

Climate Change and Emerging Infectious Disease: An Evolutionary and Practical Perspective

*Daniel R. Brooks*¹

The emergence of infectious diseases (EID) crisis costs humanity more than a trillion dollars annually and is mostly due to high probability/low impact pathogens. The Stockholm Paradigm explains how the intersection of ecological specialisation, phylogenetic conservatism and climate change lead to massive increases in EIDs. More such pathogens will emerge in the near future, threatening to create unsustainable socio-economic stress on the existing human, crop and livestock and other animal health care systems. The DAMA (Document – Assess – Monitor – Act) Protocol is designed to buy time and lower costs through the proactive approach “finding them before they find us”. DAMA activities focus on habitat interfaces where reservoirs critical for disease outbreaks to recur interact with humans and the plants and animals upon which they rely. Further cost reductions can be achieved through enlisting citizen scientists connected by cell phone apps with cooperating networks of specialists, who would assess findings and make recommendations for additional monitoring and action activities.

The 1918 Spanish Influenza pandemic infected 25% and killed 10% of the human race. And yet, it caused no significant introspection among health specialists. For a century, they have continued to follow a triad of *medicate* (the ill)-*vaccinate* (those at risk)-*eradicate* (the biodiversity responsible for transmission and persistence). The age of antibiotics, led by the discovery of penicillin, was just a decade away, Pasteur’s dictum “une maladie, un vaccin” held sway, and the increased understanding of transmission dynamics allowed us to reduce exposure and interrupt disease spread. A century later, coping with infectious diseases of humans, livestock and crops is a trillion-dollar a year enterprise – greater than the GDPs of all but 15 countries – the costs borne disproportionately by those that can least afford them. Some claim we are winning the war against disease. Others see today’s emerging disease phenomenon as something in urgent need of novel action.

Beginning in the last decade of the 20th century, health professionals and biodiversity specialists first noticed a marked increase in disease worldwide. Known pathogens were showing up in new hosts, in new places, or returning to places where we thought they had been eradicated; and previously unknown pathogens were showing up in humans, and the plants and animals upon which we depend socio-economically. And yet, their transmission modes and microhabitat preferences seemed as specialised as ever. For more than a century,

¹ Evolutionary Systems Research Group, Centre for Ecological Research, Hungarian Academy of Sciences, 8237 Tihany, Hungary and Institute for Advanced Studies Kőszeg, Hungary. E-mail: dnlbrooks@gmail.com

evolutionary biologists followed the *coevolutionary arms race* paradigm, suggesting that pathogens pursue specialised exploitation of the hosts upon which they depend for survival and which, in turn, try to eliminate the pathogens. Despite the foreboding name, the paradigm offered comfort to those attempting to cope with disease. Pathogens could not move into new hosts without evolving new genetic capacities, which would be rare. Coevolution was evolution's firewall: there would be few new pathogens, and we could manage them with medicate-vaccinate-eradicate. This is the *Parasite Paradox*: How can pathogens be specialised enough to engage in coevolutionary arms races and yet increase their range of hosts more rapidly than undirected mutation would predict?

The solution to the parasite paradox is the *Stockholm Paradigm*. It is based on two Darwinian principles. First, all outcomes of natural selection, no matter how intensely specialised, are local. A coevolutionary arms race involving one pathogen and one host at one place may not limit opportunities involving other hosts at other places. Extreme specialisation thus need not limit evolutionary potential. All hosts in all parts of the world that *could be infected* by a pathogen form the pathogen's "fundamental fitness space" while hosts that *are infected* comprise the pathogen's "realised fitness space". The greater the misfit between fundamental and realised fitness space, the greater the potential for pathogens to colonise new hosts, given the opportunity, without having to wait for new genetic capacities to evolve. Second, evolution is conservative. Pathogens and hosts must exploit resources in their surroundings in order to survive, so all species are specialised in some way. If their specialised traits are phylogenetically conservative, even distantly related species may serve as hosts for a particular pathogen and, conversely multiple pathogens may be capable of infecting each other's hosts, given the opportunity. Ecological fitting is the process of exploring newly available fitness space based on pre-existing capacities; evolutionary conservatism is a source of immense potential. For technical discussions, see Hoberg and Brooks (2008, 2015), Agosta, Janz and Brooks (2010), Araujo et al. (2015), Hoberg et al. (2015), Nylin et al. (2018) and Brooks, Hoberg and Boeger (2019).

