Emerging infectious diseases (EIDs) pose threats to conservation and public health. Here, we apply the definition of EIDs used in the medical and veterinary fields to botany and highlight a series of emerging plant diseases. We include EIDs of cultivated and wild plants, some of which are of significant conservation concern. The underlying cause of most plant EIDs is the anthropogenic introduction of parasites, although severe weather events are also important drivers of disease emergence. Much is known about crop plant EIDs, but there is little information about wild-plant EIDs, suggesting that their impact on conservation is underestimated. We conclude with recommendations for improving strategies for the surveillance and control of plant EIDs.

**EIDs of the four world staples**

Harlan [5] demonstrated that global food consumption predominantly comprises four staple crops: wheat, rice, maize and potato. This dependency has led to intense interest in the infectious diseases affecting these crops and the discovery of some key, globally emerging plant EIDs. Two important examples of staple crop EIDs with profound socio-economic implications are potato late blight and Karnal bunt.

Potato late blight, caused by the oomycete *Phytophthora infestans*, is the most important biotic constraint to potato production worldwide. This parasite probably coevolved with wild potato (*Solanum*) species, and initially emerged as a disease of the cultivated potato *Solanum tuberosum* when *P. infestans* was transported to Mexico from the South American Andes [6]. *Phytophthora infestans* has repeatedly emerged as an important disease of the potato as the parasite has moved into new countries and naïve host plants. It was introduced into the northeastern USA around 1840 and, subsequently, into Europe, where it decimated potato production, causing the Irish potato famine and the forced migration of five million people [7]. From Europe, blight was introduced into Asia, Africa and South America, with further introductions from Mexico into the USA and Canada during the early 1990s [8,9]. As new strains of *P. infestans* evolve, new outbreaks of the disease emerge, causing devastating epidemics globally; such as the virulent fungicide-resistant strain US-8, which emerged during 1992 [10].

Karnal bunt is a fungal disease of wheat, durum, rye and triticate (a hybrid of wheat and rye) caused by *Tilletia (Neovossia) indica*. The disease first emerged in Karnal, India during 1931 and was initially restricted to South Asia and Iraq [11]. In 1972, it became of global importance following its discovery in Mexico and, in 1996, it was

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**Plant EIDs by host type**

We organized plant EIDs into three major groups, based on their relationship to anthropogenic food production (Table 1). Many infectious diseases have emerged as threats to cultivated and wild plants. Although not exhaustive, in Table 1 we present a representative list of plant EIDs and discuss examples of some of those that exemplify the importance of EIDs as threats to agriculture or biodiversity conservation. We do not include protozoan or metazoan agents as the distinction between plant pests and pathogens for these groups is blurred.

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**Table 1.** Emerging plant EIDs and examples of those that exemplify the importance of EIDs as threats to agriculture or biodiversity conservation.  

<table>
<thead>
<tr>
<th>EID Type</th>
<th>Example</th>
<th>Impact on Agriculture/Biodiversity</th>
</tr>
</thead>
<tbody>
<tr>
<td>EIDs of the four world staples</td>
<td>Potato late blight</td>
<td>Threat to potato production</td>
</tr>
<tr>
<td></td>
<td>Karnal bunt</td>
<td>Threat to wheat, durum, rye, triticate</td>
</tr>
</tbody>
</table>

