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THE EVOLUTIONARY BIOLOGY OF *FUSARIUM OXYSPORUM*

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ABSTRACT

Fusarium oxysporum is an anamorphic species that includes both pathogenic and nonpathogenic strains. Plant pathogenic forms cause a wilt disease and are grouped into formae speciales based on their host range; some are further subdivided into pathogenic races. Many formae speciales are comprised of multiple clonal lineages and, in some cases, a pathogenic race is associated with more than one clonal lineage, suggesting independent origins. Although some evidence suggests one pathogenic race may give rise to another, recent derivation of a pathogen from a nonpathogen has not been documented. Most new occurrences of Fusarium wilt appear to be the result of a recent introduction rather than an independent local origin of the pathotype. Asexual propagation is the dominant influence on population structure in *F. oxysporum* and the absence of sexual reproduction is not likely to prevent this pathogen from continuing to inflict significant damage on susceptible crop hosts.

Introduction

Fusarium oxysporum Schlechtend.: Fr. is an anamorphic species circumscribed by a suite of morphological criteria (65), principally the shape of the macroconidium, the structure of the microconidiophore, and the formation and disposition of chlamydospores. Notwithstanding these unifying criteria, considerable morphological and physiological variation within *F. oxysporum* has

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long been recognized; many such variants were initially assigned specific status (88). Subsequently, Snyder & Hansen (82) proposed to consolidate all of these species (the entire section elegans) into *F. oxysporum*, a concept that has received wide acceptance. Nevertheless, it is clear that *F. oxysporum* comprises a wide diversity of strains, and the extent to which these differences are sufficient to justify recognition of separate species is by no means a settled issue. Here, the broad concept of *F. oxysporum* is applied as it is consistent with usage in most of the recent literature. There is little doubt, however, that as molecular data continue to accumulate, *F. oxysporum* will be subject to further taxonomic revisions.

Given the large number of pathogenic variants in *F. oxysporum* and the intensity with which many of them have been studied, it is not feasible to offer a comprehensive synthesis of the literature on this fungus. Here, we focus on the evolutionary biology of *F. oxysporum*, drawing primarily from recently published data to evaluate hypothetical explanations for the evolution of formae speciales and pathogenic races in this species. Even with this limitation, the literature is so extensive that our citations only provide illustrative examples and do not constitute an exhaustive inventory of relevant published works.

Ecological Context

Although *F. oxysporum* has been studied primarily because of its ability to cause diseases of economically important plant hosts, its distribution and ecological activities reflect a much more diverse repertoire. For example, *F. oxysporum* is among the more commonly isolated fungi from asymptomatic roots of crop plants (37, 40, 84). Typically, isolates obtained in this way cannot be demonstrated to be pathogenic on the plant species from which they were recovered (44). Whereas they may be incapable of causing disease, nonpathogenic strains of *F. oxysporum* are aggressive colonizers of the root cortex (1, 37, 78). That such strains do not cause a wilt disease is presumably due either to their inability to enter the vascular tissue or to a rapid response of the host that localizes the infection (26). Nonpathogenic strains are also capable of colonizing crop residue (32) and quickly reoccupying fumigated soils (58). Thus, *F. oxysporum* has a well-documented ability to persist without recourse to pathogenesis.

Both pathogenic and nonpathogenic strains of *F. oxysporum* are found in agricultural soils throughout the world, and it is these populations that have received the most attention from researchers. However, substantial populations of *F. oxysporum* also are found in many native plant communities, in soils that have never been cultivated (14, 38, 69, 81, 87). In fact, the near ubiquity of *F. oxysporum* in soils worldwide has led to its inclusion in what has been termed the global mycoflora (70). Although the ecology of *F. oxysporum* in native soils has not been extensively studied, isolates of this species are known to be closely associated with plant roots (87), as they are in agroecosytems.

Note that *F. oxysporum* is not a pathogen of plants in native situations, in spite of substantial soil populations in some areas. In particular, grasslands often support large populations of *F. oxysporum* (38, 64), yet grasses, whether cultivated or native, are not known to be hosts to pathogenic strains of this fungus (7, 12). This is consistent with a predominantly nonpathogenic relationship between *F. oxysporum* and its plant hosts.

