital-augmented production function along the lines of Lucas (1988) with disease-specific and age-specific data on morbidity and mortality of the disease of interest (cf. Bloom et al., 2004). Mortality and morbidity related to a certain disease reduce aggregate effective labor supply depending on age-specific individual labor supply, age-specific education levels, and the susceptibility of the corresponding age group to the studied disease. In addition, treatment costs reduce the amount of resources that are available for saving/investment and therefore hamper capital accumulation. The framework thus allows tracing the long-run supply-side effects of a disease (including the substitution patterns of labor and capital over time) on GDP in general equilibrium, but also disregards behavioral changes on the household side. The long-term general equilibrium nature of the approach and the missing scope for analyzing patterns of private or public response to the emergence of an epidemic limits the applicability of this approach in the case of infectious diseases. For applications to noncommunicable diseases and traffic accidents (where the approach is generally better suited) see Chen et al. (2018, 2019a, b).

- (v) No approach discussed so far is ideally suited to analyzing the effects of infectious diseases on macroeconomic outcomes. To this end, merging a detailed production function approach as described in item (iv) with behavioral effects and their repercussions on the macroeconomy and with disease dynamics according to a susceptible–infected–recovered (SIR) model in the background would be desirable. Attempts along these lines couple SIR models with computable dynamic general equilibrium models of the economy, incorporating the direct effect of the disease and disease- or policy-induced behavioral changes on aggregate production and income (cf. Chakraborty et al., 2010, 2016; Goenka et al., 2014; Brotherhood et al., 2020; Eichenbaum et al., 2020a, b; Glover et al., 2020; Krueger et al., 2020).⁶ In these frameworks, disease transmission follows an SIR pattern, household behavior follows a dynamically optimal path as in the standard business-cycle literature with endogenous labor supply, and a simplified production function is usually applied on the supply side to keep the framework tractable.⁷

This short overview shows that from a macroeconomic perspective (i) and (ii) fall short of providing an accurate picture of the economic effects of infectious diseases, while (iii) and (iv) capture some aspects well, for example, the growth effects of severe conditions that affect many countries or the overall burden of a disease if household behavioral reactions need not be identified directly. However, only an approach along the lines of (v) would, in principle, be able to capture all important macroeconomic effects of infectious diseases and the pathways by which they emerge.

4. An epidemiological economic model

In this section, we sketch a general epidemiological macroeconomic model with competing risks to analyze how the epidemiological properties of disease dynamics (rates of infection, mortality, and recovery; measures of morbidity) and the state of disease (as measured by the size of the infected population) affect individual consumption, labor supply, and protective behavior, and ultimately impact on macroeconomic outcomes.

4.1 Epidemiological dynamics

Consider a population of $N = S + I + R$ living individuals. Among these, S are susceptible (vulnerable to contracting an infection), I are infected, and R are recovered (with immunity). The disease dynamics for susceptibles are given by

$$S_{t+1} - S_t = B_t - \mu_t S_t - (1 - \mu_t) \alpha_t I_t S_t = B_t - [\mu_t - (1 - \mu_t) \alpha_t I_t] S_t, \quad (1)$$

where B_t denotes the number of susceptible newborns, α_t the infection rate, and μ_t refers to baseline mortality. The number of susceptible individuals increases with the birth of individuals without immunity and decreases with deaths from causes other than the infectious disease under consideration. The number of susceptible individuals also decreases because a fraction of still-living susceptibles become infected and therefore move from state S to state I . The dynamics of the number of infected are given by

⁶ A second line of contributions that is not always based on SIR dynamics more specifically assesses the effects of infectious diseases on economic development and in particular on the onset of the demographic transition or the emergence of a demographically induced poverty trap (Lagerlöf, 2003; Momota et al., 2005; Boucekine et al., 2009; Aksan and Chakraborty, 2014; Gori et al., 2020).

⁷ A rich literature on behavioral epidemic modeling provides alternative approaches, including network- and agent-based models (e.g., Hollingsworth et al., 2011; Manfredi and D’Onofrio, 2013; Liu et al., 2015; Enanoria et al., 2016). While these models often allow for a rich description of behavioral and epidemic dynamics, they typically do not include detailed models of the underlying economies, and the pathways by which different effects emerge are difficult to trace.

$$\begin{aligned}
I_{t+1} - I_t &= (1 - \mu_t)\alpha_t I_t S_t - \mu_t^I I_t - (1 - \mu_t^I)\mu_t I_t - (1 - \mu_t^I)(1 - \mu_t)\gamma_t I_t \\
&= \{(1 - \mu_t)\alpha_t S_t - \mu_t^I - (1 - \mu_t^I)[\mu_t + (1 - \mu_t)\gamma_t]\}I_t,
\end{aligned} \tag{2}$$

where μ_t^I denotes infection-specific mortality and γ_t is the rate of recovery, conditional on survival. The number of infected increases with the number of susceptibles who get infected at time t and decreases with general and disease-specific mortality and with the recovery of the surviving infected who move from state I to state R , in which they do not have the disease but acquired immunity.⁸ The number of recovered increases with the number of the surviving and recovered infected and decreases with the number of recovered who die from causes other than the infectious disease under consideration:⁹

$$\begin{aligned}
R_{t+1} - R_t &= (1 - \mu_t)(1 - \mu_t^I)\gamma_t I_t - \mu_t R_t \\
&= B_t - \mu_t N_t - (1 - \mu_t)\mu_t^I I_t.
\end{aligned} \tag{3}$$

The competing risks structure is an important prerequisite to capture the dynamics of infectious diseases that become chronic or recurrent and constitute a significant mortality risk in their own right, that is, diseases with a low γ_t and a high μ_t^I . This applies in particular to HIV, at least until potent treatments became available. While the economic implications of competing risks will be addressed subsequently, in the SIR dynamics they appear prominently in the dynamics of the number of infected, where individuals may exit due to death from the disease, $\mu_t^I I_t$; due to death from other causes, $(1 - \mu_t^I)\mu_t I_t$; or due to recovery, $(1 - \mu_t^I)(1 - \mu_t)\gamma_t I_t$. The latter two terms need to be weighted with survival to avoid double counting. We note, however, that this is not always explicit in the formulation of SIR models, where (under the assumption $\mu_t = 0$) the unconditional recovery rate is often stated as a single parameter, $\hat{\gamma} = (1 - \mu_t^I)\gamma_t$. While this is fine for any analysis in which the disease-specific mortality μ_t^I is held constant, care needs to be taken to account for the term $\frac{d\hat{\gamma}}{d\mu_t^I} = -\gamma_t + (1 - \mu_t^I)\frac{d\gamma_t}{d\mu_t^I}$ in case variations of this mortality rate are considered.¹⁰

To reflect hospital crowding exacerbating death rates as during the COVID-19 epidemic, several models (e.g., Acemoglu et al., 2020; Brotherhood et al., 2020; Eichenbaum et al., 2020a, b; Glover et al., 2020) assume the mortality rates $\mu_t^I = \hat{\mu}_t^I(I_t)$ to be an increasing function of infections. Although not contained in these models, similar assumptions could be applied to the recovery rate γ_t or, indeed, to general mortality μ_t , the latter reflecting the deferral of lifesaving treatments due to crowding in the health care system (Maringe et al., 2020). However, general mortality might also decline if preventive behavior reduces the probability of infection by other viruses (e.g., the common flu) or if lockdowns reduce road accidents (Oguzoglu, 2020).

Let the infection rate be given by the function $\alpha_t = \alpha(N_t, c_t^S, l_t^S, v_t^S, c_t^I, l_t^I, v_t^I)$, where c is consumption, l is labor supply, and v is preventive effort with the superscripts S and I denoting these activities for susceptible and infected individuals, respectively. As is intuitive, the infection risk rises with

⁸ More general frameworks allow for yet another transition from state R back to state S because immunity might only be short lived (as, e.g., in the case of the flu) and for a state of infection without symptoms that comes in between states S and I in which individuals might be infectious without knowing that they carry the disease as in the case of COVID-19 (see, e.g., Anderson et al., 2012; Brotherhood et al., 2020; Rowthorn and Toxvaerd, 2020).

⁹ The change in population size $N_{t+1} - N_t = B_t - \mu_t N_t - (1 - \mu_t)\mu_t^I I_t$ is easily constructed from combining equations (1)–(3).

¹⁰ Here, the first negative term captures the direct reduction due to mortality of the population from which recovered individuals are drawn, while the second term captures a selectivity effect according to which surviving individuals may also face better chances of recovery.

consumption and labor supply due to the associated social interactions and decreases with preventive effort, such as exercising restraint in risky activities; using protective devices, for example, face masks in the case of a disease like COVID-19 or condoms in the case of HIV; and taking up vaccination. Thus, we have the following derivatives: $\alpha_{c,s}, \alpha_{c,l} \geq 0$; $\alpha_{l,s}, \alpha_{l,l} \geq 0$; and $\alpha_{v,s}, \alpha_{v,l} \leq 0$. Generally, the infection rate also depends on the population size, such that $\alpha_N \geq 0$. Here, several competing specifications are in use: Assuming that a susceptible individual interacts randomly with other members of the population at a rate $\hat{\alpha}$, the infection risk is $\hat{\alpha} \times I_t/N_t$, where the probability of meeting an infected person is given by the prevalence rate I_t/N_t . The seminal SIR model by Kermack and McKendrick (1927) posits $\hat{\alpha} = \hat{\alpha}_1$, a constant rate of interaction. This implies that by lowering the prevalence rate I_t/N_t , the infection risk $\hat{\alpha}_1 \times I_t/N_t$ decreases, *ceteris paribus*, with the size of the population. By contrast, many models (e.g., Philipson, 2000; Eichenbaum et al., 2020a, b) assume that the rate of interaction is proportional to the population size, such that $\hat{\alpha} = \hat{\alpha}_2 N_t$. In this case the infection risk $\hat{\alpha}_2 I_t$ is independent of the population size. While the two approaches are (broadly) equivalent for (near) stationary populations, the assumptions about the interaction rate $\hat{\alpha}$ become less innocuous when disease mortality or responses in terms of fertility or migration lead to changes in the population size. The same applies to diseases that turn endemic within non-stationary populations. Acemoglu et al. (2020) explore a general specification of the infection function that nests the two polar cases described but allows for all intermediate forms. Studying testing and lockdown policies in the context of COVID-19, they show that while the recovered offer greater protection for the case $\hat{\alpha} = \hat{\alpha}_1$, lockdown policies tend to be more effective in settings where the rate of infection increases with the population size, as is the case for $\hat{\alpha} = \hat{\alpha}_2 N_t$.

4.2 Behavioral responses

Building on Eichenbaum et al. (2020a), we assume that individuals within each of the *SIR* groups maximize utility over their remaining life course from time t onward.¹¹ We can write the maximization problem for susceptible individuals at time/age t in terms of the value function:

$$V_t^S = \max_{c_t, l_t, v_t} \{u(c_t, l_t, v_t) + \rho(1 - \mu_t)[\alpha_t I_t V_{t+1}^I + (1 - \alpha_t I_t) V_{t+1}^S]\}, \quad (4)$$

where $\rho \in [0,1]$ is the time discount factor, $u(c_t, l_t, v_t)$ is period utility, and V_t^i denotes the value function of group $i = S, I, R$ as of time t . Period utility increases with consumption ($u_c > 0$), decreases with labor supply ($u_l < 0$) because of the disutility of work, and decreases with prevention ($u_v \leq 0$) because preventive activities typically imply restraint in what would otherwise be some utility-maximizing behavior.¹² We normalize utility in the state of death to zero.

