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1

Epidemics and Institutions

> In many cases the houses of the dead had to be shut up, for no one dared enter them or touch the belongings of the dead. No one knew what to do. Everyone, one by one, fell in turn to death's dart.
>
> —ACCOUNT OF THE BLACK DEATH IN PIACENZA BY THE NOTARY GABRIELE DE MUSSIS, 1348–56[1]

In the early 1340s, the Black Sea port of Caffa was a city of about 17,000 households from many cultures—Italian, Mongolian, Jewish, Greek, Armenian, Vlach, Trapezuntine, Circassian, Mingrelian, Sutar, Polish, Georgian, Russian, Tartar, Turkic, Arab, Iranian. Genoa, an Italian city-state, had bought the place 80 years earlier from the Golden Horde, after which Caffa flourished as a trade hub.[2] But in 1343 the khan of the Golden Horde resolved to strengthen his own authority and that of Islam. He sent an army to harry European trading posts in the Black Sea region, confiscating wares and ships, ejecting Italians from the Venetian entrepôt of Tana on the Sea of Azov and pursuing them to the

1. Quoted in Horrox 1994, 23. De Mussis, a notary (lawyer) who lived from *c*.1280 to *c*.1356 in the Italian city of Piacenza, did not personally witness the rise of plague in Caffa. His *Istoria de Morbo sive Mortalitate quae fuit anno dni MCCCXLVIII* (History of the Disease, or the Great Dying, which took place in the year of our Lord 1348) dates from between 1348 and 1356.

2. The "Golden Horde" is the most commonly used term for the Mongol (later Turkicized) khanate that was established in the thirteenth century in the north-western sector of the Mongol Empire. The name this khanate used to refer to itself was "Ulug Ulus", Turkic for Great State. The expression "Golden Horde" emerged in the sixteenth century, when Russian chroniclers began to refer to this khanate as the "Zolotaia Orda", the Golden Camp or Palace. Modern specialists sometimes refer to it as the "Kipchak Realm" or the "Jochid Realm". For reasons of intelligibility, this book uses "Golden Horde" as the mostly widely understood term for this complex medieval polity.

Genoese fortress at Caffa, which he besieged. In 1346 bubonic plague—probably originating in Mongolia, where marmots kept it smouldering—blazed up in the khan's army. In the vivid account of Gabriele de Mussis, a Piacenza notary, the epidemic broke up the besieging army but also the city of Caffa, whose inhabitants fled in all directions.[3] Other accounts describe how plague arose in the Mongolian army after the siege, diffused around Crimea to Venetian Tana, and voyaged across the Black Sea with grain shipments when trade embargoes were relaxed in 1347 after the hostilities.[4]

Over the next seven years, plague travelled by sea to Constantinople, Alexandria, Sicily, Genoa, and Venice, and thence throughout the Mediterranean, Asia Minor, and Europe. It journeyed with merchants, sailors, soldiers, refugees, pilgrims, migrants, and the rats and fleas that accompanied them.[5] It moved at a pace of 40 kilometres daily by ship, 2 by land in the warm and populous south, and 0.5 by land in the cold and desolate north, reaching nearly every society in Asia Minor, the Middle East, North Africa, and Europe by 1353.[6] This disease became the most lethal epidemic in recorded history, the Black Death, killing 33–58 per cent of the entire population in the societies it struck.

Each of the institutions analysed in this book—market, state, community, religion, guild, and family—played a role in this spectacular act of contagion. The market brought immunologically naive Italians to a region where they met carriers of plague from the Mongolian steppe and bore the infection onwards to societies where plague had been rare or absent for centuries. The state established the privileged Italian entrepôts in Crimea and brought the Golden Horde army to Caffa, setting up sieges and encampments where humans, rats, and fleas crowded together, along with their waste.[7] Local communities in Italy repulsed returning ships, sending them onwards to other communities, which in turn ejected marginal inhabitants such as paupers, beggars, Jews, and

3. Wheelis 2002; Slater 2006, 271–2, 274–7; Benedictow 2004, 44, 49–54, 60–1, 64, 69, 130, 181, 183, 212, 227; M. Harrison 2012, 3; Benedictow 2021, 164, 178–9, 187, 248, 431, 451; Favereau 2021, 248–9, 256.

4. H. Barker 2021; Favereau 2021, 249–51.

5. Plague was probably transmitted from the Black Sea region to Europe through a multiplicity of routes and mechanisms in 1347. But most scholars think that the Black Death emerged from encounters between Mongol armies and European merchants in the Black Sea region, in which military, political, commercial, and religious forces combined to create a perfect epidemiological storm.

6. On the pace of transmission, see Benedictow (2004, 229–31). Biraben (1975, 90) estimates a faster diffusion of plague, at a rate of 0.66–5 km/day on land, averaging 4 km daily.

7. As discussed earlier, this medieval polity referred to itself as "Ulug Ulus", Turkic for "Great State". On the reasons for regarding it as a "state" in the fourteenth century, see Trepavlov (2018) and Favereau (2021, esp. ch. 7).

migrants, who then carried disease into nearby villages and on to new towns. Religion created pressures in the Golden Horde, which brought a Muslim army to Caffa, and soon impelled penitent Christianity to organize superspreader gatherings in plague-struck Europe. Guilds of physicians, surgeons, and apothecaries claimed exclusive privileges over their professions, defending ancient knowledge against disruptive innovations, and limiting the supply of medical services to patients and communities. Families insisted on deathbed and funeral gatherings in the teeth of official prohibitions, as in Piacenza in 1348, where, "when one person had contracted the illness, he poisoned his whole family even as he fell and died, so that those preparing to bury his body were seized by death in the same way".[8]

But each of our six institutions also helped to mitigate this epidemic, and many others before and since. The market responded by shutting down even without being ordered to do so, as in Alexandria and Bilbais, where the caravanserais closed in 1350 not by public order but when merchants and customers refused to attend.[9] Market exchange created gains from trade and rising incomes, helping people accumulate savings so they could stay away from the market temporarily and pay taxes to finance public health measures. The state responded by providing public goods such as information, sanitation, and isolation measures, with Italian city-states appointing health boards to control contagion as early as 1348. The community deployed norms, information, and peer pressure to enforce sanitation and social distancing. Religions exhorted the faithful to obey public health regulations and donate alms to help the poor comply without starving. Doctors' guilds supplied medical advice and recruited volunteers to serve in pesthouses. The family provided a basic safety net, enabling members to stay away from the labour market for longer than if kin-based risk-pooling had been unavailable, and supporting contagion controls to safeguard vulnerable family members.

