Oomycetes, or water moulds, are fungal-like organisms phylogenetically related to algae. They cause devastating diseases in both plants and animals. Here, we describe seven oomycete species that are emerging or re-emerging threats to agriculture, horticulture, aquaculture and natural ecosystems. They include the plant pathogens *Phytophthora infestans*, *Phytophthora palmivora*, *Phytophthora ramorum*, *Plasmopara obducens*, and the animal pathogens *Aphanomyces invadans*, *Saprolegnia parasitica* and *Halioticida noduliformans*. For each species, we describe its pathology, importance and impact, discuss why it is an emerging threat and briefly review current research activities.

This article is part of the themed issue ‘Tackling emerging fungal threats to animal health, food security and ecosystem resilience’.

1. Introduction

Oomycetes, or water moulds, are fungal-like eukaryotes classified as stramenopiles, and are phylogenetically grouped with diatoms and brown algae [1–4]. They are among the most problematic group of disease-causing organisms in both agriculture and aquaculture, and represent a recurrent threat for global food security. Oomycetes cause some of the most devastating plant diseases affecting crops, ornamental plants and trees. They result in major economic losses and serious damage to natural ecosystems [5,6]. The most notorious species are members of the genus *Phytophthora*, such as the late blight pathogen *Phytophthora infestans*, known for triggering the Irish potato famine [7,8]. Other notable species include *Phytophthora palmivora* [9], causing cocoa black pod, and the sudden oak death pathogen *Phytophthora ramorum*, which emerged in more recent epidemics threatening native tree species [10]. Additional important oomycete plant pathogens include members of the *Pythium* genus and downy mildews [11,12].

In contrast with their terrestrial counterparts, aquatic oomycetes remain understudied [3,4]. This applies particularly to animal pathogenic oomycetes, such as *Saprolegnia* and *Aphanomyces* spp. [13]. Aquaculture has become one of the world’s fastest growing food sectors, where freshwater fish dominate global aquaculture production [14]. Fish farming and fisheries provide livelihood and income for an estimated 54.8 million people, with employment in these sectors growing rapidly [15]. Diseases such as epizootic ulcerative syndrome and saprolegniosis, caused by oomycetes, are a significant threat to the aquaculture industry [16].
2. Why are oomycetes so successful?

The key to pathogenic oomycetes’ success resides in their capacity to adapt to overcome host resistance and occasionally jump to new hosts. Various features of oomycete biology can explain their high evolutionary potential. One major driving force for their success is encompassed by their flexible mating system. Oomycetes can reproduce sexually (either homothallic or heterothallic), asexually or through intergeneric hybridization [17] enabling them to gain both allelic diversity (sexual and intergeneric hybridization) and to rapidly proliferate, resulting in large population sizes (asexual/clonal populations) [18]. Large population sizes compensate for the lack of sexual recombination and increase the occurrence of spontaneous mutants with enhanced fitness [18].

Genome sequencing of oomycetes has provided additional insights into their capacity to adapt to hosts. Oomycete genomes harbour large repertoire of genes encoding virulence effectors, which modulate host processes [19,20]. These effector genes are typically associated with fast evolving regions of the genome, particularly in areas enriched in repeats and transposable elements, promoting genome duplication, shuffling, increased rates of mutagenesis and gene silencing [21]. This bipartite genome organization, differentiated by slow and fast evolving regions, has led to the ‘two-speed’ genome model, where gene-sparse repeat-rich compartments serve as a cradle for adaptive evolution, underpinning infection success by enabling the pathogen to rapidly overcome host resistance, evolve new virulence determinants and even jump to new host species [22]. Another interesting aspect about oomycete genomes is that they exhibit variations in ploidy and can exist as either triploids or polyploids [23–25]. The relevance of this feature is currently not fully understood, however, polyploidy has been shown to enhance vigour and buffer mutational changes 