Similarly, the maximization problem for infected individuals at time/age t is given by

$$V_t^I = \max_{c_t, l_t, v_t} \{\tilde{u}(c_t, l_t, v_t) + \rho(1 - \mu_t)(1 - \mu_t^I)[\gamma_t V_{t+1}^R + (1 - \gamma_t) V_{t+1}^I] + \beta V_t^S\}, \quad (5)$$

¹¹ Our model generalizes Eichenbaum et al. (2020a) by considering (i) a specific preventive effort, v_t , apart from adjustments to consumption and labor supply; (ii) the multiplicity of disease and general survival risk; (iii) the presence of altruism on the part of infected individuals; and (iv) a dynamic budget constraint.

¹² While we emphasize the macroeconomic linkages and therefore follow Eichenbaum et al. (2020a) by explicitly allowing for consumption and labor supply responses to the disease, we acknowledge the large body of literature in economic epidemiology that models the choice of prevention effort, often understood as vaccination but also as social distancing (e.g., Geoffard and Philipson, 1996, 1997; Kremer, 1996; Auld, 2003; Fenichel, 2013; Toxvaerd, 2019, 2020). For surveys see Philipson (2000), Gersovitz (2011), and Chen and Toxvaerd (2014).

where $\beta \in [0,1]$ denotes the degree of altruism of the infected toward contemporary susceptible individuals and where period utility $\tilde{u}(c_t, l_t, v_t)$ is typically lower than that of susceptible individuals, that is, $\tilde{u}(c_t, l_t, v_t) \leq u(c_t, l_t, v_t)$, with additional effects relating to the marginal utilities.

Finally, we can write the life-cycle utility of recovered individuals as

$$V_t^R = \max_{c_t, l_t} \{u(c_t, l_t, 0) + \rho(1 - \mu_t)V_{t+1}^R\}, \quad (6)$$

where we note that recovered individuals who are immune to the disease have no need or incentive to engage in preventive behavior.

Assuming that physical capital k_t is the only savings vehicle and normalizing the price of consumption goods to one, individuals face the following dynamic budget constraint:

$$k_{t-1} - k_t = r_t k_t + \phi^i w_t h_t l_t - c_t - p_t v_t, \quad (7)$$

where r_t and w_t denote the interest rate and the wage rate per unit of human capital h_t (which for the moment we treat exogenously), respectively; where ϕ^i with $i = S, I, R$ refers to labor productivity of type i with $\phi^S = \phi^R = 1$ and $\phi^I \in [0,1]$; and where $p_t \geq 0$ is the price of protective goods in case these can be purchased. This would apply, for example, to condoms for the prevention of HIV infection, to face masks for reducing the risk of COVID-19 infection, or to costly vaccines.

4.2.1 First-order conditions for susceptible individuals (S types)

The choices of susceptible individuals follow from the first-order conditions:

$$\begin{aligned} u_c - \lambda_t^S + \rho(1 - \mu_t)(V_{t+1}^I - V_{t+1}^S)\alpha_{c,S}I_t &= 0, \\ u_l + h_t w_t \lambda_t^S + \rho(1 - \mu_t)(V_{t+1}^I - V_{t+1}^S)\alpha_{l,S}I_t &= 0, \\ u_v - p_t \lambda_t^S + \rho(1 - \mu_t)(V_{t+1}^I - V_{t+1}^S)\alpha_{v,S}I_t &\leq 0, \end{aligned}$$

where λ_t^S denotes the shadow price of wealth for a susceptible individual. For all conditions, the first two terms govern the choice that would be made in the absence of the disease (i.e., $I_t = 0$). Notably, under the assumption that $u_v < 0$, individuals would not engage in preventive activity. The third term then indicates how choices are adjusted if this reduces the risk of infection, that is, if $\alpha_{c,S}, \alpha_{l,S} > 0$ and $\alpha_{v,S} < 0$.

Proposition 1

Under the plausible assumption that $V_{t+1}^I < V_{t+1}^S$, susceptible individuals lower their consumption and their labor supply and engage in preventive activities in line with

- *the “stakes of infection” in terms of the present value of the expected loss in future life-cycle utility in the case of infection, $\rho(1 - \mu_t)(V_{t+1}^I - V_{t+1}^S)$;*
- *the impact of the respective activity on infection risk, $\{\alpha_{c,S}, \alpha_{l,S}, \alpha_{v,S}\}$;*
- *the prevalence of the disease, I_t ; and*
- *opportunity costs, $h_t w_t$, and prices for protective goods, p_t .*

Note that apart from the future orientation, as measured by the discount factor ρ , and the utility loss implied by the infection, the “stakes of infection” also depend on the general survival prospects, $1 - \mu_t$. This implies that population groups with poorer survival prospects (e.g., the old or the poor) have

ceteris paribus a lower incentive to invest in self-protection (Oster, 2012a). The corollary that follows is that the downward adjustments in consumption and labor supply are weaker for these groups.

The utility loss from the infection itself, $V_{t+1}^I - V_{t+1}^S$, is high if the infectious disease is associated with (i) high morbidity, implying a strong loss in period utility $\tilde{u}(c_t, l_t, v_t) \ll u(c_t, l_t, v_t)$ or productivity $\phi^I \ll 1$; (ii) high mortality μ_{t+1}^I such that survival into period $t + 2$ is compromised; or (iii) poor prospects of recovery, that is, $\gamma_{t+1} \ll 1$. By now considerable empirical evidence is available to indicate the dependency of preventive behavior on life-cycle circumstances (e.g., Kremer, 1996; Auld, 2003; Dupas, 2011; Oster, 2018; Dang et al., 2020). Income tends to be conducive to prevention by raising the stakes from income loss and the value of prevention (relative to foregone consumption), while education increases awareness and empowerment to engage in prevention. In addition, expectations about disease development have been shown to matter. Here, a belief that future infection, for example, in period $t + 1$, is unavoidable will narrow the wedge ($V_{t+1}^I - V_{t+1}^S$) and thus the stakes of infection in period t . Finally, the decision to engage in prevention and the means of prevention depend on the prices for protective measures (e.g., Cohen and Dupas, 2010) and the opportunity cost.

Remark 1

The model's structure allows considerable scope for heterogeneity across (i) diseases that differ with respect to their disutility effects, infection risks, morbidity effects, mortality effects, and survival prospects $\{\tilde{u}, \phi^I, \mu^I, \gamma, \alpha\}$; (ii) individuals that differ with respect to wealth, income, education, general mortality risk, impatience, and susceptibility to the disease $\{\lambda^S, w, h, \mu, \rho, \alpha\}$; and (iii) countries that differ with respect to income levels, price levels, and institutional settings that drive general mortality and infection risk and insure against the cost of prevention and infection $\{w, p, \mu, \alpha\}$.

While most older literature in economic epidemiology focuses on preventive effort, v , that may be costly but is otherwise not linked to macroeconomic performance, recent macroeconomic literature on the impact of COVID-19 has focused on social distancing as a means of prevention that directly bears on consumption and labor supply (see, e.g., Brotherhood et al., 2020; Eichenbaum et al., 2020a, b; Glover et al., 2020; Krueger et al., 2020). Indeed, this literature shows that the reduction in consumption and labor supply for reasons of self-protection can have large macroeconomic repercussions, as experienced in the context of COVID-19. Importantly, however, an interdependency exists between the extent of self-protection and the extent to which consumption and labor supply are reduced due to social distancing. To simplify the exposition, we assume that no cross-effects exist between the marginal utility of consumption and labor supply $u_{cl} = 0$ (as is assumed, for example, in Eichenbaum et al., 2020a, b).

Proposition 2

Self-protection is conducive to alleviating the negative impact of the epidemic on consumption and labor supply if it lowers the riskiness of these activities to an extent that overcompensates a decline in the marginal utility of consumption or an increase in the marginal disutility of labor that is associated with self-protection.

To illustrate the claim, consider the partial derivative of consumption with respect to prevention effort:

$$\frac{\partial c^S}{\partial v^S} = \frac{-[u_{cv} - \rho(1-\mu_t)(V_{t+1}^I - V_{t+1}^S)\alpha_{cSv}I_t]}{u_{cc} - \rho(1-\mu_t)(V_{t+1}^I - V_{t+1}^S)\alpha_{cS}I_t}.$$

Noting that the second-order conditions imply that $u_{cc} - \rho(1 - \mu_t)(V_{t+1}^I - V_{t+1}^S)\alpha_{c^s c^s} I_t < 0$, we obtain $\partial c^S / \partial v > 0$ if and only if $u_{cv} - \rho(1 - \mu_t)(V_{t+1}^I - V_{t+1}^S)\alpha_{c^s v^s} I_t > 0$. Under the plausible assumptions that $u_{cv} \leq 0$ and $\alpha_{c^s v^s} < 0$, that is, protection is potentially reducing the marginal utility of consumption but is also lowering the infection risk associated with consumption, the condition then implies the statement in the proposition (see Doganoglu and Ozdenoren, 2020, for a modeling application to COVID-19). Note that such an outcome is likelier the higher the stakes of infection are and the higher the prevalence of the disease is. A similar result is easily obtained with respect to labor supply. In macroeconomic terms, the proposition implies that the reductions in consumption or labor supply may be strongly mitigated if individuals have the scope to take preventive actions. This creates inroads for policymaking in enabling and fostering such options, which are related to (i) availability of vaccination, (ii) availability of condoms/face masks/insecticide-treated bednets, (iii) restraints on risky behaviors (e.g., just working/shopping but without mingling), or (iv) substitution of consumption/employment with less risky forms like food delivery instead of eating in restaurants or home office instead of commuting by public transport to work in open plan offices. Krueger et al. (2020), who generalize the Eichenbaum et al. (2020a) model of the COVID-19 pandemic to allow for substitution toward home production and home consumption, show that quantitatively this may mitigate both the human and the economic loss associated with the pandemic considerably.

Remark 2

The dependency of preventive actions on the prevalence of a disease has the important repercussion that diseases are difficult to eradicate via private actions alone (Geoffard and Philipson, 1996, 1997). This follows immediately from the fact that preventive incentives wane with declines in disease prevalence and peter out entirely once the disease is close to eradication, $I_t \approx 0$. In such a case, the further evolution of the disease toward either vanishing or re-emerging very much depends on (pure) epidemiological properties.

4.2.2 First-order conditions for infected individuals (*I* types)

The choices of infected individuals are determined by the first-order conditions:

$$\begin{aligned} \tilde{u}_c - \lambda_t^I + \beta\rho(1 - \mu_t)(V_{t+1}^I - V_{t+1}^S)\alpha_{c^I} S_t &= 0, \\ \tilde{u}_l + \phi^I h_t w_t \lambda_t^I + \beta\rho(1 - \mu_t)(V_{t+1}^I - V_{t+1}^S)\alpha_{l^I} S_t &= 0, \\ \tilde{u}_v - p_t \lambda_t^I + \beta\rho(1 - \mu_t)(V_{t+1}^I - V_{t+1}^S)\alpha_{v^I} S_t &\leq 0, \end{aligned}$$

where λ_t^I denotes the shadow price of wealth for an infected person.

Proposition 3

Under the assumption that $V_{t+1}^I < V_{t+1}^S$, infected individuals lower their consumption and their labor supply and engage in preventive activities in line with

- *the degree of altruism, $\beta \in [0,1]$;*
- *the “stakes of infection” in terms of the present value of the expected loss in future life-cycle utility in the case of infection, $\rho(1 - \mu_t)(V_{t+1}^I - V_{t+1}^S)$;*
- *the impact of the respective activity on infection risk, $\{\alpha_{c^I}, \alpha_{l^I}, \alpha_{v^I}\}$;*
- *the number of susceptible individuals, S_t ; and*
- *opportunity costs, $\phi^I h_t w_t$, and prices for protective goods, p_t .*

Infected individuals accommodate prevention concerns to the extent $\beta \geq 0$ that they are altruistic toward the susceptible. Note that entirely selfish individuals with $\beta = 0$ will not adjust their

consumption and labor supply to avoid infections and, for $\tilde{u}_v \leq 0$ and $p_t \geq 0$, will not engage in any preventive behavior. Even if infected individuals engage in some prevention for $\beta \in (0,1)$, they impose a certain (contemporary) externality on the susceptible population.

