Fungal genomes enhance our understanding of the pathogens affecting trees cultivated in Southern Hemisphere plantations

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Abstract

Forest pathogens are a major cause of forest disturbances and they have a significant economic impact on commercial forestry. Genomics is an important technology now available for studies concerning tree health, enabling researchers to better understand pathosystems and potentially to prevent future epidemics from occurring. Comparative genomics at the species level makes possible the identification of unique genomic regions and/or genes that influence the development of pathogens and their ability to cause disease. In addition, population genomics can reveal processes involved in the evolution of pathogens potentially showing how selection and/or environmental adaptation could have driven their emergence. Using these tools, important mechanisms involved in the evolution of pathogens and their hosts can be determined. Practical applications of such knowledge include the formulation of strategies for pathogen detection and surveillance, as well as breeding disease-resistant trees. These new and evolving technologies are set to ensure the long-term sustainability of plantation forestry in the Southern Hemisphere.

Keywords: forest tree pathogens, genomics, plantation forestry

Introduction

The field of genomics has significantly improved our understanding of fungal pathogens of trees, particularly non-native trees established in Southern Hemisphere plantations. Many of these advances have arisen from the relative reduction in the costs associated with genome sequencing and access to whole genome sequencing platforms and facilities (Chiu and Miller 2016). Previously, the genomes of model fungi, which included a small number of plant pathogens, received the bulk of attention as researchers sought to understand the characteristics and evolution of these organisms. However, with the wide accessibility to whole genome sequence information, researchers are increasingly applying the knowledge and lessons learnt from model organisms to non-model species (Ellegren 2014). This has led to the sequencing of the genomes of numerous non-model fungi, including pathogens of trees, and some of these are emerging as model systems in their own right.

The genomes of an increasing number of fungal pathogens of woody plants in natural ecosystems and in plantations are being studied globally (Table 1). In many cases, these genomic resources have been complemented with genome sequence information for the woody hosts (e.g., *Eucalyptus* and *Pinus* species) of these fungi (Hirakawa et al. 2011; Myburg et al. 2014; Neale et al. 2014; Stevens et al. 2016; Wang et al. 2020). Sequencing the genome of *Fusarium circinatum* (Wingfield et al. 2012) marked the genesis of genome research on fungal pathogens of non-native plantation-grown trees in the Southern Hemisphere. Subsequently, the genomes of several fungal pathogens of plantation-grown trees occurring in various Southern Hemisphere countries have been sequenced (Table 1). Access to these genome resources has provided a multiplicity of opportunities to study the evolution of pathogens and their genes and genomes, as well as their genome architecture and population biology.

A number of reviews have considered various aspects of fungal and plant pathogen genomics (e.g. Skamnioti et al. 2008; Stukenbrock and Bataillon 2012; Ma et al. 2013; Gladieux et al. 2014; Plissonneau et al. 2017; Stajich 2017; Keriö et al. 2020). Those most relevant to forest pathology have dealt with the contribution of genomics to the field (Hamelin 2012) and the impact that the availability of these genomes has had on the field of plant pathology (Aylward et al. 2017). In the current paper, we specifically considered the impact of genomic studies of fungal pathogens of non-native *Pinus*, *Eucalyptus* and *Acacia* species, which are utilized for commercial plantation forestry in the Southern Hemisphere (Table 1).

The pathogens considered in this study are *Austropuccinia psidii*, *Dothistroma septosporum*, *Fusarium circinatum*, *Teratosphaeria destructans*, *Chrysoporthe* and *Ceratocystis* species, as well as species in the family Botryosphaeriaceae (Figure 1). *Austropuccinia psidii* is the causal agent of myrtle rust on non-native *Eucalyptus* grown in commercial plantations in South America (Pérez et al. 2011; Rodas et al. 2015; Granados et al. 2017). *Fusarium circinatum* causes pitch canker on susceptible *Pinus* species and the fungus is considered one of the most important pathogens of pines (reviewed in Wingfield et al. 2008). *Teratosphaeria destructans* is a leaf and shoot pathogen of *Eucalyptus* species (Wingfield et al. 1996; Burgess et al. 2016; reviewed in Andjic et al. 2019) that was only relatively recently identified in the Southern Hemisphere, in South Africa (Greyling et al. 2016). *Dothistroma septosporum* causes Dothistroma needle blight on non-native *Pinus* in the Southern Hemisphere (Gibson 1972; Alzamora et al. 2004; Barnes et al. 2004; Rodas et al. 2016). The *Chrysoporthe* species included are pathogens of non-native *Eucalyptus* in Africa and South America (Hodges 1980; Conradie et al. 1990; van der Merwe et al. 2001; Nakabonge et al. 2006), while those of *Ceratocystis* are pathogens of non-native *Eucalyptus* and *Acacia* species in the Southern Hemisphere (Wingfield et al. 1996; Roux et al. 1999; Barnes et al.

2003; Roux and Wingfield 2009). The Family Botryosphaeriaceae includes various genera with species that occur as latent pathogens of *Eucalyptus* and *Pinus* in various regions of the Southern Hemisphere (Slippers and Wingfield 2007; Slippers et al. 2017). For instance, disease caused by the latent pathogens *Neofusicoccum parvum* and *Diplodia sapinea*, respectively, manifest as branch die-back when trees are under stress (Slippers and Wingfield 2007; Slippers et al. 2017).

The primary goal of this review was to examine how genomics research has advanced foundational knowledge about various aspects of the biology of the fungi mentioned above. A brief overview of the genomics workflow is followed with a discussion of how this field of research has contributed to the taxonomy and diagnostics of these organisms. The impact of genomic data on our understanding of their population and reproductive biology were then investigated. We also examined how genomics provided answers to questions on the ecology and evolution of these fungi. Finally, we explored how fungal genomics and comparative genomics have already and will in future facilitate improvement of our ability to manage the diseases of tree species that sustain forestry in the Southern Hemisphere.

From DNA to genome sequence

Appreciating the impact of genomics on the study of forest tree pathogens requires general understanding of how genomes are sequenced, assembled and annotated, as well as how information is obtained from genomes. Here, we do not attempt to provide an in-depth discussion of the intricacies of genomics methodologies, and rather refer readers to the Field Guide to Whole-genome Sequencing, Assembly and Annotation by Ekblom and Wolf (2014).

A broad overview of the workflow used in whole-genome sequencing projects is presented in Figure 2. The first step when designing a genome sequencing project is to decide on the fungal strain(s) that will be sequenced. For taxonomic purposes and inter species genomic comparisons, researchers typically select the type strain of the species under consideration. This is because its genome will serve as the reference for all other strains in the species. Strains for inter species comparisons might also be selected based on their unique features and where researchers wish to conduct comparative genomics studies (see below). For intra-species comparative studies, individuals are typically selected to span the broader biological, ecological and / or geographical variation within the species. This is done with the aim of capturing genomic differences that can be used for genetic studies or that might explain unique characteristics of the individuals at the genomic level.

An important step in genome sequencing is to obtain genomic DNA of adequate quality and in a sufficient quantity required for the specific sequencing platform being used. This is often challenging as many fungi produce polysaccharides and other compounds that hamper the DNA extraction and sequencing processes. Once genomic DNA has been obtained, it is fragmented (mechanically, chemically or enzymatically) in order to construct genomic libraries that are sequenced using high throughput sequencers. Often, more than one type of sequencing platform is used as these platforms differ in the length of sequences that they can read, output per run and error rate (Bleidorn 2016; Chiu and Miller 2016).