When pathogens have the opportunity to explore, we must assume they have the capacity to add hosts without the emergence of novel genetic information. This is a game-changer for emerging diseases. We have not only provided a direct link between climate change and emerging disease, we have shown that the EID crisis will get much worse before it gets better.

Throughout evolutionary history, pathogens experience alternating episodes of geographic expansion and isolation. Host ranges increase during biotic expansion and decrease during biotic isolation. This produces an oscillating dynamic with respect to both geographic distributions and host range. Species that survive episodes of widespread climate change are those that are capable of moving away from their areas of origin to areas in which conditions similar to their original ones still prevail. Episodes of climate change are always followed by extended periods of climate stability. Those periods are marked by the emergence of novel diversity in restricted geographic areas. There may be local coevolutionary arms race interactions, but susceptible hosts also remain to be exploited then next time opportunity knocks. The emerging disease phenomenon is thus more a matter of taking advantage of new opportunities than of evolving new capacities. What changes opportunities? All previous bursts of host range expansions have been associated with episodes of regional to global climate change. This links the EID crisis to the Climate Change Crisis in a surprisingly

simple and direct way. Climate change initiates movement, and movement brings susceptible hosts into contact with pathogens they have never seen before.

We have seen this before without understanding it. At least 100,000 years ago, the first waves of human population expansion were catalysed by dietary changes accompanying the invention of tools that allowed us to compete with top-line carnivores for animal prey. The improved diet fuelled a population boom that created pressure for geographic expansion. Both diet change and geographic expansion brought those earliest humans into contact with novel pathogens. Emerging disease may have been a factor limiting human populations for 90,000 years. Around 14,000 to 12,000 years ago, the Pleistocene breathed its last gasp amid rapid, abrupt and tumultuous cooling and warming episodes, ushering in our current era of unusually stable climate condition. Domestication and agriculture, along with sedentary lifestyles, emerged quickly. For the next 5,000–10,000 years, humans living a sedentary lifestyle in relative stability tended to cope with short-term but often intense climate fluctuations, and associated plagues, famine, floods, drought and conflict with abandoning their abodes to start again elsewhere.

In the past 5,000 years we learned to endure environmental changes without moving. Part of that learning involved recognising certain connections between urban life and disease. These included obvious things like poor sanitation, poor nutrition, inadequate water and proximity to certain habitats, like swamps full of mosquitoes. Our innovations decreased the possibility of outbreaks and increased the speed of mitigating them when they occurred. Part of the success of such measures was due to the fact that these early urban dwellers were coping with known diseases, not with new ones encountered as a result of colonising a new geographical area. When such measures were successful, humans were capable of staying longer in their cities. That marked the emergence of true urbanisation. But the technological innovations were themselves costly and their success was translated into ever-increasing population density. In 1918, 14% of humans lived in cities; today, the figure is greater than 50% and rising. Sixty years ago, Charles Elton warned that climate change and environmental disruption would produce a lot of biological, including human, migration. And he was correct. By the second half of the 20th century, however, the technological advantages of urbanisation had changed the traditional dynamic. Rather than spreading out into new territory, humans began to migrate in larger numbers to fewer locales.