All six institutions thus played central roles in both transmitting contagion and controlling it. But, as this book will show, institutions do not behave the same way in all societies. Every human society has markets, states, communities, religions, occupational associations, and families. But these take different forms and act differently in different times and places. Even when they take similar forms themselves, they are embedded in a different overall institutional framework, where they cooperate and compete in different ways. Societies vary in how they deal with epidemics.

This book uses the past seven centuries of history to investigate how human societies dealt with epidemic disease. It focuses on contagion. How do social institutions enable us to contract, coerce, and coordinate so that we take

8. De Mussis 1348–56, quoted in Horrox 1994, 19.
9. Dols 1977, 160, 278

account of the effects our infectious behaviour has on others? What weaknesses make an institution fail to control epidemic contagion—or even exacerbate it? How is each institution constrained by others? Do some societies have better institutions than others? Can societies learn? What does history tell us about institutional solutions to the problems posed by epidemic disease?

1. How to Think about Contagion

If you went to church in London near the end of the 1563 plague, Bishop Edward Grindal would have exhorted you to practise social distancing. This was not to save you—at least, not from plague. It was to redeem your soul. But it was also to protect others, since "all men are bound in conscience not to do any thing that by common judgment and experience may bring a manifest peril and danger to their brethren or neighbours". Bishop Grindal's exhortation was based on the idea of the "negative externality"—where some action of mine imposes costs on others on top of the costs I incur. If my getting a disease imposes costs on others, society would like me to do less of it than I would if I were acting altogether selfishly. As Bishop Grindal added, I should avoid being infected not just out of "the rule of charity" but out of collective "profit". Shunning needless interaction during plague was, he urged, "very like to be profitable for this afflicted city".[10]

Epidemics are one of the most extreme examples of negative externalities that human societies know. The classic example is pollution: my factory profits only me, yet poisons everyone. But pollution pales to insignificance beside contagion. In terms of conspicuous costs that one person imposes on others, it is hard to think of a more dramatic example than infection. Epidemics thus create one of the best laboratories for understanding how societies deal with negative externalities—situations where a choice I make inflicts costs on others on top of any I myself incur.

A major way societies deal with externalities is through institutions. Institutions are systems of rules, customs, and practices that structure the way we interact as human beings. By giving us systematic ways to cooperate, institutions reduce transaction costs—costs of search, information, bargaining, decision, policing, and enforcement. These are the costs we incur in trying to reach agreements to act together. Economists mostly focus on how institutions reduce our transaction costs in producing, consuming, and trading. But institutions also lower the costs of ensuring that I take account of the spillover costs my choices inflict on others. That means they not only help us allocate resources efficiently, achieve technological progress, increase overall output, and redistribute resources to the needy. They also help us deal with infectious

10. Grindal 1843, 271.

diseases. This book will argue that assessing institutions in terms of how well they help society cope with epidemics is no less relevant than assessing how they help us improve economic performance—and indeed that the two activities are deeply interconnected.

Different institutions help us deal with infectious disease in different ways. The market offers mechanisms for people to bargain to compensate one another for contagion or pollution. The state offers ways to compel people to stop infecting or polluting others to begin with. The local community, the guild, and the family offer low-cost ways for a group to coordinate individual decisions to benefit all its members—and perhaps even the wider society. Religion teaches ethical norms of altruism, inspiring—or even, as with Bishop Grindal, admonishing—us to care about other people. The family provides emotional motivation for individuals to protect their members by avoiding and controlling contagion. The best institutional solution to a particular instance of contagion will differ according to the attributes of the disease, the technology available to deal with it, and the social context in which it occurs. But in principle, social institutions help us deal with the problem that my infection has spillover effects that I do not take into account, giving rise to contagion that inflicts unintended costs on everyone.

In practice, institutional solutions to epidemic contagion do not always work out well. For one thing, institutions do not just reduce the costs of dealing with infectious disease. They reduce the costs of many human interactions that have nothing to do with contagion. These can take precedence, interfering with how well any institution deals with epidemics. States wage war, religions organize pilgrimages, communities eject migrants, guilds profit their members, families feed their children. These activities benefit states, religions, communities, guilds, and families in other ways. But they also worsen contagion, as we shall see.

Second, most institutions serve some groups more than others—the state favours rulers over ruled, the community privileges citizens over outsiders, religion benefits priests over parishioners and heretics, the guild profits members over interlopers, the family nurtures kin over non-kin. Even if preventing an epidemic is in the interest of society, powerful groups have interests they prioritize more. Rulers and bureaucrats prefer higher tax revenues. Communities and their citizens prefer to favour locals and eject outsiders. Priests prefer to hold religious assemblies and preach against science. Guild masters prefer to control their occupations and monopolize their customers. Families prefer to help relatives instead of complying with lockdown. Swayed by such interests, even an institution capable in principle of mitigating an epidemic can fail to do so. It can even make contagion worse.

Does this mean we should just give up on understanding or improving our institutions? On the contrary, this book will argue. The same infectious disease

works out differently in different times and places. This is not so much because of biological characteristics of microbe or human host. It comes from how biological features interact with social ones. The same institution acts differently in different societies. This book analyses how key social institutions deal with epidemics in different places and times, and explores the reasons they succeed, fail, or make things worse.

2. A Brief History of Epidemics

History seethes with epidemics. This is not surprising. For most of history, infectious disease has killed many more people than war or hunger. Counting victims is hard because identifying cause of death is complicated and records are imperfect. Minimum estimates show infectious disease causing 45 per cent of all deaths in England and Wales in 1850, 36 per cent in Britain in 1900, and 40 per cent in low-income countries in 2018.[11] In 2019, the year before Covid-19 struck, communicable diseases caused over 26 per cent of the disease burden across the world, rising to 33 per cent in South Asia and 66 per cent in Sub-Saharan Africa.[12]

Not all these diseases were epidemics. A disease is "endemic" ("within a certain people") when it is permanently prevalent in a place at a predictable level—like smallpox in seventeenth-century China or cholera in eighteenth-century Bengal. It turns into an "epidemic" ("on a certain people") when it starts to infect exceptional numbers in a particular place and time—as with plague around the Black Sea region in 1346–47 or cholera when it broke out of India after 1816. A disease becomes a "pandemic" ("pertaining to all the people") when it spreads across an entire country, continent, or planet—like the Black Death in 1347 or Covid-19 in 2020.[13] In this sense, the difference between endemic, epidemic, and pandemic disease is mainly a matter of scale.