The main output of high throughput sequencers are sequence reads (fragments of DNA sequences). The quality of the raw reads is assessed using software such as FastQC (Andrews 2010), and those of low quality are removed from the dataset. Following this step, the reads are assembled into contigs based on their overlapping sequences, and the contigs can

subsequently be joined into scaffolds. A combination of long and short read DNA sequencing technologies provides the most complete genome assemblies (Amarasinghe et al. 2020). Various software packages are available for *de novo* assembly of sequences (Khan et al. 2018). The completeness, and hence the quality, of the assembled genome can be measured based on the percentage of universal single copy genes present in the assembled genome. This is done using software such as Benchmarking Universal Single-Copy Ortholog (BUSCO) (Waterhouse et al. 2017). If the completeness of the genome sequence is sufficient, genes are identified using software packages that apply gene models to recognise genes based on the molecular characteristics of open reading frames (Yandell and Ence 2012). Software applications used for this purpose include AUGUSTUS (Stanke and Morgenstern 2005). Finally, the function of the genes are identified based on sequence similarity with genes that have previously been functionally annotated (see Yandell and Ence 2012).

In some cases it is not feasible to sequence the complete genome of a fungus. This can preclude researchers from pinpointing the exact locations of genes or contigs on chromosomes. However, the relative location of genes can be determined if genetic linkage maps are available. For example, a genetic linkage map was generated prior to sequencing the genome of the pitch canker pathogen (*F. circinatum*) by making use of the hybrid progeny of a cross between this fungus and the maize pathogen *F. temperatum* (De Vos et al. 2007). The 12 linkage groups of *F. circinatum* were then integrated with genome data to allow ordering of the genomic contigs into linkage groups or chromosomes (De Vos et al. 2014). The genome of *F. circinatum* is now assembled into 12 pseudomolecules that will in future serve as a physical map in which the exact locations of genes are known (Wingfield et al. 2018b). In a similar way, linkage maps generated for other fungal tree pathogens (e.g., *Amylostereum areolatum*, *Ceratocystis fimbriata* and *Ce. manginecans*) (van der Nest et al.

2009; Fourie et al. 2019) will greatly facilitate the chromosomal positioning of contigs and provide further insights into the locations of genes relative to one another.

Whole genome sequencing projects have shown that genome size variation is prevalent among fungal species. The genome size of most fungi falls within the 30-40 million bases (Mb) size range. However, genome sizes range from two to 1 200 Mb (Aylward et al. 2017; McTaggart et al. 2018), with the largest to date being that of the myrtle rust pathogen *Austropuccinia psidii* (McTaggart et al. 2018). The average genome size of plant pathogenic Basidiomycota (53.7 Mb) is much larger than that of plant pathogenic Ascomycota (39.4 Mb) (Aylward et al. 2017). The majority of tree pathogens listed in Table 1 have genome sizes within the 30-69 Mb size range, with the highest number (26) of genome sizes being within the 40-49 size range (Figure 3). Similar to the study of Aylward et al. (2017), the average genome size of fungi in the Ascomycota (37.93 Mb; Median: 34.60 Mb; Table 2) is much smaller than those of the Basidiomycota (120.48 Mb, Median: 61.42; Table 2). However, a more than two-fold size difference in the average genome size was observed from the analyses of the Basidiomycota genome sizes for this review when compared with those in Aylward et al. (2017) for the same phylum. This difference can be ascribed to the inclusion in this review of a greater number of rust fungi, which have very large genomes (Table 1).

Identification of fungal pathogens

Accurate identification of fungal tree pathogens is an essential first step towards developing disease management strategies and for informing appropriate biosecurity and quarantine processes (McTaggart et al. 2016). Incorrect identification of pathogenic fungi typically impedes our understanding of their biology, epidemiology and impact. For example, the myrtle rust pathogen *A. psidii* (formerly *Puccinia psidii*) that is a destructive invasive

pathogen in various parts of the world, particularly Australia, was initially identified in 2010 as *Uredo rangelii* when it was encountered in that country (Carnegie et al. 2010). Because *U. rangelii* is considered to be a less serious pathogen, quarantine procedures were discontinued in Australia shortly after it was first detected. Later, however, teliospores with the morphological characteristics resembling those of *P. psidii* were observed (Carnegie and Cooper 2011), and the fungus was then referred to as *P. psidii sensu lato* (Carnegie and Lidbetter 2012) after which it was formally transferred to the new genus *Austropuccinia* (Beenken 2017). The unfortunate misidentification of this important quarantine pathogen delayed strategies to potentially contain its spread in Australia where it now threatens highly susceptible native Myrtaceae species with extinction (Carnegie et al. 2016). Moreover, this fungus is now a serious threat to non-native *Eucalyptus* species in other countries of the Southern Hemisphere.

Fungal genomes provide useful information to develop diagnostic tools that ensure rapid and reliable identification of pathogens. Accurate identification of fungal species generally requires DNA sequence data for particular genomic regions. The sequence data of these regions, requires PCR amplification, which in turn relies on the availability of PCR primers to amplify the regions of interest. However, primers are not always available or, when they are available, might not hybridize with sufficient specificity to their target regions during PCR. Additionally, PCR primers should not be so specific to a particular isolate that false negatives might occur, or insufficiently specific that false positives occur. However, these complexities are easily resolved with the use of genome sequences, which allows primers to be designed by comparing existing primer sequences with those in the genome of the organisms under consideration. This approach is well illustrated in the study of Fourie et al. (2014) in which the genome sequence of the aggressive tree and root-crop pathogen *Ce.*

fimbriata was used to generate species-specific primers to amplify genes, which were then used to delineate cryptic species in *Ceratocystis sensu stricto.* More recently, Feau et al. (2018) published a genomics pipeline that utilises genome sequences to identify taxon-specific genome regions that can be used to design highly accurate PCR assays for the identification of plant pathogens.

Comparative genomics studies can allow for the identification of single nucleotide polymorphisms (SNPs) to aid species delineation. This approach relies on the genomes of a collection of isolates representing each of the species under investigation to ensure that the identified SNPs vary between, but not within, species. An example of the application of this technique relevant to a plantation tree pathogen can be found in a study by Fourie et al. (2014) who identified unique SNP markers for 13 *Ceratocystis* species using high throughput sequencing of restriction enzyme digested DNA. They also showed that SNPs were more taxonomically informative than the DNA sequences of protein coding genes. For instance, they could conclusively delineate a range of species of *Ceratocystis* and, based on the absence of SNPs, reduced *Ce. acaciivora* to synonymy with *Ce. manginecans*.

Another way of exploiting genome data to combat tree diseases is for developing methods to rapidly and accurately identify pathogens *in situ* in plantations or nurseries. One such a method is Loop Mediated Isothermal Amplification (LAMP) of targeted DNA (Notomi et al. 2000). In addition to species diagnostics, this technique can be used to identify genetic traits such as the mating type of fungi (King et al. 2019). LAMP relies on auto cycling strand displacement DNA synthesis by DNA polymerases under isothermal conditions with a set of four or more primers that hybridize to different parts of the genome (Notomi et al. 2000, and see Niessen 2015 for an explanation of the reaction). The amplified DNA fragments can be

observed using various methods, such as production of a white precipitate if DNA amplification has been successful (reviewed in Zhang et al. 2014). Alternatively, the amplification process can be visually monitored in real time by adding colorimetric indicators that produce a fluorescence signal if the LAMP reaction is positive (Niessen 2015 and references therein). Once a protocol has been developed, LAMP is cheaper and requires less effort than conventional PCR (Niessen 2015). Furthermore, the reaction is highly sensitive, thus enabling the amplification of genomic regions in the presence of low concentration DNA (Notomi et al. 2000).

Although LAMP has become an important diagnostic tool for fungal identification, particularly for in-the-field situations, LAMP protocols for plantation tree pathogens in the Southern Hemisphere are still in development or are yet to be developed. However, with the availability of genome data, it would be relatively straightforward to identify species-specific genomic regions and genes for designing the LAMP primers. From a Southern Hemisphere perspective, significant progress has been made for *F. circinatum.* Comparative genomics data were used to identify various candidate genes that are unique to the fungus (Maphosa et al. 2016). Also, data from a study by van Wyk et al. (2018), showed that a genomic region of 12 000 base pairs, which underpins a growth QTL, is present only in *F. circinatum* and not in its close relatives These candidate genes and the long stretch of DNA unique to *F. circinatum* is currently being used to develop a LAMP-based diagnostic protocol for this important tree pathogen.

