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What Viruses Want

Evolutionary Insights for the Covid-19 Pandemic and Lessons for the Next One

Dominic D. P. Johnson[*]

Introduction: The Utility of a Biological Approach

Politics and international relations used to be the domain of philosophers, historians, and social scientists. But in the twenty-first century, the life sciences are becoming increasingly indispensable disciplines for both the study and practice of political life. Biology in particular represents a crucial area of knowledge for political leaders to understand, and to inform policy. Why so?

First, *major global challenges are increasingly biological or ecological in nature*, notably climate change, environmental stress, biodiversity loss, resource management, population pressures, biomedical technology, global health and—not least—pandemics such as COVID-19 (see Chapter 6 by Morin et al. in this volume). Biology and other scientific disciplines are essential to understanding such problems and their effects, and to devising technical solutions.

Second, human health and behaviour themselves are increasingly understood to have important *genetic, biological, and cognitive underpinnings*, which are crucial to understanding the impacts on, and the reactions of, individual human beings (citizens and leaders alike) and the population in response to contemporary political and global challenges. We need to better understand our own biology and behaviour to meet these challenges effectively.

While insights from the life sciences are numerous and varied, they are also unified under a single, overarching theoretical framework: evolution. Evolution

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explains all life on Earth, its origins, variation, adaptations, and interactions. Indeed, as Theodosius Dobzhansky observed, 'nothing in biology makes sense *except* in the light of evolution' (Dobzhansky 1973). Evolutionary biology therefore allows one to start from first principles in addressing a given problem by drawing on a common theoretical grounding and a vast empirical record that spans billions of years and millions of organisms, among which humans are just one. This broader picture allows us to see patterns, causes, and consequences in the dynamics of many contemporary challenges, as well as in our own species, that standard social science approaches cannot (Barkow 2006; Lederberg 2000; Sagarin 2012; Sagarin et al. 2010; Sagarin and Taylor 2008; E. O. Wilson 1999). Evolutionary approaches thus bring novel insights for a variety of topics in politics and international relations, but especially for one of the most important global challenges of the century: pandemic disease outbreaks such as COVID-19.

The Problem: Non-Rational Actors and Dynamic Threats

One might think that, while obviously needing biology (and subfields such as genetics and epidemiology) to understand virus outbreaks and develop treatments and vaccines, managing a global pandemic itself is largely a problem to be dealt with by economic and political tools. However, the COVID-19 pandemic has revealed serious flaws in that view. Prior to the crisis, the United Kingdom and the United States were judged to be among the best prepared for a global pandemic. The 2019 Global Health Security Index (GHSI) had ranked the UK and the US as first and second out of 195 countries, both for overall preparedness and specifically in their capacity for 'rapid response to and mitigation of the spread of an epidemic' (Roberts 2020: 17–18). At the time of writing, in the grip of the pandemic, they stand among the worst in the world (by cases or by deaths per population).[1] Evidently, our expectations for the state apparatus that would be needed to deal with a global pandemic and who is best placed to implement it were wrong. Surprisingly, responses even from apparently well-resourced nations were too little, too late, and ineffective. Many poorer countries have managed to keep infection rates significantly lower. Different countries have varied widely in their experience and strategies, and have performed well or badly at different times and in different respects. But in general, political leaders (among others) have demonstrated a familiar tendency to underestimate the threat and overestimate their ability to deal with it (Johnson 2004; Sharot 2011a).[2]

[1] See Johns Hopkins University of Medicine Coronavirus Resource Center (https://coronavirus.jhu.edu).

[2] Successful pandemic responses clearly did not depend on the ideal of a western democratic political system, since some democracies have fared relatively poorly and some autocracies have fared relatively well. Rather, other factors seem to have proven more important for the effectiveness of pandemic

An evolutionary perspective can help to understand these failures and why a pandemic virus is so hard to defeat. If humans and states were rational actors facing a static threat, we could follow the data and the scientific advice and governments could respond to a global pandemic with effectiveness and coordination.[3] We would still have a major problem to deal with, but it would be easier to solve than the one we actually have, which is responding to a *rapidly changing* threat with a population that is *far from the ideal of rational actors*, waylaid by a variety of evolved dispositions and cognitive biases. Of course, great uncertainty means it is hard for anyone to know just how aggressively one 'should' fight the virus, given that every extra effort imposes costs and sacrifices opportunities. But the uncertainty also means that there is greater room for big mistakes, and thus greater weight on applying the precautionary principle—to err on the side of caution (Johnson et al. 2013).

Worse still, leaders themselves have struggled to understand and communicate the scientific data and advice. Of course, they have a difficult balancing act to trade off multiple competing demands and stakeholders—not least keeping the economy afloat while, at the same time, severely curtailing people's freedoms. But it is hardly uncontroversial to say that they have recurrently got a lot about COVID-19 wrong. Even well-informed opinion leaders and policymakers, who may have seen things clearly themselves, still evidently expected *other* people to behave rationally and for a long time continued to treat the virus as a *static* threat. Meanwhile, some leaders blatantly failed to recognize the gravity of the situation. Adam Roberts lamented US President Donald Trump's actions as 'serially gormless involvements in the COVID-19 crisis' (Roberts 2020: 31).

By virtue of evolution, humans are not rational actors but instinctive biological ones, with varied and contingent responses, and viruses are not a static threat but a dynamic and changing one. We need to think more about our own evolution as well as the evolution of viruses to effectively address pandemics. Politics obviously remains central, but an evolutionary perspective can help.

Plan of the Chapter

This chapter focuses on two broad insights from evolution: (1) the biology and evolution of viruses; and (2) the biology and evolution of the host—us. As for the first, *evolutionary biology* provides a framework for considering what viruses 'want', that is, the selective pressures that cause them to operate the way they do,

mitigation efforts, regardless of regime type, such as past experience, crisis leadership, and flexible institutions (Roberts 2020).

[3] As discussed by Herrmann and Isabelle in Chapter 14 of this volume, the fact that rational agents can have heterogenous initial preferences, and the externalities linked to individuals' choices and behaviours, would nevertheless make this a complex task, of course.

their adaptations, strategies, mechanisms of spread, severity, lethality, and (not least) their rapid genetic mutation and the evolution taking place right now—the evolutionary 'arms race' that underlies viruses' and hosts' strategies to outwit and stay ahead of each other.

As for the second, an evolutionary perspective also helps us understand the *hosts*—human beings—and our behaviour and responses to disease. Sometimes our biology and behaviour seems puzzling or irrational, but it can make more sense under the lens of evolution. For example, *evolutionary medicine* provides a framework for thinking about why the human body succumbs and reacts the way it does to disease and treatment. This includes, for example, how and why pathogens manipulate our biology in order to spread more effectively, and why the body's reaction—often itself an overreaction—to infection rather than the infection itself can be crucial to the consequences of disease (Wrotek et al. 2020). In addition, *evolutionary psychology* helps to explain common *behavioural* responses to disease, as evolved heuristics that, today, can have 'adaptive' or 'maladaptive' consequences. Depending on the context, such heuristics include the manifestation of over-optimism and ill-preparedness for disasters, the 'negativity bias' underlying apparent overreaction and fear, and the conflicts between self-interest and the public good. Finally, *gene-culture co-evolution* sheds light on the interaction between human evolution and the environment, and how selection pressures acting on human biology are in part shaped by the cultural traits and environment we have ourselves created. This means that changing physical and social environments have, in turn, impacted our genetic adaptations, not least in relation to disease and our responses to them.