Increasing the spatial scale of disease triggers new social challenges. Endemic disease often varies across regions of the same country. When that disease turns into an epidemic it affects places that are normally spared. These may lack biological immunity as well as social mechanisms to cope with the new disease. In turn, when a disease becomes a pandemic, it spills across

11. Alfani and Ó Gráda 2018, 137.

12. "Share of Disease Burdon from Communicable Diseases vs. GDP per Capita, 2019", Our World in Data, accessed 19 May 2024, https://ourworldindata.org/grapher/share-of-disease-burden-from-communicable-diseases-vs-gdp?tab=table&time=2019. Disease burden is measured in disability-adjusted life years (DALYs), defined as years of life lost to due to premature mortality plus years of healthy life lost due to disability. According to current estimates, infectious diseases cause around one-quarter of deaths in the world today: see "Infectious Disease", Wellcome, accessed 1 June 2024, https://wellcome.org/what-we-do/infectious-disease.

13. Pelling 2020; Alfani 2022, 6; Alfani 2023a, 3–4.

political, commercial, communal, religious, and cultural frontiers. So a pandemic often disables institutions such as state, market, community, and religion which normally help people coordinate responses to contagion. It also creates the cross-border externalities we examine in later chapters.

Differences in scale also create differences in kind. When a disease is endemic it is often limited to particular demographic and social groups—children, the elderly, the poor and homeless. When it becomes an epidemic, it penetrates deeply into new social groups. Ultimately, an epidemic or pandemic threatens everyone, though as we shall see in chapter 2 the poorest almost always suffer most.

Endemic, epidemic, and pandemic disease challenge human societies in many of the same ways. So many of the analytical points this book makes about how institutions affect epidemics apply to all infectious diseases. But the negative externalities of contagion and the positive externalities of sanitation, social distancing, and immunization become much more salient when a disease is, or threatens to become, an epidemic or a pandemic. Consequently, the actions of the institutions analysed in this book have much more acute repercussions—for good or ill—in an epidemic situation. Moreover, institutional failures make it more likely that an endemic disease will flare up into an epidemic or pandemic. This book focuses on how institutions affect epidemics and pandemics, while bearing in mind that many of the analytical points apply to all infectious diseases.

Table 1.1 shows the historical epidemics that spread most widely—that is, approached pandemic scale—over the past two millennia. In the 1,855 years between the Antonine plague (possibly smallpox) and the Covid-19 pandemic (a coronavirus), there were 15 severe pandemics, one every five generations on average. Of these, 6 (40 per cent) were bubonic plague, 2 were influenza, 1 was mixed (smallpox, typhus, measles, and influenza), and there was 1 each of ancestral smallpox, haemorrhagic fever, syphilis, cholera, HIV/AIDS, and coronavirus. Pandemics thus struck every 125 years or so over the past two millennia. These 15 pandemics were merely the most notable episodes of a constant and lethal struggle between microbes and humans in Europe, the Mediterranean basin, the Middle East, China, India, other parts of Asia, the Americas, and Africa. This was bad for people in all these times and places. But it was good for science, because it created a huge variety of contexts in which to analyse epidemic disease and human responses to it.

We do not have evidence on all epidemics. This book extends geographically and chronologically as far as reasonably reliable evidence survives. Geographically, the historical record gets fragile once we move outside Europe and the Middle East. The book therefore analyses as much as is known about China, India, and the Americas, but perforce discusses Europe and the Middle East more intensively because of the availability of archival sources, data collection,

TABLE 1.1. Major Lethal Pandemics of the Past Two Millennia

Era	Dates	Epidemic name	Infection	Regions affected	Victims (millions)	Mortality (% of population)	Source
Epidemics of late antiquity	165	Antonine "plague"	Ancestral smallpox (possibly)	Roman Empire	7–8	10–30	1, 3
Epidemics of late antiquity	249–70	Cyprianic "plague"	Haemorrhagic fever (speculative)	Roman Empire		15–25	1
Plagues (main), first pandemic	541, possibly up to 550 in N. Europe	Justinianic plague[a]	*Yersinia pestis*	Europe, Mediterranean	Up to 25–50 overall	25–50 overall (50 in Egypt and other densely populated areas)	1, 4
Post-Justinianic plagues, first pandemic	565, 627–717, 740s	Five Great Islamic plagues,[b] other post-Justinianic plagues	*Yersinia pestis*	Italy (565); Middle East, esp. Iraq, Syria, Palestine (627–717); Mediterranean (740s)			4
Plagues (main), second pandemic	1347–52	Black Death	*Yersinia pestis*	Europe, Mediterranean, Middle East, C. Asia, possibly parts of China	Up to 50 in Europe and the Mediterranean; unknown elsewhere	35–60 in Europe and the Mediterranean; unknown elsewhere	1
Plagues (main), second pandemic	1356–66	Pestis secunda	*Yersinia pestis*	Europe, Mediterranean, Middle East	Up to 5–10 in Europe and the Mediterranean; unknown elsewhere	15–20 in Europe and the Mediterranean; unknown elsewhere	6
Plagues (main), second pandemic	1625–32	Second plague pandemic	*Yersinia pestis*	Most of C. and W. Europe (excluding most of Spain and C.-S. Italy)	Up to 2 in northern Italy; up to 1.15 in France; up to 0.25 in Switzerland; up to 0.16 in the Dutch Republic; unknown elsewhere	30–35 in N. Italy; 20–25 in Switzerland; 20–25 in S. Germany, Rhineland, and Alsace (up to 40 incl. victims of famine and Thirty Years' War); 8–11 in the Dutch Republic; unknown elsewhere	6

Plagues (main), second pandemic	1647–57	Second plague pandemic	*Yersinia pestis*	Andalusia, Spanish Mediterranean, and C.-S. Italy	Up to 1.25 in the Kingdom of Naples; up to 0.5 in Spain; up to 0.33 in France; unknown elsewhere	30–43 in Kingdom of Naples; at least 25 in Andalusia; 15–20 in Catalonia; unknown elsewhere	1
Other extreme early modern epidemics	1492–1550	Columbian Exchange (New to Old World)[c]	Syphilis[d]	Europe, Asia	Up to 2–5 in Europe; unknown elsewhere	Up to 4–5 in Europe; unknown elsewhere	1
Other extreme early modern epidemics	1492–1650	Columbian Exchange (Old to New World)[e]	Smallpox, typhus, measles, influenza, etc.	American continent		Up to 80–90 in the first century (confounded with direct impact of colonization)	1, 5
Cholera pandemic	1817–1923	Cholera pandemics 1–6	Cholera	Worldwide	At least 1 (probably many more)		1
Third plague pandemic	1894–1920	Third plague pandemic	*Yersinia pestis*	China, India, Europe, USA	12		1
Russian flu	1889–90	Russian flu	Influenza	Russia, Asia, Europe	1		1
Influenza pandemic	1918–19	Spanish flu	Influenza	Worldwide	50–100	Globally 2.5–5; Italy 1.1–1.3; USA 0.65; Germany 0.37; China up to 2; British India 5–6; South Africa 6	1, 2, 6
HIV/AIDS global pandemic	1983–2025 (ongoing)	HIV/AIDS global pandemic	HIV/AIDS	Worldwide, worst in sub-Saharan Africa	75 infections, 30 deaths[f]		2

