REVIEW

Recent Fungal Diseases of Crop Plants: Is Lateral Gene Transfer a Common Theme?

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Submitted 17 September 2007. Accepted 26 October 2007.

A cursory glance at old textbooks of plant pathology reveals that the diseases which are the current scourge of agriculture in many parts of the world are a different set from those that were prominent 50 or 100 years ago. Why have these new diseases arisen? The traditional explanations subscribe to the “nature abhors a vacuum” principle—that control of one disease creates the condition for the emergence of a replacement—but does little to explain why the new pathogen succeeds. The emergence of a new disease requires a series of conditions and steps, including the enhanced fecundity of the new pathogen, enhanced survival from season to season, and spread around the world. Recently, evidence was obtained that wheat tan spot emerged through a lateral gene transfer event some time prior to 1941. Although there have been sporadic and persistent reports of lateral gene transfer between and into fungal plant pathogens, most examples have been dismissed through incomplete evidence. The completion of whole genome sequences of an increasing number of fungal pathogens no longer allows such proposed cases of lateral gene transfer to be dismissed so easily. How frequent are lateral gene transfers involving fungal plant pathogens, and can this process explain the emergence of many of the new diseases of the recent past? Many of the apparently new diseases are dependant on the expression of host-specific toxins. These are enigmatic molecules whose action requires the presence of plant genes with products that specifically encode sensitivity to the toxin and susceptibility to the disease. It is also notable that many new diseases belong to the fungal taxon dothideomycetes. This review explores the coincidence of new diseases, interspecific gene transfer, host-specific toxins, and the dothideomycete class.

The recent emergence of many new diseases.

Plant pathology is a mature discipline with references to recognizable diseases dating back to antiquity (Agrios 2005). The identification of pathogens depended on the development of pure culture methods and microscopy and (except for some classes of viruses and phytoplasma) had become a thorough, routine, and recognizably modern activity by the 1920s. We can be reasonably certain, therefore, that fungal diseases recognized after this somewhat arbitrary date were not missed earlier through lack of technical facility. The spectrum of diseases has changed quite dramatically over the last few decades (Table 1). The successive editions of the seminal textbook

Plant Pathology by G. N. Agrios dating from 1969 to 2005 naturally highlight the diseases that were the major preoccupations of current pathologists and on which current research was focused. Taking as an example the diseases of wheat, it is clear that while several diseases remained relevant or declined during the period of intensive industrial agriculture, others emerged either from low to high importance or from absolute obscurity to significant annual relevance. Confusions in nomenclature are a persistent problem and these have combined with an understandable tendency of field pathologists to ignore minor diseases to make the details of the pattern of emergence of disease highly obscure. However, there is no doubt that the spectrum of diseases is changing and that many new diseases have appeared.

The main cereal fungal pathogens to have fallen from prominence are the Ustilaginomycete group of smuts and bunts. They were regarded as the main pathogens in France in the 1750s, causing 50% crop losses (Prevost 1807). Fungicides applied to leaf and especially to seed have all but eradicated these pathogens. Prominent new cereal diseases include those caused by the dothideomycete genera *Helminthosporium* (syn. *Drechslera* and *Cochliobolus*) and *Pyrenophora* (Table 2).

Why do new diseases emerge?

It is easy to suggest plausible reasons why new diseases might emerge. The most obvious is that the host crop is new or being grown on a much wider scale than before. Such large-scale plantings, even if not monocultures, tend to be favorable for disease. Many new crop species, such as canola, avocado, and kiwi fruit, were introduced in the last few decades and essentially all crops have been grown in larger fields and more intensively than in previous decades. The second is that the crop is now being grown in areas proximal to a disease source; examples include coffee rust, rubber rust, and sudden oak death (Kluza et al. 2007; Money 2006). Third, cultivars of crops may be selected for one reason but inadvertently are susceptible to the pathogen; the best known cases of this phenomenon are Victoria blight of oat and southern corn leaf blight (further details below). Fourth, cultural conditions may favor a new disease; reduced- or zero-tillage practices, in which (fungal-infected) stubble is retained between seasons, have been widely linked to the rise of various necrotrophic diseases (Bailey et al. 2001; Bockus and Shroyer 1998; Eyal 1999; Gilbert and Woods 2001; Krupinsky and Tanaka 2001).