This chapter gives an overview of these various evolutionary insights. Each is a rather different set of ideas, even representing different subdisciplines in themselves (e.g. biology, genetics, medicine, psychology, anthropology). But they are united by the common underlying theoretical framework of evolution. In taking a brief look at them all, this chapter aims to advance the broader goal of the book in exploring how different disciplinary approaches can bring new ways of thinking about problems in politics and international relations (see the Introduction by Ackerly et al.). Evolution raises a range of new questions, and often novel solutions, which are unique to an evolutionary approach. One could write such a chapter at any time and focusing on any number of topics. But COVID-19 starkly highlights the importance of biology in the future of the globe (and the field), as well as the importance of humans not just as political actors but also as biological organisms with an evolutionary legacy and part of a complex evolving ecosystem that we ignore at our peril. Arguably, we have not appreciated the danger of our biological vulnerabilities in a newly globalized world until it was too late.

The Pathogen: An Evolutionary Perspective on Viruses

The Biology of Viruses

Viruses have been around for millions of years and long predate humans—we are a mere blip in the story. However, we haven't known about them for long. As the smallest biological entities, around 50 times smaller than bacteria (varying from just 20 to 300nm), they cannot be seen with a light microscope and eluded identification altogether until the advent of electron microscopy in the 1930s. Before that, they remained mysterious agents of disease in plants and other organisms, earning them the name 'virus'—the latin word for poison (Campbell 1993; Green et al. 1990).

Though simple, or precisely because of their simplicity, viruses are remarkable entities. Comprising just a small amount of genetic material and a protein casing, they are able to hijack a host organism's cellular machinery and turn it over to exclusively serve their own purposes. Following infection, proteins on the virus's exterior (such as the now famous 'spike protein' in SARS-CoV-2—the virus giving rise to COVID-19) bind to receptors on the host cell surface and then fuse with or inject their genetic material directly into the cell. Once inside, the host's DNA is deactivated and the cell starts producing the building blocks of more virus particles instead, replicating multiple new copies of their genetic material and synthesizing more encasing proteins. Viruses have the property of 'self-assembly', meaning that after production of the new components, the virus genes and protein casings automatically snap back together to form complete virus particles. Finally, the new viruses burst from the cell, going on to infect new cells in the same or new hosts. In a final trick, some viruses actually take on a coat of the host cell's own membrane as they escape, so that the virus looks to other host cells like one of its own. Viruses are masters of disguise and this makes them particularly hard for hosts to detect and defeat.

The clinical effects of viruses are highly variable. Some damage or kill the cells they use to replicate, others release toxins, others trigger the body's own defences to go into overdrive in fighting the virus. (The immune response can be seen as an emergency trade-off that puts the body at risk, e.g. with high fever, but in order to better fight the virus (Wrotek et al. 2020).) The damage done depends on the extent to which the host tissue is able to regenerate. Influenza virus affects the epithelial lining of the respiratory tract, the cells of which regenerate by normal cell division. Thus, an infected person can recover fully. By contrast, the polio virus infects nerve cells, which never divide and thus cannot be replaced, rendering the damage permanent.

The Evolution of Viruses

As well as understanding how viruses work, biology also sheds light on how viruses first emerged and evolved—and importantly, how they continue to evolve today. Since viruses rely on host cells to replicate, they cannot have predated other forms of life; rather, they must have evolved *from* existing organisms. They are thought to have originated as breakaway genetic material (initially like other naturally occurring 'mobile genetic elements', such as plasmids and transposons), which then acquired an independent ability to parasitize the machinery of host cells to create more copies of themselves. One tell-tale sign of viruses' origins is that they often tend to be more genetically similar to their hosts than they are to other viruses (indeed, many virus genes are more or less identical to those of their hosts), betraying an original shared ancestry with the hosts themselves (Campbell 1993; Green et al. 1990). The evolution of viruses and host organisms are deeply intertwined so that, in genetic terms, many viruses have become part of the make-up of hosts, just as hosts are part of the make-up of viruses (Ryan 2009; Villarreal 2005).

This origin also accounts for viruses' 'host range'—the number and types of different species that a given virus can infect. This depends largely on the recognition of binding proteins on the virus exterior and corresponding receptors on the host cell surface. Sometimes, common receptors found in different cells allow infection by the same virus of multiple species. Swine flu, for example, can infect both pigs and humans. Rabies, similarly, can infect rodents, dogs, and humans. Other viruses have a much narrower range, and may be limited not merely to a single species but to *specific cells* within that species. The common cold virus, for example, only affects cells of the upper respiratory tract, and ignores others. SARS-CoV-2 appears to have originated in bats, and then jumped to humans, probably via an intermediate host, such as a pangolin (Burki 2020).

One can argue about whether viruses represent 'living' things. With no cells or cellular machinery of their own (which all other living organisms have), they are 'obligate parasites' that are not able to reproduce without the help of the cells of another organism. Unlike bacteria, which can live and grow on a petri dish, viruses are inert and *cannot* replicate unless they infect a host cell. However, they have genetic material written out in the 'universal language of life' (as DNA or RNA), which contain the instructions for reproducing more copies of themselves (Campbell 1993: 354). This means that viruses evolve.

Whenever organisms replicate, mutations inevitably arise in the copying of their genetic code from one generation to the next (every time DNA or RNA is copied, there is a chance that mutations occur). Often, these mutations have no significant effect (and are thus 'neutral mutations'), or may actually be damaging to the organism (deleterious mutations). But sometimes these mutations bring novel advantages for replicating even more effectively. If so, carriers will do better than the original form and spread, along with the novel mutation. This is the engine

of evolution, and confers the ability to generate novel adaptations and, ultimately, speciation. The same is true for viruses, both in their origin and their subsequent development. SARS-CoV-2, for example, must have undergone a mutation that allowed a virus formerly confined to animal hosts to 'jump' to human hosts. The COVID-19 pandemic, therefore, is not due to a pre-existing virus as such, but to its *evolution*. As discussed later, it has evolved, and will evolve again.

On the one hand, these small changes in the genetic code are helpful for global mitigation efforts—without the accumulation of copying errors in the virus's genetic code (most of which are neutral mutations), scientists would not have been able to build a genetic family tree of how the virus has changed over time, which allows them to trace its origins and track its spread. On the other hand, mutations among pathogens represent a special danger of their own because they generate new variants to fuel an arms race of pathogen effectiveness and host defences (Dawkins and Krebs 1979; Ridley 1994). If and how—and how fast—the virus mutates ultimately affects our ability to avoid it, as well as to develop immunity and successful vaccines, and for these to stay effective for a reasonable period of time. Natural or induced immunity to the virus now does not guarantee continued immunity in the future against a virus that is changing all the time. This is why we have to develop new seasonal flu vaccines every year, which vary in their effectiveness.

What Viruses 'Want': An Evolutionary Lens on Virus Strategies

Of course, viruses don't 'want' anything, in the sense in which that word is commonly used and understood by most people. But as shorthand for how organisms are adapted to survive and reproduce by doing things that help them do so, it is a useful way of thinking about things. It allows us to think through the biology and behaviour of any organism to identify how the strategies they employ—even, or especially, if initially puzzling—may in fact be functional, biological adaptations that aid the organism in achieving its ends. It focuses our attention on the evolutionary rationale for why organisms have adapted to do the things they do. How does their biology or behaviour succeed in getting more copies of themselves into the next generation? This differentiates potentially functional characteristics from mere accidents, random noise, or empirical but unexplained trends.

We can engage the same logic for viruses. They seem nasty, vindictive pathogens because they make people ill and sometimes kill them. But viruses are biological organisms that are just doing what they do in order to survive and reproduce. In fact, their entire mode of replication is a remarkable evolutionary innovation. From the gene's eye view, as famously espoused by Richard Dawkins, viruses are notable in that they have figured out a way to escape the confines of 'orthodox' replication, in which genes have to share their passage to the future with

groups of many other genes in the chromosomes of sperm and egg, combining to transmit genes from one body to another once per generation (Dawkins 1976). Instead, virus genes bypass having to wait around for the painfully slow process of host reproduction altogether because their genetic material literally floats independently through the air to travel directly from body to body. This means that they can replicate much faster than other organisms, and without having to make all the adaptive compromises with other genes that have other priorities and duties in the body. Viruses have found a much more efficient method of replication.

