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The Stockholm Paradigm: Lessons for the Emerging Infectious Disease Crisis

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Abstract

The emerging infectious disease (EID) crisis represents an immediate existential threat to modern humanity. Current policies aimed at coping with the EID crisis are ineffective and unsustainably expensive. They have failed because they are based on a scientific paradigm that produced the *parasite paradox*. The Stockholm paradigm (SP) resolves the paradox by integrating four elements of evolutionary biology: ecological fitting, sloppy fitness space, coevolution, and responses to environmental perturbations. It explains why and how the EID crisis occurs and is expanding and what happens after an EID emerges that sets the stage for future EIDs. The SP provides a number of critical insights for changing scientific and public policy in a manner that allows us to begin coping with the EID crisis in an effective manner. It provides hope that we can anticipate EIDs and prevent them or at least mitigate their impacts.

Keywords: Stockholm paradigm, emerging infectious disease, ecological fitting, sloppy fitness space, oscillation hypothesis, coevolution, climate change, environmental perturbation

Introduction

Across the entire biosphere, encompassing wildlands and managed landscapes and the ecological interfaces created by them, emerging infectious diseases (EIDs) caused by viruses, bacteria, fungi, protists, and metazoans that infect humans, livestock, crops, and wildlife are increasing in number and socioeconomic impact. The response by health agencies—public, agricultural, veterinary, and wildlife—has been not as effective as we might wish, but it's not because of lack of effort. The shortcomings are due in large part to the fact that health specialists are guided by three expectations: (1) EIDs will be rare; (2) because they will be rare, EIDs can be handled with traditional palliative measures (medication, vaccination), or in very rare cases with crisis response; and (3) EIDs cannot be predicted, so regardless of cost, crisis response is the best we can do. The first two expectations are contradicted by experience and the third has not been taken seriously because we have not recognized the contradiction between experience and expectation in the first two cases. And all three expectations stem from a failed paradigm of pathogen-host evolution and ecology.

The belief that EIDs ought to be rare and, in any event, are unpredictable stems from a core principle of the accepted framework for pathogen evolution—pathogens are so strongly co-adapted to particular host species that they cannot change hosts unless specific genetic mutations arise—hence the rare and unpredictable—that allow a new host to be colonized. Empirical evidence supports the notion that pathogens are highly specialized with respect to their hosts, and yet comparisons of pathogen and host phylogenies indicate that pathogens have often changed hosts in evolutionary history, consistent with contemporary experience with EIDs (Brooks and Hoberg, 2000; Hoberg and Brooks, 2008, 2015; Brooks et al., 2015, 2019). These inconsistencies in the standard paradigm produced the *parasite paradox* (Agosta et al., 2010; Brooks et al., 2019).

When preparation and palliation are not effectively coping with any kind of disease, prevention becomes necessary. EIDs have achieved that status. By many estimates, more than half the species on this planet are pathogens of some form or another. EIDs are much more than just a few viruses affecting human beings that make occasional headlines. EIDs include all pathogens that affect humans and every other species—wild and domestic—upon which humans depend for survival and well-being, and includes diseases we have never seen before but are seeing now as well as diseases that we thought we had contained or eradicated that are reemerging. EIDs are also costly; conservative estimates prior to the SARS-CoV-2 pandemic assessed the combined treatment costs and production losses due to EIDs at $1,300,000,000,000 per year (Agosta et al., 2010; Boeger et al., 2022; Brooks et al., 2022; Hoberg, Boeger, Brooks, et al., 2022; Trivellone et al., 2022). Most of those costs are hidden, rolled into the cost of doing business, leading to increased costs for health care and food. But there is now clear evidence that EIDs are unsustainably expensive; at the current pace, the costs of EIDs associated with food availability, sustainability, and safety in the US will exceed the projected US gross domestic product (GDP) within 80 years (Trivellone et al., 2022).