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Corresponding author: Andrew A. Cunningham (a.cunningham@ioz.ac.uk).  
Available online 14 August 2004
<table>
<thead>
<tr>
<th>Disease and class</th>
<th>Pathogen, region and host origin</th>
<th>Hosts[^a]</th>
<th>EID temporal and geographical emergence</th>
<th>Impact on human well-being, economy or biodiversity</th>
<th>Factors driving emergence</th>
<th>Refs</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>EIDs of the four world staples</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Potato late blight</td>
<td>Phytophthora infestans; Mexico; wild Solanum spp.</td>
<td>Potato Solanum tuberosum</td>
<td>19th century (Europe); early 1990s (North America)</td>
<td>National famine, political instability and economic loss</td>
<td>Repeated anthropogenic introductions into non-native regions</td>
<td>[8,9]</td>
</tr>
<tr>
<td>Rice stripe necrosis</td>
<td>Rice stripe necrosis virus (Benyvirus); Cote D’Ivoire; Oryza spp.</td>
<td>Rice Oryza sativa</td>
<td>1991 (Colombia); recently (Central America)</td>
<td>Up to 40% yield loss, persistence of vector leads to abandonment of rice fields</td>
<td>Hypothesized anthropogenic introduction with rice germplasm</td>
<td>[63]</td>
</tr>
<tr>
<td>Rice blast</td>
<td>Magnaporthe grisea; China; rice</td>
<td>Rice, barley, wheat, pearl millet and turf grasses</td>
<td>All rice-producing areas over past century; 1996 (USA)</td>
<td>Causes production losses of US$55 million y⁻¹ in South and Southeast Asia</td>
<td>Thought to have spread worldwide by the exchange of seeds by dispersal appears to be primarily by contaminated seed</td>
<td>[64]</td>
</tr>
<tr>
<td>Karnal bunt</td>
<td>Tilletia indica; India; wheat</td>
<td>Common wheat, durum wheat and triticale</td>
<td>1972 (Mexico), 1992 (USA), 2000 (South Africa)</td>
<td>India yielded total losses of ~0.5%, but up to 89% of kernels infected in certain areas, with yield losses from 20% to 40% in highly susceptible varieties</td>
<td>Evolution of new strain that broke Q124 resistance</td>
<td>[11–15]</td>
</tr>
<tr>
<td>High Plains disease</td>
<td>Maize high plains virus (HPV); central and western USA; maize or winter wheat</td>
<td>Maize, wheat, barley and several grasses</td>
<td>1995 (Chile, Brazil and Israel)</td>
<td>In Idaho, disease affected ~750 acres of maize in initial year, with yield losses exceeding 50% in several corn fields</td>
<td>Possibly has a large distribution, but only recently recognized</td>
<td>[65]</td>
</tr>
<tr>
<td><strong>EIDs of cash crops, secondary staples and non-food crops</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Severe form of cassava mosaic disease</td>
<td>EACMV-Ug 3Svr (whitefly-transmitted begomovirus); Uganda; cassava</td>
<td>Cassava Manihot esculenta</td>
<td>1988–present (Central and East Africa)</td>
<td>Regional crop failure and famine</td>
<td>Interspecific recombination of begomoviruses possibly driven by pathogen pollution</td>
<td>[16,17,52]</td>
</tr>
<tr>
<td>Moko disease</td>
<td>Ralstonia solanacearum race 2 (biovar 1); Central America; Heliconia spp.</td>
<td>Banana Musa paradisiacal and several weeds</td>
<td>Philippines</td>
<td>Disease incidence within plantations ranged from 0.62% to 63.8%</td>
<td>Anthropogenic introduction of crop plant into non-native region</td>
<td>[39]</td>
</tr>
<tr>
<td>Tomato yellow leaf curl</td>
<td>Tomato yellow leaf curl virus (whitefly-transmitted geminivirus); Israel; tomato</td>
<td>Common bean Phaseolus vulgaris and tomato Lycopersicon esculentum</td>
<td>1999 [Spain (bean)] and 1990s [global spread (tomato)]</td>
<td>Up to 80% yield loss</td>
<td>Anthropogenic introduction of propagation material</td>
<td>[46,47]</td>
</tr>
<tr>
<td>Sugar cane orange rust</td>
<td>Puccinia benghalensis; Australia; Sugarcane Saccharum officinarum</td>
<td>Sugarcane Saccharum officinarum</td>
<td>Australia</td>
<td>Australian sugar production fell by 25% and gross value of cane production dropped 20% in 2000–2001</td>
<td>Evolution of new strain that broke Q124 resistance</td>
<td>[66]</td>
</tr>
<tr>
<td>Citrus canker, Florida</td>
<td>Xanthomonas axonopodis pv. Citri; southern Florida, USA; citrus spp.</td>
<td>Citrus spp.</td>
<td>North and central Florida, USA</td>
<td>Almost 2 million trees destroyed, yielding a loss of &gt;US$200 million</td>
<td>Probably arrived with workers or equipment that had been poorly decontaminated, or via imported infected fruits</td>
<td>[18,19]</td>
</tr>
<tr>
<td><strong>EIDs of wild plants</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dutch elm disease</td>
<td>Ophiostoma spp. (vascular wilt fungus); global; elm spp. (Ulmus)</td>
<td>Ulmus spp.</td>
<td>Repeated pandemics during 20th century (North America, Europe and Southeast Asia)</td>
<td>Repeated pandemics removed most mature trees</td>
<td>Anthropogenic introduction of pathogen and vector in imported timber. Later introduction of parental pathogen enabled hybridization to produce increased virulence</td>
<td>[22]</td>
</tr>
</tbody>
</table>
Table 1 (continued)