Genomics lies at the forefront of recent developments in biosecurity and quarantine measures (see Hamelin and Roe 2020 for detailed discussion). Earlier, McTaggart et al. (2016) have argued that a paradigm shift is needed from a name-based to a gene-based approach when

assessing the risks of pathogens. This is because genome information can be used to predict pathogen lifestyle based on the presence of genes associated with pathogenicity, or the reduction/expansion in certain gene families (Soanes et al. 2008; Ohm et al. 2012; Lo Presti et al. 2015; Haridas et al. 2020). Genomics studies have shed significant light on the evolutionary processes and mechanisms involved in fungal speciation and the evolution of new and emerging pathogens (e.g., Stukenbrock 2013; Steenkamp et al. 2018). Genomics studies have also shown that fungal genomes are dynamic and that the signature of such plasticity may predict the "adaptability" of a species or individual (Ohm et al. 2012; Zhang et al. 2018). Genome-based information is thus invaluable to infer risks associated with particular organisms, but the adoption of such gene and genome-based approaches to biosecurity would be dependent on the availability of detailed genome information for pathogens that can be used in risk assessment. Therefore, development of rapid and cost-effective genome sequencing technology and the regular release of newly sequenced pathogen genomes will greatly facilitate future implementation of this biosecurity paradigm.

Population biology

Population genetic studies provide information regarding the genetic diversity of populations. Because genetic diversity in a population changes over time, this information can be used to determine pathways of introduction and to track the movement and establishment of pathogens. Understanding the genetic variation in populations also provides insights into the potential of pathogens to adapt to new hosts and the probable durability of resistant genetic tree planting stock (McDonald and Linde 2002; Graça et al. 2011).

Most contemporary population genetic studies of fungal pathogens utilise microsatellite markers. These markers are short tandem sequence repeats distributed throughout the genome

of organisms (Levinson and Gutman 1987), they are highly polymorphic and inherited in a Mendelian manner (Levinson and Gutman 1987; Tautz 1989). Although their identification traditionally has been time-consuming and expensive, high throughput genome sequencing has facilitated rapid development of large sets of microsatellite markers for diverse organisms. For those affecting Southern Hemisphere forestry, this was initially done using high throughput sequencing of microsatellite enriched DNA libraries (Santana et al. 2009). More recently, however, the easy access to genome data has provided opportunities for the *in-silico* identification of microsatellites using genome comparisons (Hoffman and Nichols 2011). This approach has been used to identify microsatellite markers for a wide range of fungi, including pathogens of trees important to plantation forestry in the tropics and Southern Hemisphere (Cai et al. 2013; Simpson et al. 2013; Jia et al. 2015; Mercière et al. 2015; Mlonyeni et al. 2018; Varady et al. 2019). In fact, using comparative genomics, makes it possible to design these markers such that they function across several species or even across genera (Leyva-Madrigal et al. 2014; Bhat et al. 2018). For example, Nagel et al. (2020) recently developed microsatellite markers that can be amplified across species in the genera *Lasiodiplodia* and *Neofusicoccum*, numerous of which are important latent pathogens of plantation-grown trees (Slippers and Wingfield 2007; Slippers et al. 2017).

Genomics can advance population genetic studies through the application of a large number of genetic markers. Population genomics is a relatively new field that owes its emergence to the development of techniques for affordable genome sequencing (for reviews see Stukenbrock and Bataillon 2012; Grünwald et al. 2016). In contrast to traditional population genetics, which relies on a small number of neutral markers such as microsatellites, population genomics uses a large number of markers spread throughout the genome (Stinchcombe and Hoekstra 2008). In addition to providing insights into ecological,

evolutionary and demographic processes acting upon populations, population genomics has the added benefit of informing how these processes, together with speciation and adaptation, affect genomes (see Grünwald et al. 2016 for details).

Most population genomic studies on tree pathogens have focussed on pathogens important in the Northern Hemisphere. These studies show that population genomics approaches have the potential to become indispensable tools in epidemiological research (Quinn et al. 2013). Population genomics also represents a powerful tool to better understand how pathogens emerge and to discover the mechanisms involved in their adaptation to new niches or lifestyles (Branco et al. 2015, Jensen et al. 2016, Plissonneau et al. 2017). An example of a population genomic study on a tree pathogen relevant to plantation forestry in the Southern Hemisphere is that of Bradshaw et al. (2019) on the important needle blight pathogen *D. septosporum*. By analysing the genomes of 18 strains from 15 countries, they showed that there is significant genetic variation among strains. Importantly, this also extended to gene copy number variations that underpinned the observed among-individual differences in the production of the toxin dothistromin that is important to pathogenicity. Genome comparisons such as these have clearly increased our understanding of environmental and host adaptation and they provide valuable insights into the biology and potentially, the management of tree pathogens.

Genomics provides insight into the reproductive strategy of fungi

Knowledge regarding the reproductive biology of tree pathogens is fundamental to understanding their epidemiology. As is true for all fungi, tree pathogens can reproduce asexually (clonal) or sexually. One of the advantages of clonal reproduction is that co-adapted alleles are maintained in the population, allowing for the rapid spread of new

genotypes (McDonald and Linde 2002; Möller and Stukenbrock 2017). Sexual reproduction, in contrast, enables adaptive evolution by increasing the effectiveness of combining beneficial mutations in response to environmental changes (Billiard et al. 2012).

The availability of genome sequences permits the rapid identification of the genes necessary for sexual reproduction. This is particularly valuable in the case of genes involved in mating as these evolve rapidly and are thus not that easy to identify. In fungi, mating type is determined by the genes encoded at the *MAT* loci (Heitman et al. 2013). The Ascomycota have one of these loci (*MAT1*) and its structure and gene content determines the mode of sexual reproduction (i.e., homothallism or heterothallism) in a particular fungus (Wilson et al. 2015; Wilken et al. 2017). For an individual to reproduce homothallically or to "self", the locus must contain both the *MAT1-1* and *MAT1-2* genes. Heterothallic species or "obligate outcrossers" require the interaction of individuals with either the *MAT1-1* or *MAT1-2* genes at their *MAT1* locus (Wilken et al. 2017). By contrast, Basidiomycota generally have tetrapolar mating systems that need to be heterozygotic at two unlinked *MAT* loci for sexual reproduction to occur (Heitman et al. 2013).

The structure and composition of the genes at the *MAT* loci of tree pathogens provide useful signatures to elucidate their mating system and thus to understand their reproductive biology. This is important, because many pathogens do not engage in sexual reproduction in the laboratory setting, and sometimes not even in the field. An example of such a fungus is the opportunist pine canker pathogen *Diplodia sapinea* (= *Sphaeropsis sapinea*, Figure 1), and it was used in one of the first studies to employ genome data specifically to elucidate the mating system of a pine pathogen in the Southern Hemisphere (Bihon et al. 2014). This ground-breaking study in the Botryosphaeriaceae showed that *D. sapinea* has a heterothallic

mating system, and later work showed that the related fungus, *Botryosphaeria dothidea*, is homothallic (Marsberg et al., 2016). Based on comparative genomics studies, it is now known that the members of this important fungal family includes both homothallic and heterothallic species in sexual reproduction (Nagel et al. 2018). In a similar way, the mating strategy of various other Southern Hemisphere pathogens of plantation tree species have been determined. For example, members of the Ceratocystidaceae and species of *Chrysoporthe* also have homothallic and heterothallic mating strategies (Simpson et al. 2018; Kanzi et al. 2019), while *T. destructans* is heterothallic (Havenga et al. 2020).

Genomes provide an important resource for the *in-silico* search for mating type regions in fungi. Primers flanking the regions or the mating type genes can then be developed and used to rapidly screen the mating types of a population. The presence or absence of mating type genes in the population provides valuable information about the outcrossing potential of the population. This in turn is important for developing disease management strategies because a high outcrossing potential allows for rapid genetic changes in the population resulting in a possible increase in the virulence of the pathogens, or potential loss of host resistance in currently planted tree clones due to the emergence of novel genotypes. This approach for screening a population has been used successfully to identify mating types in populations of *Teratosphaeria* leaf and shoot pathogen populations from different countries (Aylward et al. 2020; Havenga et al. 2020), as well as in *Lasiodiplodia* spp. and *Diplodia* spp. (Nagel et al. 2018).

