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An Overview of Ecological and Habitat Aspects in the Genus *Fusarium* with Special Emphasis on the Soil-Borne Pathogenic Forms

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ABSTRACT

The genus *Fusarium* is diverse, widespread and commonly found world-wide. This ubiquitous fungus exists in soils, grows in and on living and dead plants and plant products, on living and dead animals; its conidia are water-borne, but in some instances may be air-borne and its chlamydospores are typically soil-borne. When a sexual stage exists, the ascospores are air-borne, some spores may be found at rather high altitudes. There are salt-water forms, living in the deep sea, some in the shells of sea turtles, causing the shells to break up. Some forms have been isolated from within the corneas of diseased eyes of humans and animals, and cited as the cause of a serious problem leading to vision loss. Species of *Fusarium* vary greatly in growth rates, substrate and optimal climatic preferences, there are those found only in the tropics, while others are the causative agent of snow mold. They contain a wide range of pigments, ranging from light tans and yellows, carmine red, green, bright blue to blue-black, some that are water soluble (quinone-type compounds), others fat soluble (carotinoids) and some that are closely bound to spore walls. Four different kinds of spores are recognized in the genus- macro- and microconidia, chlamydospores and ascospores. Sclerotia of variable sizes can also occur in culture and are brown, some blue-black and there even some white ones. Most *Fusarium* spp. grow well on typical laboratory media and are easy to culture.

Key words: *Fusarium oxysporum*, *Fusarium solani*, survival in soil, pathogenicity, disease control

PRODUCTION OF TOXINS AND OTHER HARMFUL OR DISTINCTIVE SUBSTANCES.

Some Fusaria produce potent toxins that may be present in food and feed products [64, 90, 104, 106]. The three most notorious toxin classes are the 'trichothecines' the 'zearelenones' and the 'fumonisins'. Arguably, the most dangerous of these substances are those of the trichothecine group. Humans and animals that ingest them in food products or feed develop acute clinical symptoms, such as subcutaneous hemorrhaging, alimentary toxic aleukia (ALA), deterioration of throat tissue, exhaustion of bone marrow abortion of fetuses, and frequently death ensues after eating food contaminated with these toxins.

Although several kinds of Fusaria have been implicated as producers of trichothecines, the fungi usually responsible lie within the species *Fusarium tricinctum* (sensu Snyder and Hansen), but are most likely to be cultures known as *F. sporotrichiella* by many mycologists today. This toxin producer is found on grain and seeds only sporadically. Special ecological niches appear to be of great importance in order for this fungus to establish and are likely related to moisture, humidity and other yet unknown environmental factors, because fortunately these toxin producers do not number high among the usual Fusaria isolated from soil and seed encountered as laboratory samples. However, sometimes they do occur and in alarming populations. I have personally isolated

them in abundance occasionally, once on milo maize seed obtained from Brazil; on another occasion nearly all of the *Fusarium* colonies (some 85%) appearing on soil dilution plates were those of *F. sporotrichiella*. These soil samples were from a series collected from "Udi Village" in Hunan Province China. A few years ago, cotton seed samples in shipments from Australia yielded enough of this same fungus to raise the question of the desirability of feeding the seed to dairy cattle, in that the contamination occurred unevenly throughout the seed lots, being much higher in some pockets than others.

During the Vietnam war there were reports of toxic yellow rain [148], falling down on the Hmong people of Laos. It was presumed by the large amount and the damaging effect of the strange yellow powder falling down from the sky, that it was a product of soviet biological warfare. The organism contained in the yellow rain was identified as *F. tricinctum* and confirmed by plant pathologist Eugene Smalley, University of Wisconsin. Furthermore, it appeared to be identical to, and produced the same toxin as a culture in his collection labeled "T-2". The culture had originally come from the collection of Professors Hansen and Snyder at University of California Berkeley, who had been Dr. Smalley's professors in graduate school. Thus the name T-2 toxin was given and it stuck. Later, after much investigation, it was concluded that yellow rain was a natural phenomenon and further speculation on its origin arose. Entomologists found the fungus may develop profusely in bee excrement, but whether this had been the source of the problem in Laos has never been definitely proven. It would seem to take big swarms of bees to toxify a whole area. Nevertheless, since significantly high populations of these dangerous fungi can arise sporadically, recognition of a problem, should it occur, requires vigilance.