<table>
<thead>
<tr>
<th>Disease and class</th>
<th>Pathogen, region and host origin</th>
<th>Hosts</th>
<th>EID temporal and geographical emergence</th>
<th>Impact on human well-being, economy or biodiversity</th>
<th>Factors driving emergence</th>
<th>Refs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chestnut blight</td>
<td><em>Cryphonectria parasitica</em>; (fungus); Japanese chestnut <em>Castanea crenata</em>; Eastern Asia</td>
<td>American chestnut <em>Castanea dentata</em></td>
<td>Late 19th century–present (eastern USA)</td>
<td>Regional elimination of a co-dominant species that sent ripple effects throughout ecosystem</td>
<td>Anthropogenic introduction of host and pathogen</td>
<td>[20,21]</td>
</tr>
<tr>
<td>Jarrah dieback or root rot</td>
<td><em>Phytophthora cinnamomoni</em>; (oomycete fungus); global range; isolated from &gt;900 plant species globally</td>
<td>Range of species including ones in <em>Proteaceae</em>, <em>Papilionaceae</em>, <em>Mimosaceae</em> and <em>Epacridaceae</em></td>
<td>1970s–present (Australia)</td>
<td>Threat of extinction to endemic eucalypts</td>
<td>Anthropogenic introduction and dissemination within regions</td>
<td>[67]</td>
</tr>
<tr>
<td>Florida torreya mycosis</td>
<td><em>Pestalotiopsis microspore</em>; (filamentous fungus); northern Florida, USA; Florida torreya <em>Torreya taxifolia</em></td>
<td>Florida torreya <em>Torreya taxifolia</em></td>
<td>Northern Florida, USA</td>
<td>Severe population decline resulted in host being placed on the US endangered species list and threatened with imminent extinction</td>
<td>Unknown. Hypothesized environmental factors causing normally benign fungus to increase pathogenicity. Hypothesized that fire suppression causes environment suitable for pathogenic fungus to emerge</td>
<td>[54,55]</td>
</tr>
<tr>
<td>Dogwood anthracnose</td>
<td><em>Discula destructiva</em>; (filamentous fungus); Appalachian, USA; flowering dogwood <em>Cornus florida</em></td>
<td>Flowering dogwood <em>Cornus florida</em></td>
<td>1980s present (Appalachians, USA)</td>
<td>Threatens survival of dogwood throughout its range. Hypothesized that loss of dogwood fruit production will significantly affect frugivorous wildlife</td>
<td>Anthropogenic introduction of pathogen</td>
<td>[68]</td>
</tr>
<tr>
<td>Eelgrass wasting disease</td>
<td><em>Labyrinthula zosterae</em>; (slime mould); eastern USA seaboard; eelgrass <em>Zostera marina</em> and <em>Z. caulescens</em></td>
<td>Eelgrass <em>Zostera marina</em></td>
<td>Repeated die-offs during 20th century; identified late 1980s (eastern USA seaboard)</td>
<td>Mass mortality over large areas; eelgrass able to survive only in stenohaline refugia</td>
<td>Unknown</td>
<td>[26]</td>
</tr>
<tr>
<td>Sudden oak death syndrome</td>
<td><em>Phytophthora ramorum</em>; central California coast; California bay laurel and Oregon myrtle and <em>Rhododendron</em> spp.</td>
<td>Most woody plant species in mixed evergreen and redwood forests</td>
<td>2002 (England and Poland)</td>
<td>Led to the death of tens of thousands of oak trees</td>
<td>Unknown</td>
<td>[69]</td>
</tr>
<tr>
<td>Pondberry stem dieback</td>
<td>Group of three fungal pathogens; Southeastern USA; Pondberry <em>Lindera melissifolia</em></td>
<td>Pondberry <em>Lindera melissifolia</em></td>
<td>Currently in southeastern USA</td>
<td>Might prevent endangered host from increasing its population size</td>
<td>Unknown</td>
<td>[27]</td>
</tr>
</tbody>
</table>

*EIDs are classified on the basis of their ‘emerging’ characteristics, according to Daszak et al. [2]. EIDs are those that: (i) have increased in incidence, geographical or host range; or (ii) have changed pathogenesis (e.g. hantavirus pulmonary syndrome); or (iii) are caused by pathogens that have newly evolved (e.g. canine parvovirus); or (iv) have been discovered or newly recognized [1,2].

b Not all hosts are listed.
reported for the first time in the USA [12]. Karnal bunt is a soil-, seed- (principal source of spread) and air-borne disease and it is thought that disease spread is predominantly via importation (introduction) of the pathogen. The disease decreases seed viability and flour quality and wheat consisting of 3% or greater bunted kernels is considered unfit for human consumption [13,14]. Recent interest in the disease has surrounded the potential for its use as a biological weapon and exports of wheat from many regions with Karnal bunt have been banned, leading to severe economic loss for affected countries [15].

**EIDs of cash crops**
Cash crops include secondary staple food crops (e.g. citrus fruit, bananas, coffee and cacao) and non-food crops (e.g. forestry, fodder and tobacco). These are particularly important to developing countries because they generate income, employment and foreign exchange. In many areas of the developing world, regionally important staple crops are becoming significant cash crop options, as is the case for cassava *Manihot esculenta* in sub-Saharan Africa. Cassava mosaic disease (CMD) is the most important biotic constraint of cassava production. This disease spread from its source in Tanzania during 1894, occurring Africa-wide by 1987. The disease is caused by whitefly-transmitted begomoviruses, including *African cassava mosaic virus* (ACMV) west of the Rift Valley, and *East African cassava mosaic virus* (EACMV) east of the Rift Valley [16]. In 1988, an epidemic of a highly virulent variant of EACMV (EACMV-Ug; arising from the inter-specific recombination of EACMV and ACMV [17]) began in Uganda and spread throughout East and Central Africa, causing crop losses on a scale that required international intervention to prevent widespread famine.