The Stockholm paradigm (SP) (Agosta et al., 2010; Brooks et al., 2014; Hoberg and Brooks, 2015; Brooks et al., 2019; Agosta and Brooks, 2020; Agosta, 2022) allows us to see the EID crisis as an expected outcome of climate change and anthropogenic impacts on the biosphere. The SP explains why and how the EID crisis occurs and is expanding, and what happens after an EID emerges that sets the stage for future EIDs. The SP combines the effective elements of various preexisting perspectives on evolutionary diversification into a novel and broadly integrative framework (Agosta and Brooks, 2020; Agosta, 2022). As an explanatory platform for EID, and beyond its Darwinian foundation, early insights leading to the SP can be found during the heyday of the orthogenetic movement in parasitology (e.g., Wenrich, 1935) and of the coevolutionary arms race movement of the late 20th century (e.g., Brooks, 1979; Janzen, 1985). (See Brooks et al., 2019, for a more detailed historical account.)

As a unified conceptual framework, the SP provides a number of critical insights for changing scientific and public policy in a manner that allows us to begin coping with the EID crisis in an effective manner. Above all, the SP enables us to anticipate EIDs and prevent them, or at least mitigate their impacts. Adopting this new perspective will require some rethinking on the part of those trained in the traditional paradigm.

Ecological Fitting

Ecological fitting (EF) (Janzen, 1985; Brooks and McLennan, 2002; Agosta and Brooks, 2020; Agosta, 2022) is an umbrella term for the way in which inheritance systems use built-in evolutionary capacities to cope with changing conditions. The major elements of EF are phylogenetic conservatism, co-option, and phenotypic plasticity. These are not "hidden traits" but rather well-known elements of the foundation of Darwinian evolution—the inherited capacity to cope with changing conditions by changing. As Darwin suggested, the most powerful of these is phylogenetic conservatism. Phylogenetic conservatism in resource specialization, transmission dynamics, and microhabitat

preferences allow pathogens to be highly specialized and yet flexible. Pathogens with specific host resource requirements may be capable of infecting a broad range of hosts if the specific resource is phylogenetically conservative and widespread (Janzen, 1985; Brooks and McLennan, 2002; Brooks and Boeger, 2019; Agosta and Brooks, 2020; Agosta, 2022). It is as simple and fundamental as recognizing that SARS-CoV-2 requires some level of compatibility between viral S1 and the host's ACE2 receptors for infection (Conceicao et al., 2020; Damas et al., 2020); therefore, almost all mammals must be considered at risk for infection if exposed (Boeger et al., 2022; Hoberg, Boeger, Brooks, et al., 2022).

A robust modeling platform has shown that EIDs can occur easily in this way (Araujo et al., 2015; Braga et al., 2018; Feronato et al., 2022). A second implication of EF is that pathogens exhibit pronounced specificity for certain characteristics of host species but do not exhibit specificity for particular host *species*. This specificity for characteristics has long been acknowledged by researchers working with insect-plant associations (e.g., Nylin et al., 2018), but within parasitology and disease research disciplines, the belief that pathogens are somehow specialized on particular host species persists. This belief is an archaic holdover from orthogenetic views of evolution that impedes efforts to understand how many hosts might be susceptible to a given pathogen and why (Brooks and McLennan, 1991, 1993, 2002; Brooks et al., 2019). We strongly encourage that the term *host specificity* be replaced with the term *host range* to avoid continuing confusion and misunderstanding.

Sloppy Fitness Space

The SP asserts that increasing host range is a matter of pathogens having the opportunity to be exposed to susceptible but previously unexposed hosts. For explaining EID, the SP thus places at least as much emphasis on the opportunities for pathogens to encounter susceptible but previously unexposed hosts as on the inherited capacities of the pathogen or host. In evolutionary biology, opportunity space is fitness space. *Fundamental pathogen fitness space* (FFS) encompasses all hosts all over the world in which a pathogen could survive. *Realized pathogen fitness space* (RFS) encompasses those hosts in which a pathogen is currently surviving. The greater the difference between FFS and RFS, the sloppier the fitness space is and the greater the potential for persistence during changing conditions (Agosta and Klemens, 2008; Agosta, 2016, 2022).