Given the widespread occurrence of *F. oxysporum* strains that are apparently nonpathogenic, it is reasonable to suspect that pathogenic forms were derived from originally nonpathogenic antecedents. Given the close association of these fungi with plant roots, an altered form capable of growing beyond the cortex and into the xylem could quickly exploit this capability and presumably gain an advantage over fungi restricted to the cortex. The advance of a fungus into the vascular tissue might elicit an immediate response from the host, successfully restricting the invader, or a delayed or otherwise ineffective response, eliminating enough water-conducting capacity to induce wilting (11). Alternatively, the plant might simply tolerate limited growth of the fungus within xylem vessels, leading to an endophytic association (15, 73). In this case, subsequent changes in the host or parasite could alter the relationship, such that fungal activities and/or a response of the host would result in the development of disease symptoms.

In any scenario leading to pathogenesis in a native plant community, the pathogen might be short-lived. Debilitation of the host would limit growth opportunities for the fungus, which would face renewed competition from other microorganisms following the death of the colonized plant. To the extent that the pathogen perpetuated itself on an infected host, the spatial association between inoculum produced on the dead plant and suitable infection courts on new hosts would likely militate against expansion of the infestation. Problems such as these may have limited the development of pathogenic forms prior to the advent of agriculture. On the other hand, the establishment of endophytic associations may have been less constrained.

Whereas pathogenic forms of *F. oxysporum* may not have persisted in native situations, selection for relatively intimate associations, up to and including asymptomatic colonization of the xylem, may have been common. A multitude of such strains, all capable of spawning a pathogen, may have accumulated prior to the domestication of crop plants. Over time, endophytic associations could have become, in effect, latent infections (80), where aggressive growth by the fungus was induced prior to death of the host. Wilt pathogens may have arisen simply through the selection of forms with shorter latent periods.

If pathogens have developed as specialized forms from a larger pool of generalist nonpathogenic (and possibly endophytic) strains, the former might have lost some of the ecological versatility that is the hallmark of the nonpathogens. Garrett (28) observed that, given their ability to grow within the xylem of a

living plant, at least initially without causing extensive damage, vascular wilt fungi must be relatively specialized parasites. Thus, given the necessity of survival within a living host and a lessening of the selective pressure to preserve saprophytic competence, diminished abilities to compete with nonpathogenic strains might be expected. Indeed numerous studies confirm that pathogens may, in some situations, be out-competed by nonpathogenic strains (1, 54, 78). However, these observations may reflect the range of variation among strains of *F. oxysporum* rather than a fundamental difference between pathogens and nonpathogens. Furthermore, pathogenic strains of *F. oxysporum* appear to have lost little of the ecological breadth found among nonpathogens, retaining the ability to colonize both crop residue and roots on nonhosts (8, 9, 32, 37, 44). This suggests a relatively recent origin of pathogenic forms such that insufficient time has passed for the incidental costs of specialization to be readily apparent.

Formae Speciales

To accommodate the known pathogenic variants within *F. oxysporum*, Snyder & Hansen erected 25 formae speciales (82). This designation was intended to describe the physiological capabilities of the fungi and was not a part of the formal taxonomic hierarchy. The forma specialis concept has been useful to plant pathologists because it identifies a subset of isolates that are of concern to the production of a crop susceptible to Fusarium wilt. This purpose is served, regardless of whether a forma specialis corresponds to a natural grouping of related strains. Nevertheless, the possibility that isolates within a forma specialis are unified solely by pathogenicity and are otherwise genetically heterogeneous and possibly polyphyletic in origin is of more than academic interest. The nature of the diversity comprised within a forma specialis has a direct bearing on the prospects for disease control through genetic resistance. In fact, this issue should be directly confronted during the development of genetically resistant germplasm, to ensure that the true potential of the pathogen is considered in the screening process. Understanding the evolution of pathogens in *F. oxysporum* will ultimately require a detailed characterization of the relationships between diverse pathogenic and nonpathogenic forms in this species. And formae speciales, notwithstanding uncertainty about their biological significance, constitute the logical starting point for this undertaking. Most studies reported to date have focused on an individual forma specialis, seeking to characterize the diversity therein, especially as it relates to pathogenic races (see below). The results of these studies make it possible to identify variants representative of various formae speciales that can be included in a broader study designed to characterize intra-specific relationships. Informative analyses of these relationships will most likely be based on comparisons of nucleotide sequences in conserved regions of the nuclear and mitochondrial DNA (39, 68).