Detailed genome comparisons can also provide insights into processes implicated in the evolution of fungal reproductive strategies. Comparative genomics of pathogenic fungi relevant to Southern Hemisphere plantation tree species have revealed unusual *MAT* locus

organisation, and have elucidated the mechanisms involved in the evolution of the locus. In a recent study, Kanzi et al. (2019) showed that the Eucalyptus stem canker pathogens *Ch. cubensis* and *Ch. deuterocubensis* are homothallic while their closely related sister species, *Ch. austroafricana*, is heterothallic. However, the *MAT1-2* idiomorph (or allelic version) of *Ch. austroafricana*, unlike typical *MAT1-2* idiomorphs, contained the *MAT1-1-2* gene and a truncated *MAT1-1-1* gene. The authors also found long terminal repeat (LTR) retrotransposons in the *MAT1* locus of *Ch. cubensis* and *Ch. deuterocubensis*, but not in *Ch. austroafricana*. In terms of phylogeny, *Ch. deuterocubensis* is basal to *Ch. cubensis* and *Ch. austroafricana* and harbours *MAT1-1-1, MAT1-1-2, MAT1-1-3* and *MAT1-2-1* genes at its *MAT1* locus. In contrast, *Ch. austroafricana* has only *MAT1-1-1* and *MAT1-2-1* genes in the respective *MAT1-1* and *MAT1-2* strains. Taken collectively, these data suggested that *Ch. austroafricana* became heterothallic through LTR-mediated loss of the *MAT1-1-3* and *MAT1-2-1* genes, from the homothallic ancestral locus, to form the *MAT1-1* and *MAT1-2* idiomorphs, respectively. The process likely also involved degradation of the *MAT1-1-1* gene, leaving the truncated version in *MAT1-2* idiomorph. Although not conclusive, the results of the study by Kanzi et al. (2019) and various other authors (Olive 1958; Geiser et al. 1998; Amselem et al. 2011; Duong et al. 2013), support the view that certain heterothallic fungi might have evolved from homothallic ancestors.

Genomics reveal mechanisms influencing pathogen biology

Comparative genomics involves the identification of similarities and differences between the genomes of organisms. This makes it possible to identify the genetic factors responsible for biological differences. Several studies on fungal pathogens of Southern Hemisphere plantation trees have made use of comparative genomics to shed light on carbon utilization

and fungal lifestyles, on fungal genome plasticity, and on the mechanisms involved in genome evolution and integrity.

Various groups of enzymes allow fungi access to plant-derived carbohydrates for survival in particular niches. One of these are the invertases (carbohydrate active enzymes of the glycoside hydrolase family 32: GH32) that are responsible for hydrolysing glycoside in saccharides. These enzymes enable fungi to gain access to and utilise sucrose produced by their hosts, and thereby enhance their growth (Parrent et al. 2009). Invertases may also play a role in fungal virulence (Schirawski 2015). Plant pathogens, in contrast to non-pathogens, have a higher copy number of invertase genes and consequently, there is an association between the number of invertase genes and the ability of fungi to cause disease (Parrent et al. 2009). Comparison of the genomes of *Ce. albifundus*, *Ce. manginecans*, *Ce. fimbriata* and other tree-infecting fungi in the Ceratocystidaceae which can be either pathogenic or saprobic, revealed that the gene copy number of GH32 has expanded in *Ce. albifundus* and other pathogenic species, while a loss of GH32 genes occurred in non-pathogenic members of this family (van der Nest et al. 2015). These findings showed how differences in the arsenal of carbohydrate active enzymes encoded by Ceratocystidaceae species are reflected in their different ecologies.

Comparative studies have revealed that the genomes of many pathogens are highly dynamic, often representing sub-genomic compartments that evolve at different rates (Dong et al. 2015). The slow-evolving compartment contains the core genes that are necessary for cellular and housekeeping functions, while the faster evolving compartments are typically rich in transposable elements (TEs) and genes that are involved in niche-specific or adaptive processes (reviewed in Stukenbrock 2013; Dong et al. 2015; Stajich 2017, Bertazzoni et al.

2018). As a result, these rapidly evolving compartments harbour genes that are not present in all members of a species (referred to as accessory genes), cause genome plasticity and promote adaptation to new hosts and environments (Stukenbrock 2013; Dong et al. 2015; Bertazzoni et al. 2018). These accessory genes or compartments may be interspersed with core genes where they are distributed across various chromosomes of a fungus. An appropriate example of this was recently reported for *Ce. albifundus* (van der Nest et al. 2019), which causes a wilt disease on plantation-grown *Acacia mearnsii* in several African countries (Wingfield et al. 1996; Roux et al. 2009). This study showed that the core sub-genomic compartment and or accessory compartment harbour genes that encode for products required for access to plant-derived nutrients, host-pathogen interactions, as well as signal transduction in sensing and responding to environmental conditions. These processes likely contributed to pathogenicity and host specialization of the fungus, and the genes encoding them represent relevant targets for the development of disease management strategies through genetic modification of the pathogens.

The accessory sub-genomic compartment may also be localized to specific genomic regions, such as sections of particular chromosomes and even entire chromosomes. The latter are usually dispensable for growth under certain conditions and are not necessarily present in all members of the species (Vlaardingerbroek et al. 2016; Bertazzoni et al. 2018). These types of chromosomes are particularly prevalent in the genus *Fusarium*, where they often encode genes and molecules needed for infection, disease development and virulence (Ma et al. 2010; Vlaardingerbroek et al. 2016; Waalwijk et al. 2018). The pitch canker pathogen, *F. circinatum*, also harbours at least one dispensable chromosome (van der Nest et al. 2014a; Waalwijk et al. 2018), which may explain why strains lacking it cause smaller lesions on *Pinus* species in virulence assays (Slinski et al. 2016).

Various recent studies have shown that horizontal gene transfer (HGT) plays a key role in the plasticity of fungal genomes. HGT involves the transfer of genetic material between species (Syvanen 1985, Doolittle 1999) and for many years was thought to occur only in prokaryotic organisms. However, comparative genomics has revealed that HGT can occur between prokaryotes and eukaryotes as well as among eukaryotes (Soanes and Richards 2014). In fungal pathogens, HGT has been shown to alter the genomic landscape and give rise to functional novelty, which facilitates adaption to new ecological niches (Soanes and Richards 2014; Dhillon et al. 2015). For example, a recent study of *F. circinatum* genomes showed that a 12 kilo bases genetic insert, likely acquired via multiple HGT events (van Wyk et al. 2018), encodes functions that allow the fungus faster growth at higher temperatures relative to closely related species (De Vos et al. 2011; van Wyk et al. 2018).

Other mechanisms that enhance genome plasticity involves mobile genetic elements (MGEs) such as TEs and repeated sequences. MGEs mediate genetic variation by facilitating inversions, duplication, deletions, ectotopic recombination and double stranded breaks in genomes (Gray 2000). In fungal pathogens, they are a source of genetic variation required for adaptation in the evolutionary "arms race" between pathogen and host (Rouxel et al. 2011; Möller and Stukenbrock 2017; Mat Razali et al. 2019). Comparative genomics has also revealed that these elements are major drivers of genome evolution and they impact on pathogenicity, host range and changes in the biology of fungal pathogens (see reviews by Daboussi and Capy 2003; Mat Razali et al. 2019). In the Ceratocystidaceae, for example, MGEs shaped the evolution of the GH32 gene family (van der Nest et al. 2015). Compared to other genera in this family of fungi, *Ceratocystis* species have many more MGEs in their genomes (Fourie et al 2019) and they have most likely played an important role in the evolution of the *MAT1* locus and the chromosome that harbours the locus in these fungi

(Simpson et al. 2018). Another interesting example is found in the myrtle rust pathogen *A. psidii*, which has been shown to be unusually large for a fungal genome (Tan et al. 2014; McTaggart et al. 2018). Some of the increase in genome size has been attributed to an enrichment of TEs (Tan et al. 2014). The results of the above studies suggest that TEs have played a significant role in adaptation to environment and hosts in these fungi.