Citrus canker has also caused significant economic losses worldwide. This bacterial disease, which is caused by *Xanthomonas axonopodis* pathovar. *citri*, leads to canker lesions and the shedding of fruit and leaves in citrus species, such as orange and grapefruit. The disease is thought to have emerged from Southeast Asia or India [18] and now occurs in >30 countries, including the USA, where it emerged in Florida during 1995. In the largest individual program to eradicate a plant disease, the US Government conducted a US$200 million canker eradication program during the mid-1990s, consisting of clear cutting >1.8 million infected trees or ones within a 32-m radius of an infected tree [19]. However, the disease re-emerged and it is now known that the bacterium can spread >580 m during storms.

**EIDs of wild plants**
EIDs of plants that are not cultivated have long been recognized as being significant to the ecology of economically important species. The causative agent of chestnut blight, the fungus *Cryphonectria parasitica*, is native to east Asia [20] and was introduced from Japan into North America during the late 19th century with imported Japanese chestnut *Castanea crenata*, which is resistant to this disease [20]. Within 50 years, chestnut blight had killed most mature American chestnut *C. dentata* trees within their natural range [21]. Although this bark disease does not infect the roots and epicormial regrowth still occurs, new sprouts die of blight before reaching sexual maturity, rendering this species effectively extinct [21].

Twice during the 20th century, pandemics of Dutch elm disease spread throughout North America, Europe and southwest Asia, killing most elm trees *Ulmus* spp. in these regions [22]. Both pandemics were driven by the international transportation of infected timber [22–24]. The first pandemic, which began in Europe c. 1910, was caused by *Ophiostoma ulmi*, a weakly pathogenic vascular wilt fungus. The second pandemic, which began during the 1940s, was caused by concurrent outbreaks of two novel, geographically distinct and highly virulent pathogens: *O. novo-ulmi* in Europe and Asia, and *O. americana* in North America and Britain. The origin of *O. ulmi* is believed to be Asia [25], whereas the causative agents of the second pandemic are thought to have rapidly evolved via interspecific hybridization following the global introduction of the parental pathogen [22].

An increasing number of reports describing EIDs affecting less studied species has recently been published: for example, a slime mould causing wasting disease and catastrophic loss of eelgrass *Zostera marina* [26]; a series of fungal pathogens associated with stem dieback in the endangered pondberry *Lindera melissifolia* [27]; and white crucifer rust *Albugo candida* causing mortality in remnant populations of the Humboldt Bay wallflower *Erysimum menziesii* ssp. *eurekense* [28]. Although EIDs are probably not the primary cause of decline in most cases, stochastic events, such as disease emergence with or without other environmental pressures, might lead to host extinction. For example, populations of an endangered South African shrub *Euphorbia barnardii* are under pressure from human population encroachment. Damage by domestic animals can also enable invasion of bacterial wilt pathogens, resulting in disease-induced population declines [29].

**Factors driving the emergence of plant EIDs**
Understanding emergence requires knowledge of host–parasite biology, which, for understanding many plant EIDs, will involve the use of multiple-host system models parameterized for a large number of environmental, ecological and biological factors (e.g. [30]). However, it is possible to identify broad trends in plant EID emergence by analyzing hypothesized environmental drivers. Using the methods of Dobson and Foufopoulos [30], we analyzed seven years (1 January 1996–31 December 2002) of ProMED data on plant EIDs to identify pathogen taxonomy and the most significant drivers of emergence (Box 1).

**Pathogen pollution**
The emergence of plant EIDs, similar to those of humans [1], wildlife [2] and domestic animals [4], is driven mainly by anthropogenic environmental change (such as introductions, farming techniques and habitat disturbance). For plant EIDs, these changes are those largely related to trade, land use and severe weather events (predicted to increase in frequency and severity owing to anthropogenic
Box 1. Characteristics of the pathogens and drivers of emerging infectious diseases of plants

We analyzed the major taxonomic groups of pathogens that cause emerging infectious diseases (EIDs) of plants and the factors that drive their emergence using data from ProMED reports of plant EIDs (http://www.promed-mail.org). ProMED is a global electronic reporting system for outbreaks of emerging infectious diseases and toxins, which is open to all sources, and is a program of the International Society for Infectious Diseases. Outbreak reports are submitted to a moderator who reviews the data for accuracy and posts information that is considered reliable. Corrections to inaccuracies are also posted and openly communicated. These are the most comprehensive global plant EID data currently available and ProMED has been used previously to analyze trends in EIDs of wildlife [30].

For our analyses, we reviewed data published in ProMED between 1 January 1996 and 31 December 2002. For all entries, information about pathogen, host, location, date and known or hypothesized underlying drivers of emergence was collated. Citations listed within the ProMED database were also reviewed. Duplicate entries were removed, as were diseases that did not fulfill our EID definition (see main text) or for which no driver was listed.