This evolutionary perspective is important because it helps us to make sense of the often surprising biology and 'behaviour' of viruses, to understand the conditions that might be conducive to spreading or mitigating them, and the conditions under which they are likely to evolve in benign or harmful ways. Below are some examples, before we apply the approach to COVID-19.

In their efforts to increase the probability that they get passed on and replicated, many pathogens have evolved mechanisms to change not only their own behaviour, but also to change the behaviour of their hosts. For example, while coughing or sneezing might be seen as 'annoying byproducts of the virus's activities', in fact, they are highly adaptive consequences that help to transmit the virus and can be considered as 'deliberately engineered by the virus to help it to travel from one host to another' (Dawkins 1976: 246). Viruses are more likely to survive if they have traits that facilitate their spread among people, and common symptoms of viral diseases such as coughing, sneezing, and diarrhoea greatly enhance the transmissibility of the virus, as they forcefully expel them into the environment where other people can pick them up (Ewald 1997).

Similar dynamics are at play in other viruses, such as rabies. The rabies virus is transmitted by saliva, and thus requires infected animals coming into close contact with others and biting them. That can happen via normal animal interactions, but they don't bite each other all the time. However, rabies actually changes the behaviour of an infected animal in several ways that greatly increase the probability of transmission. Symptoms of rabies include (1) foaming at the mouth, which increases the amount and readiness of saliva to infect others; (2) restless roaming behaviour, taking infected animals far beyond their normal home range; and (3) an increase in aggressiveness that makes what are 'normally peaceful and friendly animals become ferocious biters' (Dawkins 1976: 247). A rabid dog becomes unafraid and aggressive. While we think of the virus as primarily afflicting the individual dog, it also drastically alters the host's biology and behaviour to help itself get into more dogs, and thereby replicate and spread more copies of the virus. Interestingly, a corollary is that the virus does not 'want' the dog to die; rather, it wants the dog to go around infecting as many other individuals as possible before it does so. A similar logic applies to other viruses. A smart virus should not kill its host. If anything, it should devise ways to be able to infect them again in the future.

As Rafe Sagarin put it, what 'the virus wants more than anything is stability … like a businessman trying to maintain a steady clientele' (Sagarin 2012: 156).

The Evolution of SARS-CoV-2: Genetic Mapping

Knowledge of the biology of SARS-CoV-2 has obviously been vital in developing clinical treatment of the virus and vaccines, as well as in forming policies of prevention and mitigation. What is perhaps less obvious is the importance of the *evolution* of the virus in mitigation efforts and the development of policy responses.

First, there is good news. An evolutionary approach is indispensable because it has allowed what would otherwise be an impossible study of how the virus spreads. Because mutations in the virus's genetic code occur at a regular rate, the cumulative history of gradual changes can be used as a molecular clock, leaving trails of breadcrumbs behind each new lineage as it spreads around the world. As the virus accumulates slight variations over time as a result of these copying errors (most of which have little effect but, nevertheless, build up cumulative fingerprints), we can build a genealogy of the virus. This allows one to trace, via the number and pattern of mutations, the branches of the 'family tree' and the common ancestors of emerging variants of the virus. The technique has long been used to track other virus pandemics, as was done, for example, with swine flu (H1N1) (Smith et al. 2009), and there has now been a vast scientific effort to trace the changing genomes of SARS-CoV-2 worldwide (see the Global Initiative for Sharing All Influenza Data (GISAID) database, https://www.gisaid.org, and Nextstrain, https://nextstrain.org).

One important insight is that it gives an estimate of when the virus *first emerged* among humans in China—and this date is estimated to be mid-to-late November 2019 or early December (Bedford et al. 2020). Why is this important? Because there are hugely significant political questions (and conspiracies) about the origins of COVID-19 and cover-ups about the first incidences of the virus in China and other countries, and whether it might have been man-made (Burki 2020). Evolutionary biology has been crucial in putting to rest some potentially inflammatory politics, quite apart from helping to deal with the virus. It is equally vital, of course, for political leaders and influential voices to understand this biology accurately.

Another important insight is that genetic sequencing allows us to tell how the virus has *spread*—from which points of origin, between which countries and among which people, and when these occurred. Then, in combination with other data, this can reveal what interventions have been more or less successful (or unsuccessful) in permitting the spread of particular strains. For example, the spread of one early variant of COVID-19, dubbed 20A.EU1, from Spain to the

UK during the summer of 2020 is evidence that the country-to-country travel restrictions in place at the time failed to contain the virus (a combination of states' permissive travel policies and people's eagerness to continue travelling). Evolutionary geneticist Emma Hodcroft, involved in the tracking project, pointed out that one obvious policy implication is the ability to distinguish local transmission (within country) to imported transmission (from other countries), which can be crucial to effective containment, as well as when to ease lockdown restrictions in the future (Cookson and Bott, 2020).

The Evolution of SARS-CoV-2: Virus Strategies

But what about the role of evolution in the strategies that the virus employs? How good is SARS-CoV-2 in this evolutionary arms race of cat and mouse? One feature that sets it apart from other viruses is its level of deceptiveness. In other flu viruses, people tend to become noticeably sick before they become particularly infectious (with the SARS outbreak in 2003, for instance, there were no cases of people spreading infection until after the onset of symptoms; see www.cdc.gov). This made mitigation efforts relatively straightforward because infected people can be easily identified and isolated before they infect others. That radically slows onward infection. But with COVID-19, the viral load hits a peak around, or even before, people first feel unwell (He et al. 2020). That means that people are likely to have already passed the virus on before they even realize they have it. The virus can be replicating away in the body's cells without generating any symptoms at all, and without even alerting our own immune system to its presence. In the words of virologist Paul Lehner, 'This virus is brilliant, it allows you to have a viral factory in your nose and feel completely well' (Gallagher 2020). It does this by deactivating the release of interferons that usually signal the presence of an infection to the rest of the body. Applying our gene's eye view, this, of course, is highly adaptive because transmission of the virus is much more 'successful' if you are not aware you are sick and carry on going about your normal business—and thus more effectively spread it to others. It is an unusual but understandable feature of SARS-CoV-2, as Lehner highlights: 'This is a really brilliant evolutionary tactic—you don't go to bed, you go out and have a good time.' Even laboratory observation of infected cells do not reveal that the virus is there, despite tests showing they are 'screaming with virus', which Lehner calls one of the virus's 'joker cards'.

This adaptive characteristic has, of course, been one of the enduring problems of COVID-19 pandemic mitigation, as well as the reason for the need for strict self-isolation measures if one has been in contact (even unknowingly) with an infected, or possibly infected, person. As well as developing clinical treatments, we have had to devise ways to clamp down on potential hosts and detect virus cases before people develop symptoms and to track down all potential contacts even if they

were fleeting contacts with strangers. This has proven to be an immense technical, social, and political challenge. In this respect, the virus is well ahead of us in the evolutionary arms race. The stealthy infection and spread of COVID-19, arguably the greatest challenge of the global pandemic response, is—from an evolutionary perspective—exactly what the virus wants.

This biological property of COVID-19 also accounts for much of the public and political discontent about the lockdowns. It seems fundamentally illogical to stop people who *seem* well from doing things, and can still feel harsh even if one understands the problem. But this is precisely how the virus is spreading. The hidden diffusion of invisible agents among apparently healthy people is hard to grasp, yet essential to act upon if we are to achieve effective prevention. Unfortunately, it means the restriction of civil liberties of asymptomatic people, which makes for unpopular policy and public discontent. Notably, the relatively low mortality rate of COVID-19 makes this dilemma worse. The balance of risk and associated fear and responses (Blumstein 2020) would likely be different in a more dangerous virus outbreak.