These reasons refer to changes in the crop and the way it is grown. Pathogens also change. We are familiar with the concept of mutation of avirulence genes leading to the defeat of resistant cultivars. Point mutations (both missense and nonsense) and gene deletions have been implicated in the mutation

of avirulence genes in fungal pathogen races (Thomma et al. 2005). Evidence that new a disease arose by *acquisition* of a new gene function recently has been obtained (Friesen et al. 2006). Tan spot of wheat is believed to have emerged as a result of lateral gene transfer of the gene for ToxA from the related wheat pathogen *Stagonospora nodorum*. Such observations add significant credence to the possibility that lateral gene transfer might be a common event and, thus, could be the cause of many of the significant new diseases observed in the last century. The goal of this review is to highlight some recent diseases and discuss whether their emergence might be due to lateral gene transfer.

Lateral gene transfer in fungi.

The terms horizontal, interspecific, and lateral gene transfer appear to be used interchangeably to refer to the movement and fixation of DNA sequences between species. Each of these terms refers to the transfer of a gene (a nucleic acid sequence) from one isolate to another by processes other than the routine asexual cell division, sexual processes involving fusion of gametes, and parasexual exchange between compatible isolates. Moreover each term—horizontal, lateral, and, particularly, interspecific—seeks to distinguish gene transfer events *between* species from those *within* species. In view of the fact that the definition of the term “species” for fungi is still controversial and perhaps ultimately unsustainable, we prefer to use the term lateral because it sidesteps this issue.

Lateral transfer events are well established between prokaryotic species and are well established from prokaryotic sources to eukaryotic recipients (Lacroix et al. 2006; Roger and Hug 2006; Wenzl et al. 2005; Xu 2006). Examples of lateral gene transfer between eukaryotes are much less well established, though many claims have been made (Andersson 2005).

Evidence for lateral transfer comes in many forms of differing force. The most common evidence is that genes isolated from the proposed recipient are absent in closely related species but present in more distantly related species. As a result, phylogenetic trees based on this gene are considered incongruent and,

thus, incompatible with the phylogenetic tree of the species in question. Lateral gene transfer then is postulated. The difficulties with this argument are multiple. First, the orthologous gene may not have been discovered in related organisms; only when all the appropriate species have been completely sequenced is this objection finally overcome. Even when full genomes are available, there are other explanations. The genes may not be recognizably similar; they may evolve at too high a rate for orthology relationships to be obvious. Multiple copies of genes may obscure orthology and create efficient mutational opportunities through translocations and gene shuffling. Selective loss of genes in one or more lineage can create incongruent gene and organism trees. Distinguishing between the lateral gain and lineage-specific loss hypotheses certainly will become easier as more genome sequences become available, but always will be problematic.

A second source of evidence is that certain isolates of a species may lack the gene. This would be the case of a recently acquired gene or a gene that had marginal or equivocal evolutionary benefits. Essential genes, by definition, would be present in all isolates. Strongly beneficial genes would be expected to become fixed in the entire population in a time scale related to the fecundity benefit conferred by the gene, the mode of dispersal, and eco-geographical considerations. Equivocal genes may be beneficial in some circumstances and deleterious in others consistent with a patchy distribution; an example might be the fungal genes with dual avirulence and necrosis-inducing functions (Schurch et al. 2004). As for the incongruent gene/species tree argument, the patchy-within-species argument cannot be regarded as conclusive evidence on its own.

A third level of evidence equates to the founder effect used to model movements of populations across geographic regions. Populations that move to areas previously uncolonized by the species nearly always will contain only a subset of the genetic variation present in the home range. Analysis of genetic variation was the critical argument in favor of lateral transfer of *ToxA* (Friesen et al. 2006; Stukenbrock and McDonald 2007). Here, the level of variation of the gene was much greater in

Table 1. Wheat diseases mentioned in successive editions of *Plant Pathology* by Agrios^a

Edition 1, 1969	Edition 2, 1978	Edition 3, 1987	Edition 5, 2005
Bunt	Covered smut	Scab	Crown and root rot
Dwarf bunt	Dwarf bunt	Stem rust	Dwarf Bunt
Loose smut	Helminthosporium leaf spot	Take-all	Ergot
Powdery mildew	Scab	Stripe rust	Fusarium scab
Root rot	Stem rust	Helminthosporium leaf spot	Leaf and glume blotch
Stem rust	Take-all	Leaf and glume blotch	Head blight or scab
			Powdery mildew
			Leaf rust
			Root rot
			Smut
			Stem rust
			Take-all
			Tan spot

^a Diseases in bold are discussed in the text.