All pathogens are specialists with respect to the inherited elements of EF: required environmental (mostly host) resources, microhabitat preferences (called site specificity by parasitologists and tissue tropisms by disease specialists), and transmission dynamics. Equally, all pathogens are relatively generalized or specialized in proportion to how much of their FFS is RFS. A broad host range does not make a pathogen a generalist. As the number of documented hosts for SARS-CoV-2 has increased, researchers have begun to refer to the virus as an ecological generalist, despite the fact that it remains highly specialized on ACE2 and restricted to hosts having that resource. In reality, SARS-CoV-2 is an example of a pathogen that is a specialist with respect to a host resource that is phylogenetically conservative and widespread (across all mammals—an entire class of vertebrates). The pandemic is a powerful exemplar of a pathogen that has generalized in host fitness space as a result of being given many opportunities to come into contact with many susceptible but previously unexposed hosts (Boeger et al., 2022; Hoberg, Boeger, Brooks, et al., 2022). The increase in host range by SARS-CoV-2 is a classic case of ecological fitting in sloppy fitness space (SFS).

Increasing host range by EF increases the distribution of the pathogen in fitness space based on preexisting capacities. By generalizing in host fitness space, however, the chances that new variants will be able to emerge and survive increases. Although initial colonization of a new host is achieved through preexisting variation, each new host species represents a new selective regime for the colonizing pathogen population. Novel variants thus emerge *after the colonization of new hosts*, not before. The evolution of post-infection variation creates significant potential for new clades of pathogens with distinct epidemiological characteristics to emerge simply by chance. The emergence of multiple novel variants of SARS-CoV-2 following its initial emergence and subsequent geographic spread is a prime example. This aspect of the SP dynamic gives rise to the unpredictable aspects of new EIDs, including greater or lesser virulence, or even greater transmissibility, as appears to be the case for Omicron (Boeger et al., 2022; Hoberg, Boeger, Brooks, et al., 2022).

Pathogens Are Not "Enemies"

The metaphor of pathogens as enemies and agents of disease and death is powerful. But it is misleading. Pathogens are normal components of the biosphere. Their effects on humans and species that humans care about are not a personal insult to humanity; they are simply part of how pathogens make a living. It is convenient but short-sighted to try to understand pathogens by focusing on how they discomfort us and our lives. If we are to understand how to cope with the EID crisis, we must focus on the distribution of pathogen diversity and how human activities intersect with pathogens' biological "business as usual." There may

be places where particular hosts have reduced the number of pathogens compared to other places, but this does not mean there is any evolutionary process of enemy escape or enemy release, much less places that are enemy-free zones. It is simply an indication that pathogen and host fitness space are dynamic and relatively independent of each other.

Infectious Disease Is Not an Inherited Trait

Disease and death preoccupy health professionals, while survival and persistence are the focus of evolutionary biology. There is no question that diseased organisms may be less successful evolutionarily than those that are not diseased. However, there is no natural capacity called "causing disease"—"disease" is a human interpretation of a variety of natural conditions. If an infected host is diseased, it is an indication that the host represents marginal fitness space for the pathogen. The more marginal the new host, the greater the misfit, often manifested as disease. At the same time, marginal fitness space provides the strong selection for variants that are better accommodated to the new host fitness space. Such new variants may not evolve rapidly, and indeed in a Darwinian world there is no guarantee it will ever happen. Even if there is disease associated with the pathogen-host association, if both pathogen and host continue to survive—even though they do not thrive—their association will persist (Anderson and May, 1982, 1985; Araujo et al., 2015). Pathogenicity and virulence are equally not inherited traits but outcomes of living in marginal fitness space. When we speak of the evolution of pathogenicity, of virulence, or disease, we are at best speaking metaphorically; at worst, we are wasting our time.