Virus Mutations: The Next-Generation Threat

The SARS-CoV-2 virus that leads to COVID-19 is damaging and deadly enough as it is, but the much scarier possibility has always been that it mutates to become even better at what it does: more infectious, stealthier, or more deadly. Viruses, like all organisms, are under the pressure of natural selection, which favours variants that reproduce more effectively at the expense of variants that reproduce less effectively. Thus, any mutation that increases the virus's ability to reproduce or pass on more copies of itself will spread, regardless of any other consequences for the host.

Evolutionary geneticists often tend to express a word of caution here—as noted above, most mutations are neutral or deleterious to the virus. They thus tend to lead to ineffective viruses or merely lay down an additional marker for our tracing of their spread, but rarely result in a suddenly more dangerous form (e.g. because of increased transmissibility or severity). Early on in the pandemic, that may have seemed unlikely or even far-fetched. However, it has become perhaps the single most important problem, especially in a novel global pandemic with a vast susceptible population of billions of people, and a danger that an evolutionary approach has forced us to pay attention to (Korber et al. 2020; Van Egeren et al. 2020). Here's why it matters.

First, remember that a mutation can occur every time genetic material is copied in the process of replication. For humans, that occurs once a generation. For viruses, this happens millions of times even within a single infected individual—in days (it is estimated that at peak infection a single person has 10^9–10^{11} individual

virions in their body; Sender et al. 2020). The capacity for mutation (and thus evolution) is operating at a *much* faster speed than it is for humans and other animals that are confined to biological reproduction. The same is true of bacterial pathogens, which is why we are constantly losing the arms race against them in the widespread problem of antibiotic resistance—the time frame for the evolution of bacteria resistant to a new antibiotic 'is often frighteningly short' (Gluckman et al. 2011: 257). It is also important to recognize one very important thing: the selection pressure on the virus will *increase*, rather than decrease, once it is faced with vaccination programmes and an accumulating herd immunity that start fighting it directly (Van Egeren et al. 2020). The greatest danger in the arms race may be yet to come. In the 'war' against the virus so far, we have mainly been running away (in avoiding and averting it). Only now are we starting to shoot back.

Second, as mentioned earlier, mutations of the virus genome are what allowed it to move from animals to humans in the first place. It was formerly a virus that had a host range limited to other animal species (mainly bats) but was not infectious to humans. But then, there was a mutation, or a set of mutations, that together allowed it to 'jump' to humans (Burki 2020). That was not an inconsequential mutation for the human population. More such evolutionary leaps by other as yet unknown viruses lie in the future.

Third, new mutations of the virus *since* that origin have already emerged. We saw above that many of these are just small alterations that help us trace different strains, but do not necessarily change how the virus operates. But other mutations (or combinations of several mutations) represent new variants of the virus that can have different characteristics. And these are not just minor details. For example, it is estimated that the 20A.EU1 variant of coronavirus that emerged in Spain over the summer of 2020 underlay the second wave in Europe (in the autumn of 2020). It accounted for more than 80% of UK cases (at the time of first drafting in October 2020), probably in large part because of holidaymakers who travelled to Spain over the summer and brought it home with them (Hodcroft et al. 2020). Of course, since the original drafting of this chapter, we have seen a steady stream of additional new variants that are proving much more significant (to which I will return shortly).

Fourth, the virus can jump around again between species. In Denmark, coronavirus jumped from humans to mink, of which there were millions in farms sustaining the largest fur-trading industry in the world (Milne and Cookson 2020). But this is not just a problem for the animals. The virus mutated into a different strain within the mink population, and then jumped *back* to humans again. It was feared that this new variant may spread more widely among the human population and render vaccines under development less effective. The Danish government sought emergency powers to exterminate all 17 million mink in the country (the Netherlands had already eliminated their entire population). This is the kind of danger that mutations can pose when the virus becomes exposed to large and interacting populations of people and other animals.

Implications of an Evolutionary Approach

If new variants of the virus are not too different from previous ones, then immune responses among recovered (or vaccinated) individuals may remain effective even against new variants. But this is not necessarily the case. That is, of course, why the virus evolves. It does not 'want' to be recognized or defended by hosts' immune systems, and new variants that avoid detection and defences will do better than older variants to which herd immunity is building. Plus, most importantly of all, as well as merely posing a challenge to levels of immunity, new variants may be more infectious or more harmful than their ancestors. The 20A.EU1 variant mentioned above did not seem to be more infectious. But at least three other variants quickly emerged that did appear to be becoming more infectious. The first, variant D614G, emerged quite early on in February 2020, and already was suggested to be more infectious and to lead to higher viral loads than original forms of the virus (Korber et al. 2020; Volz et al. 2020). While there was debate about the extent to which it was more transmissible (Grubaugh et al. 2020), it is clear that after emergence it spread rapidly around the world, replacing and coming to dominate in even well-established populations of former variants, suggesting that it had been subject to a process of positive selection (Korber et al. 2020). It was also of concern because the variant was an amino acid change in the spike protein, which is exactly what vaccines were targeting. Fortunately, early variants still appeared to be susceptible to vaccines under development (Weissman et al. 2020). However, modelling suggests that subsequent mutations are likely to give rise to resistance in an 'evolutionary escape' (or 'vaccine escape') that will be subject to greater levels of positive selection with the roll out of vaccines and accumulating levels of recovered immunity in the population (Van Egeren et al. 2020). Korber et al. (2020) developed an 'early warning' system to closely monitor spike protein evolution as the pandemic develops.

They did not have to wait long before something more sinister came along. In December 2020, a H69/V70 variant rapidly spread to become the dominant form of the virus in the South-East of the UK, spreading quickly around the country and plunging it into a third severe lockdown (European Centre for Disease Prevention and Control 2020). Sometimes, new variants can come to dominate accidentally via founder effects (new variants spread more because they happen to emerge in areas with high rates of social mixing and transmission). However, it is believed that this variant was indeed up to 70% more transmissible than prior forms of the virus, making finely balanced disease control restrictions suddenly inadequate. Also, the mutations again related to changes in the virus spike protein, raising concerns about the efficacy of new vaccines just being rolled out and the antibodies from people who formerly had COVID-19. At the time, it was thought that the current vaccines would remain effective, as they attack several different parts of the virus. Even if not, they can be adjusted to work on similar variations (as are the seasonal flu vaccines).

Finally, of even greater concern was the 501.V2 variant that emerged in South Africa. This one had more significant changes to the spike protein, and thus could pose problems for the vaccines being deployed. Antibodies to previous COVID-19 infections may not be able to recognize new virus variants as easily. No doubt, by the time of publication things will have moved on further (at the time of copy-editing, we are again in the grip of yet another new variant—Omicron). But the point should be clear: viruses are evolving entities that do not just present a static challenge to 'solve', but a race in which it is vital to stay in for the long haul and keep up. Public policy has many demands, but an evolutionary perspective presses home that it is imperative to rapidly quash the virus to manageable levels before it can evolve into an even greater problem.

With such a large and vulnerable population with no prior immunity, there is much scope for the virus not only to spread, whether quickly, or in a long 'slow burn', but either way to evolve rapidly along the way. This is unlike other viruses to which there has been at least some prior immunity in the population, and which thus spread more slowly and to a more limited group. Even with past novel viruses such as swine flu, it was similar enough to extant viruses that people already had at least some protection from previous exposure. COVID-19 is entirely new, and no one had any prior immunity, which is why it was constantly on the verge of ripping through unprotected populations like wildfire, in an exponential increase.[4] The greater the population infected, the greater the sheer number of virus replications taking place and thus the greater number of mutations that will occur and, consequently, the higher the probability of more virulent forms emerging.