Many fungal genomes limit the spread and distribution of MGEs by employing "genome defence mechanisms", but these mechanisms themselves may also drive divergence and allow adaptation to particular niches. One such mechanism that has been more widely studied in fungi is the repeat induced point (RIP) mutation pathway that occurs only in fungi where it plays an important role in genome evolution and integrity (Clutterbuck 2011). RIP counteracts the deleterious effect of MGEs by targeting duplicated sequences and inducing C:G to T:A transitional mutations in the repeat regions, thereby disrupting MGEs and in the cases of TEs potentially deactivating them (for detailed description see Hane et al. 2015 and references therein). For example, *in-silico* analysis of *Ceratocystis* genome sequences with software such as the RIPCAL suite (Hane and Oliver 2008; Hane and Oliver 2010) showed that *Fot5* TEs were likely rendered inactive (van der Nest et al. 2015). However, RIP may also facilitate modification of genes because the process is somewhat leaky in that it also acts on regions that neighbour MGEs, and RIP may act on any repeated sequences, whether they are coding or not (Rouxel et al. 2011; Daverdin et al. 2012; Hane et al. 2015; Gladyshev 2017; Lelwala et al. 2019). Accordingly, RIP is now known to be capable of driving diversification and development of genes related to pathogenicity (e.g. Rouxel et al. 2011; Daverdin et al. 2012; Lelwala et al. 2019), thereby altering the pathogen-host interaction.

Implementation of the recently developed software "The RIPper" (van Wyk et al. 2019a) will significantly improve our capacity to study the impact of RIP on fungal genomes and the biological consequences that this might have on fungal pathogens. "The RIPper," is a user-friendly set of web-based tools for genome-wide analysis of RIP in the Ascomycota. Making use of this tool, together with other data has shown that *F. circinatum* and closely related *Fusarium* species harbour molecular hallmarks of RIP, and that their genomes are impacted by this process (van Wyk et al. 2019b). In these fungi, regions affected by RIP were gene-sparse and flanked by numerous pseudogenes flanking the regions. Here, RIP appears to drive the independent divergence of chromosomes and alters chromosome architecture, suggesting that it has played a key role in the evolution and biology of *F. circinatum* and closely related *Fusarium* species. Integration of RIP information with population genomic studies and studies designed to compare the functions encoded across genomes will undoubtedly reveal the significant role that this genome defence mechanism has played during the evolution of fungal pathogens and their genomes.

Future prospects and conclusions

Genomics is a rapidly expanding and evolving field of study adding to the arsenal of tools available to forest pathologists seeking to understand and manage tree disease problems. Population genomics is one of the areas in this field that provides the most exciting short-term future prospects. Rather than sequencing one or a few isolates of a species, multiple isolates across populations of a species can be sequenced and compared for SNPs, gene sequence and content differences, as well as variation in genome architecture. This enables researchers working on fungal tree pathogens to develop considerably more robust descriptions of pathogens. Also, to better understand the adaption of these pathogens to their environment than has hitherto been possible.

The advances made in recent genomics research are strongly dependent on the development of new sequencing technologies. These have enabled researchers to produce genome sequences, which are more complete and of higher quality than was possible in the past. These technological advances make it possible to improve the outcomes of earlier genome sequencing efforts, which often yielded highly fragmented sequences. Third generation or single molecule sequencing can now be used to sequence full chromosomes as these technologies can generate longer (more than 100 kilobase pair) sequences (Bleidorn 2016, Wolters et al. 2018). Furthermore, these next-generation technologies can be used to sequence the genomes of unculturable pathogens (Ahrendt et al. 2018, McTaggart et al. 2018), they are useful in metagenomics studies aimed at describing the community of beneficial and non-beneficial organisms that inhabit a tree host (Kemler et al. 2013; Nilsson et al. 2019), studying the ecology of fungi in forest soils (Lance et al. 2020) and screening for exotic invasive pathogens (Tremblay et al. 2018).

Association mapping, such as quantitative trait locus mapping (QTL) and genome-wide association studies (GWAS), provide important opportunities to identify loci influencing complex heritable traits (Pritchard et al. 2000; Purcell et al. 2003). These techniques are emerging in the field of tree pathology, as genetic variation can be measured across the genome (Genissel et al. 2017; Plissonneau et al. 2017). For example, seven genomic loci have been identified in the important conifer root pathogens *Heterbasidium annosum* that are involved in virulence (Dalman et al. 2013). These loci encode genes known for virulence in other fungal pathogens, as well as novel candidate virulence genes.

Functional characterization of genes is necessary to confirm the role of genetic features identified *in-silico*. Strategies employed in functional characterization include targeted gene

knockout technologies and *in planta* expression assays (Weld et al. 2006). A more recent technology is the CRISPR-Cas9 system where sequences in the genome can be altered with high precision (Ran et al. 2013). This is a rapidly emerging technology that is certain to be applied in studies seeking to manage the negative impact of tree pathogens including those affecting plantation forestry based on non-native species in the Southern Hemisphere.

Genomics as a technology provides a crucial component of systems biology studies. In such studies, the focus is not on single genes or processes, but on the behaviour and relationships of all elements in the biological system (Ideker et al. 2001; Kitano 2002a; Kitano 2002b). Systems biology integrates data generated from genome sequencing, RNA expression studies, gene regulatory networks, gene interaction studies and metabolomics into a model and can be graphically displayed (Ideker et al. 2001). Such research will enable tree pathologists to achieve an integrated view of how pathogens respond to their environments and hosts. When incorporated with the systems biology of the host, such studies would lead to important insights into pathosystems, which in turn could fortify disease management strategies.

The many technologies linked to our ability to sequence fungal genomes have already made possible considerable progress in understanding plant pathogens including those of trees. While genomics studies on tree pathogens has understandably lagged behind those on pathogens of food crops, this situation is changing rapidly and considerable work has already been conducted on tree crops grown intensively in plantations of the Southern Hemisphere. Plantation forestry based on non-native tree species in the tropics and Southern Hemisphere has been very severely affected by diseases caused by fungi (Wingfield et al. 2001; Wingfield et al. 2015c). This will continue in the future as new pathogens are increasingly moved

globally through trade in forest products (Burgess and Wingfield 2016; Santini et al. 2018; Sikes et al. 2018). The sustainability of these plantation industries will increasingly rely on the application of new technologies (Wingfield et al. 2013; Wingfield et al. 2015c) to deal with these threats.