Urbanisation became a *density trap* for humanity. Highly technological cities seem buffered from disease outbreaks. Alongside the various benefits of living in urban settings, however, are a number of risk factors. First, modern cities require a constant flow of energy, water and material goods in and out. That is, their footprint connects an enormous amount of fitness space containing an enormous number of actual and potential pathogens. Second, cities are places of high human population density and contact. Many pathogens are more likely to become established in rural areas than in cities but, once there is a disease outbreak in an urban setting, the higher the population density, the greater the likelihood of exposure to and spread of the disease. Remember what happened in 2014, the first time the Ebola virus entered an urban setting. Third, cities function through extreme division of labour with extreme inter-dependency. This means that disabling a relatively small proportion of the work force can wreak havoc with the regular operations of the city. Fourth, city-dwelling humans walk through landscapes that permit pathogens to persist daily. Cities that have attempted to “go green” by creating parklands provide nurturing environments for animals that carry zoonoses.

In 2016, a rabid raccoon from the wildlands of New York hopped a ride in someone's pickup truck, crossed the international border into Hamilton, Ontario, disembarked and infected local raccoons, which then posed a severe threat to all other susceptible species – including us – in the city. West Nile Virus arrived in North America when a single infected tourist hopped a ride on commercial airlines from Africa to New York, where local mosquitoes fed on him and transmitted the pathogen to birds living in Central Park. Those birds were the source of infection in humans living in the city, and in wild birds living outside the city, so the disease spread rapidly outward to encompass most of North America, where in a short time it established itself as part of the continent's pathogen pollution. Fifth, a population of underpaid, undereducated, undernourished people who are virtually invisible to public health services supports the wealthy standard of living that attracts people to large technologically advanced cities. They are the backbone of our urban centres where they handle the food, water, garbage, dirty laundry and hospital waste; they clean the toilets, handle and cook the food and change the dirty linen in restaurants and hotels.

In 1842, Edgar Allen Poe published a short story entitled *The Masque of the Red Death*. It told a tale of a town in which there was a smallpox outbreak. The privileged rich people in the town took in as many supplies as they would need to wait out the disease outbreak, then closed the gates to their castles and settled down to party until the plague was done. Needless to say, when the plague passed and the surviving poor people in the town opened the castle gates, they found that all the rich people were dead. Consider today's world with 4.5 times as many people and 4.6 times as many people living in cities as a century ago. Twenty pathogens in urban settings, each having only 5% of the impact of the 1918 influenza pandemic, would exact at least the same toll. And yet, humans continue migrating into new areas, coming into contact with novel pathogens. Urban-dwellers (and their domestic plants and animals) add the risk of amplified levels of local pathogens resulting from increased population density. Urban and rural populations interact directly with climate which determines where conditions are conducive for pathogen development (so called permissive environments), the structure of environmental interfaces, distribution of reservoirs, and eventually the opportunities and pathways for exposure and dissemination of EID.

As cities grew in size, they required ever-greater amounts of resources from external sources, increasing connectivity; this was the beginning of *globalisation*. Within the last 3,000 years or so we developed the ability to move large numbers of people and large amounts of goods widely about the planet. Consequently, human population is denser than previously thought possible, and this is at least partly due to globalisation broadening food and resource distribution. We have never before had this much technological capability or global connectivity. And yet, in some ways we are unusually fragile at this apex of our technological power. Even a small drought or an extreme precipitation event, direct manifestations of accelerating climate change, can hurt humans and agriculture. Droughts were at the core of the Syrian conflict that has displaced millions, and now ravage central Africa where famine and disease expand. In a hyper-connected world, this leads to ever more people concentrated in ever-growing cities, potentially exposed to more pathogens, especially in the high-risk age categories of infants and seniors.

Technologically advanced cities should be able to offset the risk of new pathogens being introduced as a result of trade, travel and migration by detecting incoming pathogens at their point of entry. In theory, yes. But programs of inspection and quarantines cost time,

money, and human resources and slow the flow of goods. Globalisation has produced a kind of *connectivity trap*, leading to increased risk and increased costs of risk assessment and intervention. Every major advance in human civilization has been accompanied by an increase in disease risk, and global climate change is increasing the opportunity space for an immense diversity of pathogens to emerge in novel places and hosts. How will we cope?