Policy Implications

For all the intense public and political discussion of COVID-19, what was striking is that the role of virus *evolution* was largely overlooked—at least here in the UK—until the rapid spread of the H69/V70 variant forced the issue (and sharply reversed government policy). An evolutionary perspective raises troubling policy questions of its own. Until that point, many people seem to have assumed a static threat which was waiting patiently for us to attack it when we were able—with treatments, vaccines, and other measures. But the reality is that the virus has always been changing and we need to think about its ongoing and future evolution as well as its current state. In weighing up options for how to deal with the virus, how might our own actions and policies impact the course of its evolution? This important question seems to be rarely, if ever, discussed (at least in the media and among

[4] There is some indication that, while we do not have antibodies, prior cold virus T cells may help fight COVID-19 (Doshi 2020).

politicians). If we could click our fingers and eradicate the virus, there would be no further evolution. The problem with a slow war is that it gives time for evolution to take place. The existence of multiple animal–human–animal jumps and numerous emerging variants suggest that this remains a significant risk. If we maintain partial and rolling lockdowns, allowing a slow burn of the virus through the global population, the potential for further evolution of the virus will stay high over a longer period of time. As well as minimizing hospitalizations and deaths and getting back to normal life and economic recovery, another important reason to move hard and fast with lockdowns and vaccination programmes is to smother the virus to manageable levels as quickly as possible before it can evolve into something different, or worse. This might even arguably be the *most* important rationale for draconian policies and infringements of civil liberties. A constant evolution of viruses happens already with seasonal flu viruses, and we are likely to face a new (but harder) ongoing battle with SARS-CoV-2 for years to come. One piece of good news is that the underlying rate of genetic change in SARS-CoV-2 is actually relatively slow compared to some other viruses (such as HIV or flu; although, of course, the problem is still the sheer number, rather than rate, of opportunities for COVID-19 infections in a population of billions). One article cautioned, back in April 2020: 'Although surveillance has not shown any mutations that would affect its transmissibility or virulence, scientists are watching closely in case they appear' (Cookson and Bott 2020). This has now happened multiple times, and evolutionary theory suggests we should not be surprised.

There is also a further problem to consider, and which governments and public health services need to remain vigilant about. As Van Egeren et al. (2020) warn, the virus will only really come under significant evolutionary selection pressure when we have mass vaccinations and the accumulation of immunity fighting back. Until recently, the virus has enjoyed a vast sea of susceptible individuals with no immunity to counter its spread. The rise of vaccinated and recovered people in populations around the world will make the virus's environment for replication much tougher, and any new potentially beneficial mutations correspondingly more advantageous relative to existing forms. Indeed, in a vaccinated world, mutations will become key to virus survival. The toughest part of the arms race may be yet to come.

The Host: An Evolutionary Perspective on Human Responses to Disease

Evolutionary Medicine

Beyond the direct application of evolutionary principles to understand and fight pathogens themselves, evolutionary theory has also given rise to a wholesale rethink of medicine at the level of the *host*—how humans succumb and respond to

disease. 'Evolutionary medicine' is a relatively new field that brings its own brand of insights for clinical medicine and public health (Gluckman et al. 2009; Nesse and Williams 1995; Williams and Nesse 1991). To simplify the story, much of medicine has treated the human being as a machine which, when it develops a fault, needs fixing. Instead, evolutionary medicine focuses on how the machine was built by natural selection in the first place, how the types of 'fault' have developed and changed over time, and how the machine might best behave and be treated to avoid them. Looking at health and disease from an evolutionary perspective—that is, in the context of the evolutionary history of human beings and our exposure to changing environments and prevailing diseases—generates different sorts of questions and novel responses. These are not instead of, but complementary to the normal mechanistic identification of causes, symptoms, and treatments, and an approach that is becoming an important part of medical training (Nesse et al. 2010; Nesse and Stearns 2008). The insights from evolutionary medicine can be classed into three main categories.

First, evolutionary medicine's 'core principle' is that *natural selection acts to promote biological fitness, not health or longevity* (Gluckman et al. 2011: 251). This is a vital nuance because, while counter-intuitive to the casual observer, after reproductive age natural selection has little interest in (there is less 'selection pressure') keeping us alive. This explains why we become so vulnerable to certain diseases as we age and why our responses to them weaken. For example, genes that promote growth in early life to become strong and resilient may have been favoured by natural selection, but later in life the same genes can contribute to obesity, diabetes, and cardiovascular disease—a phenomenon called 'agonistic pleiotropy' (Gluckman et al. 2011: 255). Selection is often absent, or not strong enough to mitigate the detrimental effects later on because if we are no longer producing offspring there are no (or lesser) consequences for our genes in subsequent generations. One implication for viruses such as COVID-19 is the susceptibility of older people to infection and severe symptoms. Long-lived individuals can sometimes have an advantage, in having built up immunity to numerous different disease pathogens over their lifetimes. However, when faced with a novel virus to which we have no prior immunity, older people are at a physiological disadvantage because they produce less varied T cells, making it harder for them to develop immunity to new pathogens by trial and error.[5]

Second, *evolutionary legacy affects our susceptibility to disease in different environments*. For example, scurvy is a debilitating and potentially fatal disease that occurs on long sea voyages or during famines, when one fails to obtain vitamin C

[5] On the other hand, and in a different domain, older people also accumulate long memories that include rare but catastrophic events that most people—including political leaders—have never experienced before and may thus discount too readily. Sometimes, elders see dangers and hardships that others cannot imagine and this biological store of knowledge could be one reason why natural selection has kept humans alive after reproduction as long as it has. Thanks to Ed LeGrand for this point.

from fresh fruit and other natural sources. Like many animals, human ancestors used to be able to synthesize vitamin C themselves. But at some point in our ancestral past, we lost the necessary enzyme for making this crucial part of our diet (probably through random neutral mutations) when fruit was a plentiful part of our environment and we didn't need to make vitamin C of our own. Other examples include short-sightedness, which is much more common today as a result of our increasingly indoor life focusing on close objects in artificial light, which distorts the growth of our eyes in childhood. The rise in mental illness is also thought to be partly a result of navigating a novel social environment that isolates us from the close and extensive family and social groups that were ever-present in our evolution (Dunbar 2003; Gluckman et al. 2011). As one example applied to the COVID-19 pandemic, imposed periods of isolation and the prevention of social and physical contact are precisely the kinds of novelty that can be psychologically harmful for a social animal like us.

Third, *evolutionary mismatch* describes the problems that can arise given that the contemporary world is very different from the one in which we evolved. Our biological and behavioural mechanisms are adaptations designed for the conditions of our past, not the present. Notably, for most of our evolutionary history, 'we largely lived in small social groups, survived on very different diets and were exposed to a much lower density of pathogens and toxins' (Gluckman et al. 2011: 252). This leads to a potential *mismatch* between the environment in which (and for which) our bodies and brains evolved and the very different social and physical environments in which we now live. This often plays havoc with the way we behave in relation to our current environment and our biological responses to it. A classic example is our taste for sugar, salt, and fat. This is an evolved disposition that was highly adaptive when such commodities were rare in our evolutionary past (and, indeed, long before that for all vertebrate species) and it was adaptive to seek them out, but in today's world of plentiful junk food the same disposition contributes to heart disease and diabetes which kill, for example, half a million Americans a year (www.cdc.gov/nchs/fastats, 2020).

Obvious insights from this evolutionary perspective for COVID-19 include, first, the shift to living in extremely large, dense, and globally interconnected groups, through which diseases can spread easily and rapidly into new susceptible populations. Human biology and social organization have a long history of cohabitation with disease (Lederberg 2000; Roberts 2020), but we are not so well equipped to deal with the novelty and scale of new global pandemics. Second, population expansion has led to large numbers of people settling in formerly natural areas in many parts of the world, increasing exposure to wild animals and the consequent emergence of zoonotic diseases. Wild animals tend to be the source of most major influenza virus pandemics, and in the future animal-to-human transmissions are expected to increase over time with burgeoning populations and declining alternative habitats, with an inevitable encroachment into

wildlife areas and an increased probability of exposure to (and evolution of) new pathogens.