Table 2. Some twentieth-century fungal plant diseases

Crop	Disease	Pathogen	Year	Reference
Tomato, Japanese pear, strawberry, tangerine, etc.	Alternaria stem canker	<i>Alternaria alternata</i>	1920s	Kodama, personal communication
Wheat	Tan (yellow leaf) spot	<i>Pyrenophora tritici-repentis</i>	1941	Friesen et al. 2006
Oat	Victoria blight	<i>Cochliobolus victoriae</i>	1946	Meehan and Murphy 1947
Gladiolus	Gladiolus flower blight	<i>Curvularia lunata</i>	1947	Horst 1979
Chrysanthemum	Chrysanthemum Petal blight	<i>Hersonilia perplexans</i>	1951	Horst 1979
Apricot	Cytosporina dieback	<i>Eutypa armeniaca</i>	1957	Carter 1957
Corn (maize)	Southern Corn Leaf Blight	<i>Cochliobolus heterostrophus</i>	1970	Ullstrup 1972
Corn	Yellow leaf disease	<i>Mycosphaerella zeae-maydis</i>	1971	Army and Nelson 1971

S. nodorum than in *Pyrenophora tritici-repentis*, consistent with a recent acquisition by the latter.

Codon usage in transferred genes could be expected to reflect the bias in the donor organism rather than the recipient. Such an analysis is more powerful when comparing distantly related organisms with strongly contrasting codon usage patterns (Garcia-Vallve et al. 2000) but also has been used to support cases involving gene transfers from a source of only moderate difference in bias (Liu et al. 2003; Temporini and VanEtten 2004).

Clustering of genes with related functions in host genomes is a common phenomenon in filamentous fungi, especially in relation to secondary metabolite genes. Such clustering has been considered to be prime facie evidence of lateral gene transfer (Walton 2000). However, it is also possible that the genes are clustered to permit co-regulation, as is believed to be the case, for example, with the quinate cluster found in many fungi (Hane et al. 2007); thus, clustering can be regarded as only circumstantial evidence for lateral transfer.

Consideration of the possible mechanism of transfer brings focus to the role of transposons. Mobilization of DNA from a donor organism is much more plausible if the gene is associated with a transposable element. Both type 1 and type 2 elements generate extrachromosomal copies with features to aid integration at novel sites in genomes. Such capabilities would give transposons much greater transfer potential than the bulk genome. These features would be replicated in the recipient genome, though the transposon may have decayed or moved on since the transfer event. In these cases, the tendency is for transposons to integrate in repetitive DNA (whether because that is where they are targeted or where they are tolerated is a mute point) (Hua-Van et al. 2000; Larrondo et al. 2007).

Finally, the context of lateral gene transfer must consider the role of supernumerary or b-chromosomes. Supernumerary chromosomes are defined as ones which are dispensable for normal growth but are associated with specific and often critical facets of the phenotype (Covert 1998; Han et al. 2001; Temporini and VanEtten 2004). The transfer of a supernumerary chromosome between isolates of *Colletotrichum gloeosporioides* has been demonstrated conclusively (He et al. 1998). Even though the isolates in this study were from the same species, this should be regarded as a clear case of lateral transfer because the isolates were vegetatively incompatible.

Oomycetes.

Among the most clear-cut cases of lateral genes based entirely on phylogenetic evidence are a minimum of four genes postulated to have moved from a filamentous fungal ancestor to the oomycete pathogens *Phytophthora ramorum* and *Phytophthora sojae* (Richards et al. 2006a,b). Although the evidence is entirely phylogenetic, it is based on the analysis of whole-genome sequences and has been thoroughly validated statistically. The timing of the transfer is not specifically addressed; however, we can surmise that it was prior to the divergence of the *Phytophthora* genomes. Interestingly, both the oomycete genomes are from twentieth-century pathogens. *Phytophthora sojae* was described only in 1958 and *Phytophthora ramorum* in 1996. However, we can be certain that the genes identified in this project, encoding a sugar transporter, a purine permease, an aromatic acid degradation gene, and a lactose utilization protein, are not the cause of the new diseases because the genes are shared by both pathogens. Most likely, these genes (and perhaps many others) were acquired close to the time that *Phytophthora* spp. evolved as plant pathogens because they are linked to the ecological requirements of osmotrophic growth (i.e., growth using dissolved substrates in the exterior environment). Such substrates would be much

more abundant in the context of the living and decaying plant substrata that now constitutes the habitat of the pathogenic oomycetes.

