GUEST ESSAY Global hosts and global pathogens: a perspective

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Abstract

Plant species are assailed by a remarkable diversity of pathogens, and these and other pests pose a serious direct risk to collections in botanic gardens as well as a potential source of pathogen escape. The high diversity of species in gardens combined with low population numbers minimises the likelihood of disease spread of specialist pathogens, but importation of novel pathogens is a constant concern. In parallel with natural systems, there is little data on pathogen loads in botanic gardens, on what accession policies minimise these and if such loads are likely to differ by country of origin or plant life form. Nevertheless, commonsense measures such as prohibiting the importation of plants in soil, shifting to seed and *in vitro* propagation, and inspection and quarantine on receiving and transferring plants should be implemented. This edition of *Sibbaldia* explores a variety of directions for improving our ability to develop strategies for dealing not just with pathogen threats, but with a more rational approach to pests and to microbial interactions that are a natural part of a plant's heritage.

Introduction

In the introductory paper to this volume on biosecurity and botanic gardens we address important general questions in relation to plant pathogens. While the focus of the volume is on plant pathogens, many of the issues that are discussed apply equally well to plant pests in general such as viruses, nematodes and insects. We begin with a consideration of the global abundance and distribution of pathogens in nature, a subject about which there is remarkably little precise data, but about which useful inferences can be made using existing databases. We then address the implications of this global abundance on the likelihood of pathogen spread in botanic gardens themselves, balancing the characteristics of these gardens that may decrease as well as increase the risk of disease spread. We focus on many of the challenges to biosecurity and emphasise that while there are straightforward and sensible approaches that can be applied to reduce disease risk, there remains a lack of knowledge about diseases in botanic gardens that urges directed research attention.

How many pathogens are there?

Every state in the US has an Extension Service that advises farmers and horticulturists, and operates 'plant clinics' where plants that show signs of disease can be sent for diagnosis, whether these signs take the form of too many aphids, too much water or spots on leaves. Fungal pathogens, because they are easily identified with an ordinary microscope,

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have been recorded extensively, and this data is now available via an online platform (Farr & Rossman, 2019). This database gives us a chance to assess, at least for fungi, the number of species of pathogens that cause disease in a particular plant species. Let us consider how many fungal pathogens attack two important plant families, the Solanaceae (tomato) and the Brassicaceae (mustard): Fig. 1 shows one of the golden rules of disease, summarised by the aphorism "diseases are like the stars: the more you look, the more you see". The major predictor of how many pathogens have been found infecting a given host species is always how well that species has been studied. This is not just true for plant fungal diseases but extends to ectoparasites on rodents (Kuris & Blaustein, 1977), helminth species in birds (Walther *et al*., 1995), infectious diseases of primates (Nunn & Altizer, 2006) and other studies of disease patterns in plants (Williams *et al*., 2011). Also evident from Fig. 1 is that crops fall on the same approximate linear trend as the wild species, showing that they are not necessarily getting more pathogens than wild species: it is simply that they have been studied more intensely. There are also curious, usually poorly understood differences in the susceptibility of different types of organisms to different pathogens, which here in Fig. 1 is seen as the susceptibility to fungal diseases of more species in the Brassicaceae relative to Solanaceae, especially when well studied. This figure also illustrates another golden rule: the number of species of pathogens that can potentially infect one host species is huge (note the log scale), and often surpasses