(*continued*)

TABLE 1.1. (*continued*)

Era	Dates	Epidemic name	Infection	Regions affected	Victims (millions)	Mortality (% of population)	Source
Covid-19 pandemic	2020–25 (ongoing)	Covid-19 pandemic	Covid-19	Worldwide	771 infections, 6.9 deaths (officially reported); 12–18 excess deaths (estimated)[g]		[7]

Sources: [1] Alfani and Murphy 2017, esp. table 1 (pp. 316–17), sources cited in main text of that article. [2] Heinrich 2021, 4. [3] Harper 2017, 18, 115; Haldon et al. 2018, 2; Duncan-Jones 2018, 44; Shabana 2021, 6. [4] Dols 1977, 17, 20–6. [5] Newson 1985, 41–2, 48. [6] Alfani 2023a, 6–7 (table 1), 26. [7] "WHO COVID-19 Dashboard", World Health Organization, accessed 3 October 2023, https://covid19.who.int/; "Estimated Excess Mortality from the World Health Organization", Our World in Data, accessed 3 October 2023, https://ourworldindata.org/excess-mortality-covid#estimated-excess-mortality-from-the-world-health-organization.

Notes: Table does not include pandemics whose characteristics and dimensions are still under-researched, e.g. some large waves of the second plague pandemic; the undiagnosed non-bubonic-plague pestilences of the 1480s–90s and 1550s; and a number of possible pandemics of influenza, yellow fever, typhus, and smallpox.

[a] Figures for Justinianic plague refer to the initial outbreak only, not to the outbreaks which make up the "first pandemic" ending in 750.

[b] The concept of "five Islamic plagues" is based on contemporary sources and has been questioned by some modern scholars.

[c] After the mid-sixteenth century, syphilis mutated into a less aggressive disease.

[d] Given data scarcity, the number of victims of syphilis provides a very uncertain upper boundary only; it is debatable whether all (or any) post-1490s pox outbreaks were caused by syphilis.

[e] Following colonization, epidemics recurred frequently for centuries where the population was not entirely eradicated, yet the greatest demographic toll took place roughly in the first 150 years.

[f] Figures as of 30 April 2021.

[g] Official figures include only confirmed cases and deaths reported to WHO; excess mortality = gap between actual deaths and expected deaths without Covid-19.

quantitative analyses, and historiographical works on epidemics. Similarly, the historical record gets very fragile for the centuries before the Black Death, so this book inevitably concentrates on the seven centuries or so since the 1340s. This book takes the view that it is counterproductive to report guesswork or unreliable research just because they extend into less well recorded eras and regions. But it also seeks to illuminate the questions, methods, and theoretical approaches that can be used once better data are collected on times and places that have yet to be analysed.

Even though this book is about human societies, microbial biology matters. Table 1.2 shows some of the diversity in how different epidemic diseases behave. Some differences are epidemiological—the share of the population that catches the disease (infection rate), the share of the infected who die (case fatality), the share of the whole population that the disease kills (population mortality). The infection rate varies from under 2 per cent for nineteenth-century cholera to nearly 100 per cent for pre-vaccination smallpox. The case fatality rate ranges from 2–3 per cent for Spanish flu and Covid-19 to 80–90 per cent for bubonic plague and Ebola. The population mortality rate ranges from under 1 per cent for cholera to 50–60 per cent for the Black Death plague variant.

Death is not the only problem. Infection matters by itself, since it reduces people's ability to work and—more important—enjoy life. Surviving infection does not lead to living in health. Most epidemic diseases leave some patients with serious sequelae—after-effects of infection—such as blindness, neurocognitive decline, or fatigue. These have repercussions not just for the individual survivor but for the rest of society.

The social distribution of infection and death also differs across diseases, as table 1.2 shows. Some epidemics infect and kill the young and those in the prime of life (like the Spanish flu), others kill the old (like Covid-19), and still others find victims across the entire age spectrum (like plague). Some epidemics show little gap between rich and poor, as with natural smallpox, Ebola, or SARS. Others are diseases of poverty—plague, typhus, cholera. Women have a slight advantage over men in fighting off most diseases, because they have stronger natural immune systems, but pregnancy greatly increases case fatality rates for many diseases, including bubonic plague, smallpox, measles, influenza, SARS, and Covid-19.

Epidemics are not a matter of simple biology. Epidemiological differences evoke differing social responses. A high infection rate alone does not necessarily attract concern if case fatality is low, since few people die. Conversely, high case fatality does not necessarily attract attention, since population mortality is low if infection rates are low. It is the combination of non-trivial infections with non-trivial case fatality that results in high population mortality and attracts social attention. A disease that attacks the rich typically attracts more resources

TABLE 1.2. Morbidity, Mortality, and Social Selectivity of Major Pandemics in History

Disease	When	Infection rate (%)	Case fatality rate (%)	Population mortality rate (%)	Sequelae	Poverty	Age	Gender
Plague	1347–52	51–67	80	50–60	Gangrene, clots, meningitis	Weak		No conclusive evidence
Plague	16th–17th century	24–53	80	20–40	Gangrene, clots, meningitis	Medium		No conclusive evidence
Plague	Pre-antibiotic		50–80		Gangrene, clots, meningitis	Strong		Pregnant women
Plague	1890s		*c*.80		Gangrene, clots, meningitis	Strong		No conclusive evidence
Smallpox	Pre-vaccination	*c*.100	30–40	10–20	Blindness, scarring	Weak		Pregnant women
Typhus	Pre-antibiotic		>50		Sepsis, seizures, neurocognition, fatigue	Strong		No conclusive evidence
Measles	Pre-vaccination		10		Blindness, neurocognition	Medium		Pregnant women
Cholera	19th century	0.7–2	50	0.5–1	Chronic enteropathy, malnutrition	Strong		No conclusive evidence
Spanish flu	1918–19	*c*.33	2.5	0.3–6.0	Heart risk	Medium	25–40	Pregnant women
Ebola	1990s–present		60–90		Fatigue, pain, blindness, neurocognition	Weak		No conclusive evidence
SARS	2003		10		Fatigue, osteoporosis, breathing	Weak		Pregnant women
Covid-19	Pre-vaccination		2.3–3.6		Fatigue, neurocognition, breathing, dizziness	Medium	>65	Pregnant women

Sources: Alfani 2023a; Alfani 2023b; Atmar, Englund, and Hammill 1992; Dixon 1962, 2, 6–7; Fleck-Derderian et al. 2020; Haider et al. 2022, l, 93; Hassett 2003, 14–15; S.-T. Liang et al. 2021, 273–4; Livi-Bacci 2006, 206, 210; Matsuo et al. 2023; E. Smith et al. 2023; Snowden 2019, 29, 84, 95, 163, 241, 334, 471, 475–6.