Pathogens Do Not Magically Appear and Disappear

Pathogens are present even when they are not producing disease or headlines, a fundamental insight explored by J.R. Audy more than 60 years ago (Audy, 1958) but largely ignored today. If a host is heavily affected by disease, that host represents marginal fitness space for the pathogen, and the SP suggests that there must be at least one other host that is not diseased. Thus, when there is disease emergence we must not simply focus on the newly diseased hosts but must immediately search for other hosts—sometimes called reservoirs but more appropriately called original hosts—which were the source of the emergence in the first place (Brooks et al., 2014; Brooks et al., 2015, 2019; Hoberg, Boeger, Molnár, et al., 2022). Likewise, when the disease emergence subsides or seems to disappear from the newly infected host, we must not waste time celebrating the demise of the pathogen. We can be certain that it still exists with the potential to reemerge if we do not take steps to limit exposure between the original host and the host of interest to us. The periodic reemergence of pathogens, such as Ebola, is an example. When there are no active Ebola cases in human beings for a month, Ebola has not disappeared from the planet, only to reappear magically in the same place or elsewhere at a later time.

The Oscillation Hypothesis: Where Does Coevolution "Fit"

Exploiting environmental resources is the essence of evolutionary specialization and explains why pathogens are so specialized with respect to required host resources, microhabitat references, and transmission dynamics. Coevolutionary interactions describe the dynamics by which pathogens exploit hosts. They may be passive (*Resource Tracking*) (Jermy, 1976, 1984) or active; if active, they may be symmetrical (*Coevolutionary Arms Race*) (Mode, 1958, 1961, 1962, 1964) or asymmetrical (*Red Queen Dynamic*) (Hamilton, 1980). These different possibilities produce different selection filters that allow novel variants to emerge or rare variants to be amplified through progressive evolutionary specialization (Brooks and Boeger, 2019; Brooks et al., 2019). They do not, however, provide an explanation for how specialized pathogen-host associations can become generalized; in other words, they lead to the parasite paradox rather than resolving it (Agosta et al., 2010).

The more intense the exploitation, the more localized the specific interactions. And the more localized the association, the smaller the amount of fundamental fitness space occupied. And if the specialized traits are phylogenetically conservative and widespread, the greater the specialization through exploitation, the sloppier the fitness space becomes and the greater the potential for increases in host range if new opportunities present themselves. Coevolutionary interactions may help create the conditions under which novel variants emerge and rare variants are then amplified, but they do not restrict the pathogen's fundamental fitness space. This is how pathogens can be highly coadapted to a particular host in a particular place and still retain the capacity to expand their host range, given the opportunity.

Pathogens, like all species, are both exploiters and explorers. They constantly exploit their immediate surroundings but retain the capacity to explore new potential fitness space. Conditions favoring exploitation should be associated with restricted host ranges and emergence of diverse novel variants; conditions favoring exploration should be

associated with increasing host range and the spread of preexisting capacities within pathogen fitness space. Alternating between exploitation-biased and exploration-biased behavior produces the evolutionary patterns of increasing and decreasing host range that provided insight leading to the *oscillation hypothesis* (Janz and Nylin, 2008). Oscillations are why some pathogens can be deemed "virulent but not dangerous" to humans because humans represent marginal host fitness space to which the pathogen cannot be easily transmitted (Guth et al., 2022), while others—such as Omicron—can be the opposite (Boeger et al., 2022; Hoberg, Boeger, Brooks, et al., 2022).

Evolution does not affect what is not happening, so no matter how intense the local co-adapted responses by pathogens to given hosts may be, they will have no direct effect on susceptible but unexposed hosts in other places. Thus, when new opportunities occur and pathogens encounter novel hosts, the locally co-adapted associations cannot function as evolutionary firewalls against expanding their host range and producing EIDs.

One implication of the SP is that coextinction of pathogens and hosts is not likely to have been a major phenomenon in the history of life (Hoberg and Brooks, 2008). Altered environmental conditions that might lead to the extinction of an original host may also create new connections in pathogen fitness space, increasing the opportunity for pathogens to be exposed to susceptible but previously unexposed hosts. We need not fear that host extinction will lead to catastrophic pathogen extinction, nor can we hope that coextinction will serve as a form of evolutionary biological control.