Similar to the incentives that susceptible individuals face, the incentive to engage in prevention increases with the “stakes of infection,” $\rho(1 - \mu_t)(V_{t+1}^I - V_{t+1}^S)$, reflecting the utility loss imposed on a contemporary susceptible individual in the case of an infection and the additional infections that are avoided at time t . Notably, the contingency of the stakes of infection on general survival has repercussions if the susceptible population is selected from groups with higher mortality (e.g., the older population, the poor, etc.). Utilitarian altruists may then have a low incentive to invest in protecting these vulnerable groups.

We assume here that individuals only care about those infections they cause in person, where a broader notion of altruism would also embrace an intertemporal component, $X_{t+1} = \rho(1 - \mu_{t+1})(V_{t+2}^I - V_{t+2}^S)\alpha_{t+1}S_{t+1} \frac{dI_{t+1}}{dI_t} + X_{t+2}$, reflecting the knock-on effect of the infections caused at time t . Given that the individuals under consideration do not *directly* cause the knock-on infections, it seems reasonable they cares less about them. However, the intertemporal term also illustrates that the size of the dynamic part of the externality may be substantial, in particular, in the case of fast-spreading infections such as COVID-19. In such cases, decisive policy interventions may well be required.

Remark 3

A richer behavioral model should account for reciprocity and trust (Alfaro et al., 2020; Doganoglu and Ozdenoren, 2020), which would make own prevention efforts depend positively on the (expected) prevention of others (Bartscher et al., 2020). The violation of prevention norms, in particular by public figures and members of the government, may erode voluntary efforts in such a setting (Fancourt et al., 2020).

Finally, the incentive to engage in prevention increases with the size of the susceptible population and thus tends to decline with disease prevalence, i.e., with I_t (but also with R_t). This implies that to some extent preventive actions of altruistic infected individuals foster disease eradication even when an incentive on the part of the susceptibles to engage in self-protection no longer exists.

Depending on the cross-derivatives between the preventive efforts of the susceptible and the infected within the function $\alpha(\cdot)$, preventive efforts may be strategic substitutes (complements) across types. In the (more likely) case that they are strategic substitutes, the following holds.¹³

Proposition 4

If preventive efforts of the susceptible and the (altruistic) infected are strategic substitutes, both groups have an incentive to underinvest in prevention when taking the other group’s efforts as given, implying that preventive efforts tend to be too low from a welfare perspective.

To consider a specific example, we build on Eichenbaum et al. (2020a) and assume that $\alpha(N_t, c_t^S, l_t^S, v_t^S, c_t^I, l_t^I, v_t^I)I_tS_t = \hat{\alpha}(N) \left[\left(\frac{c_t^S}{v_t^S} \times \frac{c_t^I}{v_t^I} \right) + \left(\frac{l_t^S}{v_t^S} \times \frac{l_t^I}{v_t^I} \right) \right] I_tS_t$. Focusing on consumption, we then have

¹³ Strategic complementarity may plausibly arise only in circumstances of uncertainty or social norms, where individuals align their own behavior with the observed behavior of others.

$$\frac{\partial c^S}{\partial c^I} = \frac{-\rho(1-\mu_t)(V_{t+1}^I - V_{t+1}^S)\alpha_{c^S c^I} I_t}{u_{cc} - \rho(1-\mu_t)(V_{t+1}^I - V_{t+1}^S)\alpha_{c^S c^I} I_t} = \frac{-\rho(1-\mu_t)(V_{t+1}^I - V_{t+1}^S)\hat{\alpha}(N)I_t}{u_{cc} v_t^S v_t^I} < 0.$$

where the second equality and the inequality follow for our specific example. Here, *reductions* in consumption to prevent disease are strategic substitutes, and this implies that the underprovision of prevention as a public good within a noncooperative Nash equilibrium is well known (Pauly, 1970), that is, individuals will consume too much. A similar result is easily verified with respect to the choice of labor supply, l_t^I , which is overprovided, and preventive effort, v_t^I , which is underprovided, a well-known result in the literature on economic epidemiology (e.g., Chen and Toxvaerd, 2014 in the context of vaccination; Toxvaerd, 2019 in a general prevention model; Farboodi et al., 2020; Garibaldi et al., 2020; McAdams, 2020; Toxvaerd, 2020 in the context of social distancing; and Kuhn et al., 2011 for a life-cycle account of externalities arising from health care choices).

While, to our knowledge, this has not yet been studied, the presence of complementarity in preventive efforts could yield important macroeconomic implications. Compare, for instance, two economies, of which only one experiences altruism on the part of the infected. Assuming that infected individuals are less productive, that is, $\phi^I < 1$, the greater reduction in labor supply on the part of the infected altruists then allows healthy but susceptible individuals to reduce their labor supply less and thus mitigate the loss in output.

One general issue with voluntary preventive actions on the part of the infected is the presumption that they know about their infection. Indeed, one prime problem of infectious diseases is that individuals may be ill-informed because they have not yet developed symptoms, or even remain asymptomatic altogether, or because symptoms may be signals for several diseases of different degrees of infectiousness or severity. These issues have turned out to be particularly salient for HIV infection, where the median lag between infection and the development of symptoms lasts for several years, and for COVID-19, where the 7–14 day incubation period is relatively long for a disease with likely airborne infection and where the large share of asymptomatic infections and difficulty of distinguishing symptoms from the common flu exacerbate the information problem. Brotherhood et al. (2020) develop a model in which altruistic individuals engage in prevention but where individuals are imperfectly informed about their illness status. They conclude that testing for COVID-19 is a prerequisite for effective voluntary prevention of the disease, which quantitative analysis bears out.

Finally, the disease itself changes infected individuals' behavior regardless of whether they engage in prevention or not: Infected individuals tend to lower consumption (ia) if the disease lowers their marginal utility from consumption, that is, if $\tilde{u}_c(c, l) < u_c(c, l)$, and (ib) if they are subject to a negative income effect (implying a higher λ_t). Infected individuals also tend to lower labor supply if (iia) they experience a higher disutility from working due to the illness, $\tilde{u}_l(c, l) > u_l(c, l)$, and (iib) if the productivity loss from illness is sufficiently large, $\phi^I \ll 1$, to overcompensate a possibly offsetting income effect that would induce individuals to work more hours to make up for the earnings loss due to lower productivity.

4.3 Human capital responses to the epidemic

Infectious disease outbreaks can affect human capital accumulation in various ways, including the occurrence of long-term physical and mental health sequelae, interrupted access to the health system, school closures, and changes in the expected benefits of attending school. Arguably, because of their long-term nature, investments in human capital, that is, education and general health, tend to be less affected by unexpected and transitory infectious disease epidemics (such as dengue or Ebola outbreaks) than by endemic infectious diseases (such as HIV and malaria in Sub-Saharan Africa). A

similar argument applies when the number of competing pathogens increases, leading to an increase in the risk of acquiring some infectious disease (of many), a scenario that is deemed likely even for richer countries in the global North. We first discuss the implications of (endemic) infection risks on educational investments before turning to health.

4.3.1 Education

For simplicity, assume that at time t individuals (or in the case of children, their parents on their behalf) can invest an amount e_t into education at some cost $\kappa_t > 0$ and thereby augment human capital $h_{t+1} = \theta^I f(e_t)$, with $f' > 0$. Here, θ^I with $\theta^I \leq \theta^S = 1$ (ignoring the recovered) denotes the health-dependent productivity of education, where Bleakley (2007, 2010), Lucas (2010), Baird et al. (2016), and others document that educational investments are less productive in the presence of infections (hookworms, malaria) during childhood. The first-order condition for the choice of e_t by a healthy susceptible individual is then given by

$$\rho(1 - \mu_t)[\alpha_t I_t \phi^I w_{t+1} \lambda_{t+1}^I + (1 - \alpha_t I_t) w_{t+1} \lambda_{t+1}^S] f' - \kappa_t \lambda_t^S = 0.$$

Optimally, the expected return to education (in value-of-wealth terms) is equated with the cost-weighted current value of wealth. Here, the expected return increases with general survival—the Ben-Porath effect—and is conditioned on the probability of getting infected. Demobilizing infections, for which $\phi^I \ll 1$, then imply a drag on the returns to education and thus the accumulation of human capital, a drag that increases with the probability of getting infected. Interestingly, however, an offsetting tendency may exist. If the infection imposes severe financial strain on the infected, we may find $\lambda_{t+1}^I \gg \lambda_{t+1}^S$ so that the threat of infection may actually provide a stimulus to educational investments for precautionary reasons. A similar argument would hold if the infection risk itself were contingent on income or education. Finally, we note that the presence of an infectious disease may raise the cost of education, as would be the case whenever concerns about transmission through the educational system reduce the supply of education (in case of full lockdown triggering an infinite increase in the price). Especially in developing countries, adult morbidity or mortality may force children to take up labor at the expense of education (cf. Gertler et al., 2004; Case and Ardington, 2006). This is tantamount to an increase in the opportunity cost of education, κ_t , paired with a negative income effect, implying an increase in λ_t^S .

For an individual who is already infected at the point of taking up education, the first-order condition reads

$$\rho(1 - \mu_t)(1 - \mu_t^I)[(1 - \gamma_{t+1})\phi^I w_{t+1} \lambda_{t+1}^I + \gamma_{t+1} w_{t+1} \lambda_{t+1}^R] \theta^I f' - \lambda_t^I = 0,$$

implying that the return to education and thus the incentive may be reduced directly if morbidity lowers the effectiveness of education, $\theta^I < 1$. We also note, however, that the mortality risk from the infectious disease is now reducing the returns to education, an issue that has been shown to be particularly relevant in the context of HIV (Bell et al., 2006; Fortson, 2011). As before, infections that curb individual productivity tend to reduce the incentive to invest in human capital, an effect that tends to be more pronounced the lower is the expectation of recovery.¹⁴

Finally, we note that in a more general model that includes fertility choices, the quality–quantity tradeoff implies that the constraint on educational investments that infections impose may decelerate

¹⁴ To the extent that educational investments not only raise human capital in the next period, h_{t+1} , but also in periods further into the future, the returns to education for the infected enter the first-order condition of susceptible individuals as higher-order returns contingent on the risk of infection.

or even stall fertility reductions (cf. Aksan and Chakraborty, 2014 for a theoretical account and application to Sub-Saharan Africa; Bleakley and Lange, 2009 for supportive evidence in the case of malaria; and Young, 2007; Boucekkine et al., 2009; Fortson, 2009; and Juhn et al., 2013 for mixed evidence in the case of HIV/AIDS).

4.3.2 Health

In a similar vein, the threat of infections may bear on individuals' behavior toward their general health and survival. Thus, assume that at time t individuals can invest an amount m_t to lower their mortality $\mu_{t+1} = g(m_t)$ with $g' < 0$. The first-order condition for a healthy susceptible individual is given by

$$\rho[\alpha_t I_t V_{t+1}^I + (1 - \alpha_t I_t) V_{t+1}^S] g' - \lambda_t^S = 0,$$

where $\rho[\alpha_t I_t V_{t+1}^I + (1 - \alpha_t I_t) V_{t+1}^S]$ corresponds to the expected utility stream over the remaining life cycle. Continuing to assume that $V_{t+1}^I < V_{t+1}^S$, high infection risk from the infectious disease lowers the value of survival and thus the incentives to invest in general health. This is a particular problem in the case of an endemic disease, such as HIV or malaria, because it stifles incentives to invest in health (cf. Oster, 2012a) with further repercussions on productivity and economic outcomes (cf. Bloom and Sachs, 1998; Bloom et al., 2019b, 2020c). Rossi and Vilar (2020) show that public anti-malaria interventions are an important lever to trigger private health investments.