3. Phytophthora infestans: the Irish potato famine pathogen

Phytophthora infestans infects potato and tomato plants, causing late blight disease. It is infamous for triggering the Irish potato famine in the mid-nineteenth century. Today, it is still a significant threat to global food security, causing severe yield losses in these crops, and economic losses of more than USD 6 billion annually [30,31]. In addition to destroying the foliage, P. infestans can infect tubers, which facilitates migration of the pathogen via the global seed tuber trade [32]. Current disease management strategies include chemical control and/or deployment of resistant cultivars. Resistance to agrochemicals, however, is common in epidemic-causing isolates [33], and due to the pathogen’s adaptability, disease resistance genes are also quickly broken down and rendered ineffective [34].

Disease outbreaks are typically caused by asexually reproducing clonal lineages [24,32,35,36]. These aggressive lineages dramatically reshape the population structure and become dominant over a short period of time [36,37]. For example, in Great Britain, genotype 13_A2, first detected in July 2005, surged to more than 75% of the P. infestans population by 2008 (figure 1) [35]. Subsequently, 13_A2 was displaced by another genotype, 6_A1, which increased in occurrence from 20% in 2010 to 80% in 2011 (http://www.fwi.co.uk/academy/lesson/potatoes-understanding-blight1). In addition, some P. infestans clonal lineages have become pandemic. 13_A2 was detected for the first time in South India in 2008, where it caused severe late blight epidemics in tomato, often resulting in 100% crop loss [38]. The emergence of this genotype in the Indian subcontinent has led to an increase in late blight incidence on tomato and potato, with epidemics reported in subsequent years following its introduction. The P. infestans genome was sequenced in 2009 [8], which established this species as a model system, pioneering studies of oomycete effectors and evolution [8,40]. Notably, the study of P. infestans effectors have significantly moved forward our understanding of host processes targeted by the pathogen, forming strong foundations for plant–microbe interaction studies [19,41]. Furthermore, effectors have emerged as powerful tools to rapidly identify new resistances in potato germplasms.

4. Phytophthora palmivora: the pathogen of tropical plants

The P. palmivora is a plant pathogen that infects more than 200 plant species in the tropics, including dicots and monocots [7]. Economically important hosts include cocoa, coconut, pineapple, rubber tree, durian, citrus, papaya and oil palm. Disease outbreaks in these crops impact the livelihood and nutrition of millions of people globally. Annual losses due to diseases caused by P. palmivora, such as oil palm bud rot and cocoa black pod, range from USD 250 million to over USD 1 billion, respectively [9].

Rainy season and high humidity favour pathogen sporulation leading to rapid disease progression and production of oospores. Disease spread from the soil into the canopy is often initiated through rain splash. Once the roots, leaves and fruits in the lower canopy are infected, the pathogen quickly spreads to the upper canopy by insects, wind and through wind-driven aerosols [7,42]. Additionally, transport of infected nursery plants, improper disposal of infected material, irrigation with zoospore-contaminated water, changing temperatures and global trade all contribute to the spread of P. palmivora. This makes it a serious threat to the economy of many developing countries, where cultivation of highly productive perennial crops is intensifying.

The broad host range of P. palmivora has enabled research using model plants such as Medicago truncatula [43], Hordeum vulgare (barley) [44] and Nicotiana benthamiana [45], moving forward the characterization of the molecular mechanisms involved in P. palmivora colonization. Notably, the process of root colonization by this pathogen is used to study similarities and differences between pathogenicity
Table 1. Features of oomycete pathogens highlighted in this paper, including the primary host, genome size, repetitive DNA content (as a percentage of genome size), number of protein coding genes, percentage of predicted secreted proteins and a summary of why the pathogen is a (re-)emerging threat. n.d., no data.