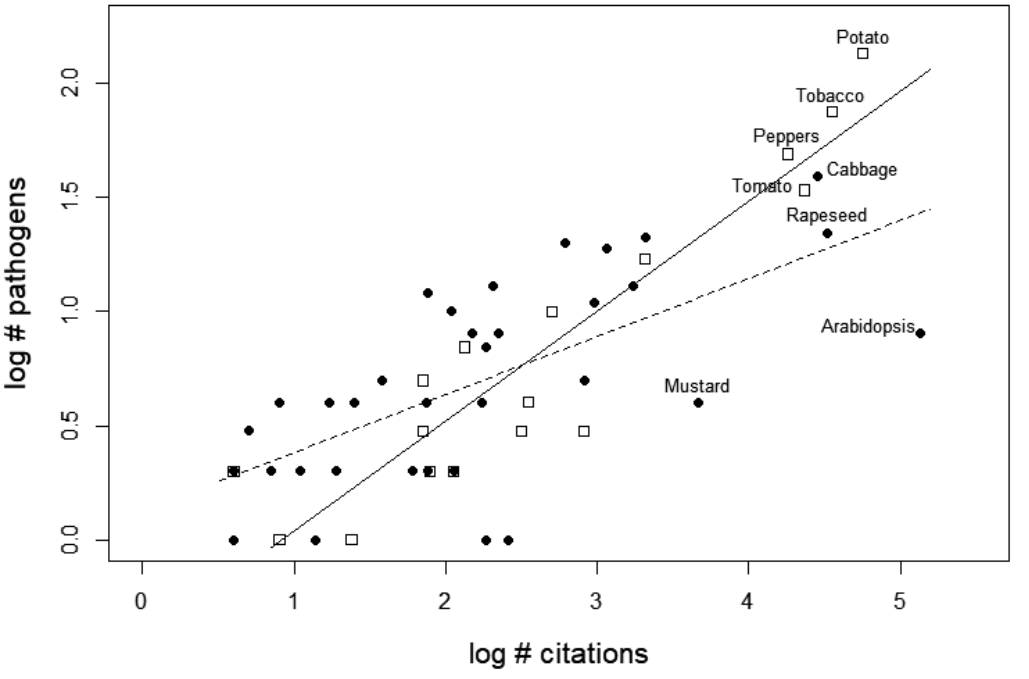

Fig. 1 Number of fungal pathogens per species plotted against the number of citations for that species in *Biological Abstracts*, both scaled to $\log_{10}(x+1)$. Squares are species in the tomato family (Solanaceae) and the filled circles are species in the mustard family (Brassicaceae). Each point represents a plant species in the USA, with the four most extreme points in each family representing the indicated major crops in these families (Farr & Rossman, 2019). The slopes of the lines are significantly different (P=0.0053).

a hundred for fungal diseases alone. In humans, the best-studied species, over 1,400 pathogens or parasites have been identified (Woolhouse & Gowtage-Sequeria, 2005) and, of these, nearly 300 appear in the Gideon database[3] which includes the diseases most frequently encountered in humans.

If we reverse the question and ask how many hosts are infected by a particular pathogen, the picture that emerges is that most pathogens and parasites tend to infect relatively few hosts. The data supporting this is not easy to obtain because generally hosts are not screened for all their pathogens, and not all pathogens are easy to identify. However, insects that are parasitic are readily identifiable to species, and an analysis of insects that are parasitic (Price, 1980) showed that 80 per cent were restricted to fewer than five host species, with the majority having only one host. Only a fraction of 1 per cent were parasitic on more than 50 species of host. Even in groups that are known to be broad generalists, specialism is relatively common. In species of the plant pathogen *Phytophthora*, a comprehensive review from 1996 (Erwin & Ribeiro, 1996) and the recent USDA fungal database (Farr & Rossman, 2019) show a bimodal distribution similar to that of insect parasites, but with many more extreme generalists (Table 1).

These sources allow more detailed analysis of the principle that the more one looks, the more one sees, as well as other interesting trends (Fig. 2). Of the 161 *Phytophthora* species recognised today (USDA APHIS, 2019), only 42 had been described by 1991. Prior to 1930, more generalists were described than specialists (defined here as infecting fewer than five host genera), while nearly all the species described between 1930 and 1990 were specialists, such that in the later review (Erwin & Ribeiro, 1996) the two groups are approximately equal (Table 1). The post-1990 boom in the number of species (Fig. 2a) is no coincidence: DNA 'barcodes' were popularised for phylogenetic analysis and identification of fungi and oomycetes in that year (White *et al.*, 1990), and this new

Host range of parasite	Number of insect parasite species with a given host range	Number of *Phytophthora* species 1996 with a given host range	Number of *Phytophthora* species 2019 with a given host range
1	494	12	39
2	154	2	24
3	89	3	13
4	71	3	8
5–10	112	5	21
11–50	46	11	21
>50	4	9	17

Table 1 The number of parasite or pathogen species that have a given host range (i.e. the number of host species in which the parasite has been found to cause disease). The left-hand column shows the host range, and the other columns show how many insect parasite species (Price, 1980) and how many *Phytophthora* pathogen species have a given range. For *Phytophthora*, the host range is as recorded in two studies: Erwin & Ribeiro (1996) and Farr & Rossman (2019).