For an infected individual, the first-order condition reads

$$\rho(1 - \mu_t^I)[\gamma_t V_{t+1}^R + (1 - \gamma_t) V_{t+1}^I] g' - \lambda_t^I = 0,$$

where high mortality from the infectious disease and poor prospects of recovery tend to lower the value of general survival and thus the incentives to invest in general health. Again, this is a particular problem in the case of an endemic disease, such as HIV or malaria.

We conclude by drawing attention to the important role of complementarities in this context. In particular, we have shown that both the return to educational investments and the return to investments in general health tend to decline with (i) the risk of acquiring an infectious disease and with (ii) the morbidity and (iii) mortality associated with this disease. Conversely, recall from the previous subsection that both general health, that is, a lower μ_t , and human capital, h_{t+1} , tend to raise the stakes of infection and the value of lowering the morbidity and mortality associated with the infection and improving the recovery. Overall, this implies that efforts aimed at preventing and treating the infectious disease are complementary to investments in education and general health, suggesting the scope for virtuous and vicious cycles depending on socioeconomic status within economies and on the state of economic development across economies (cf. Lagerlöf, 2003; Chakraborty et al., 2010, 2016; Azomahou et al., 2016).

5. Macroeconomic repercussions of epidemiological dynamics and behavioral responses

The production side of the economy is represented by a production function according to which the inputs aggregate physical capital, K_t , and aggregate human capital, H_t , combine to produce aggregate output Y_t (or the GDP of a country):¹⁵

$$Y_t = F(K_t, H_t).$$

¹⁵ For Malthusian economies, explicit consideration may also be given to land as a fixed factor of production. For an analysis in the context of the Black Death see, for example, Jedwab et al. (2020a).

Aggregate human capital is the product of aggregate labor supply, L_t ; average human capital, h_t ; and average disease-specific productivity, ϕ_t , such that $H_t = h_t\phi_tL_t$. Average human capital, in turn, is determined by the quantity and quality of schooling when young and by work experience. In more elaborate models, a breakdown of the population to age cohorts can be made to the extent that older persons might have more experience but might have received less schooling in the past (cf. Bloom et al., 2020b). Assuming a closed economy implies that aggregate output equals aggregate income. Aggregate output/income, in turn, can be consumed or saved such that aggregate capital accumulation is given by

$$K_{t+1} = Y_t - C_t - G_t + (1 - \delta)K_t,$$

where C_t is aggregate private consumption; G_t is aggregate private or public resource use for education or health; and δ is the rate of depreciation of physical capital. The following subsections describe the main macroeconomic effects of the disease and efforts to prevent infections.

5.1 Human capital and output

By causing morbidity and mortality and thus a reduction in the labor force, any epidemic will have a direct impact on human capital, which, as we will outline, may be accompanied by possibly even larger indirect effects through behavioral adjustments in labor supply and education.

(i) Disease-specific morbidity and mortality reduce human capital and thereby output *directly* in the short run and, in the case of mortality or scarring, also in the long run. The extent to which human capital is reduced very much depends on the particular age pattern of the epidemic in terms of incidence, morbidity, and mortality (cf. Bloom et al., 2020b in the context of noncommunicable diseases). Compare, for instance, the impact of COVID-19 and that of HIV. In terms of severe morbidity and mortality, COVID-19 tends to be more concentrated among older adults and thus does not trigger a large direct loss in labor supply.¹⁶ HIV/AIDS is different, mainly because heterosexual sex is its dominant transmission mechanism, which means it mainly affects young or prime-age workers. Note, however, that considerable variation may occur across countries, where, in the case of COVID-19, the prevalence of severe cases tends to stretch to much younger age groups in some countries (cf. Dowd et al., 2020; Kass et al., 2020 point out that obesity may increase the incidence of severe cases among younger ages in the United States). (ii) Additional short-run reductions in labor supply may arise *indirectly* from the voluntary or mandated restrictions aimed to curb infection risk in the workplace, which tends to be the prime mechanism in the case of COVID-19. (iii) *Long-run* reductions in human capital arise if the epidemic stifles the incentives to invest in human capital.

5.2 Private consumption

Here, the following channels matter. (i) Aggregate consumption tends to be reduced *directly* through mortality and possibly through morbidity. The extent to which this is true tends to depend on the age pattern of the epidemic, in particular, of mortality in combination with the age structure of consumption (Kuhn and Prettnner, 2018). (ii) Voluntary or mandatory restraints on consumption to avoid infections lead to an *indirect* reduction in consumption. (iii) Foregone consumption (due to illness or voluntary restraint) translates into additional savings, and, perhaps more importantly, individuals are induced to engage in precautionary saving due to uncertainty. While an increase in savings may enhance long-run economic growth, the implied short-run shortfall of demand may, in the presence of rigidities, create severe unemployment (Guerrieri et al., 2020). (iv) The extent to which short-run

¹⁶ Bloom et al. (2020e) assess the often unmeasured and disregarded economic contributions of older adults, such as care for their grandchildren.

earnings losses (due to illness or shutdown) translate into reductions in consumption strongly depends on whether or not individuals have access to government insurance programs and transfers and to the capital market. (v) Expenditure is reallocated away from consumption to expenditures aimed to prevent or treat the disease. Such reallocation may occur directly in the case of private out-of-pocket payments for prevention or treatment or publicly in the case of tax-financed public prevention or treatment programs and indirectly as increases in health-insurance premia. Again, the extent to which private payments tend to curb consumption is prone to vary with the coverage of public health care, with the individual’s income, and with access to the credit market. In regard to increases in public expenditure, the impact through the commensurate tax increases may well be delayed. In particular, for shock-like epidemics, such as COVID-19, much of the current expenditure flowing into testing-and-tracking measures and the development of treatments and vaccines is prone to be financed through debt, which would be repaid only later.

5.3 Expenditure on health, education, and infrastructure

A priori, one would expect a strong increase in expenditure targeted to preventing and treating the disease. The extent to which these activities curb savings and capital investments is not initially clear. While current prevention and treatment costs can be understood as a form of consumption, the extent to which this lowers savings depends on the extent to which treatment costs are financed out of other private consumption or other expenditures (e.g., for infrastructure or education). Regarding the expenditures for developing future treatments or vaccines, they may be considered research and development (R&D) activities, which may well lead to the development of profitable new goods despite lowering the accumulation of capital. Here, a more thorough analysis would call for a multisector growth model, as is considered, for example, in Jones (2016), Kuhn and Pretzner (2016), or Frankovic and Kuhn (2018). Further impacts to the structure of public expenditure may arise if epidemics in which mortality is concentrated among particular age groups change the age structure of the population, which then requires readjustments of the transfer system.

As a result of the relative changes previously described (which depend on the disease under consideration, on the country that is modeled, etc.), goods and factor prices are likely to change. For example, a disease that strongly reduces the working-age population implies rising wages in the long run (such as in the case of the Black Death; see also Jedwab et al., 2020a). As another example, the supply chain disruptions that lockdowns and social distancing cause will put upward pressure on consumer prices, whereas the reduction in consumption due to these measures will work in the opposite direction. The total effect then depends on the relative strength of these effects, which, in turn, depends on the items under consideration.¹⁷

5.4 Key economic pathways

The effects of epidemics feeding through multiple macroeconomic transmission channels makes drawing general conclusions difficult. Table 1 summarizes some of the key pathways as linked to certain properties of an infectious disease and provides examples of diseases for which these pathways are particularly prominent.

Table 1: Key economic pathways and features of disease (assuming absence of effective treatments)

	Short-run impact		Long-run impact		
	Direct loss of labor supply	Behavioral or policy-related shock to	Reduction in return to human capital	Reduction in productivity of education	Structural changes: (i) Technical

¹⁷ Balleer et al. (2020) document these forces for the case of Germany during the first stages of the COVID-19 pandemic and find deflationary demand-side effects to dominate in the short run.

		consumption and labor supply			progress, sectoral change (ii) Trading patterns (iii) Stalling development
Disease properties / context	Epidemic; high infection rates (α) among working-age individuals; high mortality/morbidity ($\Phi \ll 1$); low rates of recovery, including scarring (γ) or tendency to become chronic	Epidemic; high infection rates (α) due to casual social encounters; unknown/unavailable means of prevention; high uncertainty	Endemic (i.e. high prevalence); high adult mortality (μ^f) and/or childhood diseases that involve scarring (including in-utero effects)	Endemic; high childhood infection rates; high loss in educational productivity ($\theta^f \ll 1$) due to morbidity and/or high parental morbidity/mortality	(i) New epidemics turning endemic in rich countries; (ii) true pandemics; (iii) endemic diseases with strong complementarities
Examples	1918 Influenza, HIV/AIDS (when endemic), tuberculosis, malaria	SARS, MERS, Ebola, COVID-19	HIV/AIDS, 1918 Influenza, tuberculosis	Malaria, hookworms, HIV/AIDS (orphanhood)	(i) & (ii) COVID-19 (iii) HIV/AIDS, malaria, tuberculosis

Broadly, we may categorize diseases according to their macroeconomic impact as follows: (i) Diseases that lead to a direct large-scale loss of labor, as they affect prime-age workers (rather than older adults or children). The impact increases with the extent of mortality and morbidity and, in the latter case, is exacerbated to the extent that recovery is impossible, for example, due to scarring. This implies a short- to mid-term supply-side shock to labor, leading to a short-term recession and mid-term adjustment issues due to an excessive capital intensity. (ii) Diseases that are transmitted through nonspecific social interaction in the workplace or during leisure (including most airborne pathogens and smear infection) have the potential to trigger precautionary reductions in consumption and labor supply and thus stronger short-run recessions unless other means for protection exist. Significant short-run reductions in labor supply and consumption are also the consequence of uncertainty, as associated with new diseases or incoherent policy responses (Baker et al., 2020).¹⁸ (iii) Endemic diseases with high infection risk over the life cycle, high morbidity and mortality, and poor chances of recovery tend to reduce the return to human capital and thereby stifle its accumulation. This will compromise long-run growth and development. (iv) Diseases that affect children tend to reduce the productivity of human capital accumulation and thus, again, stifle its accumulation. In addition, if these diseases lead to scarring, they may also directly trigger long-run productivity loss.

Finally, (v) various long-run structural effects are conceivable. These tend to differ by the stage of economic development. For rich and advanced economies, the emergence of new pandemic diseases and their possible transition into an endemic stage may trigger strong technological or sectoral responses.¹⁹ Here, the potential to face large, frequent, or unpredictable losses to labor induces firms to substitute capital for labor in the production process and thus may emerge as a driver of automation (Prettner and Bloom, 2020). Both workers and firms have incentives to rely on technologies that enable social distancing by working from home. Sectors that rely on social interaction either because it is inherent or for reasons of economies of scale (e.g., culture, entertainment, hospitality, sports, tourism)

¹⁸ Specifically, Baker et al. (2020) show that about 50% of the output loss in the course of the COVID-19 epidemic may be due to uncertainty.