A question of central importance regarding the evolution of pathogens in *F. oxysporum* is the frequency with which pathogenic forms have arisen and the antiquity of the event(s). Thus, if the appearance of a pathogenic *F. oxysporum* was a rare event, all currently known pathogens may trace their origins to a single ancestral genotype and the reservoir of potential pathotypes would be limited to strains associated with this single lineage. If the ancestral pathotype is of ancient origin, contemporary pathogens may have a long history of association with their hosts and may even have co-evolved therewith. It would then be reasonable to expect that isolates pathogenic to the same species share a recent common ancestor and, in general, that formae speciales would constitute more or less natural groupings. Alternatively, pathogenic forms may be affiliated with an array of distinct lineages or clades. If this is so, then happenstance, rather than co-evolution, may explain most of the host-pathogen combinations now recognized; heterogeneous and polyphyletic formae speciales might be common, although monophyletic groupings would not be unexpected.

The currently available data are not sufficient to critically evaluate hypotheses concerning the big picture of pathogen evolution in *F. oxysporum*. In principal, it should be possible to consider related questions at the level of individual formae speciales, such as whether a forma specialis is monophyletic. In practice, however, this is problematic because it is not known, a priori, which formae speciales are most closely related, rendering the selection of an outgroup somewhat arbitrary. Of course, formae speciales that are nearly devoid of genetic diversity (e.g. 83) seem very likely to be monophyletic, although the outgroup dilemma remains. One approach to this problem is to examine formae speciales that are pathogenic to related host species, on the assumption that pathogens of closely related taxa are themselves closely related. For this reason the formae speciales causing wilt diseases on crops in the Cucurbitaceae (seven have been described) are an attractive group to consider.

A close relationship between cucurbit-infecting formae speciales is indicated by the similarities in mtDNA reported by Kim et al (48, 49), based on a study of five formae speciales: *F. o. cucumerinum, F. o. lagenaria, F. o. luffae, F. o. melonis*, and *F. o. niveum*; these are pathogens of cucumber, calabash gourd, vegetable sponge, muskmelon, and watermelon, respectively. Based on RFLPs, a total of 14 mtDNA haplotypes were recognized, with unique haplotypes within each forma specialis. However, one pattern generally occurred most frequently for each forma specialis. Two mtDNA haplotypes were associated with four of the five formae speciales. The mtDNA haplotype most common in *F. o. niveum* also was associated with one or more isolates from every other forma specialis, except one, *F. o. luffae*. The absence of the common haplotype in this group may simply reflect a small sample size: Only two isolates were examined.

Based on the differences in mtDNA haplotypes, both cluster analysis and parsimony analysis indicated that all five formae speciales were closely related and, in some cases, isolates of different formae speciales were genetically more similar than were isolates of the same forma specialis (49). Within this group, *F. o. niveum* was the most homogenous of the formae speciales (48) and *F. o. cucumerinum* was the most diverse. Five different mtDNA haplotypes were detected among the nine isolates of *F. o. cucumerinum*. Whereas some isolates of *F. o. cucumerinum* grouped with the rest of the formae speciales under one branch, three isolates of this forma specialis consistently separated out as unique and quite distant from the remaining formae speciales. While most formae speciales grouped together on strongly supported branches, these three isolates of *F. o. cucumerinum* and one isolate of *F. o. melonis* (from Mexico) were consistently far removed from the rest. Because many isolates of the different formae speciales were closely related and grouped together under a single branch, it might suggest that formae speciales within the Cucurbitaceae have a monophyletic origin, that is, they have been derived from a common ancestral type. On the other hand, many of the formae speciales are themselves apparently polyphyletic.