One might ask why it is that, after all the millions of years of evolution, the human body seems so vulnerable to so many different kinds of disease. But the answer itself also comes from evolutionary biology. First, pathogens are evolving too, so there is an arms race in which the evolution of our defences has to constantly adapt to counter the evolving strategies of disease pathogens, or when we move into novel environments (as with the constant battle to develop a new seasonal flu vaccine each year). Second, natural selection can't solve every problem, and must make trade-offs in which susceptibility to a given disease may be a necessary risk of pursuing some other important evolutionary function (such as sickle-cell anaemia reducing the blood's oxygen-carrying capacity, but at the same time providing protection against malaria). Third, since selection does not maximize health and longevity, but rather maximizes reproduction, many diseases result from a sacrifice of resources that might otherwise have been allocated to greater defence and countermeasures against pathogens.

Evolutionary Psychology

An important question arising from an evolutionary perspective is the extent to which we are adapted to deal not just with the *biological* challenges of the modern world, but also with the *cognitive* challenges of the modern world. The basic story here is that, ideally, we would act as rational actors, but we don't. One might expect general intelligence and rational thinking to be able to observe a problem accurately, evaluate the evidence in an even-handed manner, and act accordingly to minimize harms and maximize returns. However, a large research literature now shows that, empirically, humans fail to conform to this rational actor model of behaviour in numerous ways (Fiske and Taylor 2013; Gilovich et al. 2002; Kahneman 2011). Instead, even when the information we have is accurate, we are subject to a host of evolved cognitive heuristics and biases that lead us to make decisions that violate, sometimes quite radically, the expectations of rational choice. There are long lists of such biases, which have been identified and replicated under controlled conditions in the lab, as well as observed at work in the real world, where they contribute to significant decision-making failures from financial and policy disasters to crises and wars (Jervis 1976, 2017; Tetlock 1998, 2005). One important application is to think through how cognitive biases may affect our responses to the current and future pandemics. Cognitive biases are especially prominent when we face a blizzard of conflicting or confusing information (as we have about so many aspects of COVID-19 and governmental advice) and have to rely on our gut instincts instead of perfect information and calculation.

Before we come to specific examples, if these biases are such a liability, why do we have them? This is where evolutionary psychology comes in. Human physiological and psychological traits evolved to deal with the adaptive problems of our evolutionary past, not with the often very different kinds and scale of problems that we experience today, especially those that occur in international relations and events such as global pandemics (Barkow et al. 1992; Buss 2005; Pinker 2002; Wright 1994). The human lineage spent 99% of its history in a Pleistocene environment of small kin groups of hunter-gatherers, and only 1% in the post-agricultural environment of large, urbanized societies with sophisticated institutions and technologies. As a result, we should not necessarily expect evolved psychological mechanisms to trigger in appropriate circumstances, or to cause their intended effects. (In fact, many human dispositions extend much further back in the evolution of not just humans, but also *ancestral* species that preceded us many millions of years ago, cementing them into our, and many other, vertebrate physiological systems (Blumstein 2020).) Even when they *are* triggered by an appropriate stimulus (i.e. an evolutionarily relevant one), in modern settings the behaviour they give rise to may nevertheless cause inappropriate or costly outcomes (since the prevailing costs and benefits of the consequences of our behaviour have also changed). This phenomenon helps to explain, for example, the stubborn persistence of self-interest, nepotism, status seeking, addiction, and crimes of passion, even when those behaviours will come back to bite us and we know it (Burnham and Phelan 2001; Frank 1988; Giphart and van Vugt 2018; Kenrick and Griskevicius 2013).

Unfortunately, many of our evolved cognitive mechanisms appear to have been severely detrimental to efforts to mitigate the COVID-19 pandemic, both among the public and among political leaders as well—first in recognizing the danger, second in responding to it, and third in coordinating activities to fight it.

First, human brains are arguably ill suited to fully grasp the *threat* itself and the consequent implications of a global pandemic. As the virus initially spread in China and on to other regions, it is remarkable to think back to the slow reactions among governments around the world. They could have been even worse, of course, but national responses were widely regarded as having been complacent and slow. Historically, this turns out to be quite a common phenomenon. States repeatedly fail to recognize and prepare for impending dangers, especially novel dangers with which they are unfamiliar (Betts 1983; Johnson and Madin 2008; Sagarin 2012).[6] Several cognitive biases contribute to doubt or disbelief about the severity of new dangers, such as the widespread 'optimism bias' (Sharot 2011b), 'cognitive dissonance' (Cooper 2007; Festinger 1957), and the 'exponential-growth prediction bias', which, in the case of COVID-19, means that people fundamentally fail to appreciate the potential rapidity of its spread (Banerjee et al. 2020).

[6] Although, of course, states can sometimes be overly fearful as well; see Thrall and Cramer (2009).

There also seems to be a survivorship bias when it comes to addressing diseases, as we 'tend to underestimate past threats and disasters because we know that humankind survived them' (Roberts 2020: 8). In short, several important cognitive biases appear to have been at work in hindering our ability to act quickly.

Second, people's *responses* to the virus have also been hampered by common cognitive biases. The UK government (among others) set up a behavioural insights team specifically to anticipate how people would likely respond to the pandemic and restrictions on freedom, and how policy could be developed to counter common biases and maximize compliance (www.bi.team/our-work/covid-19). The major dynamic among afflicted populations has, fortunately, been a general acceptance of the problem and cooperation with government lockdowns and restrictions on behaviour. But such draconian rules have not always been agreed with or followed, and there have been multiple and sustained breaches, as well as organized protests and resistance. Much of the violations (and compliance) are due to people's perfectly rational assessment of their own level of risk, and making corresponding trade-offs in deciding what they can and cannot (or should and should not) do. The primary problem with that has been that people—especially younger ones—are assessing their *own* immediate risk (perhaps correctly), but not the risk that their behaviour poses to others, the longer-term consequences of sustained risk-taking, or the cumulative risk if everyone does the same thing. We tend to discount others and the future. At least part of the problem in achieving and enforcing lockdown rules, therefore, has been a natural disinclination, amplified by self-interest and cognitive dispositions, to change one's behaviour because of invisible agents—especially if one feels fine.

In ancestral environments, we were subject to lower and fewer novel pathogen loads, within a much lower population, at a lower density, and with more activity outdoors. While diseases were common, we are not well adapted to perform and sustain the kinds of behaviours necessary to fight a global pandemic (when completely novel diseases were introduced into susceptible populations in history, such as by Spanish conquistadors in South America, the consequences were catastrophic). The need for constant social distancing, perhaps most notably, especially with close friends and family, is not just inconvenient but flies in the face of our instinctive social nature.[7] Moreover, the agents of disease themselves are intangible. While we have deeply ingrained natural disgust mechanisms that react to obvious signs of unsanitary or infected objects, people, and food (Curtis et al. 2011), we do not have an evolved repulsion for invisible agents of disease such as viruses. George Murdock surveyed 186 Indigenous societies around the world and found all of them to attribute illness not to the work of microscopic pathogens, but

[7] For an animal behaviourist perspective on this exact point, see Daniel T. Blumstein. "Banning Sociality Is Costly Our obligate sociality makes us vulnerable to necessary pandemic controls." Psychology Today, 29, 2020.

rather to the malicious work of supernatural agents (Murdock 1980). We have to fight against the grain of human nature to prevent ourselves from engaging in social contact and conceiving of biological agents of disease. People go about their business—often seeking out contact and without masks—appearing remarkably unafraid, perhaps itself an adaptive characteristic but one that carries new dangers in unusual times (Sharot 2011a).