¹⁹ The impulse for such changes is equally present within developing economies. However, due to their different sectoral structure and/or limitations in the resources to drive technological and organizational changes, they are likely to be followers. However, developing countries may be particularly vulnerable to sectoral changes, such as reshoring or reductions in tourism that originate in rich countries.

may face significant within-sector transformation (development of online culture, sports, entertainment) or shrinkage. Barrero et al. (2020) study such reallocations in the context of COVID-19. Large-scale epidemics may also turn out to be a driver of institutional change, for example, the construction of surveillance and early-warning systems or tracing and tracking and treatment facilities. In turn, the experience of an epidemic and the perceived success or failure of public policy responses are prone to shape the level of trust within societies and toward its institutions, governments, and experts (cf. Aksoy et al., 2020a, b; Brück et al., 2020, in the context of COVID-19). Finally, pandemics may give rise to deglobalization and/or reshoring, especially from the perspective of advanced economies with their extensive reliance on global supply chains. In contrast, from the perspective of poorer countries, infectious diseases pose a risk of stalling or even reversing economic development. As we have argued before, this is particularly true when diseases become endemic and when complementarities stifle investments into both education and health, possibly accompanied by a stalling fertility decline.²⁰

5.5 Distributional impacts

We highlighted earlier that heterogeneity both within societies and across societies is prone to shape the susceptibility to infectious diseases, the behavioral responses, and the outcomes. While such heterogeneity is reflected in the empirical studies of individual behaviors toward epidemics (discussed earlier), systematic economic research on how epidemics affect distribution within a society is surprisingly scarce.

For epidemics such as the Spanish Flu that yield a large negative effect on labor supply, one would expect at least a temporary increase in wages and reduction in the interest rate. While some historical evidence exists for both (cf. Garrett, 2009; Karlsson et al., 2014; Jordà et al., 2020), these authors have mostly avoided drawing inferences on the distributional implications. Boucekkine and Lafargue (2010) simulate the evolution of the income distribution within an overlapping generations economy that is subject to epidemic shocks to adult lives. Endogenizing the acquisition of human capital, they find that the presence of (recurring) epidemics may lead to an increase in the population share of the unskilled if orphans have poor access to education.

More extant literature describes how vulnerability to various infectious diseases is shaped by the income distribution and by poverty. While poverty and socioeconomic deprivation clearly raise vulnerability to several epidemics such as tuberculosis (cf. Duarte et al., 2018 for a survey) and malaria (e.g., Teklehaimanot and Mejia, 2008), the link is more complex for HIV/AIDS (e.g., Fox, 2012), where across Sub-Saharan African countries wealthier (poorer) individuals experienced higher infection rates in poor (wealthier) economies and where inequality itself was a driver of overall infection rates. On the premise that vulnerability to epidemics is related to poorer expected economic outcomes, this literature suggests the distributional consequences but again does not study them. A considerable amount of recent evidence has emerged for the link between poverty and deprivation and COVID-19. While the analyses by Jung et al. (2020) and Tubadji et al. (2020) suggest higher vulnerability in deprived areas in the United States and the United Kingdom, Palomino et al. (2020) and Galasso (2020) find that the disadvantaged are hit harder by lockdown measures and face poorer labor market outcomes. Considering survey data from six countries, Dang et al. (2020) also find evidence for the loss of labor income and reductions in saving among lower income strata. We conclude by noting that the underlying distributional impacts of epidemics—whether real or perceived—have the scope to

²⁰ Epidemic or endemic diseases can also be conceived as drivers of migration. While Beach et al. (2020) discuss migration in the context of the 1918 influenza pandemic, this appears less evident in the context of more modern epidemics, although arguably wherever infectious diseases stall economic development they contribute to migration incentives.

undermine social cohesion. This is in particular true when it comes to the apportionment of “guilt” and “blame” (scapegoating) in a crisis environment (cf. Jedwab et al., 2020b for a survey).

6. Policies and their effects on disease dynamics and economic outcomes

Reflecting the wide differences among infectious diseases in mode and risk of transmission, contagiousness, morbidity, mortality, duration, medical preventability, and treatability, policy approaches aimed at containing or even eradicating infectious diseases and mitigating their economic consequences tend to differ widely. In the following, we concentrate on highlighting a few exemplary approaches, touching on different aspects and stages of epidemics. Specifically, we consider policies aimed at (i) containing and controlling an epidemic through lockdown (of consumption and labor supply) and testing; (ii) the equitable provision of treatment; (iii) the prevention or eradication of the disease; (iv) the need for global policymaking in relation, for example, to the timely reporting of outbreaks and coordinated containment; and (v) macroeconomic stabilization in case of epidemic-induced recessions.

6.1 Lockdown and testing

Lockdown and testing policies have been at the forefront of the first wave of the COVID-19 pandemic, but strikingly have received relatively little attention before. Eichenbaum et al. (2020a) model lockdown policies as a tax on consumption in the budget constraint. In our case this would amount to

$$k_{t-1} - k_t = r_t k_t + \phi^i h_t w_t l_t - (1 + \tau_t) c_t - p_t v_t + \bar{\tau}_t,$$

where the tax rate τ_t is set to yield a desired reduction in consumption activities, and where $\bar{\tau}_t$ represents a lump-sum redistribution of the “tax proceeds.” Given that Eichenbaum et al. (2020a) do not allow for the accumulation of assets, that is, $k_{t-1} - k_t \equiv 0$, a forced reduction in consumption necessarily implies a forced reduction in labor supply, l_t . They find that even if individuals engage in private prevention by cutting labor supply and consumption, lockdown measures, that is, increases in τ_t above zero, tend to raise welfare by greatly curtailing the death toll even if they magnify the economic recession.

Collard et al. (2020) analyze the optimal timing of the intensity of lockdown. They find that optimal containment provides for relatively rigorous lockdown early on, which should subsequently be gradually relaxed with the progression toward herd immunity. They note, however, that measures should remain in place to counter excessive incentives on the part of individuals to rush back into activity. Notably, they find that the timing pattern very much hinges on whether or not the disease is associated with the risk of mortality peaks due to hospital congestion. If these are not present, the implementation of lockdown will be more gradual and less rigorous, as individual incentives for self-protection are strong enough or even excessive due to their failure to account for the external benefits from contributing to herd immunity. However, this result is overturned if individual failure to account for contributing to the risk of hospital congestion calls for strong and early lockdown. The analyses by Eichenbaum et al. (2020a) and Brotherhood et al. (2020) mirror these patterns.

Pointing out the lack of observability of infection with COVID-19, Eichenbaum et al. (2020b) consider a setting in which individual prevention efforts depend on their state of information. Assuming that a certain fraction of the population is (routinely) tested and thus fully informed about its infection status, they show that testing without quarantining identified infected individuals may, effectively, worsen outcomes, as in the absence of altruism those who are informed about an infection drop precautionary reductions in their activity. Combined with quarantining, a testing strategy yields considerable welfare

gains, however. Bloom and Glied (1991) point out similar welfare issues about testing in the context of HIV/AIDS.

Brotherhood et al. (2020) consider a similar setup, which, like Eichenbaum et al. (2020b), features young and old individuals, differentiated disease states—mild/feverish, which could also be flu, and severe/hospitalized—and altruism on the part of the infected. They show that testing triggers additional preventive effort on the part of altruists who are revealed to be infected and is, indeed, a prerequisite for targeted quarantine.²¹ They also show that testing should be targeted at the young, who, in the expectation of a mild course of disease, tend to be more reckless in their behavior while uninformed, whereas quarantine measures should be targeted at the old. Acemoglu et al. (2020) broadly confirm these findings within a social planner context. Outcomes can be improved further if strict quarantine is to some extent replaced by measures that allow within-age-group mingling but not across groups.

Glover et al. (2020) consider transfer payments to support individuals who are under lockdown to amount to a costly redistribution. They show that optimal lockdown policies not only trade off the future gains from a more favorable distribution of disease states against the instantaneous cost of output, but also against the cost of redistributing income. The costs of transfers thus constrain mitigation measures.

As a bottom line, while lockdown measures impose large macroeconomic costs to begin with, they are typically warranted on welfare grounds.²² The literature also tends to agree that they should be introduced early to achieve maximum leverage in terms of controlling disease progression and, in the absence of treatments or vaccinations, should subsequently be adjusted in a way that contains the disease at “manageable” levels while at the same time containing the loss in output and consumption. Here, sub-group-specific variations in the extent of quarantine tend to be much more effective and thus conducive to containing the macroeconomic cost. They require the implementation of an effective testing and tracking system.²³

6.2 Treatment

Effective treatments accelerate recovery, in terms of our model an increase in γ_t , or a reduction in morbidity or mortality, that is, an increase in ϕ_t^I and a decrease in μ_t^I , respectively. Apart from the immediate benefits to patients, treatments can have large macroeconomic value, especially for diseases such as HIV that offer poor (or, indeed, no) prospects for natural recovery and that are progressively debilitating and ultimately fatal. The macroeconomic gains to the development of effective antiretroviral treatments for HIV have accrued through two channels: First, by restoring productivity and curbing mortality, treatments have mitigated much of the loss in labor supply from prime-age workers (e.g., Bor et al., 2012; Thirumurthy et al., 2012). Second, by raising the return to human capital, the availability of treatments also fosters educational investments (Thirumurthy et al., 2012). Similar arguments apply to the provision of treatments for malaria and other tropical diseases.

²¹ In contrast to Eichenbaum et al. (2020a), Brotherhood et al. (2020) model age- and condition-specific quarantine by varying a “preference” parameter for teleworking and home-based leisure activities. This is also the way they model altruism, which thus has a “warm glow” nature rather than being based on the beneficiaries’ utility.

²² We have not touched here upon two additional important costs of extended lockdown related to (i) schools, which may strongly inhibit the accumulation of human capital, and (ii) health care providers who are not essential for the treatment of the infection, which may lead to rising mortality and productivity loss due to delayed diagnostics and treatment of other conditions (Maringe et al., 2020).

²³ Related analyses, leading to broadly similar results, include Alvarez et al. (2020), Bairoliya and Imrohorglu (2020), Berger et al. (2020), and Bethune and Korinek (2020). Caulkins et al. (2020) provide a rich analysis in which they show rather different policies in respect to the timing of lockdown to yield similar outcomes.

The availability of treatments also bears on risk-taking behavior, as for example Chan et al. (2016) and Greenwood et al. (2019) illustrate in the context of HIV. While changes in sexual practice in the context of HIV would have little macroeconomic impact, the arrival of treatments for diseases such as COVID-19 that spread easily through casual social interaction would have large effects on labor supply and consumption. Eichenbaum et al. (2020a) show that even the prospect of a treatment could vastly reduce social distancing and thus the economic recession that comes with it, albeit at an increase in the loss to life before the treatment becomes available.²⁴

Providing treatments raises a policy challenge if budget or capacity constraints imply the treatments are in insufficient supply, which is especially likely in the case of widespread and endemic diseases such as HIV or malaria or in the case of new pandemics such as COVID-19 (e.g., Bloom et al., 2020f; Emanuel et al., 2020). Anderson et al. (2012) study the nontrivial rationing problem that arises in a setting in which a limited supply of treatments needs to be allocated among several subpopulations who are subject to a susceptible–infected–susceptible (SIS) type of disease, such as malaria, where recovered individuals turn susceptible again. They show that, counter to common policy practice, minimizing the disease burden (= loss per infected \times number of infected) requires that treatments be concentrated on the subgroup(s) with the lowest share of infected individuals. The rationale behind this seemingly counterintuitive allocation lies with the SIS dynamics, where treating a limited number of infected protects a large number of healthy individuals from infection, which in turn bestows future protection on those treated. Technically, this amounts to a (dynamic) nonconvexity of the allocation problem, which is known to generate corner solutions. Bell and Gersbach (2009) come to a similar conclusion in a model in which a budget-constrained policymaker can allocate funds to treatment and educational subsidies in the face of an HIV epidemic. They find that targeting support to a limited number of households is optimal when the objective is to enhance long-run growth perspectives. They also show that if mortality is high, households should receive both treatments and educational subsidies; this double support, however, reduces the number of recipients even further. While these are stark conclusions, note that neither framework includes equality concerns in its objective.