This work, based as it is on multiple complete genome sequences, provides clear evidence for lateral gene transfer. The provision of new genome sequences may reveal that such cases are the rule rather than the exception. We can now move on to discuss the evidence that any new diseases may be due to recent lateral gene transfer events.

Pyrenophora tritici-repentis and *S. nodorum*.

The case of the fungal to oomycete transfer most likely dates back many millions of years. The transfer of the *Tsn1* gene has been functionally linked to the emergence of the latter pathogen in the last few decades (Friesen et al. 2006). *Pyrenophora tritici-repentis* is the cause of a necrotrophic foliar disease of wheat significantly called either yellow or tan spot. Although the pathogen was described reliably by microscopic examination of its spores and observed as a rare and minor grass and cereal pathogen from the 1920s, it was not until 1941 that it was observed to cause the currently characteristic severe tan lesions. The disease became progressively more regular and damaging through the 1960s and 1970s. Now, it regularly ranks as one of the top three or four foliar pathogens in many areas. The tan spot symptom is ascribed to the production and secretion by *Pyrenophora tritici-repentis* of a series of proteinaceous toxins, of which ToxA is the most important. ToxA is a small peptide that induces necrotic lesions in suitable cultivars of wheat and is produced by approximately 80% of isolates.

Evidence that the *ToxA* gene was transferred laterally from *S. nodorum* was based on phylogenetic and population level evidence. First, the gene was present in a genome sequence of *S. nodorum* but is absent from all other sequenced genomes and current databases. There is unequivocal incongruity between the *ToxA* gene phylogeny and the organisms in which it can be found. Second, the gene is not present in all isolates of *Pyrenophora tritici-repentis*, consistent with a recent acquisition. Third, the pattern of very high sequence variation in *ToxA* in *S. nodorum* and very low in *Pyrenophora tritici-repentis* again was consistent with recent acquisition (Stukenbrock and McDonald 2007). Fourth, the gene was associated with a transposable element-like genomic feature.

Although this evidence is extensive, it does not illuminate the mechanism. It is possible that *S. nodorum* was not the donor but that both organisms received the gene from a third species. Support for this idea comes from a more detailed analysis of the geographic origin of the *ToxA* alleles in *S. nodorum* isolates (Stukenbrock and McDonald 2007). *S. nodorum* is believed to have a center of origin alongside wheat in the Fertile Crescent (Stukenbrock et al. 2006). Analysis of sequence variation suggests that isolates from areas adjacent to the Fertile Crescent have low sequence diversity compared, strikingly, with the collection of isolates from South Africa. Wheat and barley are the only known hosts of *S. nodorum* and these crops were not brought to South Africa until the 1600s, along with European colonization. We can assume that *S. nodorum* traveled the world along with its hosts on seed or straw samples. Although we must acknowledge that the number of isolates is still small and the worldwide coverage is not complete, the overall low frequency of *ToxA* in *S. nodorum* and its maximum diversity in South Africa is suggestive of lateral acquisition by *S. nodorum* when wheat first was cultivated on the Cape. There is no current information on the likely source of the *ToxA* gene; however, it could be from any taxon with which wheat and its pathogens come into contact. These suggestions are intriguing and paint an alarming but irreversible picture of

the consequences of global migration in the age of exploration. At the very least, it is gratifying that these suggestions are feasible to test.

The suggested lateral acquisition by *S. nodorum* and transfer to *Pyrenophora tritici-repentis* is only the first step in the development of new disease. The lateral acquisition of the gene must be accompanied by its integration in the genome so that it can survive mitosis and cytokinesis. We have very little information about this phase except that the juxtaposition of a transposase sequence might supply transfer and integration machinery.