[3] See https://gideononline.com

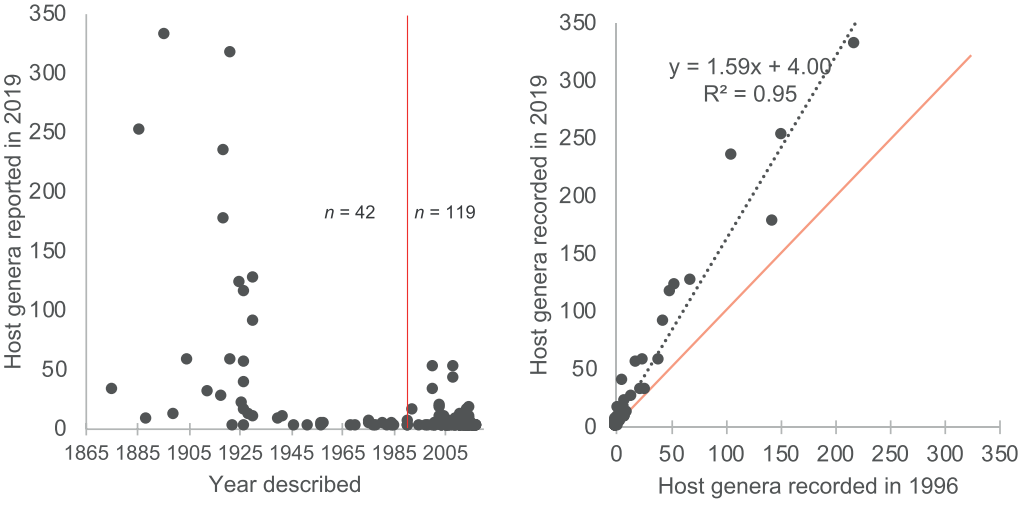

Fig. 2 Temporal aspects of host relationships for *Phytophthora* species. Fig. 2a (left): the number of hosts currently reported (Farr & Rossman, 2019) by year of first species description, with a vertical line at the year 1991. Fig. 2b (right): graph illustrating the increase in the number of hosts currently recorded from those recorded in a previous comprehensive review (Erwin & Ribeiro, 1996). The solid red line has a slope of 1 and intercept 0, which would be expected if there was no change.

way of looking has allowed the definition of many species that previously had been lumped. Over roughly the same timescale there had also been an increased interest in plant diseases in natural ecosystems (Burdon, 1987; Gilbert, 2002), which opened up new systems for study. The number of hosts per pathogen increases over this later time period (Fig. 2b), but so does the number of specialist species described (Table 1; Fig. 2a).

This analysis shows that any one host species has many parasites that are often specialised to that host, and this number appears to be increasing as systematic studies of parasites become more thorough. It is therefore likely that the number of species that cause disease (what we loosely call pathogens or parasites) greatly exceed the number that are free living. What 'greatly exceeds' means in concrete terms is not known, and may depend on the scale and thoroughness of the study (Bordes & Morand, 2009), but detailed studies on salt marsh communities have shown that there are almost as many species of visibly identifiable macro-parasites as species of free-living animal hosts, and this number would greatly increase if less easily detectable pathogens such as bacteria and viruses were included (Dobson *et al.*, 2008). Pathogens are often microscopic, and their symptoms may not be obvious (or the host may already be dead and unidentifiable!), but they are a major part of our biodiversity. Most of our information on the prevalence and diversity of pathogens comes from databases which are gathered for understanding pathogens important in medicine, agriculture and forestry. They are not an assessment of the relative abundance or impact, and we do not have perfect knowledge of the pathogens that could cause disease on every host species, the degree of damage they cause or their potential to transmit disease to other hosts. It is important to note that the above data does not imply that every individual in all populations of a

species will be diseased by all its pathogens, because all pathogens certainly do not occur in every population, nor in every individual in a population.

Another key consideration is knowing the geographic distribution of pathogens that might pose risks not just for botanic gardens but also for agriculture and forestry. Obviously, knowledge of a pathogen's presence in a given geographic region can lead to appropriate precautions in collection and importation, but unfortunately gathering such knowledge is difficult and is rarely done except as a follow-up to prevent further spread of pathogens that have already caused serious outbreaks.

While perfect knowledge of pathogen distribution and abundance is unlikely, the International Plant Sentinel Network (IPSN), as reviewed by Marfleet & Sharrock in this issue, is a framework that harnesses the unique species assemblies of botanic gardens in order to make available this sort of information at least in the community of botanic gardens. We simply do not know the number of pathogens per plant or per plant species in botanic gardens, whether this is affected by accession route or origin, and how it relates to plant life form or planting density.