<table>
<thead>
<tr>
<th>pathogen</th>
<th>important hosts (P, plant; A, animal)</th>
<th>common disease name (host)</th>
<th>why is it a (re-)emerging threat?</th>
<th>genome size (Mb) (％ repetitive)</th>
<th>protein coding genes (％ secreted)</th>
<th>references</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phytophthora infestans</td>
<td>P—potato, tomato</td>
<td>late blight</td>
<td>new aggressive genotypes emerged in the UK (2008 and 2011) and India (2008)</td>
<td>240 (7.4)</td>
<td>18 155 (8.7)</td>
<td>[8]</td>
</tr>
<tr>
<td>Phytophthora palmivora</td>
<td>P—cocoa, coconut, pineapple, rubber tree, papaya, oil palm</td>
<td>bud rot (oil palm), black pod (cocoa), fruit rot (papaya)</td>
<td>outbreaks regularly destroy oil palm and cocoa plantations worldwide</td>
<td>n.d. (n.d.)</td>
<td>n.d. (n.d.)</td>
<td>—</td>
</tr>
<tr>
<td>Phytophthora ramorum</td>
<td>P—oak, tanoaks, rhododendron, beech, larch</td>
<td>sudden oak death (oak)</td>
<td>outbreaks destroyed forests in North America and Europe during the last 20 years</td>
<td>65 (17)</td>
<td>14 451 (10.5)</td>
<td>[27]</td>
</tr>
<tr>
<td>Phacopara obscurus</td>
<td>P—Impatiens species</td>
<td>Impatiens downy mildew</td>
<td>outbreaks occurred in nurseries worldwide within the last decade</td>
<td>70 (n.d.)</td>
<td>n.d. (n.d.)</td>
<td>[28]</td>
</tr>
<tr>
<td>Aphanomyces invadans</td>
<td>A—carp, perch, tilapia, snakeheads, salmonids, estuarine</td>
<td>epizootic ulcerative syndrome</td>
<td>regularly kills farmed and wild fish worldwide, threatening food safety in Asia and Africa</td>
<td>71 (n.d.)</td>
<td>15 248 (n.d.)</td>
<td>b</td>
</tr>
<tr>
<td>Saprolegnia parasitica</td>
<td>A—fish (salmon), amphibia, crustaceans, aquatic insects</td>
<td>saprolegniosis</td>
<td>threatens fish farming in Europe, America and Asia, as well as endangered amphibia species worldwide</td>
<td>63 (17)</td>
<td>17 065 (5.7)</td>
<td>[29]</td>
</tr>
</tbody>
</table>

*aIncluding putative effectors.

bSee http://www.ebi.ac.uk/ena/data/view/GCA_000520115.
and symbiosis at the molecular level [46]. Publically available genomic resources are currently limited, which impairs research progress.

5. Phytophthora ramorum: the sudden oak death pathogen

*Phytophthora ramorum* is a devastating oomycete pathogen that causes sudden oak death disease [47]. It has a wide host range, affecting more than 100 plant species from 40 different genera. These include environmentally and economically important tree species such as oaks, tanoaks, rhododendron, European beech, Japanese larch and many woody ornamental plants [47–49]. Disease symptoms are host specific, but can vary from necrotic lesions in leaves, to shoot dieback and bleeding cankers on the stem [10,50].

In Europe, *P. ramorum* is mainly present in ornamental nurseries or gardens, however, in the UK this pathogen has caused significant tree losses and landscape-scale epidemics [48]. Despite the known reports of *P. ramorum* in Europe [51] and North America [52–55], predictive models suggest that the pathogen might be adapted to larger areas in Africa, Australia and South America. Therefore, potential of invasion and further spread of the pathogen is possible [56]. In addition, given that diagnosis is primarily based on visual inspection, disease symptoms can be masked by agrochemicals, increasing the likelihood of pathogen intrusion into susceptible areas by the ornamental plant trade market [57].