Major taxonomic groups of pathogens causing plant EIDs

Figure Ia shows that viruses, fungi and bacteria are the major pathogens causing plant EIDs. Viruses cause just under half (47%) of the reported plant EIDs that we reviewed, which is a similar percentage to that for human (44% [70]) and wildlife (~43% [30]) EIDs. However, bacteria cause a lower proportion (16%) of plant EIDs compared with human (30% [70]) or wildlife (~30% [30]) EIDs and fungi represent a higher proportion (30%) of plant EID pathogens when compared with those of humans (9% [70]) or wildlife (<10% [30]).

Factors cited as the drivers of emergence of plant EIDs

As with wildlife EIDs [30,34], pathogen introduction is the most important driver of plant EIDs (Figure Ib) (Box 2). Weather conditions are also important, which might be related to the sensitivity of plants to humidity and moisture levels and the responses of plant pathogens to weather events (Box 3). Although we are not aware of recombination being cited as a cause of disease emergence in humans or wildlife (with the notable exception of influenza viruses and HIV-1 [71]), this process was identified as the cause of emergence for 2% of the plant diseases that we reviewed.

Factors cited as the cause of disease emergence by pathogen group

Analysis of the factors cited as the cause of disease emergence for the three most significant taxonomic groups of pathogens (bacteria (Figure Ic), fungi (Figure Id) and viruses (Figure Ie)) shows that, although introduction is the most, or second most, important driver for each pathogen group, the percentage of EIDs driven by introduction declines proportionately with size of pathogen, being lowest for fungi and highest for viruses. Weather conditions are major drivers of bacterial and fungal plant EIDs, but are relatively unimportant for plant EIDs that are caused by viruses, where changes in vector populations are the most important driver after pathogen introduction. Interestingly, although agricultural changes (Box 4) were identified as important drivers of plant EIDs caused by fungi and viruses, they were not mentioned as drivers of bacterial diseases.
Box 2. Pathogen pollution: the major driver of emerging infectious diseases of plants

The involvement of pathogens in the human-mediated loss or modification of biodiversity is being increasingly recognized and has been termed ‘pathogen pollution’ [2,34]. It is defined as the anthropogenic movement of pathogens (parasites) outside their natural geographical or host-species range. There are several ways that pathogen pollution can occur, but, in each case, anthropogenic change results in a parasite crossing an evolutionary boundary, such as geographical or ecological separation [34]. The introduction of alien pathogens or hosts that leads to disease emergence is the most commonly cited driver of emerging infectious diseases (EIDs) of wildlife, and a previously underestimated form of anthropogenic environmental change [2,30,34]. However, recent analyses provide counterintuitive data for the role of invasive species in the spread of alien pathogens. Torchin et al. [72] and Mitchell and Power [73] compared the assemblage of parasites infecting animals and plants (respectively) in their native European range to the assemblages present in the same species in their naturalized range in North America. Both host groups showed markedly lower pathogen diversity in their naturalized range, supporting the ‘enemy release hypothesis’ for invasive species (i.e. that ‘escape’ from the effects of natural ‘enemies’ is an explanation for the success of the introduced species). The usually small number of individuals introduced during each invasive event results in a lower probability of either co-introduction of any given parasite, or of that parasite becoming established. Given these data, why is disease introduction the most significant driver of emerging diseases in plants and animals? We propose that the increasing volume of globalized trade has driven increased frequency of introduction events. This is combined with a heightened impact that introduced pathogens often have on naïve susceptible host populations [34]. The result is a series of high-profile introduced emerging diseases. Given the predicted continued rise of global air travel and trade volume, the number of introduced emerging diseases is likely to also increase [74].

Introduction of alien plant pathogens occurs through trade in plant products, germplasm, grafts and live plants. The international trade in seeds, worth an estimated US$40 billion–80 billion y\(^{-1}\) [35], is an efficient means of pathogen pollution. Over 2400 microorganisms occur in seeds of 383 genera of plants [36] and it is estimated that up to one-third of plant viruses might be seed-borne in at least one of their hosts [37].

A pathogen can be introduced without disease emergence (defined as an increase in disease incidence, geographical range or pathogenicity) occurring until a second factor is introduced. For example, in the absence of factors that facilitate pathogen invasion, disease occurrence and disease spread, such as vectors, some introduced pathogens might remain localized with limited, or no, disease impacts on the host plant species. The introduction of vectors, such as arthropods, can lead to the emergence of plant diseases both directly and indirectly. For example, Citrus tristeza virus (CTV) was probably introduced into South America between 1927 and 1930, but it was the subsequent introduction of an efficient aphid vector, Toxoptera citricidus, from Asia, which led to the emergence of CTV as an economically important disease in the New World. By 1950, over six million citrus trees had been destroyed in a single state of Brazil (São Paulo) [38]. Pierce’s Disease (PD), caused by the bacterium Xylella fastidiosa, has been present in California, USA, for over a century. In 1997, a new vector, the blue-green sharpshooter Graphocephala atropunctata, was introduced into California, leading to the rapid spread of PD and its emergence as a commercially important disease with the loss of >US$6 million in South California alone during 1999.

In many cases, plants introduced to new regions for agriculture are infected by endemic pathogens of other species. Moko disease of banana is caused by the bacterium Ralstonia (Pseudomonas) solanacearum, which evolved in Heliconia species in Central America.