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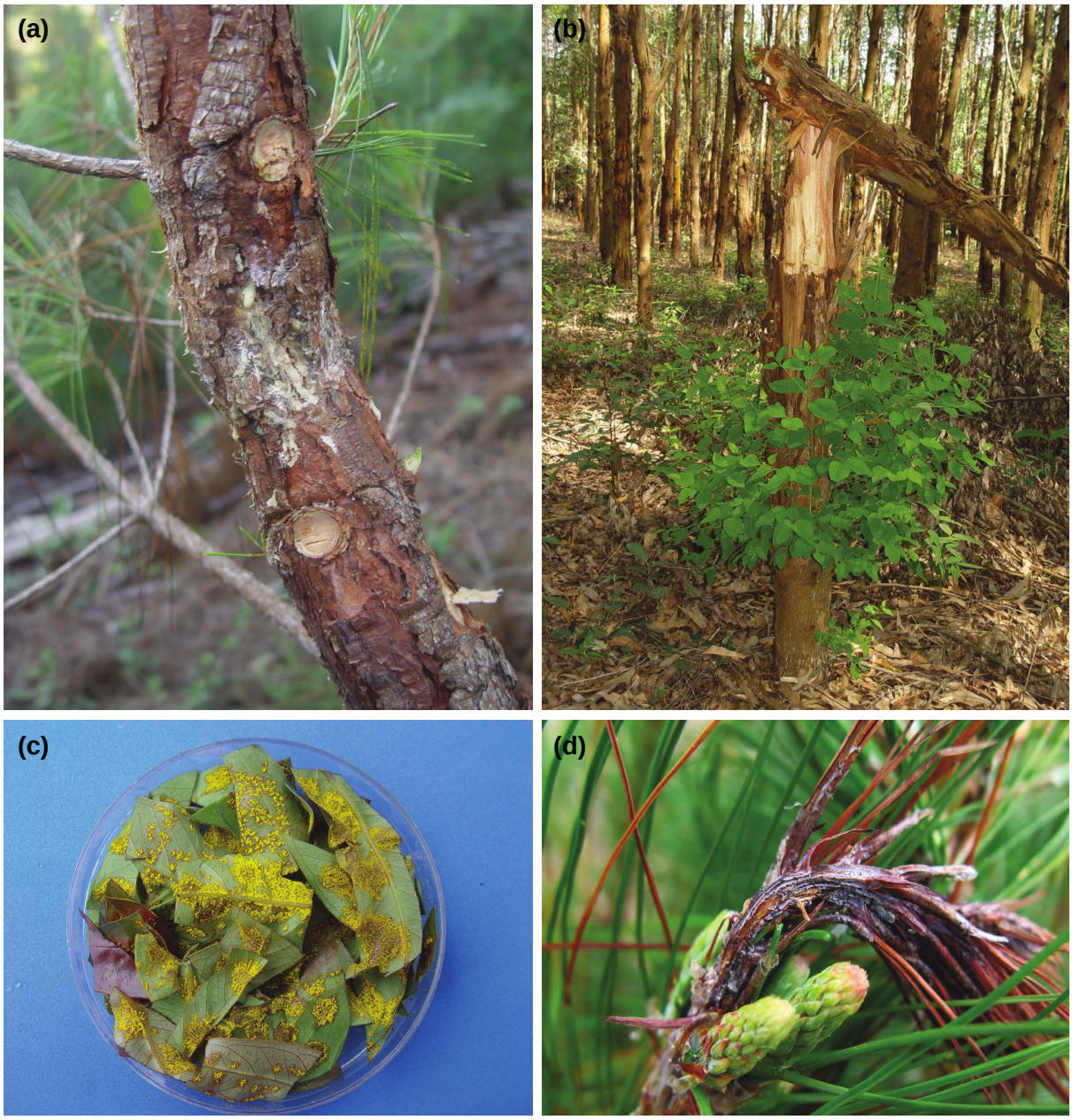

Figure 1: Signs and symptoms associated with infections by selected pathogens of *Pinus* and *Eucalyptus* grown in plantations of the Southern Hemisphere. (a) Pitch canker caused by *F. circinatum* on *P. patula* (arrow indicates pitch exudation in response to infection). (b) Chryphonectria canker on base of *Eucalyptus* sp. stem caused by *Chrysoporthe cubensis*. (c) *Eucalyptus* leaves infected by *Austropuccinia psidii* (arrow shows uredosori and yellow spore masses produced by the fungus). (d) Shoot infection on *P. patula* caused by *Diplodia sapinea*.

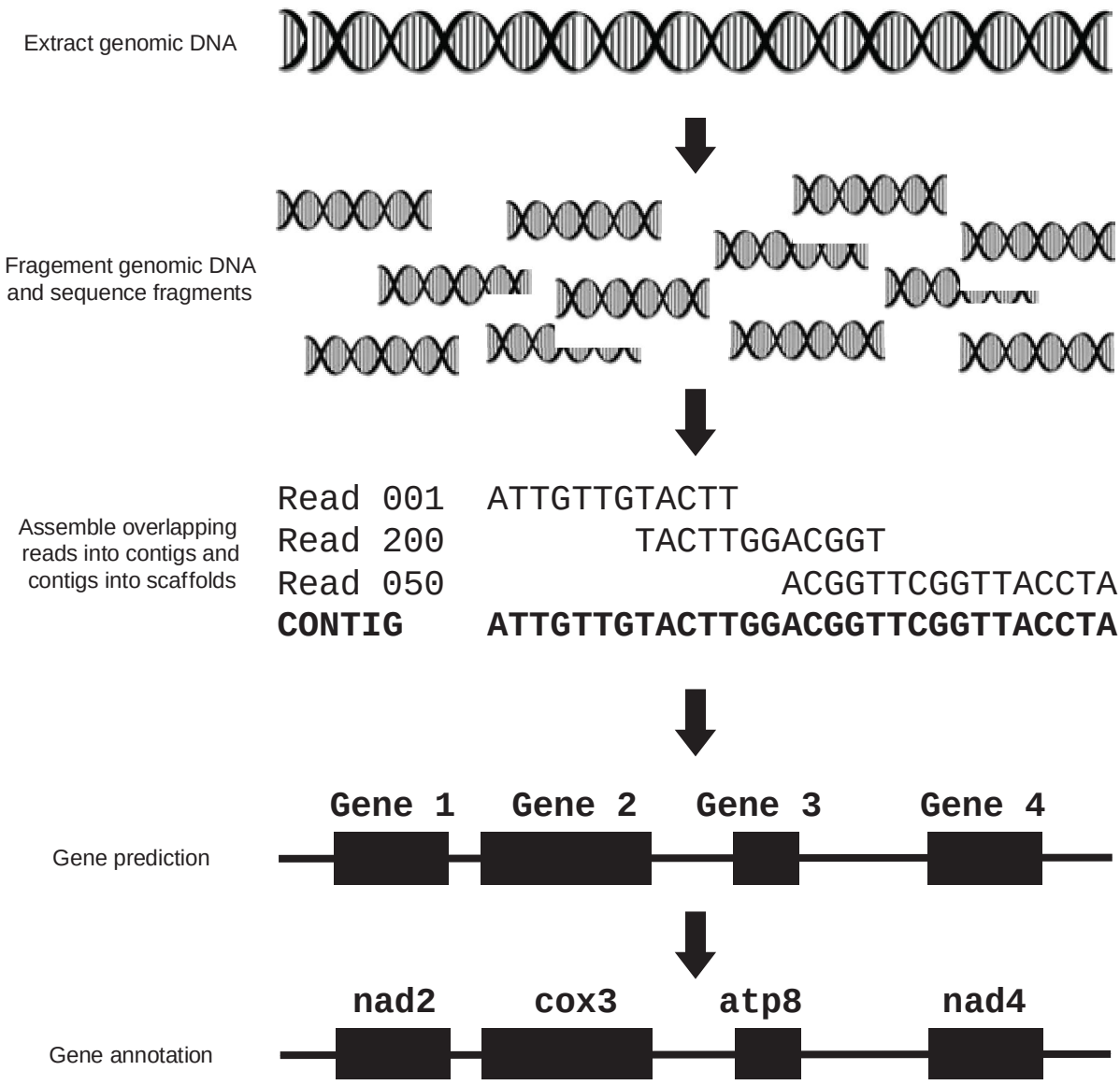

Figure 2: Overview of the steps taken during a whole genome sequencing project.