Third, there is a severe problem of *coordination and cooperation*. An effective population response requires subordinating one's self-interest for the greater public good. Despite (or sometimes precisely because of) government policies, there are strong incentives for individuals to continue moving around and meeting people to maintain their welfare and livelihoods, avoid losing their job or their clients, and see and help friends and family, not to mention having fun and doing the things they want to do. This is a classic collective action problem. While the virus would most effectively be stamped out if everyone put their self-interests aside and contributed 100% to the public good, people have strong incentives acting in the opposite direction. This is exacerbated if one sees others violating the rules or enjoying exceptions. The collective action problem is already a 'problem' as it is because, as well as the uncertainty about whether the collective goal will be achieved at all, the decision to participate oneself means that one may end up wasting time, effort, or resources in contributing to a lost cause. Moreover, having contributed, one is also left in a worse position compared to others who did not expend any of their time, effort, or resources (Olson 1965; Ostrom 1990). Of course, this is why leadership and rules become vital in such cases to ensure or enforce cohesion, rather than to merely expect or hope for it. But it doesn't always succeed. Especially in times of crisis like this one, 'Trust in leadership is essential because struggles against infectious diseases necessitate a degree of individual sacrifice for the social good' (Roberts 2020: 32). Pandemic restrictions in particular need to be perceived as clear, having a solid rationale, and working towards a definite goal. If they don't, self-interest prevails. In short, while we can see the logic of coordinated action and how it would be more effective if everyone commits to it, at the same time, we have powerful dispositions to look after ourselves and our families first. These are not just random psychological tendencies; they are deeply entrenched aspects of our evolved psychology (Lieberman et al. 2007; Pinker 2002). Rational logic and politicians imploring us to follow the rules only goes so far in a deeply social animal.

Before ending this section, it is worth highlighting three important corollaries. First, all of the above tended to imply cognitive biases acting among the public at large. However, cognitive biases apply just as much to leaders and policymakers as they do to everyone else. As well as cognitive biases affecting public reactions and compliance, therefore, they also contribute to explaining the policies (or the lack of policies) of state decision-makers (see, e.g. Jervis 2017; Sears et al. 2003; Tetlock 2005). As one obvious example, several high-profile politicians in multiple

countries came under fire or had to step down because they breached the very lockdown rules their own governments had imposed (top English, Scottish, and New Zealand lawmakers travelled to be with family in violation of lockdown restrictions). Human instincts are a powerful force and evolved incentives often override our more rational side, whether consciously or not (T. D. Wilson 2004). This applies to us all.

Second, some biases can work in opposition. For example, above I suggested that people are primarily self-interested. But, of course, there are also strong dispositions that, under certain circumstances, make humans remarkably prosocial as well (Cronk and Leech 2013; McCullough 2020; Ridley 1996). This means we must be careful to identify the *conditions* that give rise to one bias rather than another. Above, I mentioned overconfidence as a potential contributor to ill-preparedness in the face of the pandemic. But in other respects we also suffer from a 'negativity bias', which can explain instances of overreaction and fear (Baumeister et al. 2001; Tierney and Baumeister 2019). Therefore, we need to consider the different contexts and conditions under which each bias emerges, and how they may interact. Positive and negative biases, for example, can apply simultaneously but with respect to different objects: optimism bias appears to be prominent in evaluations of the *self* or one's future prospects, whereas the negativity bias tends to come to the fore when people are evaluating *others*' behaviour or the environment—including especially the risk of disease and contagion (Rozin and Royzman 2001). When both biases arise in combination, this can be a recipe for disaster, for example, inflating the threat of some external agent but, at the same time, overestimating our own ability to overcome it (Johnson and Tierney 2019). This may help to explain instances of both fear and complacency surrounding the pandemic response.

Third, many of the biases mentioned above are well known in social psychology and behavioural economics. One could thus accept their role without invoking any evolutionary origin. What exactly does an evolutionary perspective add? An evolutionary perspective is helpful because it sheds light on why we have these biases in the first place (their ultimate causes), what the biological mechanisms and triggers for the biases are (their proximate causes), and thus the physical and social conditions under which we can expect them to be amplified or reduced, and—consequently—how they can best be mitigated. Furthermore, an evolutionary perspective also forces us to consider the adaptive significance of our cognitive biases. What are they *for*? Applying an evolutionary lens reminds us that cognitive biases should not always be seen as detrimental. After all, they evolved because they helped us make good decisions, not bad ones. Thus, in the right (evolutionarily relevant) contexts they may, in fact, steer us to make more effective decisions rather than disastrous ones, especially in fast-moving situations when we have to act with poor information under uncertainty, without the luxury of time or resources to wait on perfect decision-making. Instead, we have to rely on instinct (Gigerenzer and Brighton 2009; Haselton et al. 2009; Johnson 2020). Arguably,

for example, the negativity bias may have been helpful in the COVID-19 pandemic because, by making at least some people *overly* fearful of contagion, even if irrational, it increased the efficacy and compliance of social distancing and national lockdowns.

Cultural Evolution

Up until now, I have focused on aspects of virus or human behaviour that have evolved by biological natural selection. However, humans and human societies are not bound merely by genetics. For many thousands of years, humans have also adapted to their physical and social environment through *cultural* adaptations (such as tools, clothes, rituals, social institutions, and so on). While these are 'cultural' phenomena, over time they nevertheless undergo the same dynamics of Darwinian selection as do genetic adaptations—it is just that, rather than genes being differentially selected, it is *ideas* and *innovations* that are differentially selected, with beneficial ones spreading at the expense of less effective ones (Creanza et al. 2017; Mesoudi 2011). Of course, like genes, sometimes new cultural traditions are neutral and have no positive or negative effect, and just remain in the population through drift (a lack of counter-selection). But the point is that cultural adaptations can also often exert an important positive impact on survival and reproduction (as well as feeding back to create new environments for subsequent *genetic* adaptations; Diamond 2005). The major difference between biological and cultural evolution is that cultural adaptations can arise and spread much faster, passing from mind to mind on the scale of days, compared to genetic adaptations, which have to wait many years for each new biological generation to turn over (and only then be tested by selection).

Because regional environments and local ecologies are so different around the world (from arctic tundra and desert to mountains and rainforest), different populations have evolved many cultural as well as genetic differences in their responses to the prevailing challenges of health and disease. Just as many diseases only (or mainly) occur in some parts of the world, so some *genetic* adaptations to diseases are more prevalent in some populations rather than others. There are many examples, from the distribution of malaria and sickle cell anaemia (the latter protects from the former) to lactose intolerance and the domestication of cattle (this inability to digest lactose in milk occurs in the absence of the latter tradition).[8] But many *cultural* characteristics have also arisen in response to disease, from permitted or

[8] Populations that domesticated cattle in Europe and Africa significantly changed their nutritional environment and developed genetic mutations to digest lactose, but populations that did not domesticate cattle did not. In traditional medicine, lactose intolerance was seen as an 'abnormality' or 'syndrome', but an evolutionary perspective sees it as a perfectly normal biological state in populations that did not domesticate cattle (Gluckman et al. 2011).

banned types of food and rituals of food preparation, to social norms surrounding illness and cleanliness (Murdock 1980; Murdock and Wilson 1978).

One set of recent studies suggests that cultural variation in social organization is itself an adaptation to local levels of disease pathogens, which may have relevance (at least as one interesting hypothesis to explore) to different populations' susceptibility or responses to COVID-19. Researchers have long noted systematic and consistent differences across cultures in their levels of 'collectivism' versus 'individualism'. This was a general distinction made in early comparisons of cultures by Max Weber, and collectivism–individualism was later explicitly developed as a cross-cultural variable by Geert Hofsteder and others, and has become, according to some advocates, 'a paradigmatic element of culture that, in the eyes of many social scientists, is fundamental to any understanding of cross-cultural differences' (Fincher et al. 2008: 1283; Hofstede 1980; Triandis 1995). The basic idea is that collectivist cultures (such as Brazil, India, Russia, and Japan) are more likely to emphasize the in-group over out-groups. By contrast, individualist cultures (such as the United States, the UK, France, and Germany) Of course, there is much variation within countries and among individuals, but the collectivist–individualist distinction captures a broad trend (Triandis 1995: 2).[9] (Ma-Kellams et al. 2011) still do this as well, but to a lesser extent, elevating instead individual interest and out-group opportunities.