Box 3. Climate change as a driver of emerging infectious diseases of plants

The complexities of climate change, and the biotic responses to this, makes prediction of the future impact of climate change on emerging infectious diseases (EIDs) of plants difficult, but broad trends can be surmized. Global circulation models predict that high latitudes and elevations will warm to a greater degree than the global mean warming, and that winter and nocturnal minimum temperatures will continue to increase [42]. A changing climate is likely to bring changing patterns of climate variability, including extreme meteorological events, such as precipitation anomalies and greater temperature variations [31,42]. Climate change can lead to disease emergence through gradual changes in climate (e.g. through altering the distribution of invertebrate vectors or increasing water or temperature stresses on plants) and a greater frequency of unusual weather events (e.g. dry weather tends to favour insect vectors and viruses, whereas wet weather favours fungal and bacterial pathogens) [75]. Thus, climate change can lead to the emergence of pre-existing pathogens as major disease agents or can provide the climatic conditions required for introduced pathogens to emerge. Matter et al. [41] suggested that milder winters, higher nocturnal temperatures and higher overall temperatures will enable increased winter survival of plant pathogens, accelerated vector and pathogen life cycles, and increased sporulation and infectiousness of foliar fungi. Because climate change will enable plants and pathogens to survive outside their historic ranges, Harvell et al. [41] predicted an increase in the number of invasive pathogens. The ranges of several important crop insects, weeds and plant diseases have already expanded northward [76]. Range expansion of the grey leaf blight of corn, caused by the fungus Cercospora zeae-maydis, was first noticed during the 1970s and, in the past two decades, has become the major cause of corn yield loss in the USA [76]. Extreme weather events include spells of unusually high temperature, high rainfall and long periods of drought. Increased drought might result in loss of corn yield. Aflatoxin, a compound that lowers corn quality and which is a health risk to humans, is related to drought conditions and its concentration is raised during crop-water deficits, which favour the growth of the fungus Aspergillus flavus (the producer of aflatoxin) in the weakened crop [76].
Climate change
Climate change has been linked to the emergence of human [40] and wildlife [41] EIDs, but little is known of its impact on plant EIDs. Global climate change is predicted to change the distribution and abundance of arthropod vectors and to increase the frequency of unusual weather events, one of the major drivers of plant EIDs (Box 1). Although examples of climate change driving the emergence of plant EIDs have not yet been clearly identified [41], projected levels of climate change [42] are likely to lead to disease emergence in plants (Box 3).

Agricultural change: intensification, diversification and globalization
Changes in agriculture have resulted in a series of plant EIDs affecting cultivated and wild plants. Similar to the agricultural changes that drive human [1] and wildlife [2] EIDs, the EIDs of cultivated plants are principally driven by increased globalization, socioeconomic development and technological advances (Box 4). In many developing countries, declining market prices for staple crops and the availability of year-round irrigation has promoted increased intensity and acreage of nontraditional crop plants. Export crops have diversified to include fruits, flowers and vegetables, resulting in an increasingly complex spatiotemporal mosaic of nontraditional crops and conditions that promote pathogen pollution.

Harlan [5] stated that global crop production for human consumption principally comprises only four staple foods: wheat, rice, maize and potato. We revisited Harlan’s analysis, using data from the United Nations Food and Agriculture Organisation (http://www.fao.org) and found that, for each five-year period over the past 30 years, >40% of global food crop production has consisted of these staples. In some areas, this has led to a loss of genetic diversity in crop production and to an hypothesized increase in disease susceptibility [10]. For example, in 1969, 85% of US maize produced was of the same variety, one that was susceptible to Southern corn leaf blight and yellow corn leaf blight. In 1970, outbreaks of these diseases destroyed 17% of all US maize crops [43].

Host-pathogen evolution
Gene-for-gene coevolution of host and pathogen systems can lead to disease emergence. The gene-for-gene hypothesis [44] states that, for every gene for virulence in the pathogen, there is a corresponding gene for susceptibility in the susceptible plant species. Bean common mosaic virus (BCMV) causes the most widely distributed disease of the common bean Phaseolus vulgaris. Over seven BCMV strains exist and interact with specific recessive genes in some bean genotypes in a gene-for-gene fashion. Disease emergence occurs when the virus strain has matching pathogenicity genes or when bean genotypes lack resistance to the virus. However, a single nonspecific dominant gene effectively prevents chronic systemic infection of all bean genotypes by known BCMV strains. BCMV cannot become seed borne in bean genotypes with the dominant gene. BCMV has a restricted host range outside P. vulgaris and the monoculture of bean cultivars having monogenic dominant resistance should result in the eradication of the disease.

The introduction of an avirulent mutant (strain 1) of Tomato mosaic virus in England to protect tomato seedlings from more-virulent strains (cross-protection) resulted in the emergence of virulent forms of strain 1 in all commercial plantings where the mutant had been released [45]. The Sardinia species of Tomato yellow leaf curl virus (TYLCV) complex was first reported infecting tomatoes in Spain during 1992 [46]. In 1997, the Israel (type) species of TYLCV had emerged in tomatoes in southern Spain, where it jumped host to the common bean, resulting in a disease incidence as high as 80% in some bean fields [47].