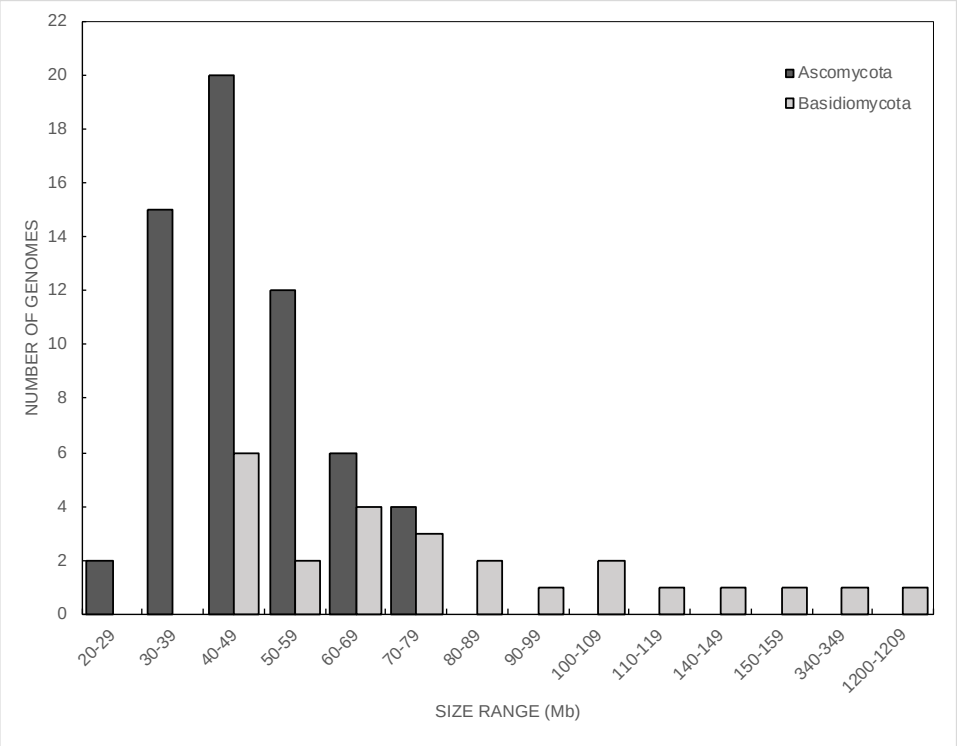

Figure 3: Number of genomes within different genome size ranges for Ascomycota and Basidiomycota.

Table 1: List of fungal forest pathogens for which genome sequences are publicly available (Species names in bold are fungal tree pathogens relevant to Southern Hemisphere forestry).

Species	Host	Disease type[a]	Year sequenced	Genome size (Mb)	Type of article[b]	Reference
			Phylum Ascomycota			
Atropellis piniphila	*Pinus* spp.	Canker	2016	43.94	Unpublished	JGI project proposal ID: 1999
Botryosphaeria dothidea	Broad host range	Dieback of woody hosts	2016	43.50	Research article	Marsberg et al. (2017)
Bretziella fagacearum	*Quercus* spp.	Oak wilt	2016	26.74	Genome announcement	Wingfield et al. (2016b)
Calonectria aciculata	*Eucalyptus* spp.	Leaf blight	2019	61.60	Genome announcement	Liu et al. (2019)
Calonectria crousiana	*Eucalyptus* spp.	Leaf blight	2019	58.10	Genome announcement	Liu et al. (2019)

Table 1 (continued)

Species	Host	Disease type[a]	Year sequenced	Genome size (Mb)	Type of article[b]	Reference
Calonectria fujianensis	*Eucalyptus* spp.	Leaf blight	2019	61.5	Genome announcement	Liu et al. (2019)
Calonectria henricotiae	*Buxus* spp., *Sarcococca* spp., *Pachysandra* spp.	Leaf blight	2016	49.05	Research article	Malapi-Wight et al. (2016)
Calonectria pseudonaviculata	*Buxus* spp. *Sarcococca* spp. *Pachysandra* spp.	Leaf blight	2016	54.97	Research article	Malapi-Wight et al. (2016)
Calonectria pseudoreteaudii	*Eucalyptus* spp.	Leaf blight	2018	63.70	Research article	Ye et al. (2018)
Celoporthe dispersa	*Eucalyptus* spp.	Canker	2019	40.00	Genome announcement	Liu et al. (2019)
Ceratocystis albifundus	*Acacia mearnsii*	Wilt	2014	27.15	Genome announcement	van der Nest et al. (2014b)
Ceratocystis eucalypticola	*Eucalyptus* spp.	Wilt	2015	31.26	Genome announcement	Wingfield et al. (2015b)
Ceratocystis fimbriata	*Eucalyptus* spp.	Wilt	2019	29.41	Research article	Santos et al. (2020)

Table 1 (continued)

Species	Host	Disease type[a]	Year sequenced	Genome size (Mb)	Type of article[b]	Reference
Ceratocystis harringtonii	*Populus* spp.	Canker	2016	31.06	Genome announcement	Wingfield et al. (2016b)
Ceratocystis manginecans	*Acacia mangium*	Wilt	2014	31.70	Genome announcement	van der Nest et al. (2014b)
Ceratocystis smalleyi	*Carya* spp.	Canker	2018	27.31	Genome announcement	Wingfield et al. (2018a)
Chrysoporthe austroafricana	*Eucalyptus* spp.	Canker	2015	44.67	Genome announcement	Wingfield et al. (2015a)
Chrysoporthe cubensis	*Eucalyptus* spp.	Canker	2015	42.62	Genome announcement	Wingfield et al. (2015b)
Chrysoporthe deuterocubensis	*Eucalyptus* spp.	Chrysoporthe canker	2015	43.97	Genome announcement	Wingfield et al. (2015b)
Corinectria fuckeliana	*Pinus* spp., *Fagus* spp., *Betula* spp., *Abies* spp., *Picea* spp., *Ulmus* spp.	Canker	2019	39.00	Genome announcement	Salgado-Salazar and Crouch (2019)

Table 1 (continued)

Species	Host	Disease type[a]	Year sequenced	Genome size (Mb)	Type of article[b]	Reference
Corynespora cassiicola	*Hevea brasiliensis*	Leaf necrosis	2018	44.85	Research article	Lopez et al. (2018)
Cryphonectria parasitica	*Castanea* spp.	Canker	2010	43.90	Unpublished	JGI project proposal ID: 2099
Cryptodiaporthe populea	*Populus* spp.	Canker	2016	56.94	Unpublished	JGI project proposal ID: 1999
Cytospora chrysosperma	*Populus* spp.	Canker	2016	36.55	Unpublished	JGI project proposal ID: 1999
Davidsoniella virescens	*Acer saccharum*	Dieback	2015	33.65	Genome announcement	Wingfield et al. (2015b)
Diplodia sapinea	*Pinus* spp.	Tip blight, canker, dieback	2014	36.97	Research article	van der Nest et al. (2014b)
Diplodia scrobiculata	*Pinus* spp.	Shoot blight	2015	35.85	Genome announcement	Wingfield et al. (2015a)
Dothistroma septosporum	*Pinus* spp.	Needle blight	2012	30.21	Research article	De Wit et al. 2012
Elytroderma deformans	*Pinus* spp.	Needle cast	2017	50.48	Unpublished	JGI project proposal ID: 1999

Table 1 (continued)

Species	Host	Disease type[a]	Year sequenced	Genome size (Mb)	Type of article[b]	Reference
Endoconidiophora laricicola	*Picea abies*	Dieback	2016	32.78	Genome announcement	Wingfield et al. (2016a)
Endoconidiophora polonica	*Larix decidua* *Picea abies*	Blue stain	2016	32.46	Genome announcement	Wingfield et al. (2016a)
Entoleuca mammata	*Populus* spp.	Canker	2016	47.20	Unpublished	JGI project proposal ID: 1999
Fusarium circinatum	*Pinus* spp.	Canker	2009	43.92	Research article	Wingfield et al. (2012)
Fusarium euwallaceae	Broad host range	Dieback	2017	48.27	Genome announcement	Ibarra-Laclette et al. (2017)
Fusarium pininemorale	*Pinus* spp.	Canker	2017	47.78	Genome announcement	Wingfield et al. (2017)
Geosmithia morbida	*Juglans* spp.	Canker	2016	26.50	Research article	Schuelke et al. (2016)
Gremmeniella abietina	*Pinus* spp., *Abies* spp., *Picea* spp.	Canker	2016	38.81	Unpublished	JGI project proposal ID: 1999
Grosmannia clavigera	*Pinus* spp.	Blue stain	2009	30.00	Research article	DiGuistini et al. (2011)