Some pathogens kill large numbers of humans, by infecting them or by killing their food sources. Most reduce human productivity and agricultural production, increasing costs associated with public and agricultural health. The socio-economic impact of these latter pathogens is staggering – at least a trillion dollars a year and rising. The planet is a minefield of evolutionary accidents waiting to happen – and they are happening daily. Anthropogenic changes occur faster than landscapes can cope with them, increasing with both population growth and movement and with globalisation. In the large, slow world of 1918, deaths from disease were the biggest concern. In today's small, fast world, loss of productivity due to disease may be even more significant as death in socio-economic terms.

No matter how well we come to cope with them after they happen, high probability/low impact pathogens will keep coming and we will always face the health scenario of “death by a thousand cuts”. Each new EID exacts a cost and persists as *pathogen pollution* after its initial acute outbreak, always having the potential to break out anew. A pathogen can be disseminated globally on time scales of days, if not hours. The Stockholm Paradigm predicts that EID will continue so long as climate change perturbations continue, and that will continue indefinitely. We must adopt policies that buy time and lower costs, so humanity can cope with a future characterised by a complex host and geographic disease mosaic. Humanity can and must anticipate and mitigate the effects of these EID. The Stockholm Paradigm embodies a plan to do just that.

The danger is great
The time is short
We are largely unprepared
But we can change that

The DAMA (Document–Assess–Monitor–Act) protocol (BROOKS et al. 2014) is both comprehensive and highly focused. DAMA *documentation* focuses on the intersection of *means of transmission* – primarily soil, water, food and biting arthropods – and *reservoirs* – the more virulent a pathogen is in a local situation, the more likely it is to have a reservoir that often escapes our attention because it is not being impacted to the same extent, if at all. The best place to find reservoirs is in critical habitat *interfaces* – between wildlands and agricultural lands, agricultural and urban settings, and in wildlands settings within urban areas. *Assessments* are designed to: 1. anticipate the arrival of known pathogens, given what we know of anticipated climate change, migration and trade connections; 2. identify known pathogens before they produce disease; and 3. identify close relatives of known pathogens that might cause disease. Assessments produce recommendations about *monitoring* pathogens of significance, and *actions* to mitigate their impact – making their arrivals less certain and mitigating their impacts where they occur. DAMA projects would also take advantage of self-interest and emerging cell phone apps to get local folks (citizen scientists) involved in

documenting what is going on literally in their own backyards, reporting that information to national and international initiatives in which specialists can assess what they report, and suggest additional monitoring and mitigation (action) activities.

As yet, no one has attempted to implement the DAMA protocol as we describe it above. A number of initiatives are under way and we encourage them. The EID crisis is more than a select number of viruses infecting humans; if our health services are overwhelmed by coping with ongoing unanticipated outbreaks of high probability/low impact pathogens in humans, crops and livestock, regardless of our technological advances in the past century, we will be unable to respond adequately to low probability/high impact outbreaks like the 1918 Spanish Influenza pandemic.

The Stockholm Paradigm has provided a direct link between climate change and emerging disease. This means that EID must be considered yet another threat multiplier in discussions of the anticipated impacts of climate change. Chief among these is the recognition that climate change breeds conflict, which breeds migration – of humans and non-human hosts, as well as a universe of pathogens – which often breeds more conflict.

We believe climate change poses a great danger to humanity, that there is little time to act, and we are not sufficiently prepared to cope. And in no case are we acting with a sufficient sense of urgency. The DAMA protocol does not compel anyone to act when confronted with news of an impending disease threat. But some things are clearly indicated. Let us update and paraphrase Elton (1958).

We must make no mistake, we are facing a set of circumstances

that could eliminate technological humanity from this planet.

The terrible irony is that we know what to do to stave off that possibility. This may be the last chance technological humanity has to preserve itself. If our civilisation emerged during a unique period of climate stability, we may not get a second chance to re-build. The good news is that we can do this, and it will be economically feasible, because it will reduce the need for crisis response, which is unsustainably expensive. The bad news is that coping with the EID crisis – or any other aspect of climate change – will not be cheap and life will never be the same. Everyone thinks this is someone else's responsibility and it will not happen to them. But everyone is at risk. But think of your children – 70% of humans will live in cities by 2050, and very few urbanised children can survive in a non-technological world.