Interspecific hybridization of pathogens brought together by human activity has resulted in the emergence of several virulent EIDs of plants, including O. novo-ulmi and O. americana (the causative agents of virulent Dutch elm disease) and EACMV-Ug (the causative agent of highly virulent CMD). A similar occurrence of hybridization between pathogens brought together by human activity has led to the emergence of a new Phytophthora pathogen of alder (Alnus spp.) in Europe [48]. The emergence of new pathogen strains owing to hybridization between agents that are not naturally sympatric is a phenomenon repeatedly reported for plant EIDs, and is a notable difference compared with human and animal EIDs. This might reflect an insufficient understanding of
EIDs of humans and animals, or a truly unique feature of plant EIDs.

**Impact of Plant EIDs**

**Human health and welfare**

The latest available data [49,50] suggest that pre-harvest pest and disease damage in the eight most important food and cash crops accounts for ~42% of the attainable crop production, or a production value of > US$300 billion. Although crop production is affected by established as well as emerging diseases, epidemics caused by plant EIDs infecting new hosts with little resistance or tolerance to the emerging pathogen can be particularly devastating. Across Africa, >200 million people depend on cassava as their staple food (http://www.fao.org). In Uganda, cassava production plummeted from a high of 3.5 million tons during 1989 to 2.25 million tons during 1996 as a result of the emergence of EACMV-Ug, with losses estimated at US$60 million y^{-1} between 1992 and 1997 [51]. In the north and east of Uganda, the epidemic led to famine. Food insecurity resulting from this EID still threatens regions of East, Central and West Africa [52].

In addition to threatening food security directly, plant EIDs have the potential to negatively impact human health and wellbeing via other mechanisms. During the 1990s, epidemics of watermelon leaf curl disease in Sudan drove farmers into urban areas. Failure to maintain economically viable and sustainable agricultural systems displaces poor rural families into marginal environments (e.g. forests or urban slums) where they encounter new diseases (e.g. Venezuelan hemorrhagic fever, cholera and dengue fever), and become foci for the spread of new human EIDs. The negative impact of plant EIDs on biodiversity might reduce the potential for the discovery of new pharmaceuticals, new crops or disease-resistant strains of existing crop species as rare strains of crop plant or wild plants are lost. The increased use of pesticides to combat EIDs can have toxic effects on humans and ecosystems. Before the 1990s, metalaxyl was used as a therapeutic fungicide to manage potato late blight. Following metalaxyl-resistant potato blight emergence in the USA and Europe, fungicides are now used as a prophylactic and primary management tool, with a predicted 15–25% increase in fungicide use [53].

**Biodiversity**

EIDs have not coevolved with the host or ecosystem in which they emerge and, as such, are more likely than are endemic diseases to pose a threat to biodiversity through biomass loss, changes in host species complements and via the extinction of host species. As with domestic animal diseases, such as brucellosis and rabies, which can threaten wildlife populations [2], diseases of cultivated plants are likely to threaten wild plants, but this is a poorly studied area of research. During the 1950s, the Florida torreya Torreya taxifolia population, endemic to northern Florida, crashed, apparently as a result of infectious disease introduced with plantation conifers [54]. By the 1990s, a lack of seed production and the possible persistence of the causative pathogen resulted in a >99% decline in the population [55]. The continued survival of this species is reliant on ex situ propagation.

The consequences of the Dutch elm disease pandemics are profound, with the second pandemic (which began

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**Box 5. Surveillance for emerging infectious diseases of plants**

Global surveillance and reporting of plant emerging infectious diseases (EIDs) requires an international coalition that does not yet exist. Currently, the United Nations Food and Agricultural Organization (FAO) mandates countries to report plant pests and diseases to the International Plant Protection Convention [79], but economic consequences often hinder accurate reporting. There is a lack of standardization in testing protocols for seed-borne diseases and significant variation in seed protection treatments among countries. Strengthening the global phytosanitary system (i.e. international agreements on the inspection and quarantining of plants to prevent the spread of pathogens and disease) requires improved information, standardized seed testing, improved access to seed treatment protocols and revised pathology research priorities [38], but funding for adequate diagnostic facilities and programs is often lacking. For example, a disease of rice in Colombia was sequentially attributed to soil compaction, aphid damage and nematode infestation before the true cause of the disease was determined; the emergence of *Rice stripe necrosis virus* [63]. The economic, environmental and social costs of incorrect diagnoses were high. Farmers doubled tillage operations in response to the diagnosis of soil compaction and applied >1000 tons of pesticides at concentrations up to six times higher than recommended in response to the diagnoses of aphids and nematodes. Crop losses were estimated at US$10 million–15 million y^{-1} for the Eastern Plains region of Colombia, with US$4 million y^{-1} in pesticide costs in addition to the environmental impact of the pesticide use. By contrast, the total cost of equipment, materials, personnel and laboratory space used in the investigation to determine the true cause of the newly emerged disease, was estimated at US$40,000 [63].