Relationships inferred from similarities in DNA fingerprints also demonstrated a close relationship among formae speciales pathogenic to cucurbits (the same formae speciales examined by Kim et al except that *F. o. momordicae* was included and *F. o. Luffae* was not) (63). In this work, analysis of the differences in DNA fingerprints separated the cucurbit pathogens from six formae speciales pathogenic to plant species outside the Cucurbitaceae. Among the cucurbit formae speciales, four were resolved into distinct clusters but isolates of *F. o. melonis* formed two separate groups, one of which appeared closer to *F. o. lagenaria* than to the other grouping of *F. o. melonis*. Thus *F. o. melonis* appeared to be polyphyletic whereas the other formae speciales did not.

Consistent with the genetic similarities among cucurbit-infecting formae speciales are numerous reports of overlapping host ranges among these pathogens. This includes the extensive studies of Armstrong & Armstrong (6) and a number of more recent reports (30, 57, 59). Perhaps the most significant case of cross pathogenicity between species was reported by Gerlagh & Blok (30), who found *F. oxysporum* from wilted cucumber also to be pathogenic to both muskmelon and watermelon. This led to the suggestion that several formae speciales be grouped together as f. sp. *cucurbitacearum* (30). On the other hand, considerable evidence attests to the host specificity of isolates associated with the various formae speciales causing wilt diseases in the Cucurbitaceae. To some extent this discrepancy can be attributed to differences in test conditions, such as the age of the inoculated plant and the method of inoculation (6, 57); under field conditions, most formae speciales are host specific. Nevertheless, it is

reasonable to conclude that diversity exists within some formae speciales with respect to host range, which may be a further indication that these pathogens share a common ancestry.

Somewhat ironically, although the close relationship among the cucurbit wilt pathogens is suggestive of co-evolution with the corresponding host taxa, individually many of the formae speciales appear polyphyletic. However, in this case, the groupings may be polyphyletic simply because it is difficult to clearly draw dividing lines among closely related isolates and not because the formae speciales constitute highly heterogeneous groupings. Thus it is, perhaps, the cucurbit-infecting lineage, rather than the constituent formae speciales, which constitutes a monophyletic grouping. Confirmation of this would require a phylogenetic analysis including an appropriate outgroup.

Unlike the situation for the cucurbit wilt pathogens, isolates pathogenic to a single host, banana, are remarkably divergent. Koenig et al (53a) examined 165 isolates of *F. o. cubense*, cause of Fusarium wilt of banana, using anonymous, single-copy RFLP loci. Seventy-two RFLP haplotypes were identified, but the five most common haplotypes represented nearly half the collection. Based on parsimony analysis, isolates fell into two major groups with seven strongly supported clades, as well as a number of unresolved clades. The two largest lineages of *F. o. cubense* were genetically more similar to an isolate of another forma specialis, *F. o. niveum* (intended to be an outgroup), than they were to each other. They were also as genetically distinct from each other as either was to three isolates of *F. o. lycopersici* (also intended as an outgroup). Thus, it has been concluded that *F. o. cubense* may be polyphyletic in origin and that pathogenicity to banana is an independently acquired phenotype. Of course, this does not necessarily preclude co-evolution as it is possible that the divergence within *F. o. cubense* reflects the presence of both co-evolved and more recently selected pathogenic forms.

The Origin of Pathogenic Races

Variation in virulence within a forma specialis has been categorized by assigning pathotypes to pathogenic races. Races are defined by their differential interaction with host genotypes (7), which, in some cases, are cultivars known to carry one or more major genes for resistance (79). The extent of variation within a race and the nature of the relationships between races was poorly understood until recently. Insight into these issues, gleaned from a wealth of new data, now makes it possible to postulate how novel pathotypes in *F. oxysporum* are generated.

In general terms, the appearance of Fusarium wilt in a new location, or the appearance of a new pathogenic race, as might be evidenced by a previously resistant cultivar succumbing to disease, could be explained in one of two

ways. Either the new pathotype was introduced or it originated locally. The latter may be explained either by derivation from a preexisting pathogen, the classic case of selecting for a virulent race by growing a resistant cultivar, or by selecting from the local population of nonpathogenic strains of *F. oxysporum*. The literature offers some evidence to support each explanation for the origin of new pathogenic races.