The next step is for the gene to be expressed and confer a selective advantage on the strain carrying the gene. In the case of plant pathogens, the key evolutionary pinch points are sporulation and interseasonal survival. Recent research on *S. nodorum* and older work on *Pyrenophora tritici-repentis* suggests that each pathogen expresses several toxins, each of which contributes quantitatively to disease (Liu et al. 2006; Liu et al. 2004a,b). *ToxA* seems to be particularly important in the interaction of *Pyrenophora tritici-repentis* and wheat; however, for both pathogens, the toxin interacts with the wheat gene *Tsn1* that encodes an apparent receptor (Lu et al. 2006; Manning and Ciuffetti 2005; Manning et al. 2007). Plants expressing the receptor *Tsn1* respond to the purified toxin. Fungal isolates expressing *ToxA* cause enhanced disease symptoms on wheat lines expressing *Tsn1*. Thus, it is easy to imagine the scenario whereby the pathogen strain acquires and expresses the *ToxA* gene and thereby exhibits enhanced sporulation, better enabling it to re-infect wheat that season or to survive to the next season.

ToxA apparently confers a selective advantage only on wheat expressing *Tsn1*. The presence of a dominant disease sensitivity gene is paradoxical. Although it would seem to suggest that *Tsn1* confers some balancing advantage, none has yet been discovered. The possibility that the inadvertent introduction of *Tsn1*-ve wheat lines may have contributed causally to the explosion of tan spot since the 1950s was investigated in Western Canada (Lamari et al. 2005). However, sensitive wheat lines have been planted widely since the late 1800s, well prior to the emergence of the disease. The emergence of tan spot in the 1960s and 1970s coincided with the plantings of *Tsn1* wheat but cannot be causally linked.

A further contributing factor may be required. A general rise in the prevalence of necrotrophic foliar pathogens has been linked to agronomic practices called no-till, zero-till, or minimum tillage. The practices avoid the use of plowing as a method of weed control and instead rely heavily on herbicides. The retention of stubble is actively encouraged because it promotes drought tolerance and minimizes wind erosion of soil. Such conditions are tailor-made for foliar necrotrophs because it encourages the maintenance and build up of inoculum from season to season. It may be that the emergence of tan spot and other diseases required a combination of conditions, two of which may be no-till practices and *Tsn1* wheat lines.

Two other toxins, *toxB* and *toxC*, have been described for tan spot (Effertz et al. 2002; Friesen et al. 2005). Little is known about the origin of these toxins; however, it is intriguing that the multiple copies of *ToxB* are associated with transposons (Martinez et al. 2004).

***Cochliobolus* spp. diseases.**

The story of the famous Southern corn leaf blight epidemic is qualitatively similar to that of tan spot. Prior to 1970, the pathogen *Cochliobolus heterostrophus* was a regular but unimportant disease of corn (maize). However, in 1970 and 1971, a major epidemic occurred which resulted in 20 to 30% losses (Ullstrup 1972). This new disease was due to the emergence of

a new race called race T, that produced a polyketide toxin called T-toxin (Yoder 1980). Race T isolates appeared to harbor an extra 1.2 Mbp of DNA (Chang and Bronson 1996). Although these factors are suggestive, strong evidence for lateral gene transfer is lacking. First, the potential donor of the locus has never been identified. Second, the *Tox1* locus is a highly complex bipartite genetic element which induces a reciprocal translocation in crosses with race 0 and which involves highly AT rich and repetitive DNA (Turgeon and Baker 2007). Thus, although a role for lateral gene transfer cannot be ruled out, the emergence of Southern corn leaf blight is not a simple case of gene acquisition. Rather, complex processes of gene shuffling and translocation also caused this new pathogen.

Texas cytoplasmic male sterile maize also was found to be susceptible to *Phyllosticta maydis* (syn. *Mycosphaerella zeae-maydis*), causing the disease yellow blight of maize (Arny and Nelson 1971). Pathogenicity was related to production of PM toxin (Yun et al. 1998), the biosynthesis of which is encoded by a gene cluster orthologous to the T-toxin biosynthetic locus *PKS2* (Baker et al. 2006). There is no direct evidence of lateral gene transfer as yet.