Species	Host	Disease type[a]	Year sequenced	Genome size (Mb)	Type of article[b]	Reference
Huntiella omanensis	*Mangifera indica*	Sudden decline	2014	31.50	Genome announcement	van der Nest et al. (2014a)
Hymenoscyphus fraxineus	*Fraxinus* spp.	Dieback	2017	62.28	Research article	Stenlid et al. (2017)
Lecanosticta acicola	*Pinus* spp.	Needle blight	2017	28.42	Unpublished	NCBI accession AWYC00000000
Leptographium longiclavatum	*Pinus* spp.	Blue stain	2014	28.90	Research article	Ojeda et al. (2014)
Leptographium procerum	*Pinus* spp.	Root decline	2014	28.6	Genome announcement	van der Nest et al. (2014a)
Marssonina brunnea	*Populus* spp.	Leaf spot	2012	52.00	Research article	Zhu et al. (2012)

Table 1 (continued)

Species	Host	Disease type[a]	Year sequenced	Genome size (Mb)	Type of article[b]	Reference
Neonectria punicea	*Pinus* spp., *Fagus* spp., *Betula* spp., *Abies* spp., *Picea* spp., *Ulmus* spp.	Canker	2019	43.20	Genome announcement	Salgado-Salazar and Crouch (2019)
Ophiognomonia clavigignenti-juglandacearum	*Juglans cinerea*	Canker and branch dieback	2019	52.60	Research article	Wu et al. (2019)
Ophiostoma bicolor	*Picea* spp.	Blue stain	2017	25.30	Research article	Lah et al. (2017)
Ophiostoma novo-ulmi	*Ulmus* spp.	Wilt	2013	31.78	Research article	Cuomo et al. (2007)
Ophiostoma ulmi	*Ulmus* spp.	Wilt	2013	31.50	Research article	Khoshraftar et al. (2013)
Raffaelea lauricola	*Persea* spp.	Wilt	2019	34.60	Research article	Vanderpool et al. (2018)
Raffaelea quercivora	*Quercus* spp.	Wilt	2017	26.41	Research article	Vanderpool et al. (2018)

Table 1 (continued)

Species	Host	Disease type[a]	Year sequenced	Genome size (Mb)	Type of article[b]	Reference
Raffaelea quercus-mongolicae	*Quercus* spp.	Wilt	2017	27.00	Genome announcement	Jeon et al. (2017)
Septoria musiva	*Populus* spp.	Leaf spot and canker	2012	29.35	Research article	Ohm et al. (2012)
Septoria populicola	*Populus* spp.	Leaf spot	2012	33.19	Research article	Ohm et al. (2012)
Taphrina betulina	*Betula* spp.	Host tissue deformities	2019	12.50	Genome announcement	Heneghan et al. (2019)
Taphrina populi-salicis	*Populus* spp.	Leaf blister	2017	13.21	Unpublished	JGI project proposal ID: 1999
Teratosphaeria destructans	*Eucalyptus* spp.	Leaf and shoot blight	2018	32.32	Genome announcement	Wingfield et al. (2018b)
Teratosphaeria gauchensis	*Eucalyptus* spp.	Canker	2019	30.27	Genome announcement	Wingfield et al. (2019)
Teratosphaeria zuluensis	*Eucalyptus* spp.	Canker	2019	28.71	Genome announcement	Wingfield et al. (2019)

Table 1 (continued)

Species	Host	Disease type[a]	Year sequenced	Genome size (Mb)	Type of article[b]	Reference
			Phylum: Basidiomycota			
Armillaria borealis	Broad host range	Root rot	2018	71.69	Unpublished	JGI project proposal ID: 1974
Armillaria ectypa	Bryophytes	Saprophyte	2019	40.60	Unpublished	JGI project proposal ID: 1974
Armillaria fuscipes	Broad host range	Root rot	2016	53.00	Genome announcement	Wingfield et al. (2016a)
Armillaria gallica	Broad host range	Root rot	2017	85.34	Research article	Sipos et al. (2017)
Armillaria luteobubalina	*Eucalyptus* spp.	Root rot	2019	97.11	Unpublished	JGI project proposal ID: 1974
Armillaria mellea	Broad host range	Root rot	2013	58.35	Research article	Collins et al. (2013)
Armillaria nabsnona	Broad host range	Root rot	2018	62.72	Unpublished	JGI project proposal ID: 1974
Armillaria ostoyae	Broad host range	Root rot	2017	60.11	Research article	Sipos et al. (2017)

Table 1 (continued)

Species	Host	Disease type[a]	Year sequenced	Genome size (Mb)	Type of article[b]	Reference
Armillaria solidipes	Broad host range	Root rot	2017	58.01	Research article	Sipos et al. (2017)
Armillaria tabescens	Broad host range	Root rot	2019	74.88	Unpublished	JGI project proposal ID: 1974
Austropuccinia psidii	Species of Myrtaceae	Foliar rust, stem blight	2018	1 200.00	Research article	McTaggart et al. (2018)
Chondrostereum purpureum	Species of Rosacea	White rot	2019	41.20	Research article	Reina et al. (2019)
Coniferiporia sulphurascens	Species of Pinaceae	Laminated root rot	2017	39.34	Research article	Chung et al. (2017)
Cronartium comandrae	*Pinus* spp.	Canker	2013	68.60	Unpublished	NCBI accession AUZW00000000.1
Cronartium quercuum f.sp. *fusiforme*	*Pinus* spp., *Quercus* spp.	Gall and canker	2014	76.57	Research article	Pendleton et al. (2014)
Cronartium ribicola	*Pinus* spp.	Canker	2013	94.33	Unpublished	NCBI accession AWVX00000000

Table 1 (continued)

Species	Host	Disease type[a]	Year sequenced	Genome size (Mb)	Type of article[b]	Reference
Ganoderma spp.	Broad host range	Root rot	2013	39.52	Research article	Binder et al. (2013)
Heterobasidion annosum	Broad host range	Root rot	2017	33.10	Research article	Choi et al. (2017)
Heterobasidion irregulare	Broad host range	Root rot	2012	33.65	Research article	Olson et al. (2012)
Heterobasidion parviporum	Broad host range	Root rot	2018	37.76	Research article	Zeng et al. (2018)
Melampsora allii-populina	*Populus* spp.	Foliar rust	2015	335.73	Unpublished	JGI project proposal ID: 662
Melampsora larici-populina	*Populus* spp.	Foliar rust	2011	101.10	Research article	Duplessis et al. (2011)
Melampsora medusae f. sp. *deltoidis*	*Populus* spp.	Foliar rust	2017	139.73	Unpublished	JGI project proposal ID: 1999
Melampsora medusae f. sp. *tremuloidae*	*Populus* spp.	Foliar rust	2017	145.19	Unpublished	JGI project proposal ID: 1999

Table 1 (continued)

Species	Host	Disease type[a]	Year sequenced	Genome size (Mb)	Type of article[b]	Reference
Phellinus noxius	Broad host range	Root rot	2017	31.60	Research article	Chung et al. (2017)
Quambalaria eucalypti	*Eucalyptus* spp.	Shoot and leaf dieback	2018	23.50	Genome announcement	Wingfield et al. (2018b)

[a] Disease type based on the descriptions of the major symptoms.

[b] Genome announcement is a short article describing the sequencing and assembly of the genome. A research article is more descriptive, and usually more analysis has been performed to describe the genomic features. In the case of no publication, the genome sequence is available in public databases, but no article or announcement has been published by March 2020. For the latter, relevant project or sequence accessions are provided in the Reference column (JGI = Joint Genome Institute, US Department of Energy, Lawrence Berkeley National Laboratory, California; NCBI = National Center for Biotechnology Information, US National Library of Medicine, Maryland).

Table 2: Genome size characteristics of fungal forest tree pathogens.

Phylum	**Average (Mb)**	**Median (Mb)**	**Smallest size (Mb)**	**Largest size (Mb)**
Ascomycota	37.93	34.60	12.50	63.70
Basidiomycota	120.48	61.42	31.60	1200.00
All genomes	62.88	40.30	12.50	1200.00