We first consider the case for pathogen introductions. Several formae speciales have now been studied in sufficient depth to gain an understanding of the relationship between infestations in disparate locations. Many of these offer evidence for the widespread occurrence of an individual strain that corresponds either to a unique genotype or to a narrow range of clonally related genotypes (i.e. a clonal lineage). This suggests that movement of preexisting strains can account for the development of new infestations. Several examples will serve to illustrate the evidence supporting this interpretation.

The importance of pathogen dispersal as opposed to independent origins may be indicated where little or no diversity is evident within a pathotype. For *F. o. apii*, the cause of Fusarium yellows of celery, race 2 is found throughout North America, and all isolates examined to date have been associated with the same vegetative compatibility group (VCG) (17, 85; see 55 for a recent review of the use of VCGs in studies of fungal populations). Thus the available evidence suggests that a single strain has been moved to the various locations where Fusarium yellows of celery now occurs. This conclusion was strengthened by the fact that VCG phenotyping detected considerable diversity among the nonpathogenic strains of *F. oxysporum* recovered from celery roots (16), indicating that the association of only one group with the cause of disease in celery did not simply reflect a lack of sensitivity of the marker.

The use of vegetative compatibility (VC) to infer inter-isolate relationships is premised on the assumption that all vegetatively compatible isolates are clonally related, which in turn assumes the following: (*a*) that sexual reproduction is either rare or entirely absent and therefore does not have a significant influence on population structure, and (*b*) that considerable attrition has occurred since the last occurrence of outcrossing, making the existence of isolates with the same VC genotype in otherwise different genetic backgrounds very unlikely. If these assumptions are valid then isolates associated with the same VCG would also share a common "residual" genotype (72). Even so, the occurrence of somatic mutations would, over time, lead to genetic differentiation among vegetatively compatible isolates that would not, therefore, constitute true genetic clones. Isolates associated with the same VCG would, however, belong to the same clonal lineage. Empirical evidence confirming the correspondence of a VCG to a clonal lineage may be obtained by using various measures of genetic relationship to show that affinity groups, based on these criteria, tend to mirror

groupings based on VC. For example, in the case of *F. o. albedinis*, the cause of Bayoud disease of date palm in Northern Africa, 44 isolates from throughout the known range of the disease were associated with a single VCG and were also unified by similarity in both mitochondrial and nuclear DNA (83). Thus the data support a correspondence between VCG and a clonal lineage that, for this pathogen, appears to account for all known occurrences of the disease. Co-occurring *F. oxysporum* isolates not pathogenic to date palm were clearly separable from the pathogen by both VC and randomly amplified polymorphic DNAs (24).

In contrast to the wilt pathogens considered thus far, many formae speciales comprise considerable diversity and are associated with multiple VCGs. Here too it is often possible to show a close relationship between isolates responsible for infestations in multiple locations. For example, in *F. o. cubense*, which is comprised of at least 17 VCGs (53, 71; HC Kistler, personal communication), certain race/VCG phenotypes are found in many different locations. This suggests that recent movement of the banana wilt pathogen is responsible for many of the known infestations. A good example is race 4/VCG 0120, which has been recovered from Taiwan, Australia, the Canary Islands, and South Africa (71). Finer scale resolution based on multilocus nuclear DNA haplotypes confirms the widespread occurrence of individual genotypes (53a).

Similarly, in *F. o. melonis*, race1/VCG 0134 is known to occur in Europe (43), Central Asia (Z. Banihashemi, personal communication), North America (43), and South Africa (W Schreuder, personal communication). Molecular markers, including mitochondrial (42) and nuclear DNA haplotypes (4, 77), confirm that VCG 0134 corresponds to a clonal lineage. Thus, recently identified infestations of race 1/VCG 0134 in California (B Gwynne, RM Davis & TR Gordon, unpublished information), Maryland (43), New York (90), and South Africa are very likely the result of introductions from areas where the pathogen was already established.

Although movement of strains as a result of human activities is clearly the dominant influence on the establishment of new infestations of Fusarium wilt, de novo origin of pathogenic forms is also possible. Several lines of evidence support this view. First, in many formae speciales, there is a complex relationship between pathogenic races and VCGs (or clonal lineages). That is, a given race may be associated with more than one VCG and some VCGs are associated with multiple races. One interpretation of these relationships is that a given pathogenic race has had multiple origins and that different pathogenic races may be closely related.