6.3 Prevention and eradication

In particular for diseases in which private prevention by way of behavioral constraint leads to possibly large losses in economic output on top of individual utility losses, the introduction of effective vaccines or other medical or nonmedical prophylaxis is a panacea when it comes to containing or even eradicating an infectious disease.²⁵ Several issues arise in this context, however, which may require further policy intervention.

First, whenever access to or utilization of vaccines or protective devices imposes a disutility or monetary cost on the individual, underutilization typically occurs, as individuals do not internalize the full benefits their immunity bestows on others. Chen and Toxvaerd (2014) summarize the conditions for such an outcome and review the literature. Thus, appropriate subsidization of the price of prevention, p_t , or even payments for uptake are required (see, e.g., Geoffard and Philipson, 1996, 1997; Gersovitz and Hammer, 2004, 2005; Gersovitz, 2011); alternatively, prevention measures may need to be mandated at least for a part of the population. As outlined earlier, the problem becomes particularly prominent if the prevalence of the disease is low: Geoffard and Philipson (1997) show that

²⁴ Rowthorn and Toxvaerd (2020) study joint optimal prevention and treatment policies within a microeconomic SIS framework where recovered individuals turn susceptible again. They find that multiple equilibria may arise but that a decentralized allocation always tends toward too little prevention.

²⁵ As another means of insuring against the welfare loss from pandemics, governments may stockpile antibiotics. Attema et al. (2010) and Megiddo et al. (2019) study the option value of such policies based on calibrated models for the Netherlands and the United Kingdom, respectively, and find that stockpiling is a preferred option if the risk of a serious outbreak is sufficiently high.

as long as the full population cannot be covered, even compulsory vaccination programs may not be sufficient for eradication. Another problem is that reductions in the infection rate that result from introducing or improving preventive measures may be neutralized or even overturned through increases in risky behaviors similar to the case in which treatments become available. Toxvaerd (2019) shows that such disinhibition may not only boost disease prevalence, but also lead to a net reduction in welfare following an improvement in the effectiveness of prevention.

Second, barriers to prevention may be particularly high within developing countries and relate to both severe income and credit constraints and to a lack of information and education. Here, the subsidization or free provision of protective goods, such as condoms against HIV/AIDS or bednets against malaria and the provision of information on preventive measures have been shown to overcome barriers to prevention by poor households (e.g., Cohen and Dupas, 2010; Ashraf et al., 2013). Micro credits and insurance against income shocks have also been shown to play a role (Tarozzi et al., 2014; Burke et al., 2015). Education, especially girls' education, is considered to boost preventive behavior, although evidence in the context of HIV/AIDS remains mixed (cf. Alsan and Cutler, 2013; Behrman, 2015; Duflo et al., 2015). Finally, the empowerment of girls and women is considered to be conducive to disease prevention (Bandiera et al., 2020).

Third, the development of new vaccines and medical treatments for hitherto unknown pathogens, or for pathogens that become immune to current treatments, requires large upfront investments into R&D. Due to the extensive testing required for safety and efficacy, the development of medicines is a particularly lengthy process fraught with uncertainty. This implies that medical R&D absorbs large quantities of resources and raises funding issues. In particular, if carried out by private companies, the innovating firms will have to enjoy market power for them to secure the returns to their investments. This gives rise to the well-known problem of an undersupply of prevention and the possible strategic exclusion of poor consumers for the purpose of extracting profit (e.g., Kessing and Nuscheler, 2006; Forslid and Herzing, 2015). An additional issue arises when it comes to the eradication of a disease. Even if this is possible, private firms may be reluctant to provide the necessary supply for this, as they would eliminate their own future market. Geoffard and Philipson (1997) argue that such a setting calls for a deficit-financed eradication program, where future generations who benefit from the eradication would be taxed in line with their benefits.²⁶ Similar to this, Kremer et al. (2020) are propagating advance markets for new vaccines, where companies agree to sell at marginal cost but markup is topped up by pre-committed donors.

Analysis of the macroeconomic implications of large-scale medical research into the prevention of infectious diseases is scarce. What is known from the literature on medical and lifesaving innovations in general is that although their development may curb economic growth, this typically enhances welfare (Jones, 2016; Frankovic and Kuhn, 2018; Fonseca et al., 2020).

6.4 Global issues

In the context of pandemics (e.g., the COVID-19 outbreak) or of cross-country dispersion of endemic diseases (e.g., malaria or HIV), coordinated responses are essential for containment and eradication (Laxminarayan, 2016). This applies in particular to the timely reporting of local outbreaks; Laxminarayan et al. (2014) show empirically that reporting incentives depend on the circumstances: for example, the availability of common pools of treatments or vaccines would encourage reporting for the purpose of having early access, whereas their lack may discourage reporting for fear of trade

²⁶ Kremer and Snyder (2015, 2018) point out yet another distortion due to the (typical) presence of market power in the pharmaceutical industry, which is that for many diseases the private returns to investments in treatments are likely to exceed those from investments in prevention. They calculate that the deadweight loss from the provision of lucrative HIV treatments rather than vaccines may amount up to 62% of the global gain from curing HIV (Kremer and Snyder, 2018).

sanctions (cf. Malani and Laxminarayan, 2011; Saak and Hennessy, 2018 for models of epidemiological reporting games). Finally, initiatives toward the global eradication of infectious diseases lead to similar free-riding incentives at the country level as they do at the individual level, the difference being that countries are large players that can also enter negotiated agreements (e.g., Barrett, 2003). In this context, an optimal policy for a rich country may entail the provision of financial aid to a poor country in exchange for the poor country to shut down. This might be a particularly successful policy to prevent a pandemic when the initial outbreak occurs in a poor country that has difficulties in coping with shutdowns.

6.5 Macroeconomic stabilization policies

To soften the short-run economic impact that is predominantly caused by behavioral and policy reactions to the spread of an infectious disease and by rising uncertainty and supply chain disruptions, macroeconomic stabilization policies can be useful. Expansionary fiscal²⁷ and monetary policies, which have been applied on a large scale in response to the COVID-19 pandemic (see, for example, International Monetary Fund, 2020), are widely agreed to have reduced its adverse macroeconomic effects. However, in case of severe and fast-spreading epidemics, such as COVID-19, appropriate policy responses are often difficult to enact. The reason is that such pandemics affect aggregate demand and aggregate supply via different pathways within a rather short period of time. *A priori* what the relative importance of these two dimensions are and whether deflationary or inflationary pressures will prevail are unclear (cf. Balleer et al., 2020; Baqaee and Farhi, 2020; Brinca et al., 2020; Brodeur et al., 2020; Guerrieri et al., 2020 for discussions). For example, if supply chain disruptions predominantly cause the economic downturn (cf. Acemoglu and Tahbaz-Salehi, 2020), expansionary demand-side policies might be ineffective in stabilizing employment and could cause inflationary pressures. If, by contrast, uncertainty leads to a fall in investment and consumption, demand-side policies would be comparatively effective. A similar ambiguity applies to many of the lockdown measures that bear down on service markets, where again both demand and supply are affected.

Apart from these considerations, when COVID-19 broke out, many strongly affected countries faced comparatively high debt burdens (such as Italy and Spain), which reduced the scope for fiscal policy responses, and interest rates were comparatively low already, such that traditional expansionary monetary policies ran against the zero lower bound constraint. Thus, other more unconventional policies like massive amounts of quantitative easing and helicopter money have also been proposed (see, e.g., Céspedes et al., 2020; Didier et al., 2020; Elenev et al., 2020 for discussions of such policies and their effects).

At this point noting a potential conflict of objectives between expansionary economic policies that are mainly aimed at increasing consumption and labor supply and health policies that actually aim to reduce the interaction of people along the lines discussed previously is important. In this case the optimal timing of policies is of crucial importance: opening up the economy too soon and providing massive economic stimulus could actually increase the spread of infectious diseases as the experience with COVID-19 in many countries at the time of writing (July 2020) plainly makes clear.

7. Macroeconomic effects of and policies toward specific infectious diseases

²⁷ Note that expansionary fiscal policies can come in many forms. While infrastructure investments are a typical example, they require a long time period to be implemented and might come too late in case of a pandemic-driven recession. Other measures such as wage subsidies, expansions of unemployment insurance, equity injections, and loan guarantees would work more quickly (Céspedes et al., 2020; Didier et al., 2020; Elenev et al., 2020).

We now provide an overview of selected major outbreaks of different diseases since World War II. Table 2 summarizes various diseases and outbreaks, the main affected world regions, the time period, and the number of infections and deaths as estimated by various sources.

The late 20th and early 21st centuries have seen various infectious disease epidemics and pandemics. Many remain ongoing, such as HIV/AIDS, which had claimed 32 million lives by the end of 2018 according to UNAIDS (2019); malaria, which caused about 400,000 deaths worldwide in 2018 according to the World Health Organization (2019a); and tuberculosis, with 1.5 million deaths in 2018 according to World Health Organization (2019b). For comparison, the all-cause mortality in 2017 was close to 56 million deaths according to the Institute of Health Metrics and Evaluation (2018). While HIV/AIDS and tuberculosis can be found worldwide, these diseases are endemic mostly in poor countries. The economic consequences of these diseases are substantial and discussed further in later sections, but fortunately, progress against HIV/AIDS and malaria has occurred over the last two decades. This cannot be said, however, for dengue fever, which apart from the emergence of new diseases (e.g., Ebola, SARS, MERS, Zika, COVID-19) is on the rise (cf. Bloom et al., 2018; Bloom and Cadarette, 2019). In the case of some diseases, the prospect of re-emergence exists should containment efforts be relaxed, which is particularly true for poliomyelitis. Unfortunately, in this context, efforts to rein in COVID-19 through social distancing and lockdowns might lead to reductions in vaccination against other diseases, which could foster their re-emergence. This could be, for example, because visits to health centers for vaccination are reduced and resources otherwise used for vaccination against these diseases dry up (cf. Hogan et al., 2020).²⁸

Table 2: Important infectious diseases, notable outbreaks over the last decades, and their human toll

Disease	Region	Time	Infections and Deaths
HIV/AIDS	Worldwide	1981–ongoing	75 million people infected and 32 million people have died as of the end of 2018 (UNAIDS, 2019)
Malaria	Mainly Africa, South Asia, and South America	Ongoing	228 million cases in 2018 (down from 251 million in 2010) and 405,000 deaths (down from 858,000 in 2010), according to the World Health Organization (2019a)
Measles	Worldwide	Ongoing	140,000 deaths in 2018 globally with the vast majority of deaths among children below the age of five (World Health Organization, 2019d)
Cholera	Frequent outbreaks reported mainly in Africa and South Asia	Ongoing	2.86 million estimated cases per year and 95,000 estimated deaths (Ali et al., 2015)
Hepatitis	Worldwide	Ongoing	1.34 million estimated deaths in 2015 (World Health Organization, 2017)
Dengue fever	Africa, the Americas, the Eastern	Ongoing	390 million cases estimated per year in 2015 (World Health Organization, 2020c), with incidence increasing strongly

²⁸ COVID-19 has interrupted many routine immunization programs. For example, WHO, UNICEF, and Gavi have reported that the pandemic is forcing 80 million children to at least temporarily forego vaccine protection against one or more disease-causing pathogens. This corresponds to roughly 60% of the world’s annual birth cohort. Such disruption threatens to elevate rates of vaccine-preventable diseases and their physical, mental, and cognitive health sequelae across the life cycle and to deprive individuals, families, and societies of the full health, economic, and social benefits of vaccines, which are often considerably larger than the narrow benefits of avoiding infection by the target pathogen and related medical care costs (see Bloom, Fan, and Sevilla (2018).