Notes: Infection rate = % of population that contracts the disease; case fatality rate = % of cases that end in death; population mortality rate = % of population that dies of the disease; sequelae = post-infection conditions resulting from previous infection with the disease; poverty = strength of association between poverty and susceptibility to the disease; age = age-group(s) most strongly affected by the disease; gender = whether women or men are more strongly affected by the disease.

than one that hits just the poor. A disease that afflicts the very young or very old attracts less concern than one that strikes down householders and taxpayers in the prime of life. Historically, a disease that killed pregnant women attracted less attention than one that killed men of military age—hence state smallpox immunization for nineteenth-century soldiers, which long predated universal vaccination mandates. The biological features of different epidemic diseases thus already create differing institutional incentives to contain them. These biological features interact with the capacities of each institution and the interests of those who dominate its use, shaping responses to contagion.

Epidemics have afflicted large zones of the globe repeatedly across many centuries of recorded history, as tables 1.1 and 1.2 show. Indeed, they remain an active and present threat into the present day, as shown by recent and emerging diseases such as HIV/AIDS, SARS, and Covid-19. Yet we must recognize one big fact. The risks of epidemics across the globe as a whole declined enormously over the past seven centuries, and did so at an accelerating rate. Acute episodes of infectious disease weakened, they occurred less frequently, and they infected and killed fewer people when they did take place.[14] By the later seventeenth century, epidemic infection and mortality were lower than in the mid-fourteenth century. Between the late seventeenth and the late nineteenth century, they declined faster. By the early twenty-first century they had fallen even more spectacularly.

"Epidemiological transition" is the term demographers give this development, during which societies are supposed to make a happy exodus from pandemics of infectious disease into the sunlit uplands of non-communicable ailments and longer life expectancy. The epidemiological transition model holds that human populations pass through three stages of mortality and morbidity. In the first stage, "the age of pestilence and famine", societies suffer high and fluctuating mortality, in which infections cause most deaths and epidemics are common. In the second stage, "the age of receding pandemics", mortality declines, pandemics are fewer and smaller, medical knowledge advances, and infections gradually recede. The third and happy stage, "the age of degenerative and man-made diseases", is one in which mortality is low and still decreasing, infectious disease is rare, and most deaths are caused by cardio- and cerebrovascular ailments, metabolic diseases, cancers, injuries, stress-related disorders, and dementia. Epidemiological transition theory sees key changes occurring between 1670 and 1850, when colossal pandemics largely disappeared from western Europe, followed by accelerating improvement around 1900, as many lethal epidemic diseases declined simultaneously and were supplanted by non-communicable ailments.[15]

14. Omran 1971; Santosa et al. 2014; Mackenbach 2020; Mackenbach 2021; Alfani 2022, 33–4.
15. Omran 1971.

The idea of epidemiological transition is a stimulating point of departure, but we now know it does not tell the full story of the war between man and microbe. The concept was invented in 1971 and postulated a universal development based on the experience of a small number of modern, rich countries, whose history was assumed to prefigure the future of poor ones. The past 50 years have shown that this view was too hopeful. Even in Europe, infectious diseases did not all follow the same transition, but rather rose and fell, as shown in table 1.3. Infectious diseases, including epidemics, continue to display this typical rise-and-fall pattern to this day.[16]

Even more seriously, not all societies followed the same epidemiological pattern. The onset, speed, direction, and pattern of mortality developments display huge gaps across societies.[17] The elimination of infectious disease is still far from complete. Some old diseases came back because controls lapsed, as with tuberculosis and whooping cough. Others became much harder to fight because of antibiotic resistance, as with staphylococcal infections. Serious new infections emerged, such as Legionnaires' disease, AIDS, SARS, Ebola, and Covid-19.[18]

Sadly, no uniform epidemiological transition model applies to all societies. It might be argued that at least we can all accept that infectious diseases declined on average between around 1670 and 1900, and that this was mainly caused by advances in scientific and medical knowledge. Only up to a point. Knowledge about microbes and scientific approaches to medicine did ultimately play a key role in reducing epidemic mortality to the level we enjoy today. But their influence was much slower, later, and more partial than optimistic analyses would have it—for reasons this book will discuss. Scientific approaches to medicine gained force only in the final decades of the nineteenth and the early decades of the twentieth century, largely failed to control the 1918 flu pandemic, were still imperfect as late as HIV/AIDS, and are not universally accepted in most societies to this day—as became evident during Covid-19. Scientific knowledge about contagion, sanitation, social distancing, immunization, microbes, and antibiotics certainly gave us better technology for limiting epidemics. But knowledge was not enough. In many societies, new ideas and techniques for tackling epidemics were widely rejected. Why?

Social institutions, this book will argue, played a decisive role. New ideas and practices always create winners and losers. Institutions often enable the losers to block new knowledge, even though accepting it would benefit society at large. Market, state, community, religion, medical associations, and familial

16. Mackenbach 2020; Mackenbach 2021.
17. See the survey in Santosa et al. (2014).
18. Mackenbach 2020; Mackenbach 2021.

TABLE 1.3. Rise and Fall of Infectious Diseases in Europe, Ordered by Timing of Decline

Disease	Rise and fall?	Start of rise[a]	Start of fall[b]
Plague[c]	Rise and fall	1347	17th century
Typhus	Rise and fall	Late 15th century	17th century?
Smallpox	Rise and fall	6th century	18th century
Malaria	Rise and fall	16th century	18th century
Cholera	Rise and fall	1829–37	1846–60
Three intestinal infections (dysentery, typhoid fever, paratyphoid)	Rise and fall	6500 BCE[d]	Mid-19th century
Tuberculosis	Rise and fall	18th century	Mid-19th century
Puerperal fever	Rise and fall	18th century	Mid-19th century
Four childhood infections (scarlet fever, measles, whooping cough, diphtheria)	Rise and fall	18th century	Late 19th century
Syphilis	Rise and fall	Late 15th century	Early 20th century
Pneumoconiosis	Rise and fall	19th century	Early 20th century
Pneumonia	Fall only	N/A	Early 20th century
Influenza	Rise and fall	16th century	1918–19
AIDS	Rise and fall	Early 1980s	Mid-1990s
Covid-19	Rise and fall	2020	2021–23

Sources: Mackenbach 2020, 46–7; Mackenbach 2021, 1201.