In some cases, the close relationship between pathogenic races implied by their association with the same VCG has been corroborated by other measures of genetic relationship. For example, VCG 0134 of *F. o. melonis* is associated

with all four known races in this forma specialis. Furthermore, race 0, race 1, and race 1,2 all are associated with identical mtDNA (42) and nuclear DNA haplotypes (77). The close relationship between three different races may indicate that relatively simple genetic changes can lead to a change in cultivar specificity, i.e. one pathogenic race can give rise to another.

A similar situation has been described for *F. o. lycopersici*, wherein races 1, 2, and 3 all are associated with VCG 0030 (20), and affinities based on RFLPs in nuclear DNA confirm the association of this VCG with a clonal lineage (22). Here again, a close relationship between the races implies the derivation of one from another. Moreover, the association of one race with many VCGs, such as race 2 of *F. o. lycopersici* or race 2 of *F. o. melonis*, may indicate the genetic changes that generate a particular virulence phenotype have occurred on multiple occasions. Of course, genetic changes affecting vegetative compatibility are also a real and not mutually exclusive possibility. The association of multiple VCGs with the same clonal lineage is suggestive of VCGs arising secondarily through somatic processes (22).

In addition to the essentially circumstantial evidence based on studies of natural populations, some mechanistic data support the view that simple genetic changes can generate altered pathotypes. Both chemical (13) and transformational mutagenesis (47) have been shown to alter host specificity in cucurbit-infecting formae speciales of *F. oxysporum*. Bouhot (13) used nitrosoguanidine to render a watermelon-specific strain of *F. o. niveum* pathogenic to muskmelon, and Kim et al (47) transformed a race 2 isolate of *F. o. niveum* into a race 0, while maintaining the VCG of the original strain. The simplest explanation for such observations would be an effect mediated by a single gene.

The evidence presented thus far speaks only to the possibility of one pathotype being derived from another. There is no documentation that it has actually occurred, but such might be inferred from the spatial association of putative progenitor and derivative pathotypes in two recently described situations: *F. o. melonis* in Israel (46) and *F. o. lycopersici* in California (20, 21).

Race 2 of *F. o. melonis* represents a relatively recent discovery in Israel, whereas race 0 has a longer history in this country (66, 67). Race 0 is widespread in Israel, whereas race 2 has a more localized distribution, co-occurring with race 0 in some areas. Race 2 and race 0 are both associated with VCG 0138. In principal, race 2 of *F. o. melonis* could have been introduced to Israel from other parts of the world where this race is well established. However, in no other location has race 2 been associated with VCG 0138, as it is in Israel. Thus it is possible that the race 2 now found in Israel is of local origin, perhaps representing a mutant form of race 0. Of course, the case for this scenario could be made considerably stronger by showing that a finer measure of genetic

similarity, such as DNA fingerprinting (51), confirms the close relationship suggested by VC.

In California, race 3 of *F. o. lycopersici* was first identified in a field where race 2 was also known to occur (RM Davis, personal communication). Isolates of both race 2 and 3 from this field were found to be associated with the same VCG (20) and also were similar based on isozyme phenotypes (21) and RFLPs in the nuclear DNA (K Elias, personal communication).

Although the above evidence is consistent with selection of new pathogenic races from preexisting pathogens, it is not necessarily the case that this has occurred in a step-wise fashion, in concert with the introduction of new resistance genes. In *F. o. melonis*, for example, races capable of overcoming the host resistance gene FOM-1 were shown to be widespread in France prior to the cultivation of FOM-1 containing cultivars (13). Thus the eventual demise of the resistant cultivar required only an increase in the population size of a variant that was already present; perhaps an indication the mutations that generate such new pathotypes are a common occurrence.

With regard to the origin of new pathotypes, the most insidious prospect from the perspective of disease management would be the selection of plant pathogenic *F. oxysporum* from local populations of nonpathogenic strains. The existence of both pathogenic and nonpathogenic strains in *F. oxysporum* indicates that the derivation of one from the other must be considered a possibility. The question is whether such conversions occur often enough to be of concern or instead are rare events of significance only on geologic time scales.