	Mediterranean, Southeast Asia, Western Pacific		over the past decades; 40,500 estimated deaths per year according to GBD 2017 Causes of Death Collaborators (2018)
SARS	Worldwide	2002–2004	8,096 infected and 774 deaths (World Health Organization, 2020a)
H1N1 (swine flu pandemic of 2009)	Worldwide	2009–2010	18,500 confirmed deaths and 201,200 estimated deaths (Dawood et al., 2012)
MERS	Worldwide	2012–ongoing	2,494 confirmed cases and 585 deaths by the end of November 2019 (World Health Organization, 2020b)
Rabies	Africa, Asia, Central and South America	Ongoing	59,000 estimated deaths in 2017 (World Health Organization, 2020d)
Tuberculosis	Worldwide	Ongoing	10 million new infections and 1.5 million deaths in 2018 (World Health Organization, 2019b)
Ebola (Western African Epidemic of 2014–2016)	Mainly Africa (Guinea, Liberia, Sierra Leone)	2014–2016	15,261 total confirmed cases and 11,325 total deaths (Centers for Disease Control and Prevention, 2020a)
Zika	Worldwide (mainly the Americas)	2015–2016	51 reported deaths as of 2019 (Cardona-Ospina et al., 2019)
Poliomyelitis	Previously worldwide, as of 2020 endemic transmission only in Afghanistan and Pakistan	Ongoing	33 reported cases in 2018, down from 350,000 estimated cases in 1988 (World Health Organization, 2019c). If the disease is not fully eradicated, a global relapse and 200,000 new cases per year within 20 years are projected.
Influenza	Worldwide	Ongoing	389,000 estimated global deaths per year over the time frame 2002–2011 (Paget et al., 2019)
Yellow fever	Tropical and subtropical areas in South America and Africa	Ongoing	200,000 estimated cases and 30,000 estimated deaths per year (Centers for Disease Control and Prevention, 2020b)
Japanese encephalitis	Southeast Asia and Western Pacific	Ongoing	100,308 estimated cases and 25,125 estimated deaths in 2015 (Quan et al., 2019)
COVID-19	Worldwide	2019–ongoing	Over 22 million confirmed cases and 800,000 deaths globally as of August 20, 2020

Table 3 displays the disability-adjusted life years (DALYs), years of life lost (YLLs), years lost due to disability (YLDs), and the economic impact of those diseases in Table 1 for which the World Health Organization (2016) provides corresponding data. In addition, we use a rule of thumb proposed by the World Health Organization (2011) to compute a range of the economic burden of these diseases in column 5 of Table 3. The economic burden is expressed in billions of purchasing power adjusted international dollar (ID) with a base year of 2017. We observe a particularly high economic burden

related to HIV/AIDS (949–2,847 billion ID), malaria (591–1,774 billion ID), and tuberculosis (817–2,452 ID).

Table 3: DALYs, YLLs, YLDs, and economic impact of selected infectious diseases in 2016

Disease	DALYs	YLLs	YLDs	Economic impact in billion purchasing power adjusted ID (base year: 2017)
HIV/AIDS	59,951,098	55,922,099	4,028,999	949–2,847
Malaria	37,368,766	35,628,487	1,740,279	591–1,774
Measles	7,957,226	7,935,125	22,101	126–378
Hepatitis	7,464,904	7,185,133	279,771	118–354
Dengue fever	3,099,919	2,119,014	980,905	49–147
Rabies	1,570,559	1,570,423	136	25–75
Tuberculosis	51,642,599	48,804,779	2,837,820	817–2,452
Yellow fever	739,346	739,245	101	12–35

Notes: We include those diseases from Table 1 for which the World Health Organization (2016) provides data on DALYs, YLLs, and YLDs. YLL measures the mortality impact of a disease and YLD the morbidity impact. By definition, the relation $DALY=YLL+YLD$ holds. The economic impact of the diseases is calculated based on the rule of thumb suggested by the World Health Organization (2011) that the economic burden of one DALY is equivalent to between one and three years of lost per capita income. For the calculations we used global averages because for some countries the data for specific diseases are missing in the World Health Organization (2016) dataset. Per capita income in the year 2016 is taken from the World Bank (2020) and refers to purchasing power adjusted ID with a base year of 2017.

In the following subsections, we discuss the economic consequences of HIV/AIDS, malaria, tuberculosis, influenza, and COVID-19 in more detail. The discussion is based on the pertinent economic literature on the economic consequences of these diseases.

7.1 HIV/AIDS

Having first emerged during the 1980s as a sexually transmitted disease affecting in particular sexually promiscuous communities in industrialized countries, HIV/AIDS quickly spread worldwide and subsequently turned into an endemic disease in Sub-Saharan Africa, where it continues to impose a large burden on welfare and economic development. Economic research on HIV/AIDS has covered many areas, including prevention incentives and labor supply effects (some of this quoted elsewhere in this paper). Here, we focus on a selection of macroeconomic-oriented works studying the two key pathways for HIV/AIDs (cf. Table 1), namely the reduction in labor supply and the consequences of high mortality (during its early stages) on human capital investments and fertility.

Young (2005) estimates a Beckerian child quality–quantity tradeoff model relying on South African data and uses the resulting parameter estimates to simulate the effects of HIV on per capita GDP. In his framework, HIV reduces fertility to such an extent that the corresponding increase in human capital accumulation due to fertility reduction overcompensates the HIV-induced reduction in human capital of orphaned children. Based on these results, Young (2005) concludes that the overall effect of the HIV epidemic on South Africans who do not suffer from the illness is actually positive. In contrast to these results, Over (1992), Cuddington (1993), Corrigan et al. (2005), and Bell et al. (2006, 2007) calculate a comparatively large *negative* effect of HIV on the long-run level of per capita GDP, but in models based on exogenous fertility. Exogenous fertility switches off the main channel that Young (2005) considers in his analysis.

While Young (2007) corroborates his earlier results that HIV reduces fertility and thereby fosters development, Fortson (2009) finds the negative effect of HIV/AIDS on fertility to be lower than shown by Young (2005). In addition, Juhn et al. (2013) show that the epidemic has no effect on the fertility of noninfected women (an important channel for the results of Young, 2005 to emerge), and Kalemli-Ozcan and Turan (2011) and Kalemli-Ozcan (2012) even find a positive effect of HIV on fertility. In a similar vein, Fortson (2011) estimates that the effect of HIV on human capital accumulation tends to be more severe than Young (2005) shows because of the Ben-Porath mechanism that higher life expectancy induces investments in education (Ben-Porath, 1967; Cervellati and Sunde, 2013), which Young (2005) disregards in his analysis.

Azomahou et al. (2016) and Gori et al. (2020) build on these insights and construct models of the impact of HIV on economic growth that are more in line with the empirical findings and that feature a richer set of mechanisms than the one Young (2005) addresses. Allowing for an effect of the Ben-Porath mechanism on human capital accumulation and endogenizing the saving choice of individuals, Azomahou et al. (2016) find that HIV does not have a large short-run effect (which is consistent with the empirical findings of Bloom and Mahal, 1997 and Mahal, 2004), but that the adverse long-run growth effects can be sizeable and reduce per capita GDP by about 3%.

To summarize, while relevant mechanisms could lead to a positive economic impact of HIV/AIDS, as suggested by Young (2005, 2007), these effects are empirically rather weak and likely to be dwarfed by adverse economic effects that Young (2005, 2007) does not consider in his analysis.

7.2 Malaria

Malaria is another disease with a strong impact on labor supply through both mortality and morbidity, but, to the extent that children become infected, also through reductions in the productivity of education (cf. Table 1). To estimate the economic impact of malaria, Gallup and Sachs (2001) regress the average growth rate of 75 countries between 1965 and 1990 on an initial malaria index and control variables. According to their results, a 10% reduction in the malaria index predicts a sustained annual increase in economic growth of 0.3 percentage points per year. Extrapolating beyond the variation in their sample, Gallup and Sachs (2001) calculate that malaria eradication would imply a rise in per capita income growth of 2.6 percentage points on average. More recent contributions confirm a positive economic effect of malaria eradication, but conclude it is quantitatively much smaller (Lucas, 2010; Berthélemy and Thuilliez, 2015).

Bleakley (2010) relies on malaria-eradication programs in Brazil, Colombia, Mexico, and the United States around 1955 to identify the causal impact of malaria exposure in childhood on adult labor productivity. He finds that disease eradication in the worst-hit Latin American countries boosts incomes by 25%, while the figure for the United States (with a lower malaria exposure) was 12%. While Bleakley (2010) finds mixed results on the effect of malaria eradication on years of schooling, he finds a positive effect on literacy. Lucas (2010) corroborates this result, also finding a positive effect on literacy and on years of schooling based on data from the malaria eradication campaigns in Paraguay and Sri Lanka.

For a discussion of optimal malaria treatment strategies and how they change in the presence of increasing resistance to drugs and with a change in the time horizon, see Laxminarayan (2004). Smith et al. (2013) elaborate on the benefits of malaria eradication for countries when other countries do not follow such a strategy and show that disease eradication can nevertheless be successful and lasting. For the problems that require using the COI approach to estimate the burden of malaria see Chima et al. (2003).

7.3 Tuberculosis

Given its large disease burden, as measured in DALYs, and resulting economic burden, which of the diseases considered in Table 3 is second only to HIV/AIDS, tuberculosis has received surprisingly little attention from economic analysis. While being associated with high levels of debilitating morbidity, 95% of DALYs lost result from premature loss in life years (see also Laxminarayan et al. 2007 on this point). Thus, the channels through which tuberculosis affects the economy are similar to those for HIV/AIDS, namely a loss in labor supply and a reduction in the return to human capital (cf. Table 1).

Employing a combined VSL and full income approach, Laxminarayan et al. (2007) evaluate the economic burden for the 10-year period 2006–2015 for 22 high (disease) burden countries in which tuberculosis is endemic. They find it to range between 2006 USD 3.33 billion in Zimbabwe to 1,175 billion in China. Strikingly, they also find that although high-burden countries in Sub-Saharan Africa account for one third of the fatalities, they only account for one tenth of the burden. Laxminarayan (2007) also assess the economic benefit of extending novel multicomponent (DOTS) treatment programs that include directly observed treatment and short-term chemotherapy and find that the benefits exceed marginal costs by a factor of 15 across the 22 high-burden countries.

Verguet et al. (2015) apply extended cost-effectiveness analysis (ECEA) to assess the impact of universal public finance of tuberculosis treatments in India that is aimed at increasing uptake while at the same time insuring households against catastrophic expenditure. ECEA incorporates the benefits of financial protection into conventional cost-effectiveness analysis of medical treatments. The authors find that health gains and insurance value accrue primarily to the poor, suggesting that universal public finance may play a redistributive role. Verguet et al. (2017) use a simulation model to assess the impact on the incidence of catastrophic costs for households of expanding aggressive treatment strategies against tuberculosis in India and South Africa and facilitating access to treatments through intensified case finding in South Africa. They find that more aggressive treatments would reduce the incidence of catastrophic costs by 6–19%, predominantly benefiting the poor, whereas more intensive case finding would lower catastrophic costs by 5–20% but only after a period of 5–10 years.

7.4 Influenza

That seasonal outbreaks of influenza have become a part of everyday life in many parts of the world obscures both the human and economic toll of “the flu,” which is rising annually even in the absence of deadly pandemic outbreaks such as the one in 1918 (see also Beach et al., 2020). As a “commonplace” disease, the pathways are somewhat diluted, although major outbreaks are likely to trigger strong behavioral and policy-induced reductions in labor supply and consumption (cf. Table 1).