In many ways, these cases parallel Northern corn leaf spot caused by *Cochliobolus carbonum* (Walton 1996). Pathogenicity is largely due to HC-toxin, a cyclic tetrapeptide. As for T-toxin, the biosynthetic gene initially appeared to be a simple single locus, *TOX2*. More detailed studies revealed that the locus is complex, with a least five genes present in multiple copies involved in its biosynthesis (Ahn and Walton 1998). Furthermore, the genes are unstable and can be easily lost (Pitkin et al. 2000). The absence of the genes in strains that do not make HC-toxin is suggestive of lateral gene transfer, but no supporting evidence is available (Walton 2000). However, so called Helminthosporium leaf spots were apparent on wheat from the nineteenth century and there is no clear evidence that the disease is new.

A third member of the *Cochliobolus* genus is a clear example of a new disease. *Cochliobolus victoriae* causes Victoria blight of oat and first was noticed in 1946; the host-specific toxin victorin was detected a year later (Meehan and Murphy 1947). The disease is accounted for entirely by the production of the toxin (Sweat and Wolpert 2007; Wolpert et al. 2002). The emergence of the disease is ascribed to the introduction of oat cultivars carrying the *Vb* gene which confers resistance to crown rust (Litzenberger 1949). Victorin is a cyclic pentapeptide presumably produced by enzymes encoded by an nonribosomal peptide synthetase (Nikolskaya et al. 1995). Although Victoria blight of oat is a twentieth-century disease and is due to a host-selective toxin, evidence is lateral gene transfer is absent. First, the genetic basis of victorin biosynthesis has not been determined and strains of the fungus lacking the production have never been obtained. Therefore, although it seems likely that progenitor strains of *Cochliobolus victoriae* must have existed, they have never been obtained. These may be sought as mild pathogens in oat cultivars lacking *Vb*.

***Alternaria alternata* pathotypes.**

Alternaria isolates produce a series of necrotrophic diseases on a range of dicot crops: pear, apple, strawberry, and tomato, to cite just a few. These diseases emerged in the 1920s (M. Kodama, *personal communication*). Different pathotypes of *Alternaria alternata* produce 1 of at least 12 host-specific toxins (Hayashi et al. 1990; Thomma 2003) and these toxins are all the products of gene clusters (Ito et al. 2004). The AKT, AMT, and ALT toxin biosynthesis clusters all are located on small, conditionally dispensable chromosomes (Hatta et al. 2002). Phylogenetic analysis based on housekeeping nuclear genes indicates that all the strains have a common origin but

that each strain has a unique toxin biosynthesis cluster. The incongruity of the toxin gene phylogenetic trees and the bulk genome trees is the primary evidence for lateral transfer of these genes. The presence of the genes on small CD chromosomes provides a means by which the genes could transfer between the isolates (Tanaka et al. 1999). The most likely scenario is that nontoxigenic strains exist as mild pathogens or saprophytes. Acquisition of the toxin biosynthetic cluster would enable the strain to cause a more damaging disease on the appropriate host, thereby favoring the survival of both the host genome as well as the toxin biosynthetic cluster.

Other twentieth-century diseases.

Another new disease that is dependant on host-specific toxins is *Corynespora cassiicola*, the cause of Corynespora leaf fall of rubber (Barthe et al. 2007). The toxin cassiicolin is a small protein that has its three-dimensional structure determined. *Curvularia lunata* appeared in 1947 (Horst 1979) as a blight of gladiolus and now is best known as a producer of cytochalasin B. However, there is too little molecular work in the literature on these pathogens upon which to base even the most speculative hypotheses, although it is notable that both are dothideomycetes.

Conclusions.

The emergence of new diseases is a topic of the utmost importance for crop protection as well as other areas of biology. The evidence that a new disease can be ascribed to a new molecular form of a pathogen, as opposed to movement of a pathogen from one host to another or from one locality to another, is dependent on describing changes in the genetic structure of entire pathogen populations before and after the emergence of the new disease. Thus, it is highly unlikely that conditions would exist in which direct proof could be obtained. There are very few historical collections of live fungi; most are herbarium material. Furthermore, more mycological herbaria restrict themselves to clear-cut examples of major pathogens. We have seen that evidence of emergence of a disease often requires knowledge of weakly pathogenic progenitor strains. The Broadbalk collection is perhaps unique in offering samples (albeit killed, preserved samples) of the fungi associated with a wheat crop over the last 150 years (Bearchell et al. 2005). Polymerase chain reaction techniques now can be applied to understand pathogen dynamics over this time scale. It will be a challenge to relate genotypic history to the phenotypes of such pathogens. The thoughts expressed in this review highlight the value of long-term longitudinal collections of plant material and their potential value in charting not only the emergence of new diseases but also more subtle shifts in pathogen populations as they evolve to overcome new resistance genes, fungicides, and the changing climate.