Are plant pathogens good or bad for the planet?

If plants and animals are abundant and good for the planet, are pathogens also good for the planet? When at a cocktail party either of us confesses that our research is on plant disease, the first question is nearly always "How are you trying to cure it?" The presumption is that the disease should be got rid of, but if diseases are that bad, why are there so many of them around? Even Linnaeus was puzzled as to why God would have created "harmful insects", and he resolved this puzzle with the insight that they were "nature's police" (Wilcke, 1760), maintaining the balance of nature by keeping species numbers within limits. His ecological reasoning was probably sound. One of the hypotheses governing invasion biology is that the escape of invasive species from the pests and pathogens in their native habitats is a major contributor to the severity of invasions (Keane & Crawley, 2002). The growing consensus is that while pathogens have a detrimental effect on individuals (by definition), at the community level they probably increase species diversity, and in ecosystems they contribute positively to functions such as nutrient cycling and productivity (Gilbert 2002; Hudson *et al.*, 2006).

A more nuanced view of disease, beyond the simple dichotomy of good and evil, is clearly called for. A more accurate picture may be represented by Fig. 3 (purple line), where pathogens can be seen to be either highly detrimental or beneficial when moving from their impact on individuals to their effects on ecosystems. This individual-to-ecosystem continuum also parallels the continuum where there is greater immediate human concern for the human than for the natural world (Fig. 3. blue line), with perhaps agriculture and forestry taking an intermediate role representing populations (crops) or communities (managed forests).

Whether a pathogen has a detrimental effect therefore depends not only on its interactions with the host within which it is found, but also on the larger ecological context within which we view the pathogen. It may also depend on the time scale from which we view the situation. The impact of a disease epidemic on our lives, whether on our personal health or our crop yields, can

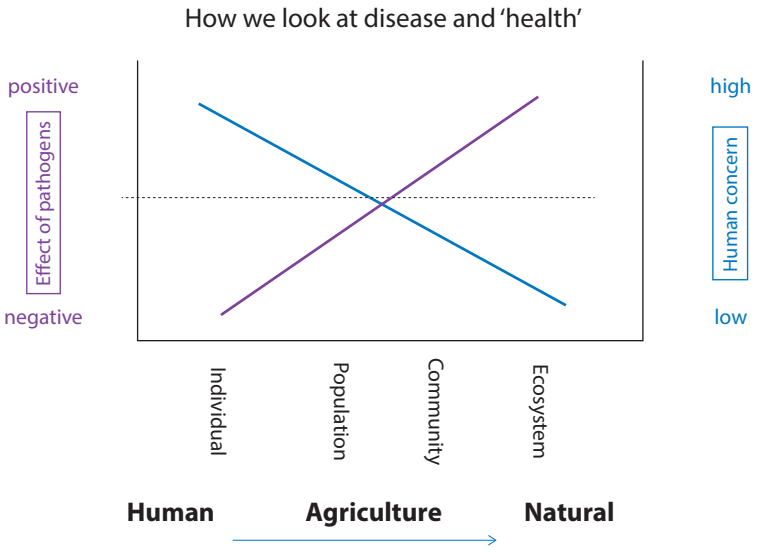

Fig. 3 The purple line illustrates that whereas pathogens may have detrimental effects at the level of the individual, they may also have beneficial effects at the level of the ecosystem in terms of promoting species and genetic diversity and contributing to ecosystem services. The blue line illustrates that human concern is greatest at the level of pathogen effects on individuals (including themselves) and least at the level of their ecosystem effects, as reflected in the resources devoted to controlling pathogens in these different contexts.

indeed be desperate and demand immediate attention, whereas in the natural world epidemics may occur, but are likely to be transient over the long term because other species may then attack the pathogen, or because there is evolution of resistance. A species severely depleted by a pathogen may be replaced by others that perform similar ecosystem functions.