[a] Approximate start of rise in Europe

[b] Approximate start of fall (or peak year) for north-western Europe only

[c] Second pandemic only

[d] In Europe, the Neolithic or first agricultural revolution started in the Aegean around 6500 BCE

institutions all have the capacity to accept and disseminate scientific approaches to epidemic disease, as we shall see. But they also have the capacity—and often create the incentive—to hinder these approaches. As this book will show, science seldom had a direct effect on epidemic outcomes. Rather, its acceptance and adoption were mediated by the institutional framework. Science, medicine, and technology greatly improve our capacity to limit infectious diseases. But how we use this capacity depends on social institutions.[19]

19. Santosa et al. 2014; Mackenbach 2020, 6–7, 76, 288–91; Mackenbach 2021.

3. What Do We Mean by Institutions?

"Institution" means different things in different contexts. In ordinary language, we use it to refer to specific organizations—the London Stock Exchange, the US government, the United Nations, the Derbyshire village of Eyam, the Catholic Church, the American Medical Association, the British royal family. But social scientists use "institution" to refer to a system of rules, customs, and practices governing how we interact in society—in North's famous formulation, "the rules of the game in a society or, more formally . . . the humanly devised constraints that shape human interaction".[20] The London Stock Exchange is a specific example of an abstract institution: the market. The US government is a specific example of the state, as is the United Nations—a supranational state institution. The Derbyshire village of Eyam, with its legendary communal action against the 1665 plague, which we analyse in chapter 4, is a specific example of a local community. Likewise, the Catholic Church is a specific manifestation of the institution of religion, the American Medical Association a modern instance of a corporative occupational group or guild, and the British royal family a famous example of the institution of the family. This book uses "institution" in this second, abstract sense, to refer to a system of rules and practices governing how we interact in society.

Some scholars argue that institutions encompass both external rules and internal preferences. This book draws a distinction between the two: internal preferences are part of culture, while external rules make up institutions. Human action, as this book thinks about it, involves both culture and institutions: people try to satisfy their preferences subject to a set of constraints. This book sees culture as contributing to the preferences, while institutions are part of the constraints. Of course, on the informal end of the spectrum, institutional rules shade into customs, norms, expectations, and preferences—that is, into culture. Almost certainly, there are two-way causal links between the systems of rules a society ends up with and the preferences held by its members.[21] But this book focuses on the constraints social institutions create for human choices via external rules, customs, and practices which do not take place primarily inside people's minds.

Why do institutions matter for epidemics? Surely disease is just a game between humans and nature—albeit a violent and lethal one? Microbes try to kill us, our immune systems try to kill them, and one side survives to play again. Institutions, by contrast, are games purely among humans. Human beings try to cooperate or compete, institutions channel how we do so, and the rules of that game decide who survives to play again. What do the two games—natural

20. North 1990, 3.

21. See the discussion in Ogilvie (2007, 660–1) and Vollrath (2019, pt. 4).

and institutional—have to do with each other? This book shows that the rules of the institutional game among humans change the outcome of the natural game between us and microbes.

Which institutions matter? Probably they all do, but which matter more? Human societies are multifarious, and we do not have evidence on them all. Even for one society, we do not know everything about all its rules, customs, and practices. So this book focuses on half a dozen key institutions: the market, the state, the community, religion, the occupational association, and the family. All six were central to most societies over the past two millennia, though in widely differing forms. All six set the rules for how humans interacted with microbes down the centuries, and still do in most societies to this day.

Our first institution is the market, the name we give to the set of rules, customs, practices, and procedures that people use to buy and sell. According to William Laud in 1637, plague epidemics were caused by people's "greediness to receive into their houses infected goods".[22] This point of view was echoed in 2021 by Dani Rodrik, who claimed that the Covid-19 pandemic would have been milder "if we had spent a little bit less time opening up our borders to trade and investment and doing the bidding of multinational corporations and banks".[23]

As these views illustrate, and as chapter 2 explores in detail, the market is often blamed for making epidemics worse. The market causes contagion, the argument goes, both when it fails and when it succeeds. The market fails when market prices do not take account of the costs individual decisions inflict on others, creating negative externalities such as contagion. Market prices also fail adequately to reward individuals and firms for creating public goods such as health information, sanitary infrastructure, vaccinations, and other medical innovations, which produce positive externalities by controlling infection. But even when the market does exactly what it is good at, facilitating voluntary exchange, it exacerbates contagion because it encourages trade and migration, along with the movement of microbes carried by goods and people.

Yet vigorous market institutions, as chapter 2 shows, also help mitigate epidemics. Measures to control contagion are costly. Border guards, street patrols, corpse inspectors, and other public servants must be paid. Sewerage and water systems must be built and maintained. Quarantined citizens have to be provisioned, or they will break out to find work or food. Medical innovations must be diffused and implemented. The Pfizer vaccine against Covid in 2021 involved assembling 280 components made by 86 suppliers in 19 countries, which would have been impossible without the market transactions and

22. Quoted in Slack 1985, 22.
23. Rodrik 2021, 69.

global production chains denounced by Laud and Rodrik.[24] To pay for public anti-contagion measures requires strong fiscal capacity, which is historically much higher in societies experiencing market-driven economic growth.[25] Communities, religions, occupational associations, and families, too, have more resources to tackle contagion in market societies, where economic growth and per capita incomes are higher.

Chapter 2 investigates how the market has interacted with epidemics down the centuries, and analyses those aspects of market activities that exacerbated or ameliorated the damage epidemic disease inflicts on human well-being.

A second key institution in tackling contagion is the state, a set of practices imposing compulsory rules over people in a territory, claiming priority over the rules of other organizations, and backed by legitimate coercion.[26] In March 1348, very soon after the Black Death reached Europe, the Large Council of the Republic of Venice set up a subcommittee of three, whose task was "to consider diligently all possible ways to preserve public health and avoid the corruption of the environment".[27] In 2020 during Covid-19, Francis Fukuyama declared that the key to dealing with a great pandemic is "whether citizens trust their leaders, and whether those leaders preside over a competent and effective state".[28]

The state has many features that deal with epidemics, as we see in chapter 3. A high-capacity state possesses policy levers enabling it to collect and diffuse information, provide sanitary infrastructure, and subsidize medical innovations—all helping to internalize contagion externalities. States also offer welfare support to motivate and enable poor citizens to comply with anti-contagion measures, instead of being compelled to leave home to buy food or earn a living.