We believe conflict resolution is possible, but it will require cooperation on a scale humanity has never attempted. We must recognise that reducing our risk requires that we help our neighbours – even neighbours with whom we have disagreements – reduce their risk. Individual security requires mutual security, from local villages to countries. This must be a permanent policy. We cannot defeat a common foe if we are at war with ourselves.

*There is no more neutrality in the world.
You either have to be part of the solution,
or you're going to be part of the problem – Eldridge Cleaver (1968).*

References

- AGOSTA, S. J. – JANZ, N. – BROOKS, D. R. (2010): How specialists can be generalists: resolving the “parasite paradox” and implications for emerging infectious disease. *Zoologia*, Vol. 27, No. 2. 151–162. DOI: <https://doi.org/10.1590/s1984-46702010000200001>
- ARAUJO, S. B. L. – BRAGA, M. P. – BROOKS, D. R. – AGOSTA, S. J. – HOBERG, E. P. – VON HARTENTHAL, F. W. – BOEGER, W. A. (2015): Understanding Host-Switching by Ecological Fitting. *Plos One*, Vol. 10, No. 10. e0139225. DOI: <https://doi.org/10.1371/journal.pone.0139225>
- BROOKS, D. R. – HOBERG, E. P. – BOEGER, W. A. (2019): *The Stockholm Paradigm. Climate Change and Emerging Disease*. University of Chicago Press. DOI: <https://doi.org/10.7208/chicago/9780226632582.001.0001>
- BROOKS, D. R. – HOBERG, E. P. – BOEGER, W. A. – GARDNER, S. L. – GALBREATH, K. E. – HERCZEG, D. – MEJÍA-MADRID, H. H. – RÁCZ, S. E. – TSOGTSAIKHAN, A. (2014): Finding Them Before They Find Us: Informatics, Parasites and Environments in Accelerating Climate Change. *Comparative Parasitology*, Vol. 81, No. 2. 155–164. DOI: <https://doi.org/10.1654/4724b.1>
- ELTON, Ch. S. (1958): *The Ecology of Invasions by Animals and Plants*. London, Methuen. DOI: <https://doi.org/10.1007/978-1-4899-7214-9>
- HOBERG, E. P. – AGOSTA, S. J. – BOEGER, W. A. – BROOKS, D. R. (2015): An integrated parasitology: revealing the elephant through tradition and invention. *Trends in Parasitology*, Vol. 31, No. 4. 128–133. DOI: <https://doi.org/10.1016/j.pt.2014.11.005>
- HOBERG, E. P. – BROOKS, D. R. (2008): A macroevolutionary mosaic: Episodic host-switching, geographic colonization, and diversification in complex host-parasite systems. *Journal of Biogeography*, Vol. 35, No. 9. 1533–1550. DOI: <https://doi.org/10.1111/j.1365-2699.2008.01951.x>
- HOBERG, E. P. – BROOKS, D. R. (2015): Evolution in action: climate change, biodiversity dynamics and emerging infectious disease. *Philosophical Transactions of the Royal Society B*, Vol. 370, No. 1665. DOI: <http://dx.doi.org/10.1098/rstb.2013.0553>
- NYLIN, S. – AGOSTA, S. J. – BENSCH, S. – BOEGER, W. A. – BRAGA, M. P. – BROOKS, D. R. – FORISTER, M. L. – HAMBÄCK, P. A. – HOBERG, E. P. – NYMAN, T. – SCHÄPERS, A. – STIGALL, A. L. – WHEAT, C. W. – ÖSTERLING, M. – JANZ, N. (2018): Embracing Colonizations: A New Paradigm for Species Association Dynamics. *Trends in Ecology and Evolution*, Vol. 33, No. 1. 4–14. DOI: <https://doi.org/10.1016/j.tree.2017.10.005>