Evolution of Invading Forest Pathogens via Interspecific Hybridization

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Abstract

Traditional morphologically-based fungal species concepts have tended to go hand in-hand with a perception that fungal species are genetically 'fire-walled' units between which almost no gene flow occurs. Also, prior to 1990, known examples of interspecific hybridization in fungi were very rare. However, observations on the internationally invading Dutch elm disease pathogens suggested that intense ecological disturbance events, including introductions or invasions, could result in hybridization. Since this could also lead to changes in a pathogen's aggressiveness, host range or other fitness attributes, it has considerable implications for the health of forests and natural ecosystems.

A conceptual model for hybridization between introduced and resident plant pathogens was developed (Brasier 1995). This was based upon an assumption that barriers to gene flow might be weaker, or even non existent, between fungal species that had evolved in geographic isolation; and that human influences have increase the chances of such species coming into contact. The potential for hybridization would also depend on many other factors, such as the frequency of niche contact; the precise nature of any genetic barriers to hybridization; the degree to which the two genomes can successfully recombine; and the ability of any resulting hybrids to compete with the 'parent' species. Possible outcomes range from the acquisition of a single gene by one parent to a full species hybrid incorporating the genomes of both parents (reviewed in Brasier 1995) Subsequently, at least eight examples of 'ongoing' hybridization have come to light (reviewed in Brasier 2000, 2001). Seven of these involve economically important forest pathogens.

Among these are the Dutch elm disease pathogens. *Ophiostoma novo-ulmi* — responsible for the current DED pandemic — has migrated across the northern hemisphere in the past 30-40 years. It has rapidly replaced *O. ulmi*, responsible for the first pandemic (see Brasier 2001). During this process the two species have come into close physical contact in the bark around scolytid vector breeding galleries of diseased elms. *O. ulmi* and *O. novo-ulmi* probably originated in different geographic locations in Asia (Brasier and Mehrotra 1995) and are strongly, but not totally, reproductively isolated. It is now known that rare and transient (unfit) hybrids occur between the two species when they come into contact (Brasier *et al.* 1998). This allows *O. novo-ulmi* — which initially spreads at epidemic fronts as a genetic clone — to acquire new vegetative compatibility (vc) and mating type genes from *O. ulmi. O. novo-ulmi* probably discards any 'unwanted' *O. ulmi* genes it receives (Abdelali, Brasier and Bernier 1999). As a result, the *O. novo-ulmi* clones at the epidemic fronts rapidly become highly diverse in terms of vc types and mating type. This in turn probably allows the pathogen to control the spread resist deleterious fungal viruses that might otherwise have brought about its demise.

Another example is the new *Phytophthora* killing riparian and shelterbelt alders across Europe. This comprises a swarm of heteroploid hybrids between *P. cambivora*, and a *Phytophthora* close to *P. fragariae* (Brasier, Cooke and Duncan 1999). Neither of these is an aggressive pathogen of alder whereas the hybrids, especially the commoner hybrid types, are highly aggressive and specific to alder (Brasier and Kirk 2001) Since *Phytophthora* has not previously been recorded as a pathogen of alder the hybrids may therefore have acquired the ability to exploit a new host — a possibility demonstrated earlier with laboratory generated *Phytophthora* hybrids. ITS sequences and AFLPs of genomic DNA indicate the hybrids are recently evolved and still evolving. How their evolution will proceed is uncertain. Whether they pose a comparable threat to North American alders is unknown.

An equally important example is that of the hybrid *Melampsora* tree rusts *(*resident *M. medusae x* 'introduced' *M. occidentalis*) that have appeared on poplar varieties clones in the Pacific Northwest that were bred for resistance to *M. medusae* (Newcombe *et al.,* 2000). The *Melampsora* hybrids can attack *Populus deltoides*, the commercial 'resistant' clones and even the local *P. trichocarpa*. Furthermore, as a result of the hybridization *M. medusae* pathogenicity genes could be transferred into the local *M. occidentalis* population.

Such examples probably represent the tip of an iceberg. Interspecific hybridization between forest pathogens is a poorly monitored process. It may be accelerating as a

result of human mediated ecological disturbances. In particular, the increasing international movement of plants and their associated pathogens that will promote hybridization; and phenomena such as host stress, ecosystem stress and access to novel hosts which will enhance the survival of hybrids. Certain fungal groups such as *Phytophthora* species, tree rusts and insect-associated ascomycete pathogens might be more likely to give rise to hybrids.

Plant health legislation is often hailed as our first line of defence. However, many hybrids or introgressants are unlikely to be detected by conventional, mainly morphologically based diagnostic methods of international quarantine. Molecularly based tools will be needed, while some of the old 'lists of scheduled organisms' will either need to be modernised or discarded. Furthermore, the issue of how to formally taxonomically define a hybrid or swarm of hybrids in a way that has legal utility for quarantine legislation or is of practical value in diagnosis has yet to be adequately addressed by mycologists. Present day fungal nomenclature offers little of value in this respect. It too may need to be modernised to allow formal definition of particular genotypes and formal description of fluid, still evolving populations that may or may not approximate to species.

Even a small genetic modification to a pathogen as a result of a hybridization event, such as the acquisition of a single gene conferring a different host specificity or a different climatic tolerance, could have a profound effect on its behaviour. This is well illustrated by the recent demonstration that, when the *O.novo-ulmi* cerato-ulmin gene is artificially transferred into the common bark saprotroph *O. quercus,* the latter becomes a potential vascular wilt pathogen (Del Sorbo *et al* 2000).

Through opportunities for hybridization, introduced pathogens have a potential impact far beyond the initial disease outbreaks that they cause. Each introduced pathogen is a dangerous, uncontrolled and open-ended experiment in evolution; and a gamble with the long-term stability of our forests and other natural ecosystems.

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