On the other hand, there is such a thing as state failure—and not just in failed states. In 1353 Giovanni Boccaccio described how the Florentine state, one of richest and most sophisticated in the world, failed during the Black Death, when "no learning nor measure was of any use, such as the clearing of the city of much refuse by officials, who were appointed for that purpose, and the prohibition of any sick person from entering and many counsels given for the preservation of health".[29] In 2020, the editor-in-chief of the *Journal of the Royal Society of Medicine* declared uncompromisingly that in England, a rich modern democracy, "the government has indulged in a level of state negligence that may be unprecedented".[30]

24. Jecker and Atuire 2021, 597.

25. Besley and Persson 2009.

26. For overlapping definitions to this effect, see Weber ([1922] 1978, 54–5) and Tilly (1990, 1–2).

27. Cipolla 1976, 11.

28. Fukuyama 2020.

29. Quoted in Henderson 2019, 2.

30. Abbasi 2020, 419.

The state fails to deal with contagion, as chapter 3 shows, because of inaccurate information, low fiscal capacity, flawed bureaucratic capacity, *Realpolitik*, and poor incentives. It also exacerbates epidemics as it pursues its other interests. European states historically allocated 50–90 per cent of fiscal capacity to military purposes.[31] The resulting wars, as at Caffa in 1346, exacerbated epidemics through sieges, campaigns, camps, colonies, persecution, prisons, and refugee flows. State expenditure on civilian purposes such as sanitation, social distancing, quarantines, public information, vaccination programmes, or welfare support accounted for a vanishingly small share of public spending well into the twentieth century, and continues to be inadequate in many poor economies to this day.

Chapter 3 explores state capacity, state failure, and state motivation in dealing with contagious diseases. It identifies and analyses those features of the state that have historically helped or hindered societies in managing contagion during epidemics.

A third institution that influences epidemic contagion is the local community—the set of rules, customs, and practices connecting people living in spatial proximity within a town, village, or neighbourhood. During the 1631 plague epidemic, the Florentine village of Pinzidimonte imposed an autonomous community lockdown, incarcerating a number of families in their dwellings and intimidating everyone else out of "frequenting the churches, the streets, work, and everywhere they were threatened".[32] During the Covid-19 pandemic, some scholars lauded similar communal autonomy in China, where "civil society organizations took responsibility of isolating residents in every community".[33]

Community "social capital", as we see in chapter 4, fostered collective action to monitor and penalize individual choices that might transmit infection, ranging from waste disposal to breaking quarantine to neglecting immunization. Communities also provided much charitable relief and informal assistance among neighbours, enabling poor local residents to comply with anti-contagion measures during epidemics.

But communities can also fail. In 1353 Boccaccio described how in Florence during the Black Death "one citizen fled after another, and one neighbour had not any care of another".[34] Even in communities where solidarity survives, it is not always deployed in a good way. In 1630 the community of Prato excluded non-locals from the communal pesthouse, even when the grand-ducal government in Florence pointed out that "by eradicating the disease outside the walls, its eradication within is made easier".[35] In Indian villages during

31. Hoffman 2015, 315.
32. Quoted in Henderson 2019, 144.
33. S. Zhang et al. 2020, 216.
34. Quoted in Alfani and Murphy 2017, 333.
35. Quoted in Cipolla 1973, 123.

the Covid-19 epidemic, Hindu mobs violently attacked Muslims whom they accused of "corona jihad", and Muslim families concealed Covid-19 infections and neighbours' attacks for fear the village would throw them out.[36] Community trust supports exclusion of outsiders and ejection of marginal groups, whose banishment spreads contagion. Communities ration welfare relief, denying it to local minorities, attenuating their incentive and capacity to comply with anti-contagion measures. Communal social capital organizes collective resistance to public health measures, as in the popular anti-vaccination riots that erupted in many villages and towns across the globe between 1796 and the present day.

Chapter 4 analyses the strengths and weaknesses of community institutions. It seeks to identify those features that enable communities to internalize contagion externalities, but also those that make local communities collapse in the face of an epidemic or even organize activities that exacerbate the calamity.

Religion is a central institution of every human society, comprising not just a system of beliefs about spiritual beings but also a set of rules, customs, and practices governing human relationships with the spiritual world.[37] In Boston in 1721, the Puritan minister Cotton Mather proselytized for smallpox immunization in the name of religion, claiming that "Almighty GOD in His great Mercy to Mankind, has taught us a Remedy, to be used when the dangers of the Small Pox distress us."[38] During the 2020 Covid-19 pandemic, the *Scientific American* blog argued that "religion and science can complement one another, as indeed they are already doing by reinforcing public health messages during the current pandemic".[39]

Religious institutions played an important role in dealing with epidemics, as we see in chapter 5. Religions used moral suasion to motivate the faithful to comply with public health measures, adopt medical innovations, and coordinate social distancing or immunization. They exhorted their adherents to make charitable donations in the name of sacred beings and directly organize hospitals and medical care, increasing people's capacity and motivation to comply with anti-contagion measures.

But religions, as we shall see in chapter 5, also interact with epidemics in malignant ways. They facilitate contagion by mandating religious assemblies, pilgrimages, and religious wars. They preach opposition to public health measures such as quarantine or vaccination. Religions deny care to some of the neediest victims by discriminating against those it categorizes as sinners or unbelievers, reducing their ability to comply with anti-contagion measures.

36. Ellis-Petersen and Rahman 2020.
37. See the discussion in Seabright (2024).
38. Mather and Boylston 1721, 18–19.
39. Barmania and Reiss 2020.

Chapter 5 explores how religions use their moral authority and worldly power when epidemics strike. It analyses which features of a religion make it more able and willing to control contagion, but also those characteristics that lead religions to resist public health measures and persecute victims, hobbling epidemic control.

The occupational association or "guild" lays down norms, rules, and practices governing a specified branch of production activities in a particular locality. This type of institution has existed for centuries across the globe, and is especially widespread in medicine. Guilds, "colleges", and "faculties" of physicians, surgeons, and apothecaries governed their professions from before the Black Death into the nineteenth century, and were succeeded by the medical associations that regulate health-care activities to this day. Unsurprisingly, medical associations played a major role in dealing with epidemics. In the Dresden plague epidemic of 1680, the authorities canvassed the local surgeons' guild to provide expert personnel for the plague isolation hospital.[40] In the summer of 2021, the Royal College of Physicians of Edinburgh emphasized its role during the Covid-19 pandemic as "an advocate for our Fellows and Members and, ultimately for the patients that we serve".[41]

Guilds and associations of medical practitioners, as we shall see in chapter 6, contributed to managing epidemics. They advised governments on public health measures and provided skilled volunteers. They donated guild funds to charitable and medical projects, improving the capacity and incentive of poor people to comply with anti-contagion measures. They regulated medical training, quality, and knowledge, addressing information asymmetries between experts and ordinary people.

But medical associations also dealt with epidemics in less beneficent ways. They exacerbated contagion by erecting non-merit-based entry barriers, creating shortages of medical expertise and making contagion advice unaffordable for the poor. They used their authority to oppose knowledge and practices that threatened their professional privileges, blocking innovations that promised to limit epidemic contagion.