Climate Change and EID: Creating Opportunities for Exploration

The SP highlights the fact that the presence of a pathogen in a host is a result of both capacity and opportunity. No matter how specialized an association between a specific pathogen and its localized host may be, there are other possibly susceptible hosts elsewhere that have not yet been exposed to the pathogen, perhaps simply because it is geographically too distant. The capacity for expanding into new hosts, therefore, is always present, waiting for something to create new opportunities, unleashing the exploratory capacity of pathogens to drive EF in SFS.

Oscillations in host range do not occur at random but rather in bursts correlated with alternating environmental perturbations and environmental stability, and every study we have been able to do on this shows that such bursts are always associated with episodes of regional or global climate change (Hoberg and Brooks, 2008; Hoberg et al., 2017; Brooks et al., 2019). Climate change may trigger opportunities for pathogens to expand their host range simply by allowing species to move (for a recent example, see Kafle et al., 2020); if a pathogen lives somewhere wet and it becomes dry, it will move away and vice versa. Such movements bring pathogens into contact with hosts that are susceptible and have never been exposed to them before.

The SP demonstrates a direct relationship between climate change and EID. The emerging infectious disease phenomenon is an aspect of global climate change in a simple yet fundamental way, and that is that climate change initiates movement among species. Environmental perturbations catalyze biotic expansions as species leave areas where conditions have changed to an extent that they are no longer inhabitable. In effect, they create massive numbers of invasive species. That gives opportunistic species like pathogens more opportunities to be opportunistic, increasing the odds that they will encounter susceptible hosts that have never been exposed before.

Environmental perturbations create new episodes of expansion based on preexisting capacities (*ecological fitting in sloppy fitness space*). Those episodes are manifested by biotic expansions creating geographic and ecological mosaics and increased host range (Hoberg and Brooks, 2008). Environmental stability creates new episodes of isolation and specialization in which novel variants can emerge, aided by founder effect. Biotic isolation creates geographic and ecological mosaics of exploiters and decreased host range (Hoberg and Brooks, 2008). As geographic and host range expands, the pathogen generalizes in fitness space; once established in a new host or new place under stable conditions, the pathogen specializes in fitness space and new variants will emerge.

There is no inherent directionality in host range changes; that depends on multiple factors that influence ecological opportunities for exposure between pathogens and susceptible hosts. Host range changes can be achieved directly or by a stepping-stone dynamic, for example, from wildlife to domestic animals to humans and vice versa (Araujo et al., 2015; Boeger et al., 2020; Morens and Fauci, 2020; Hoberg, Boeger, Brooks, et al., 2022) and fast-evolving pathogens, such as viruses, retain the capacity to retrocolonize a previous host species (Feronato et al., 2022). Terms such as pathogen spillover, spillback, host switches, host jumps, and host changes all refer to singular outcomes of host colonization and represent particular portions of the more inclusive SP dynamic.

Human Civilization Created New Opportunities for EID

In the past 15,000 years, humans have modified the face of the earth in ways that created isolated habitats within a matrix of wildlands. These modifications created isolated spaces—agricultural landscapes, urban landscapes, green spaces within urban landscapes—within which humans and their domestic species lived, bordered by interfaces with the wildlands. For almost all of that time, no one recognized that such anthropogenic changes were altering opportunity space for pathogens. Pathogens maintained by humans within those new spaces existed in conditions that allowed novel variants to emerge and rare variants to be amplified. Those variants could be transmitted into the wildlands across the habitat interfaces created by humans, and they could be transmitted from one part of the human landscape to another by cooperative efforts involving trade and travel as well as noncooperative efforts involving warfare and colonization (Brooks et al., 2019; Brooks et al., 2022; Trivellone et al., 2022). We have now emerged as the planet's primary ecological super-spreaders of disease (Boeger et al., 2022; Hoberg, Boeger, Brooks, et al., 2022) (Figure 1). Poultry and livestock, crops, anthropophilic rodents, and wild boar are additional examples of ecological super-spreaders. Bats, interestingly, are not in themselves super-spreaders. Their nocturnal habits and highly isolated roosting behavior keeps them isolated from other hosts. But their high diversity of pathogens makes them excellent sources of pathogen emergence via the stepping-stone dynamic.