But *why* does collectivism and individualism vary among cultures? A variety of theories have emerged, tending to focus on the sociological consequences of different regional practices, such as the legacies of farming wheat versus rice, for example. But Thornhill and colleagues suggest that it may also be a social response to disease. He and his colleagues see the collectivist–individualist dimension of

[9] Triandis defines *collectivism* as:

> a social pattern consisting of closely linked individuals who see themselves as parts of one or more collectives (family, co-workers, tribe, nation); are primarily motivated by the norms of, and duties imposed by, those collectives; are willing to give priority to the goals of these collectives over their personal goals; and emphasize their connectedness to members of these collectives.
>
> (Triandis 1995: 2)

Note that collectivism is similar but not identical to ideological conservatism, although the latter is itself is also known to be strongly associated with conformity and out-group hostility (Jost et al. 2003). By contrast, *individualism* is defined as:

> a social pattern that consists of loosely linked individuals who view themselves as independent of collectives; are primarily motivated by their own preferences, needs, rights, and the contracts they have established with others; give priority to their personal goals over the goals of others; and emphasize rational analyses of the advantages and disadvantages to associating with others.
>
> (Triandis, 1995: 2)

Scholars have challenged whether it represents a simplistic continuum or is multi-dimensional (Singelis et al. 1995; Taras et al. 2014). Nevertheless, while the terms themselves 'are used by many people in different parts of the world and are given different meanings', in cross-cultural psychology a large body of research identifies a number of factors that distinguish between these two broad types of orientation across cultures (Nisbett 2003; Oyserman et al. 2002).

behaviour as a 'species-typical, evolved sensitivity to information inputs from the immediate environment' (Fincher et al. 2008: 1284). In other words, genetic dispositions combined with developmental plasticity allow human psychological and behavioural mechanisms to tune their level of collectivism–individualism to match the demands of their environment (and in other work, they show that this seems to be associated with the types of political systems and religions that have emerged as well). However, what demands of the environment are we talking about? Thornhill has found that the strongest statistical predictor of collectivism–individualism around the world is the prevalence of disease pathogens in the local environment. In short, the stronger in-group/out-group bias of collectivist cultures may have arisen, at least in part, as a defence mechanism to avoid the transmission of diseases from other groups in areas of high pathogen prevalence.

The idea that the in-group/out-group bias serves as an adaptation to reduce the risk of disease transmission has been theorized for some time (e.g. Haselton and Nettle 2006), and Thornhill's studies have suggested some empirical support. The same adaptive logic that groupish behaviour may have evolved (or may now serve) to avoid disease transmission is well supported in biology more generally, as it occurs across many taxonomic groups. This suggests that, along with many other species, over our evolutionary history we have been subjected to significant disease risks that, when severe enough, 'select social animals, like humans, for an adaptive xenophobia' (Rozsa 2000: 396). Above, we considered the conflict between self-interest and group-interests undermining lockdown restrictions in the global pandemic, and to some extent the same tension has arisen between different groups within and between countries. In times of crises, when people are threatened, the in-group/out-group bias becomes stronger. They are more likely to look out for themselves and their families, but also for members of their in-group with which they identify and often rely on to thrive and survive, and to become especially wary of out-groups (Hewstone et al. 2002; Inglehart et al. 2006).

While somewhat speculative, it is interesting that the poor performance of the pandemic response among some notable rich, western individualist countries, such as the United States and the UK, falls into line with this thesis—it was arguably harder for them to restrict individual liberties and close down their people and trade to the outside world. Meanwhile, many collectivist countries fared better, as measured by deaths per population (WHO COVID-19 Dashboard, https://covid19.who.int, January 2021). Broader statistical analyses conducted so far also support this association (Jiang et al. 2020).

Conclusions: Using Evolution to Help Us

Without evolutionary biology, we would be seriously in the dark about important questions on COVID-19, including: how did the virus emerge, how has it spread,

how is it changing, and how might it best be countered? This is all the more important in a global pandemic, where global networks, international travel, and a vast susceptible population means rapid analyses of the virus geneology are vital to understanding how it is spreading and how it can best be stopped. But there is a dark side too. An evolutionary perspective suggests that many of our policies in the global mitigation efforts are playing into the hands of the virus. Evolution teaches us that organisms are always evolving and adapting, and we are not in a static fight but a dynamic one that we need to win quickly because it will get harder

Table 2.1 Some implications for pandemic mitigation efforts involving (a) the virus and (b) the host (and note these are considerations, not policy recommendations).

Target	Issue area	Political approach	Evolutionary approach
(a) Virus	**Restrictions**	Limit (to keep the economy going)	Maximize (not just to reduce spread but to reduce the opportunity for further mutation)
	Speed	Measured (to balance competing demands)	As fast as possible (to reduce positive feedback in spread and mutation)
	Travel	Lax (allow travel corridors and holidays where possible)	Strict (danger not just of spread, but also of seeding of new variants across borders)
(b) Host	**Assessment**	Implicit expectation of rational decision-making and behaviour (and thus that people will accept and follow information, advice, and guidelines)	No expectation of rational decision-making and behaviour (rather, a persistence of overconfidence, risk-taking, and other cognitive and behavioural biases)
	Social behaviour	People should follow social distancing if the logic is explained	People are unlikely to comply strictly with social distancing, given our intensely social nature (thus, augment other measures to compensate)
	Culture	Societal responses largely determined by wealth, resources, and regime type	Societal responses also determined by cultural characteristics (individualistic, rather than collectivist societies may need to act differently to achieve similar results)

Note: The insights above are not always unique or exclusive to an evolutionary perspective (they may arise from other disciplinary approaches as well), but the point is that an evolutionary perspective *emphasizes their importance* beyond what it already is. Moreover, in combination they suggest a new way of approaching the problem.

the longer the fight goes on and the greater the opportunity for the evolution of new virus variants—which will get stronger, not weaker. If the virus does not mutate too quickly, does not evolve into more virulent forms, and remains vulnerable to vaccines, then we will get away with a lot of political blundering. But we are not only fighting the consequences of evolution for the virus, we are also fighting the consequences of evolution for ourselves.

We have often been puzzled by our own responses. Not least, our slow and grudging recognition of the problem in the first place, our underestimation of its impact and our overestimation of our ability to avoid it or deal with it, our ill-adaptedness to lockdown restrictions, and the impediments to collective action. If we were rational actors, those would indeed be a puzzle. But the puzzle evaporates when we recognize humans not as rational actors, but instead as the products of natural selection with an evolutionary legacy of our own, which has given rise to an array of evolved dispositions that powerfully influence the way we think and behave. These manifest themselves differently in different cultural and political contexts, giving rise to a range of responses and successes and failures. But some fundamental traits appear to present some common dangers for us all. This may explain why we have been here before. Effective responses to global disasters—and pandemics in particular—require considerable preparation, monitoring, scale, and speed, well beyond the levels needed to address more quotidian challenges and threats. As Rafe Sagarin lamented long before COVID-19, 'By the time new diseases are visible to most health programs, and they marshal the resources to respond, it's too late' (Sagarin 2012: 75).

Many times during the crisis, people have pointed to critical things that need to be in place for an effective response, whether better leadership, more information, greater economic bail outs, deeper international cooperation, and so on. But to a large extent, and ever more so as the virus changes over time (we are at the beginning of a marathon, not the end of a sprint), we also need to apply an evolutionary lens to make sense of the peculiarities of both the virus and its host, and to understand its spread and the responses of human bodies and minds (Table 2.1 captures some key policy insights of this evolutionary approach). To paraphrase Dobzhansky (1973), much about the pandemic—in the behaviour of both the virus and its host—does not make sense except in the light of evolution.

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