Chapter 6 examines the benefits of medical associations during epidemics, along with the costs they imposed. It seeks to identify the features of a medical association that facilitated contagion control, and those that made it more likely that it would seek its members' advantage at the expense of everyone else.

The family, as an institution, coordinates how a group of relatives resides, reproduces, nurtures, consumes, and produces. In Leiden in 1484 during the plague, Govert die Ketelboeter's daughter hastened to cleanse her own family

40. Schlenkrich 2002, 39–40.
41. A. Thomas et al. 2021, S10.

of infection by hanging bedclothes over a town bridge, exposing the neighbourhood to contagion.[42] In England in 1808 thousands of women scrambled to get their children vaccinated, while anti-vaxx activists scoffed at how "mothers fly to [vaccination] as they have done to Ching's Lozenges for the cure of worms".[43] In 2020 during the Covid-19 pandemic, the Pew Research Center ascribed high infection rates in some areas to family patterns "where many people live together, [so] the risk of contagion is heightened if anyone in the household falls ill or becomes an asymptomatic carrier of the coronavirus".[44]

The family influences epidemic contagion in many ways—through residential arrangements, death-related obligations, female autonomy, migration flows, and the balance between familial and societal responsibilities. All these activities, as we see in chapter 7, interact with epidemic disease. Social scientists and demographers stress the distinction between nuclear-family systems with weak kinship links and extended-family systems with strong kin obligations. Contagion, it is argued, is better controlled in nuclear-family systems where households are small, contain fewer generations, involve weaker kin relationships, relax death-related obligations, empower females to make household health choices, direct migration flows in epidemiologically safe channels, and foster prosociality beyond the family.

By contrast, extended-family or clan systems are thought to exacerbate epidemics. In kin-intensive systems, contagion is worse because households are large, interact with wider networks of relatives, mandate kin attendance at deathbeds and funerals, deprive women of health autonomy, unleash migration flows by solitary males in epidemiologically primitive conditions, and foster "amoral familism".

These effects of families on contagion make sense in theory. Chapter 7 examines whether they prevailed in practice—either universally or at all. The family is always embedded in a wider institutional system that influences whether any particular family form acts beneficially or harmfully in response to the external shock of an epidemic. The same, it turns out, applies to every other social institution.

4. The Road Behind and the Way Ahead

Epidemics are so shocking that many see them as natural or supernatural retribution. Historically, people saw plagues as divine punishment for man's sins against God. In the modern era, we think of epidemics as a reckoning for sins against nature: reproducing too fast, ceasing to hunt and gather, inventing

42. Coomans 2021, 223.
43. M. Bennett 2008, 506.
44. Kramer 2020.

agriculture, moving into towns, eating animals, globalizing trade. Our unnatural choices are blamed for creating environments where microbes to evolve to attack us.[45] These concerns alert us to natural constraints on human life.

But they neglect other constraints. People do not just submit to nature passively. We also respond actively. We act not just as individuals but in groups, societies, and even sometimes globally. We develop social rules, customs, and practices that constrain and facilitate how we coordinate and compete over how we respond to nature, including to microbial attacks.

This book analyses these societal responses. It asks how different institutions channel our behaviour in times of mass disease, enabling us to respond collectively to natural challenges—for good or ill.

One thing it finds is that history yields no universal laws of resilience. But it does reveal systematic ways of understanding variegated outcomes. In recorded history, epidemics affect different societies differently. Why such divergence?

Institutions, this book argues, play a major role. To foster resilience to epidemic disease, we must understand how rules and practices inside societies respond to natural shocks. But the present is often too close for understanding. History, precisely because it is more variegated and further away, can help.

What, then, is the best institution for dealing with epidemic contagion? The answer is a combination of institutions, chapter 8 argues, interlinked in an interdependent institutional framework, each playing to its strengths and checking the others' weaknesses. Why is this diverse and interdependent institutional framework so important?

First, we need multiple lines of approach. Fighting epidemics requires resources, coercion, monitoring, exhortation, expertise, nurturing. Each human institution is good at mobilizing some of these, but no institution is good at them all. A social framework in which multiple institutions coexist has a better chance of tackling the multiplicity of challenges posed by contagion.

Indeed, as later chapters show, each institution has special strengths. The market can allocate resources efficiently and foster economic growth, generating resources that individuals, families, governments, communities, guilds, and religions need to control contagion. The state and the community can provide coordination, monitoring, and regulation to internalize the negative externalities of contagion and the positive externalities of disease control. Religion can provide moral suasion and exhortation, motivating people to care about each other. Medical associations can monitor training, quality, and information, solving information asymmetries between producers and consumers of epidemiological expertise. The family can reduce risks of coresidence, death

45. McNeill 1976; Diamond 1997; Harper 2017; Arenas 2021.

practices, and migration, empower women, and protect vulnerable relatives by supporting prosocial behaviour.

But every institution has weaknesses. The market can convey price signals motivating people to work, trade, or socialize without taking account of how they affect others. The state can reject or falsify information and wage wars that consume resources and spread contagion. The community can make collective health decisions that favour powerful locals and harm society beyond its borders. Religion can mandate superspreader events and preach against science. Medical associations can impose entry barriers, limit competition, and block epidemiological innovations. The family can unleash risky residential, funerary, and migration choices, suppress female autonomy, and mandate kinship obligations that ignore societal effects.

As history shows, each institution takes different forms in different societies. The state, for instance, may have features in one society which give it greater capacity than the state in another society in the same historical period facing the same epidemic disease. Alternatively, the state may have the same capacity in two different societies, but different incentives, leading one state to devote all its capacity to war, while another allocates more to public health. Sometimes the difference arises not only out of features of that specific institution but also out of how it is embedded in the surrounding institutional framework. Do communities, religious institutions, or medical associations limit the tendency of the state to allocate resources to war or, alternatively, encourage it to allocate resources to public health? This is another reason controlling epidemics needs a combination of institutions—to make up for failures by other institutions and curb their most harmful actions.

The history of epidemics illuminates institutional combinations that work well to address contagion, as chapter 8 argues. It does not usually explain, though, how to achieve such happy institutional equilibria. But even revealing the features of an institutional framework that managed to control an epidemic is worthwhile, and creates the basis for future research on how a society might bring such a framework into being.

This book does not show that certain institutions—or even certain institutional frameworks—are bad or good. It does identify the strengths that enable a particular institution to help people devise and adopt effective ways of tackling epidemics. It also detects the weaknesses in each institution that make things work out badly. It demonstrates how diverse and interdependent institutions in a framework can work together to affect epidemic contagion—for good or ill. It identifies the features which have made institutional frameworks better at coordinating responses to epidemics and better at devising innovations to improve societal learning.

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