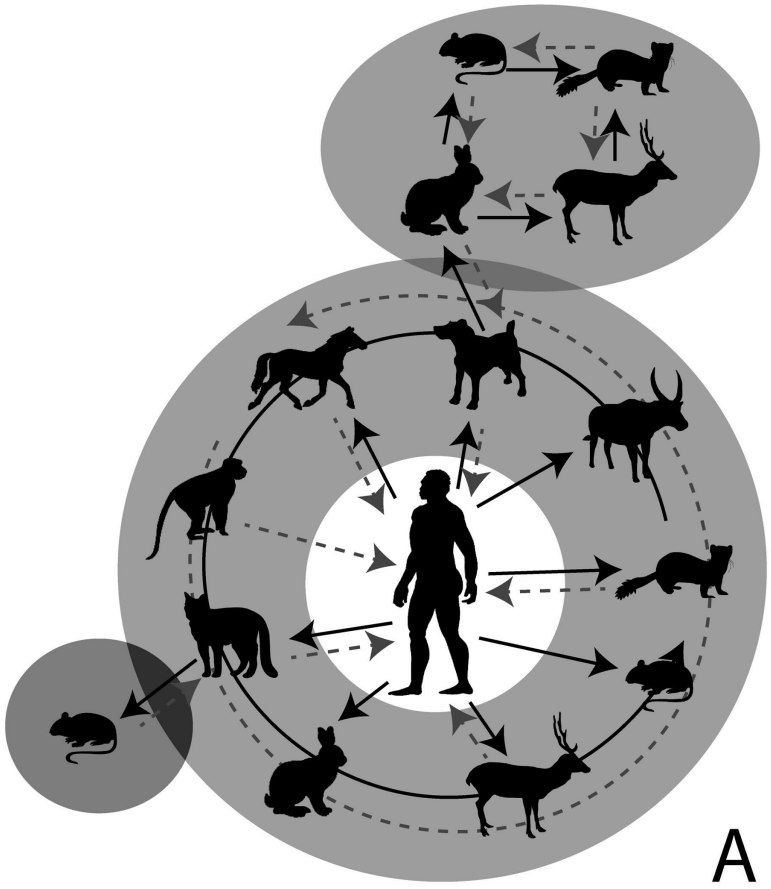
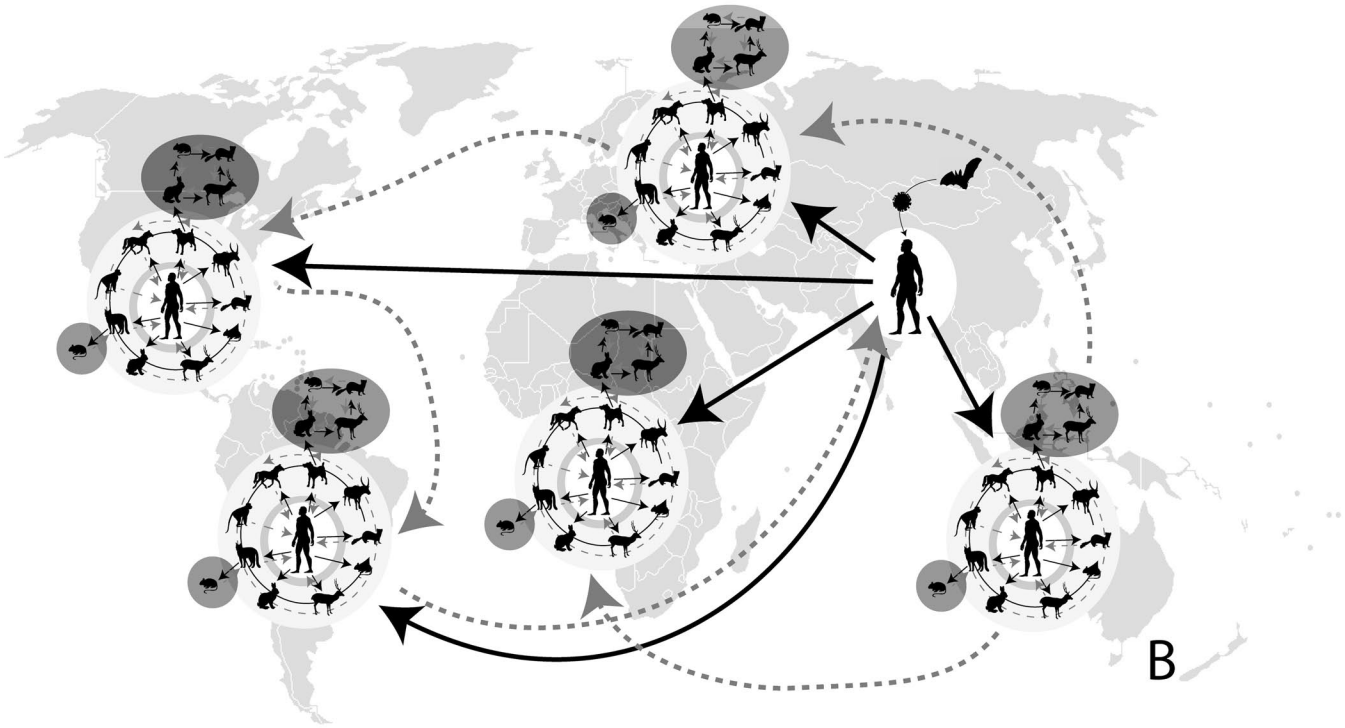