In the long term, pathogens may generate and maintain species and community diversity, but this does not obviate the fact that destructive epidemics, sometimes due to human-mediated introductions, do occur and result in negative effects, at least on the scale of human lifetimes. For example, the potential loss of *Fraxinus excelsior* (ash) from British woodlands due to the combined effects of *Hymenoscyphus fraxineus* (ash dieback) which we are now seeing and *Agrilus planipennis* (emerald ash borer) which we will almost surely see within a decade or so would have a negative impact on the communities they support (Mitchell *et al.*, 2014; Valenta *et al.*, 2017). There is evidence that loss of *Notholithocarpus densiflorus* (tanoaks) from coastal mixed evergreen forest in California results in a less diverse mycorrhizal community below ground (Bergemann & Garbelotto, 2006) in addition to shifts in the above-ground plant community (Metz *et al.*, 2012). In the United States, climate and land-use driven epidemics of bark beetles have contributed to catastrophic wildfires and resulted in a huge loss of stored carbon (Ghimire *et al.*, 2015).

In the long run, the communities and ecosystems will probably recover but not within a time scale that any human now alive will see. Pathogen introductions have had negative effects on animal populations as well as on their associated communities. Pathogens have been catastrophic for

amphibian diversity, with the movement of two fungal species having contributed to extinctions and mass declines worldwide (Scheele *et al.*, 2019), while *Myotis lucifugus* (North American little brown bats) are facing population extirpations from another introduced fungal pathogen (Frick *et al.*, 2010). In the United Kingdom, the replacement of red squirrels by introduced grey squirrels, which are more destructive to trees and contribute to the decline of oaks, has been aided by squirrel pox, which grey squirrels tolerate but red squirrels do not (Chantrey *et al.*, 2014).

Even 'rare' pathogens can have a substantial impact on the abundance of their hosts. The naturally occurring fungal disease anther-smut had a strongly negative impact on the regional abundance of its host, even though only about 20 per cent of the populations were diseased, and then only 20 per cent of the individuals in these populations had the disease (Antonovics, 1999, 2004). Randomly sampling any individual to examine would have resulted in only a 4 per cent chance of finding anther-smut on it, easily leading to the erroneous conclusion that because it is rare the disease is having little impact on its host!

While we can agree on some broad generalisations about pathogens and their hosts, the danger of pathogens is ever present not only because of their huge diversity and potential for evolution, but also because human-mediated processes such as global trade and ecological disturbance expose human activities to pathogens that might otherwise remain largely confined to natural populations.

Where do botanic gardens fit in?

Among the many activities of botanic gardens, an important component is the opportunity to champion the natural biodiversity of plants to the public. This involves introducing plants from nature into the human environment. However, botanic gardens cannot easily be characterised on the continuum between the human and natural world. They are not urgent needs but long-term treasures. It is more accurate to see them as providing functions, one could say transactions, across this human-to-natural interface rather than being at one set point on the continuum between them (Fig. 4).

Two characteristics of botanic gardens set them apart from agricultural and natural systems: a high plant diversity and a substantial process of plant movement often at an international level. Because of these features, there is a temptation to panic and

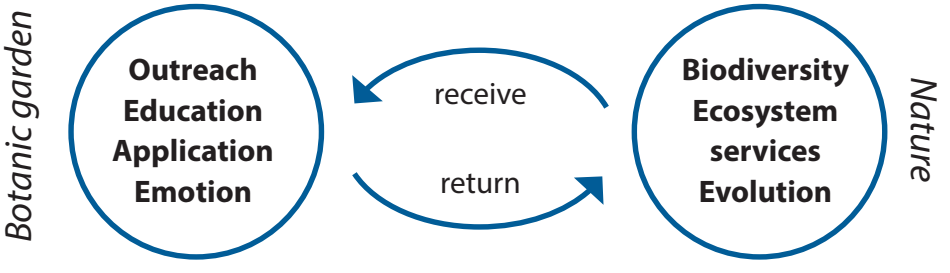

Fig. 4 Illustration of how botanic gardens act as a bridge for transactions between human populations and natural populations. The purposes of these transactions include research, education and enjoyment, as well as a better understanding of nature including its conservation.

view botanic gardens as a potential breeding ground for a soup of alien diseases. However, there are several good reasons for thinking that plant disease epidemics may be less frequent in botanic gardens than in nature, and certainly less than in agricultural and managed forest systems.

In botanic gardens, plant species diversity is very high. For example, there are currently 13,750 plant species in the living collections at the Royal Botanic Garden Edinburgh (RBGE), more than four times the number growing wild in the British Isles. However, the numbers per species are generally low, with often only one or two exemplars, and they are generally planted in relatively small patches. For most specialised pathogens, the numbers of hosts in a botanic garden would be too small to sustain a pathogen population that was highly specialised on that host species. This is in great contrast to crops, where often one species is grown at a high density over a large area, and epidemics, when they do occur, can be severe.