One way of evaluating the likelihood that pathogens were derived from nonpathogens is to look for similarities between pathogens and nonpathogens where they co-occur. For the most part, such studies have shown nonpathogens to be different from the pathogen. For example, based on vegetative compatibility, both *F. o. vasinfectum* (45) and *F. o. spinaciae* (25) were different from root-colonizing isolates of *F. oxysporum* not pathogenic to the plant from which they were isolated. Woudt et al (89) reported that nonpathogenic isolates of *F. oxysporum* associated with cyclamen were similar to pathogenic isolates (i.e. *F. o. cyclaminis*) based on polymorphisms in the intergenic spacer region (IGS) of the nuclear rDNA. However, the nonpathogens were differentiated from the pathogens by both VC and DNA fingerprints (89).

In most cases, *F. o. melonis* also is clearly distinct from nonpathogens recovered at the same location (3, 33, 34, 35). However, several exceptions have been noted. In particular, some nonpathogens appear closely related to co-occurring isolates of *F. o. melonis* based on one or more of the following: vegetative compatibility (3), mtDNA haplotype (35), or IGS haplotype (4).

Most nonpathogenic isolates that are vegetatively compatible with *F. o. melonis* can be shown, by other measures, to be dissimilar. For example, in Maryland, nonpathogenic isolates associated with *F. o. melonis* VCGs 0131 and 0134 were identified. However, based on the nucleotide sequence in the intergenic spacer region, the nonpathogens did not cluster with vegetatively compatible pathogenic isolates (5) or any other isolates of *F. o. melonis*. Thus, in this case, pathogenic and nonpathogenic isolates may be vegetatively compatible owing to a coincidental sharing of alleles at the loci influencing vegetative compatibility rather than a clonal relationship. If this is so, it violates a critical assumption for the use of VCGs as markers for important phenotypic characters such as pathogenicity. However, at least for *F. o. melonis*, exceptions to the association between VCG and pathogenicity to muskmelon appear to be rare.

In California, eight nonpathogenic isolates from a single field were found to have a mtDNA haplotype identical to the *F. o. melonis* isolates recovered from the same location (35). However, based on DNA fingerprints, the nonpathogens did not appear closely related to the pathogen. Whereas 15 or more strongly hybridizing bands were evident in restriction digests of *F. o. melonis* isolates (77), nonpathogens showed very limited homology to the probe (TR Gordon, unpublished information). Identical mtDNA among isolates with very different nuclear DNA may reflect inter-isolate transfer of mtDNA through hyphal anastomosis (36). Alternatively, the isolates in question may trace their origin to a common female parent, in which case subsequent clonal propagation must have coincided with considerable divergence in their nuclear genomes.

The divergence of non-outcrossing lineages might be accelerated through the activity of transposable elements, of which some have been documented in strains of *F. oxysporum*, including *F. o. melonis* (19). More important, the proclivity of transposable elements to insert themselves into genes, which may thereby be inactivated (18), provides a plausible explanation for the alteration of host specificity within clonal lineages; i.e. inactivation of an avr gene that would affect virulence to specific host genotypes.

Whether a significant number of nonpathogenic strains are but a transposon insertion away from causing Fusarium wilt is unknown, but at least one possible example can be cited. A nonpathogenic isolate recovered from a field in Maryland with a history of Fusarium wilt was identical to *F. o. melonis* based on VC, mtDNA haplotype (3), and IGS haplotype (4). Thus it could be distinguished from *F. o. melonis* only by its inability to induce symptoms on inoculated seedlings in the greenhouse. Perhaps clonal descendants of this isolate will eventually manifest virulence to muskmelon. Alternatively, the isolate in question could be a disabled pathogen. In either case, it is of interest to know how this isolate differs from the pathogens. Techniques such as

RFLP subtraction (76), designed to identify regions of sequence unique to one member of a pair of closely related genotypes, might be profitably applied here.