Figure 1. Dynamics of a pandemic from the perspective of the Stockholm paradigm, exemplified by COVID-19.
A. Diagrammatic representation of circulation of a pathogen with humans as ecological super-spreaders, involving transmission among realms of urban, peri-urban, and wildlife species (circles and ovate spaces). Overlaps between realms (represented by darker gray) represent interface zones, where pathogen exchanges may occur between realms—a process which may vary spatially, temporally, and at local scales because of inherent characteristics of the mammalian assemblages (e.g., diversity, behavior of peri-urban species, environmental characteristics) and humans (e.g., culture, traditions, economics).



B. SARS-CoV-2 emergence in Asia was likely associated with stepping-stone dynamics apparently involving a species of mammal (yet to be definitively identified) that bridged the ecological distance, providing the opportunity between the donor (bats) and the recipient species (humans). Initial stages of the pandemic were driven by human movements around the planet, spreading the virus across regions and continents (solid arrows). Connectivity mediated by humans disseminated or inserted SARS-CoV-2 into new systems of exploration, initially into urban and peri-urban realms and subsequently forming a complex network of transmission and emergence also involving the wildlife realm. Emergence across new realms, with distinct geographic and environmental contexts, resulted from multiple trajectories (events) of expansion and exploration over time, with subsequent potential for isolation and exploitation spatially and temporally, processes that have been demonstrated empirically. These dynamics are postulated in origins of novel variants (under different regimes of selection and isolation) of the pathogen. Given opportunity, those variants (including Delta and Omicron) became disseminated among susceptible mammals, driving secondary retrocolonization in humans. Continued expansions linked to globalized travel by humans (dashed arrows) during the course of the pandemic resulted in subsequent spread of each successive new variant and continued cycles of oscillation.