To overcome current obstacles, we suggest a three-tiered reporting system that coordinates existing reporting networks (Table I): (i) reports from official, FAO-recognized country coordinators; (ii) substantiated reports from recognized authorities (e.g. regional plant protection organizations and professional societies, Table I); and (iii) unsubstantiated reports that can then be investigated by the official, FAO-recognized country coordinator. Importantly, surveillance and reporting should be expanded to include EIDs of wild plants as well as cultivated plants.

**Table I. Reporting services relevant to plant EIDs**

<table>
<thead>
<tr>
<th>Reporting service</th>
<th>Publication and/or web site</th>
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<tr>
<td>European Plant Protection Organization (EPPO)</td>
<td>Free on-line reporting service, EPPO Reporting Service (<a href="http://www.eppo.org">http://www.eppo.org</a>)</td>
</tr>
<tr>
<td>North American Plant Protection Organization (NAPPO)</td>
<td>NAPPO Newsletter (<a href="http://www.nappo.org/Newsletter/letters_e.htm">http://www.nappo.org/Newsletter/letters_e.htm</a>)</td>
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<tr>
<td>Plant Health Early Warning System (PHEWS)</td>
<td>(<a href="http://www.1000papers.com/Agriculture/agrinet/phews.html">http://www.1000papers.com/Agriculture/agrinet/phews.html</a>)</td>
</tr>
<tr>
<td>British Society for Plant Pathology (BSSP)</td>
<td>Free on-line service, New Disease Reports (<a href="http://www.bssp.org.uk/ndr">http://www.bssp.org.uk/ndr</a>)</td>
</tr>
<tr>
<td>American Phytopathology Society (APS)</td>
<td>Plant Disease – Disease Notes (paper and on-line services, by subscription only)</td>
</tr>
<tr>
<td>University of California system</td>
<td>Journal, <em>California Agriculture</em>, New Pests and Plant Disease articles (<a href="http://californiaagriculture.ucop.edu">http://californiaagriculture.ucop.edu</a>)</td>
</tr>
<tr>
<td>ProMED-plant</td>
<td>Free on-line reporting service (<a href="http://www.fas.org/promed">http://www.fas.org/promed</a>)</td>
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during the 1940s) resulting in the loss of hundreds of millions of elms and causing closed canopy forests to become open habitat [22], leading to changes in bird community composition [56]. The deaths of mature elms resulted in increased mortality rates of sympatric trees, such as paper birch Betula papyrifera, as, for example, they became more exposed to storm damage. The American chestnut was an important component of the southern Appalachians [57] before its removal by chestnut blight. This disease-induced effective extinction has had serious consequences for the ecology of the region [58]. Aggregations of dead wood from affected trees have environmental repercussions even decades after the initial epidemic, such as influencing the structure and function of Appalachian streams [59].

Plants represent food and habitat to other plant and animal species. Where such relationships are exclusive, the potential for knock-on extinctions as a result of disease-related declines exists. An outbreak of wasting disease in the marine eelgrass Zostera marina, caused by a pathogenic slime mould Labyrinthula zosterae [26], resulted in >90% loss of eelgrass cover in the North Atlantic Ocean from 1930 to 1933. Although some eelgrass populations survived in low-salinity refugia, a host-specific eelgrass limpet, Lottia alveus, became extinct shortly thereafter [60]. Ironically, population loss owing to other reasons can lead to threats of extinction of both host and parasite assemblages. The palm Dictyosperma album var. conjugatum was once widespread on the Indian Ocean island of Mauritius until deforestation and other factors extirpated the population. The species survives only on a 151-ha offshore islet, Round Island, as a single individual. A host-specific scale insect Asterolecanium dictyospermae was recently described from this tree, surviving as a seriously endangered remnant population [61].

**Addressing the threat of plant EIDs**

The past few decades have seen the development of an increasingly secure global system of plant health inspection and quarantine (phytosanitary system) that balances agriculture protection against international trade [36]. However, our understanding of the risk of pathogen introductions is still poor and there is a need to improve surveillance strategies (Box 5). EID research must change from passive cataloguing to analysis and interpretation of the factors contributing to disease emergence. Spatial and temporal analyses of anthropogenic drivers of EIDs, coupled with an understanding of pathogen biology, evolutionary and environmental factors are required to predict and prevent emergence [62].

That there are few reports of EIDs affecting the conservation status of wild plants might reflect a lack of interest of researchers in wild plant pathogens compared with those pathogens affecting economically important crops or charismatic wildlife species. However, in addition to the impacts on biodiversity, wild-plant EIDs can threaten economically important species, and research on EIDs of both cultivated and wild plants requires better funding and should be given higher priority by the relevant policy and funding authorities. Reducing the threats and impacts of plant EIDs will require novel approaches to integrated research and long-term commitments from the scientific and political communities alike.

**References**


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