Nonetheless, botanic gardens and arboreta often function as nurseries, and there may be larger plantings of common ornamental species. For example, *Buxus* spp. (box) hedges are the subject of a fascinating story of a disease's decline and subsequent uptick associated with introduction of a new causal species (see Sharp *et al.*'s paper in this issue). Furthermore, links and sharing among gardens can act to increase the effective population size of species that are rare in any one location and may create transmission corridors for pathogens. In some cases, curatorial decisions can be made to decrease host density in order to control specialist pathogens to protect the collection as well as the environment. For example, the Fort Worth Botanic Garden in Texas uprooted and spread out its rose collection to suppress the transmission of rose rosette virus (McConnaughey, 2018).

The high species diversity in botanic gardens provides an advantage in the face of a specialist pathogen, but it also brings species together that would not normally interact, and there is a higher chance of cross-species transmission, particularly when closely related species are displayed together. The factors that determine whether a microbe can interact with a potential host are not necessarily fixed (Heath, 1981; Best *et al.*, 2010; Antonovics *et al.*, 2013; Bettgenhaeuser *et al.*, 2014), and host jumps are well documented over large phylogenetic distances (Raffaele *et al.*, 2010; McTaggart *et al.*, 2015) as well as over more recent timescales (Slippers *et al.*, 2005; Brasier & Webber, 2010; Gladieux *et al.*, 2015). Most microbes have rapid life histories, and evolutionary meaningful changes can occur over months or a few years (Gilbert, 2002; Gladieux *et al.*, 2015).

Just as the diversity in gardens may have a positive influence on cross-species disease transmission, international movement seems highly dangerous, and is recognised to be so in international permitting requirements and quarantine regulations. For example, European statutory measures are already in place to control *Xylella fastidiosa,* a serious bacterial pathogen of grapevines and olives, and involve destruction of host plants within 100 m of an infected plant (Commission Implementing Decision (EU) 2015/789, 2015).

However, introduction of alien species can be carried out in ways that reduce risk (see Hayden's and Summerell & Liew's papers in this issue). As pointed out above, it is highly unlikely that an individual will harbour more than a handful of all the pathogens that could affect the species, and when living plants are moved from nature into a botanic garden, it

is likely that many of their pathogens will be left behind. However, as far as we know, the thoroughness of this process has never been quantified. Additionally, most accessions in a botanic garden are started from seed. For example, of RBGE's 1,329 new accessions in 2018, around 1,040 (or over 75 per cent) were grown from seed. Most pathogens are not transmitted in seeds, but there have been important and consequential exceptions (Franić *et al.*, 2019; Liebhold *et al.*, 2012; Santini *et al.*, 2013; Matsushita & Tsuda, 2016; Skelton *et al.*, 2019) so care and quarantine are still important. Moreover, insect pests commonly attack seeds and fruits, and the latter additionally carry maternal tissue that may be infected.

One of the largest risks comes from pathogens in soil associated with the roots of transplanted or potted plants, and it is perhaps no coincidence that many of the papers in this issue are concerned with managing soil-borne transmission. This is because even a gram of soil may contain thousands of microorganisms including soil-borne pathogens, nematodes and seeds of invasive plants (McNeill *et al.*, 2011). Green *et al*., in this issue, document the high diversity of *Phytophthora* pathogens that can be found in soil in garden environments, and how historic links among gardens are reflected in those assemblages, while, also in this issue, Frankel *et al*. present a case study of transmission of these pathogens from and among conservation nurseries.

Challenges

One of the most basic challenges is simply establishing the identity and prevalence of pathogens in botanic gardens. This remains an important research goal. In a botanic garden there is also added information on the numbers and ages of the individuals involved, and they may be observed with greater care and frequency. Fungal and oomycete pathogens, especially if sporulating, are often identifiable to family or even genus by light microscopy, but this is impossible with viruses or bacteria. The use of DNA sequence data is central, but linking sequence data to pathogen identification may still not be straightforward. There is the additional problem of translating sequence data to pathogen effect and the development of "pathogenicity prediction tools" to identify which sequenced groups are likely to be harmful or not (MacDiarmid, 2013).