The risk space for EID is thus far larger than imagined, even recently (Carlson et al., 2022), and is continuously replenished and expanded in time by evolution (Araujo et al., 2015; Hoberg and Brooks, 2015; Braga et al., 2018; Brooks and Boeger, 2019; Brooks et al., 2019; D'Bastiani et al., 2020; Feronato et al., 2022). The biosphere is replete with a growing number of evolutionary "accidents waiting to happen" (Brooks and Ferrao, 2005), which are pathogens circulating in ecosystems and managed landscapes globally (Carlson et al., 2022). The existence of these pathogenic species that are ready and able to expand their host ranges explains why traditional approaches for coping with EID have failed. Responding only after the fact for any emergence, no matter how rapidly, is ultimately ineffective and unsustainably costly (Brooks et al., 2019; Brooks et al., 2022; Trivellone et al., 2022). Even adequately managed EID may recycle in the risk space and reemerge as distinct lineages with unique epidemiological features. Quarantine lockdowns create restricted host space, amplifying the intensity of coevolutionary interactions and allowing novel variants to emerge and rare variants to be amplified. Should the lockdowns be relaxed before transmission rates drop to below sustainable levels within the lockdown area, the effect of the relaxation will be a mini–biotic expansion event, with the rapid spread of novel variants. These events have been a recurring global theme during the SARS-CoV-2 pandemic.

Conclusions

The emerging infectious disease crisis represents an immediate existential threat to modern humanity. Current policies aimed at coping with the EID crisis are ineffective and unsustainably expensive. They have failed because they are based on a scientific paradigm that produced the parasite paradox. The Stockholm paradigm resolves the parasite paradox. Pathogens are ecologically specialized, but those specializations are phylogenetically conservative. A wide range of pathogens may have similar transmission dynamics and microhabitat preferences within hosts. There may be many susceptible but unexposed hosts needing only a change in geographic distribution or trophic structure to acquire a new pathogen (Brooks et al., 2019). Throughout evolutionary history, climate perturbations allow pathogens to oscillate between exploring fitness space that is inherently sloppy (Agosta and Klemens, 2008; Agosta, 2016; Agosta and Brooks, 2020), encountering a diverse assemblage of susceptible hosts and exploiting hosts during periods of stable environmental conditions (Araujo et al., 2015; Hoberg and Brooks, 2015; Braga et al., 2018; Brooks et al., 2019; D'Bastiani et al., 2020; Feronato et al., 2022; Trivellone et al., 2022). New pathogen-host associations resulting from changing opportunities set the stage for *subsequent* emergence of genetic innovations. Empirical evidence from deep- and shallow-time phylogenetic studies show clearly that environmental change and associated geographic expansion are correlated with host range expansion that leads to emerging diseases (Brooks and Ferrao, 2005; Hoberg and Brooks, 2008; Brooks et al., 2015; Brooks et al., 2019; Carlson et al., 2022; Guth et al., 2022).

The evidence is manifest, but failure to take notice of the long-term evolutionary dynamics of pathogens has led to short-sighted assumptions about the nature of EIDs, which, in turn, has limited our efforts to cope with them. That, in turn, has limited our ability to fully understand the emerging disease crisis as an evolutionary phenomenon and to produce effective measures for coping with it. Evolution leads to indefinite survival in an ever-changing world because it generates and stores vast amounts of *evolutionary potential* in living organisms and the ecosystems they form. They express only a fraction of that potential in any given place at any given time; the rest is what gives the biosphere a built-in capacity to persist in the face of a changing environment by changing itself (Agosta and Brooks, 2020).

The SP produces a clear message for a world experiencing accelerating global climate change: If humanity is to survive indefinitely into the future, people must use that potential to adopt public policies that better mimic the biological systems that produced both climate change and the EID crisis. The risk space is immense, and novel hosts are easily accessible when environmental perturbations—anthropogenic or not—occur. By underestimating the risk space, we have fooled ourselves into thinking that crisis response can be sustainable, even in the face of massive evidence to the contrary. But this dire depiction contains real hope. If expanding host range, the essence of EID, is based on preexisting capacities, we can use that information to anticipate and prevent, or at least mitigate the socioeconomic impacts of, EID. Reducing the cost of EID to sustainable levels is the goal of the DAMA protocol (Brooks et al., 2014; Brooks et al., 2019; Hoberg, Boeger, Molnár, et al., 2022), the public policy extension of the SP.

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