Future Prospects

In 1971, Booth (12) recognized 79 formae speciales of *F. oxysporum*, and ten years later (7) a similar compilation by Armstrong & Armstrong reflected only minor changes. Collectively, these pathogens give *F. oxysporum* an impressive host range. With the exception of grasses and most tree crops, few widely cultivated taxa are not hosts to a pathogenic form of *F. oxysporum*. Recently described formae speciales are pathogenic on less extensively cultivated food crops such as lettuce (41) and basil (23) or ornamentals such as Acacia (27). Recent history would therefore suggest that whereas most crops potentially susceptible to *F. oxysporum* incited diseases have already been exploited by this pathogen, new host-pathogen combinations will continue to be recognized. This will likely result principally from the selection of existing variants that are brought into proximity with a susceptible host as a consequence of agriculture expanding into previously noncultivated soils, the adaptation of existing crops to new regions, and the domestication of new plant species. Increases in global commerce and travel may also enhance the opportunities for new host-pathogen combinations to become established.

In addition to the recruitment of new hosts, changes in cultivar specificity among known pathogens can be expected to continue as new genes for resistance are deployed. For any given forma specialis of *F. oxysporum* the number of characterized races is not large, when compared to pathogens such as *Puccinia graminis* (74) and *Phytophthora infestans* (2). Nevertheless, many examples suggest that where new genes for resistance to Fusarium wilt are incorporated into widely planted cultivars, new races will eventually be recognized. As already described above, this appears to have occurred frequently within clonal lineages, presumably through simple genetic changes.

Alterations occurring within a clonal lineage might result from single gene mutations, possibly mediated by insertion of transposable elements, or by loss of chromosomes or chromosome segments (52). That the latter is not an unusual occurrence is suggested by the great diversity of electrophoretic karyotypes that has been described for *F. oxysporum* (10, 50, 60). Inter-isolate exchange of chromosomes or segments thereof could occur as a result of parasexuality (75), leading to novel genetic combinations. Parasexuality is a phenomenon that has been demonstrated to occur in numerous filamentous fungi in the laboratory but has not been confirmed to be of significance under natural conditions. Although parasexual exchanges have been reported to occur between vegetatively incompatible isolates of *F. oxysporum* (61), it seems likely that such exchanges would be far less frequent than those involving isolates associated with the same VCG.

Furthermore, assuming that vegetatively compatible isolates were clonally derived from a recent common ancestor, they would already have identical alleles at most loci, minimizing the prospects of generating novel genetic combinations. Of course, a rare event might lead to a significant recombinant that served to rescue a unique allele from an otherwise unsuitable genetic background.

It is commonly assumed in the biological sciences that sexual reproduction is essential to the long-term viability of a species: not only to provide phenotypic variation on which selection may act but also to mitigate the effects of Muller's ratchet (62) by cleansing the genome of deleterious sequence alterations. Furthermore, phylogenetic evidence indicates that asexual lineages in fungi tend to be relatively recent derivatives of taxa associated with perfect forms (56). Thus, fungi that adopt an exclusively clonal mode of propagation are expected to be less successful.

It is therefore appropriate to inquire whether or not the capability to reproduce sexually still exists in *F. oxysporum*. It is apparent from numerous studies that clonal propagation is the dominant influence on population structure, but this does not preclude the possibility of cryptic sexual reproduction (29). Numerous observations might be explained by recent outcrossing, such as multiple VCGs associated with the same mtDNA haplotype (34, 35) and the identification of genetically distinct isolates sharing a VC phenotype (3). However, these observations could also be explained by somatic mutations affecting VC. Similarly, apparently high levels of both phenotypic and genotypic diversity also are suggestive of a sexually reproducing population, but these indications do not constitute a compelling argument for sexuality, for reasons that have been presented elsewhere (31). To these may be added the possibility that diversity within the broadly defined taxon termed *F. oxysporum* may actually be attributable to multiple phylogenetic species (K O'Donnell, personal communication). Finally, a test for linkage disequilibrium (86) recently undertaken for *F. o. cubense* confirmed the nonrandom association of alleles expected in the absence of sexual reproduction (53a).

On balance, therefore, the available evidence inveighs against an important role for sexual reproduction in *F. oxysporum*. However, even if sexuality is no longer part of the genetic program in this species, changes resulting only from the selection of existing variants and a "fine tuning" of clonal lineages will probably be sufficient to provide a continuing challenge to the successful cultivation of crops susceptible to Fusarium wilt. As such, a better understanding of the asexual mechanisms (52) by which variants arise in this species should be a high priority in future research on this important group of plant pathogens.

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