*Phytophthora ramorum* occurs as four clonal lineages [58] that have resulted in the emergence of new diseases in forest frequency 

![Figure 1](image-url). An emerging clonal lineage reshaped *P. infestans* populations in Great Britain. (a) Multilocus genotyping of *P. infestans* isolates from 4000 late blight infection sites over 11 years. This survey revealed that the late blight epidemic that started in 2006 was due to the aggressive 13_A2 isolate that became dominant in only 3 years. The number of surveyed isolates and dominant genotypes of each mating type are indicated. Genotypes with low frequency are grouped as ‘misc’. The shading between bars indicates mating types. (Figure and legend are adapted from Cooke et al. [35].) (b) The 13_A2 isolate has rapidly spread to other continents and caused devastating economic losses especially in developing countries such as India and Egypt [38]. Dots represent an approximate of sampling sites for years 2013 and 2014 according to EuroBlight data and recent reports [39].
ecosystems within Europe and the USA [51,53,59]. The
_P. ramorum_ genome was sequenced in 2006, a few years after
the pathogen was first detected, providing novel insights into
its biology [60]. Recent genome sequencing of additional
isolates provided further genetic information to track the
spread of the pathogen [61]. However, our understanding of
its emergence and adaptation remains relatively limited [62].

6. _Plasmopara obducens_: the downy mildew
of _Impatiens_ species

_Plasmopara obducens_ is an obligate biotrophic pathogen of horti-
cultural plants from the _Impatiens_ genus. It causes the _Impatiens_
downy mildew foliar disease, which results in wilted and
defoliated plants that die within weeks of disease onset [63].
Commercially grown varieties of _Impatiens walleriana_ are sus-
ceptible to _P. obducens_, making _P. obducens_ a clear and present
threat to the cultivation of _Impatiens_ spp. worldwide [64].

Within the past decade, outbreaks have been reported
in North America [65,66], the Hawaiian Islands [67], Europe
[68–70], Asia [63] and Australia [71]. Such epidemics were
associated with important economic losses [67]. In the USA,
the spread of _P. obducens_ caused great concern and resulted
in the establishment of a research consortium [72]. Cultural
practices and chemical applications limit the spread of
the disease, however, they are not permanent solutions, as
mefenoxam-insensitive _P. obducens_ isolates have already been
identified [72]. Additionally, _P. obducens_ is a persistent patho-
gen; its ability to spread by wind and survive in soil means
disease eradication will be difficult.

The lack of information on the genetics and the structure of
_P. obducens_ populations, as well as on the factors that led to the
establishment of global epidemics, impair the development of
effective mitigation strategies. To fill this knowledge gap,
Salgado-Salazar et al. [28] recently released a draft genome
sequence of _P. obducens_, along with a set of genetic markers.
These molecular resources may help in determining both
the population structure and genetic factors that control
infection processes.

7. _Aphanomyces invadans_: the fish epizootic
ulcerative syndrome pathogen

_Aphanomyces invadans_ causes epizootic ulcerative syndrome, a
fish disease listed on the World Organisation for Animal
Health website (http://www.oie.int/en). _Aphanomyces inva-
dans_ is pathogenic on several economically important fish,
including carp, perch, tilapia, snakeheads, salmonids and estu-
arine fish species [73–75]. It was first reported from farmed
ayan, _Plecoglossus altivelis_, in 1971 in Japan [76]. Since then it
has spread rapidly throughout Asia and into some areas of
Africa.

_Aphanomyces invadans_ has been responsible for large-scale
mortalities of farmed and wild fish in more than 20 countries
across four continents [77]. In Asia and Africa, the disease has
negatively impacted the livelihood of fish farmers and fisher-
men and, in some cases, threatened the sustainable food
supply for local populations, who depend on fish as a relatively
affordable source of animal protein [74]. Experimental infection
studies have demonstrated that _A. invadans_ can produce severe
pathological changes in several European salmonid and catfish
species [78]. Hence, it is possible that it could cause serious pro-
blems in European tilapia, salmon and trout aquaculture
industries and in numerous native and wild fish species.