Unlike in many crop and horticultural organisations, the diverse accessions in botanic gardens do not come with a wealth of prior background information and knowledge that forms the basis of disease diagnosis. A plant introduced from the wild may become sickly or may develop symptoms, but it is often hard to be specific about the cause or even whether it is an organism co-introduced with that plant. Many of the projects of the IPSN (see Marfleet & Sharrock's paper in this issue) aim to facilitate identifying the causes of disease, but this paper also highlights the difficulty, time and expense that can be involved in accurate identification.

This is additionally so because plants harbour a large diversity of micro-organisms that produce no disease symptoms and may be relatively harmless or even beneficial. Distinguishing such 'endophytes' from true pathogens requires extensive experimental work based on culture and inoculation and is complicated by the fluidity with which microbes may be endophytic in one context and pathogenic in another.

An understudied factor in the context of botanic gardens is whether there are negative consequences of importing plants without their commensal partners: can

plants be healthy without their endophytic and mycorrhizal associates? Species-specific bacterial and fungal soil communities have been demonstrated to be important in the establishment of translocated *Wollemia nobilis* (Rigg *et al.*, 2017) and fungal endophytes have been shown to improve the vigour of glasshouse-grown, fungicide-treated plants of conservation concern in Hawaii (Zahn & Amend, 2017). Ultimately, a more nuanced and informed approach, rather than complete exclusion of microbial associates, may be desirable, but again this requires the kind of precise knowledge about plant microbial associations that we do not yet have.

Another challenge is preventing pathogen escape from botanic gardens themselves. The risk factors involved in disease escape from botanic gardens has rarely been assessed quantitatively, and worst-case scenarios dominate the discussions. The classic example of this is *Cryphonectria parasitica* (chestnut blight) which killed 3.6 million ha of *Castanea dentata* (American chestnut) in the space of 50 years (Anagnostakis, 1987). It was first observed at the New York Zoological Garden on American chestnuts and was hypothesised to have been imported on *Castanea mollissima* (Chinese chestnuts), perhaps as part of the collections from China on behalf of the USDA. This story is repeated in lectures and textbooks on plant disease. However, recent genetic work suggests that the *C. parasitica* genotypes that eventually established in the US came not from China but from Japan as well as from another unsampled population (Dutech *et al.*, 2012). So even in this oft-cited example of a putative escape from a botanical garden, it remains unclear exactly which imports were responsible for the epidemic. The fungus does not cause noticeable damage to the species on which it was probably imported; it was only after being transmitted to American chestnuts that it emerged as a noxious disease.

With human diseases, there is the tendency to attribute the origin of a disease to a country other than one's own. Similarly, a botanic garden could easily be blamed for any local disease outbreak. A sticky and unanswered question is the 'ethics of looking'. As shown above, we know that when we look for pathogens, we tend to find them. Looking for pathogens is undoubtedly good practice, but when we find them, are we going to be unjustifiably blamed for their occurrence? For example, the New Zealand flatworm was first recorded in Scotland in 1965 at RBGE, and while it can't be proven if this was a first occurrence or merely a first record, there is strong evidence that the flatworm's early spread in Scotland was through the horticultural trade (Boag & Yeates, 2001).

Conclusion

We have strongly argued that botanic gardens should not be viewed as rampant breeding grounds for a soup of alien diseases. However, they can still import and spread pests and pathogens, so precautions are called for. Expectations are also not evidence; nor are they precise probabilities and the reality is that we currently do not have the data to make quantitative assessments of risk that pathogens pose to botanic gardens or the degree to which botanic gardens may contribute to the dissemination of plant diseases. It could even be said that the main enemy right now is not any one pathogen, but our ignorance of the threats we are facing.

Botanic gardens receive nature, give back to nature and express nature in an accessible and aesthetically pleasing human context. How a botanic garden functions

in the context of a global threat from plant pathogens is an increasingly urgent concern, and it is important to assess the strategies that can be best adopted to meet those threats. Even in the absence of precise measures of disease risk, there are sensible measures that any garden can take to minimise risks, for example, to prohibit the importation of plants in soil (if not already prohibited by the plant health authority), to shift propagation to seed and *in vitro* methods, and through inspection and quarantine periods both when receiving and transferring plants to other sites. While not a complete elimination of transmission risk, these measures will inevitably reduce the number of pests and pathogens imported with collections. The case studies in this issue show that botanic gardens are anxious to establish evidence-based and rational procedures whereby they can reduce the opportunities for transmission of pathogens from wild-collected plants.

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