Smith et al. (2009) study various scenarios of a (hypothetical) influenza pandemic and its impact on the year 2004 U.K. economy. They identify annual losses ranging between 0.5% and 4.3% of GDP depending on the assumptions about fatality. They find that losses fall to between 0.13% and 2.3% for a prepandemic vaccine and to between 0.3% and 4.3% for a matched vaccine (single dosage). In a related analysis, Keogh-Brown et al. (2010b) study the economic impact on the United Kingdom of an influenza outbreak similar to the 1957 or 1968 one and find a relatively moderate GDP loss of 3.35% during the first quarter of the outbreak and 0.58% during its first year. These figures increase to 9.5% and 2.5% once behavioral reductions in consumption are accounted for. A more severe outbreak with 1% mortality would then lead to GDP losses of 29.5% and 6%, respectively. Keogh-Brown et al. (2010a) and Smith et al. (2011) extend these analyses to cover pandemics across European economies and the global economy and come to results within a similar order of magnitude.

Ward (2014) studies the value of an expanded influenza vaccination program against a U.S. background. She finds considerable gains in terms of medical cost savings and avoided earnings losses albeit subject to decreasing returns as coverage extends to nontarget groups. She also identifies strong

external benefits. Hollingsworth et al. (2011) study optimal mitigation strategies for an influenza pandemic akin to the 1918 one. Given inherent tradeoffs between different policy targets, they highlight a need for prespecifying policy priorities.

7.5 COVID-19

In the absence of complete and reliable evidence, much of the analysis on the economic burden of COVID-19 remains somewhat tentative. Referring to recent modeling and evidence throughout all parts of this paper, we focus here on a selection of studies that illustrate the scale of the issue. Corresponding to the very strong behavioral and policy-induced reductions in consumption and labor supply that are rooted in the fast spread of a new and dangerous pathogen (cf. Table 1), these studies focus to large extent on the impact of lockdown measures.

Eichenbaum et al. (2020a) merge an SIR model with a simple general equilibrium macroeconomic model sharing many of the features of the model proposed in Sections 4.1 and 4.2. Consumption goods are produced with labor only, and both production and consumption entail the risk of infection. Individuals fail to internalize that their own decisions to reduce their individual infection risk also reduces the infection risk of others, which justifies governmental interventions to create a stronger economic downturn. The model does not feature other preventive activities or individual heterogeneities.

Eichenbaum et al. (2020a) show that their baseline SIR model of the COVID-19 pandemic in the United States, without behavioral reactions and economic feedbacks, leads to 215 million persons being eventually infected and 2.2 million eventually dying. In and of itself, this leads to a relatively mild recession with aggregate consumption dropping by 2% from peak to trough. Behavioral reactions of individuals and the corresponding general equilibrium feedback mechanisms lead to a more severe recession, with the aggregate consumption drop being more than 9% from peak to trough but with fewer people becoming infected and the death toll falling from 2.2 million to 1.74 million. Causal econometric evidence from Korea corroborates these simulation results, where Aum et al. (2020) show that even in the absence of lockdown policies, labor supply drops by 2% to 3% in response to COVID-19 outbreaks.

Krueger et al. (2020) extend the model of Eichenbaum et al. (2020a) to allow for heterogeneity in consumption goods and in labor supply. Some goods can be consumed at home without the need to interact with others and thereby without the risk of infection (cf. Leibovici et al., 2020). By the same token, some types of work can be done in a home office (cf. Dingel and Neiman, 2020). Krueger et al. (2020) show that this reasonable extension changes the outcomes drastically in the sense that—as compared with the model without heterogeneity— 80% of the economic decline is mitigated, while, at the same time, 80% of deaths are avoided without any governmental intervention.²⁹ When addressing governmental containment measures, which they model in terms of taxes on consumption, Eichenbaum et al. (2020a) find that the optimal policy is to raise the tax over time in line with the infection rate. In their optimal scenario the tax rate rises from 2.3% in the first period to 47.3% in period 40. This leads to a much deeper drop in aggregate consumption (by 21% instead of 9%), but saves 600,000 lives in the long run.

In a similar structured model, involving, however, heterogeneity in age, uncertainty about disease state, and the possibility of testing, Brotherhood et al. (2020) examine the implications of a rich set of lockdown policies for the calibrated U.S. economy. They show that while voluntary reductions in social

²⁹ The results depend on the assumed elasticity of substitution and on the size of an autonomous infection risk. The decline in the losses to both economic performance and lives relative to a setting without substitution are nevertheless substantial in all scenarios.

interactions halve mortality and lead to an average loss of GDP of 3% in the laissez-faire economy, testing (all symptomatic individuals) and quarantining (positive cases) policies have the potential to reduce mortality by an order of magnitude and recuperate a significant share of the GDP loss. While these effects diminish for imperfect or incomplete testing, they prove to be welfare superior throughout. Universal lockdown policies tend to trigger a substantive decline in GDP. Even a mild lockdown shutting down 25% of activity for one month will lead to an additional loss of GDP by 2% relative to the benchmark without saving a substantial number of lives. Strikingly, the cost of a life saved stands at USD 116 million and thus about tenfold the conventional VSL figures (Viscusi and Aldy, 2003).³⁰ In contrast, a very strict lockdown, shutting down 75% of activity for 35 weeks leads to a drop in GDP of 46% relative to the benchmark but virtually eliminates all deaths. In this case, the cost of one life saved stands at only USD 16 million and thus close to the VSL.³¹ In line with other authors (e.g., Acemoglu et al., 2020), they find that lockdowns that are targeted at the old are helpful in lowering the GDP loss per life saved.

As these studies are cast against the backdrop of rich, industrialized economies, Alon et al. (2020) and Arellano et al. (2020) model a COVID-19 outbreak from the perspective of developing and emerging economies. Alon et al. (2020) take explicit account of the fact that developing economies tend to have younger populations, lower fiscal capacity, a larger informal sector, and a lower health care capacity. Their simulation results show that uniform lockdowns are considerably less effective in developing economies than they are in developed economies: they only save about half as many lives per unit of GDP lost. Conversely, the effectiveness of lockdowns targeted at the elderly population are considerably more effective. The finding that the tradeoff between livelihoods and lives lost underlying these findings hinges on a country's income level is supported by empirical evidence in Decerf et al. (2020). Taking a perspective on lockdown in emerging economies, Arellano et al. (2020) show that the risk of a debt crisis may seriously inhibit lockdown policies and conclude that relaxing the tradeoff between health and debt crises makes a strong case for debt relief.

8. Conclusions

We assess the effects of modern infectious disease epidemics and pandemics on macroeconomic performance by (i) describing the role of health in driving economic growth and well-being; (ii) discussing standard frameworks used to calculate the economic burden of diseases; (iii) sketching a theoretical framework to analyze the tradeoffs and the macroeconomic repercussions in the case of epidemics; (iv) discussing policy interventions; and (v) providing an overview of various infectious diseases of the late 20th and early 21st centuries, their human toll, and their economic effects.

In general, the economic effects of epidemics depend to a great extent on heterogeneities in three dimensions: (i) disease-specific heterogeneities in terms of mortality, morbidity, infectiousness, and prospects for recovery. The economic impact through the loss of labor is particularly high in the case of diseases that predominantly affect prime-age workers rather than older adults or children. The impact is also exacerbated if recovery from a disease is not possible (as in the case of HIV/AIDS) and if diseases are transmitted through nonspecific social interactions, which has the potential to trigger larger reductions in consumption and labor supply and thus more severe short-run recessions. Endemic diseases with high infection risk, high morbidity and mortality, and poor chances of recovery tend to reduce the return to human capital and stifle its accumulation, which compromises long-run

³⁰ Hall et al. (2020) estimate that individuals would be willing to forego 41% of their yearly consumption on average to avert death from COVID-19 if the average COVID-19 mortality rate stands at 0.81%.

³¹ Ichino et al. (2020) consider the tradeoff between lives saved and loss in GDP for different lockdown policies in the case of Italy. Somewhat in contrast to the findings by Brotherhood et al. (2020), they show that from some point further increases in the strictness of the lockdown are no longer effective in saving lives and only lead to a further loss in GDP.

growth and development by sustaining poverty traps. (ii) Heterogeneities at the population level in disease susceptibility, such as the population share of older adults who might be more susceptible to or be more severely affected by a disease. This has repercussions on infection dynamics and on the extent to which medical resources are needed and lockdown measures need to be imposed. In this context, the share of poor people who are at a higher risk of infection because they often need to rely on public transport and often cannot work from home is also highly relevant. Poor individuals often have no (or inadequate) access to health care, which prevents them from seeking diagnosis and treatment with further repercussions on transmission. Finally, poor individuals must often work even when sick, for example, because they do not have substantial savings to afford a long-term income loss or because they have to support a large family. Here, a lot depends on the extent to which social security systems can help to reduce the income loss of sick individuals and the extent to which poor people get access to diagnosis and treatment. (iii) Cross-country heterogeneities are decisive in the sense that richer countries might be able to sustain lockdown measures and social distancing for a longer time period than poorer countries, in which many people depend on their ability to participate in market activities. Optimal lockdown measures will therefore often be very different in rich countries than in poor countries.

Overall and given these heterogeneities, some countries can cope comparatively well with epidemics like HIV/AIDS, while for other countries such a disease implies a poverty trap from which they cannot escape without massive assistance from other countries and donors. By the same token, a disease like COVID-19 might come with enormous economic costs in rich countries but health systems might by and large be able to cope with the disease. In poor countries, however, health systems might be overwhelmed quickly (at much lower infection rates) such that death rates would increase, potentially also for other diseases, for which treatment is postponed. For example, vaccination against other dangerous infectious diseases, such as poliomyelitis, might drop because the programs cannot be sustained in the face of another epidemic, leading to a re-emergence of the disease. Overall, an epidemic that might mainly create economic hardship in rich countries could potentially lead to humanitarian catastrophes in poor countries.

Heterogeneities and inequality not only affect the spread and the economic impact of epidemics, but there is also the reverse channel of epidemics hitting people with lower incomes particularly hard. This is because (i) working from home as a coping mechanism is often not feasible for many low-income jobs; (ii) poor access to health care and insurance schemes and reliance on public transport increase susceptibility to the diseases and the potential costs when falling ill; (iii) the increased incentive to invest in automation in the course of epidemics predominantly lead to a replacement of routine and low-wage jobs (Prettner and Bloom, 2020); and iv) while efforts to re-shore economic activity in the face of supply chain disruptions and travel restrictions (e.g., in the production of medical supplies) might be seen to present an opportunity for domestic workers, re-shoring tends to be associated with more automated production, such that low-income persons would not benefit (Krenz et al., 2020) and global inequality could rise. A particular challenge for policymakers is therefore to reduce the inequality-increasing tendencies of epidemics when designing policies to cope with epidemic-induced economic downturns.

We conclude on the note that while this paper has focused on the impact of epidemics on the economy, an important reverse channel also exists relating to the impact of economic activity—and in particular trade and labor migration—on the transmission of diseases (e.g., Oster, 2012b; Adda, 2016; Qiu et al., 2020). Furthermore, the likely origin of the recent outbreaks of COVID-19, Ebola, and Zika—but also more distantly of HIV/AIDS—in the transmission of pathogens from animals to humans (zoonosis) has raised issues about the reverse pathway from economic development to the risk of epidemics. While disease ecologists have studied intensively the relationship between habitat loss and

loss in biodiversity and the spread of zoonotic infections (e.g., Lloyd-Smith et al., 2009; Johnson et al., 2015), much remains to be learned on the economic development aspects of the problem (Dobson et al., 2020).

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