Despite its economic and social importance, little is known
about the biology of the _A. invadans_ pathogen. In an effort to
address this gap in knowledge, the first draft genome sequence
has been completed at the Broad Institute (http://www.ebi.ac.
.uk/ena/data/view/GCA_000520115). These sequences will
enable studies on the biology, evolution, biodiversity, genetics,
virulence/pathogenicity and biochemistry of _A. invadans_.

8. _Saprolegnia parasitica_: the saprolegniosis
water mould

_Saprolegnia parasitica_ causes saprolegniosis on various fish
species. In addition to fish, species of amphibians [79],
crustaceans [80] and aquatic insects [81] are also highly suscep-
tible to _Saprolegnia_ infections. This is conclusive evidence that
_Saprolegnia_ species are major killers of populations of amphi-
bians globally, threatening some already highly endangered
species [82–84]. _Saprolegnia parasitica_ is a major problem in the
fish farming industry in Europe, Chile, Canada and Asia.
At least 10% of all hatchled salmon [13] and over 10% of all
eggs succumb to _Saprolegnia_ infections according to estimates
[4,13].

Until 2002, _S. parasitica_ was kept under control with appli-
cations of malachite green. However, the use of malachite
green has been banned worldwide due to its toxicity
[85–87], resulting in a dramatic resurgence of _Saprolegnia_
infections in salmon aquaculture. At present, fish farmers
are struggling to control this pathogen. Current control
methods involve treatments with formalin-based products,
which are also expected to be banned in the EU in the very
near future [88–90]. Therefore, sustainable prevention and
control measures are desperately needed.

Recent studies have elucidated an induced native immune
response and a suppressed adaptive immune response in fish
infected by _S. parasitica_ [91]. In addition to distinct bioche-
mical pathways that were described during fish–_Saprolegnia_
interaction to establish infection [92], the genome sequence
of _S. parasitica_ [29,93] has also shed light on the molecular
mechanisms of infection, broadening our understanding of
infection mechanisms.

9. _Halioticida noduliformans_: the abalone
tubeerd mycosis pathogen

_Halioticida noduliformans_ is a marine pathogen of abalone
(Haliotis sp.), on which it causes abalone tubercle mycosis, and
mantis shrimp (Oratosquilla oratoria). It is a member of the
Haliophilales, an early diverged lineage in the Oomycota
phylum [94]. _Halioticida noduliformans_ was first discovered in
2004 in South Africa [95,96] and is a threat for abalone in com-
mercial aquaculture, which constitutes more than 93% of the
global abalone market [97].

Between 2004 and 2006, several outbreaks in commercial
aquaculture farms in South Africa and Japan caused up to
90% mortality among spat and up to 30% mortality among
older animals [96,98]. Pathogen control is challenging [96].
Indeed, single _H. noduliformans_ isolates can infect at least three
abalone species from South Africa, Mexico and Japan [96].
Researchers from Japan and South Africa have provided insights into the distribution of *H. noduliformans* outbreaks and the molecular phylogeny of *Haliclona* species [95,96,98,99]. Additionally, Greeff *et al.* [100] have established a rapid and sensitive qPCR assay for species-specific detection and quantification of *H. noduliformans* in abalone tissue. However, important resources like genome sequences and gene expression data are lacking. Identification of natural hosts and determining whether these hosts act as reservoirs for *H. noduliformans* will be vital to successfully control this disease.

10. Five ways to tackle emerging or re-emerging oomycete threats

Outbreaks caused by oomycete diseases are a clear and present threat to food security and to natural ecosystems. Although there is a general awareness about these outbreaks, there are gaps in the community capacity to deliver effective short- and long-term response plans. Research showed that oomycetes evolved the ability to infect plants and animals independently of other eukaryotic microbes, and therefore, likely developed unique mechanisms of pathogenicity [101]. The last few decades have seen significant progress in understanding the biology and molecular basis of host infection by oomycete pathogens. Notably, genomic studies have revealed fundamental concepts that link genome architecture, pathogenicity-related proteins and evolution, key factors that drive disease emergence and pathogen success [22]. Here, we highlight five main research areas that need to be sustained or reinforced to help us tackle emerging and re-emerging oomycete threats:

— **Genomic resources.** Sequencing of plant oomycetes has pioneered our understanding of pathogenicity and has provided downstream tools that are essential for mechanistic research. In addition, as more genomes are sequenced, comparative genomic studies will help elucidate infection mechanisms across multiple isolates and species, providing critical information on conserved infection strategies. Oomycete pathogenomics have greatly impacted our understanding of pathogenicity and has been critical information on conserved infection strategies.

— **Genetic manipulation of hosts and pathogens.** Host genetic improvement can be accomplished by precise genome editing techniques, such as the CRISPR/Cas9 technology [103]. CRISPR/Cas9 was successfully implemented in tomato to enhance resistance against an oomycete pathogen [104]. To date, mechanistic studies dissecting oomycete–host interactions have been limited to genetic manipulation of the host species due to the lack of efficient gene replacement methods in oomycetes. Recently, Fang & Tyler [105] implemented the CRISPR/Cas9 system in oomycetes and were able to rapidly and efficiently edit the *Phytophthora sojae* genome. Further implementation of reverse genetic tools in other oomycete species is now needed.

— **Surveillance.** Surveillance consists of the assessment of disease incidence and virulence characterization (via pathotype surveys). In agriculture, pathogen surveillance is crucial to establish national and international policies, government investment and strategies in plant protection, plant breeding, seed distribution and pathology. Surveillance remains a critical aspect of disease control, since understanding the pathogen population allows rapid deployment of control methods. In addition to managed agricultural ecosystems, pathogen surveillance should also include natural habitats and potential reservoirs, as these areas generate new inoculum for re-infection.

We have entered a new era in incursion reporting, where social media provide an efficient and rapid platform for data dissemination and collaborative efforts to tackle new disease emergence and spread. By communicating disease significance to the public and building up awareness of these destructive diseases, a community effort for surveillance can be achieved. This was successfully demonstrated in Australia, after the incursion of myrtle rust [106] and in the recent actions surrounding the outbreak of wheat blast in Bangladesh [107]. Websites such as http://rusttracker.cimmyt.org and http://euroblight.net are great examples of successful first steps to coordinating global disease surveillance efforts.

— **Field pathogenomics.** In addition to tracking movement and variation of pathogens (surveillance), tackling emerging diseases will not be successful without concurrently understanding the genetics of host resistance. Field pathogenomics adds highly informative data to surveillance surveys by enabling the rapid evaluation of pathogen population structure and host genotype [108]. This has already been successfully implemented with oomycete and fungal pathogens of potato and wheat crops, and will be applicable to other pathogens as more genomic information is generated [35,109].

— **Databases.** Committing to long-term data recording, linkage between databases, free flow of information and faster exchange of information are necessary for updating epidemiological data. Data should be made rapidly and publically available to maximize the combined and coordinated efforts of the scientific community. For instance, scientists recently released websites to share sequencing data generated as an immediate response to outbreaks such as ash dieback (https://geefu.oadb.tsl.ac.uk/) and wheat yellow rust in the UK (http://yellowrust.com/) and wheat blast in Bangladesh (http://wheatblast.net). Additionally, social media websites, such as Plant Village (https://www.plantvillage.org), allow for free sharing of crop disease information in real time. These websites not only become platforms for scientists to interact, establish collaborations and coordinate among themselves, but also provide information for the general public and farmers to directly follow and participate in research efforts. They allow the world to connect, share and collectively find solutions to emerging oomycete threats.

Fighting agriculturally important pathogens is a long-term ongoing battle. Just like the constantly morphing flu virus, oomycete pathogens continuously evolve new races to evade host resistance. Agricultural scientists, veterinarians,
breeders and farmers need to remain vigilant and work together in reporting and tackling oomycete pathogens to achieve successful control.

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