The evidence of lateral gene transfer is compelling in the case of *S. nodorum* to *Pyrenophora tritici-repentis*. This is the only case we know of where both the donor and recipient of a transferred gene can be identified with any degree of certainty. In all the other cases, the evidence relies essentially exclusively on the presence of DNA in the new pathogen which is absent in related taxa. As has been noted above, there are many other explanations for this phenomenon, including gene loss and gene change. In the case of *Cochliobolus heterostrophus*, the evidence for simple gene transfer has been weighed and found to be an insufficient explanation for the emergence of RaceT. In this case, a more complex pattern of translocation and gene shuffling is needed to explain the observations. Nonetheless, although lateral transfer may not be a sufficient explanation, it has not been ruled out as a part of the process. In the cases of the other pathogens mentioned, there seems to be too little evidence available to critically evaluate the varying hypotheses.

It is the variability, plasticity, and flexibility of fungal genomes that is striking (Zolan 1995). The presence of dispensable chromosomes and the ability to transfer between isolates is well established (Covert 1998; He et al. 1998; Johnson et al. 2001; Miao et al. 1991). The emergence of race T *Cochliobolus heterostrophus* clearly involved translocations. The ability of fungi to evolve new forms is likely to be as much a consequence of genomic plasticity as of gene acquisition per se.

Nonetheless, there can be no doubt that the flood of fungal genome sequences in the pipeline will add credence to the many existing claims of lateral gene transfer and add many new examples; a picture in which significant proportions of the genomes are found to be laterally acquired seems more rather than less likely. A key requirement will be to obtain genomic information from multiple strains of a pathogen, to include examples of each pathotype as a minimum.

The organisms mentioned above are not randomly spread across fungal taxa. The last two decades have seen the mass introduction of gene-sequence data to studies of fungal phylogenetics, creating what appears to be a natural taxonomy based on evolutionary history of the fungi (James et al. 2006; Robbertse et al. 2006). The fungi described above, which combine recent emergence with host-specific toxins, overwhelmingly belong to the Dothideomycetes, a new class broadly replacing the Loculoascomycetes (Hane et al. 2007; Schoch et al. 2006). The collocation of fungal pathogens causing new diseases and using host-specific toxins in this new class is intriguing. Of course, not all Dothideomycetes are pathogens, and many do not seem to use host-specific toxins. Nonetheless, it seems prudent to ask what features of this class might have made them able to acquire laterally transferred genes. This ability is most likely to require side-by-side growth. Many fungi have the ability to form either mycelial anastomoses or conidial anastomoses (Roca et al. 2005), potentially allowing them to acquire genes from other fungi. All the pathogens in this class are nonobligate, allowing them to grow both on living and dead tissue. Many also would have the ability to grow on species related to their normal host. Therefore, there is endless potential for lateral gene transfer. However, these considerations apply to many fungal taxa, not just the dothideomycetes. An important feature of the life cycle of *S. nodorum* grown in Mediterranean climates is the ability to form asexual pycnidia during active plant growth in winter and spring as well as sexual pseudothecia during summer and autumn on stubble surviving from the previous year's crop (Arseniuk et al. 1998; Bathgate and Loughman 2001; Solomon et al. 2006a,b). Such features are shared by many of the dothideomycetes and would allow the exposure of the fungal mycelium to many potential partners for genetic exchange. Although the mechanism of interspecific transfer remains unknown, it is not hard to find both a motive and a weapon.

Some of the subject matter—effectors and interspecific gene transfer—recently has been elegantly reviewed (Van der Does and Rep 2007), to which the reader is referred for further insights and perspectives.

ACKNOWLEDGMENTS

Work in the authors' group is funded by the Australian Grains Research and Development Corporation. The thoughts expressed in this review have benefited significantly from discussions with many colleagues, especially Tim Friesen and Bruce McDonald.

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