Zearelenones are another group of Fusarium toxins. They are of a female hormonal nature, related to estrogens. When present in feed they can cause malformations of the female animal's reproductive system. Also they are emetics and cause vomiting. Most commonly these toxins are produced by *Fusarium roseum* (sensu Snyder & Hansen), especially 'Graminearum' (now mostly known as *F. graminearum*). It may be significant, given their ability to manufacture hormonal substances, that these common homothallic pathogens found on cereal crops, where they often produce copious quantities of their sexual stage, *Gibberella* perithecia, in the field or *in vitro*. These can be observed *en masse* macroscopically as small black/blue-black bodies on corn and small grain, kernels, stalks or stems.

Fumonisins are yet another toxin group from *Fusarium*-infected seed and grains They are capable of causing irreversible damage to the brain and to other organs. They can cause equine encephalomalaca in horses, also known as the "hole in the head disease". They have also been cited as the cause of esophageal cancer in humans. Certain members of the species *Fusarium moniliforme* are the pathogens responsible for the production of these poisons occurring on many of the grains, fruits, and vegetables which the fungi invade and colonize. Perhaps most notably these fungi are the cause of ear rot and stalk rot of corn and it is on this crop where fumonisins most often have been found. The appearance of a light pinkish-colored, slightly fuzzy growth on kernels, especially near the tip end of the ear, are indicative of the presence of these fungi. Needless to say, such ears should be avoided, not eaten.

Besides these products made by *Fusarium* spp. and known to be toxic to humans and animals, there are other distinctive substances derived from fusaria that can be detrimental. Perhaps, best known of these are fusaric acid and the gibberellins. Fusaric acid, a powerful toxin to plants, may be responsible for some of the symptoms seen in wilt diseases. It is one of the few toxins to plants that has been demonstrated to be produced *in vivo* in the host plant and *in vitro*, in fungal cultures. However the level of the toxin found in the host plant tissue is not correlated with disease severity. In some heavily colonized diseased plants almost none of the substance is to be found, whereas it well might occur in plants without significant damage. As discussed later virulence may involve additional entities and conditions. Gibberellin was the name given by Yabuta [150] to a purified substance of culture filtrates of *Gibberella fujikuroi*, isolated from diseased rice plants. *G. fujikuroi* is the botanical name of the sexual stage of the strains of *F. moniliforme* that caused the "foolish rice disease", bakanae-byo, known in Taiwan/Japan since Hori's l898 observation [121]. Infected rice plants grow tall, but are weak and spindly and yield poorly. Nevertheless, at one time gibberellins, especially gibberellic acid, were heavily investigated as possibly useful growth hormones and as seed germination stimulatants. However, it was eventually concluded that their use was limited because they did not promote strong healthy growth.

Another problem, less publicized ergo less well-known, is that of "gushing beer", caused by a factor produced in barley infected with *F. graminearum* (and to a lesser degree by similar fusaria). This is the same fungus that produces the zearelenone toxins. In infected plants in the field a chemical factor is produced on the grains and remains through the malting, brewing, aging and bottling processe. When a bottle is opened the contents shoot upward, sometimes several feet in the air, releasing most of the beer that the bottle held. This is due to the fungal

production of a carbonic anhydrase enzyme, which does not allow carbon dioxide to remain in solution as carbonic acids. The culprit, the pathogen causing a head blight disease of the barley kernels, can be detected rather easily by washing off a sample aliquot of the grain lot, in which the conidia typical for this fungus can be observed, since they should be profuse in the sample if there is a possibility of the problem. Pink kernels are also a clue. Good maltsters historically were known to shun such seed lots offered for sale. Because this gushing factor is particularly strong in the same kind of fungi that produce zearelenones, it would be interesting to know if the two problems were in any way related.

Besides various diseases and toxins caused by Fusaria, there are beneficial effects of the genus. Probably most notably is their reputed ability to break down crop residues to form humus in cultivated soils. In such fields *Fusarium* spp. are certainly well represented as soil and refuse inhabitants, being among the most numerous of the soil fungi. Microbial decomposition of the residues from previous crops is agronomically essential in soil-building for subsequent crops, and soil-borne Fusaria are undoubtedly a part of this process.

DEFINITION: WHAT MAKES IT A *FUSARIUM?*

Taxonomically, *Fusaria* have long been described among the imperfect fungi of the family, Tuberculariaceae. This family was known for producing conidia in tubercules, which consist of masses of masses of branched conidiophores, sporodochia. In culture these sporodochia build up and are seen macroscopically as light colored raised bodies on the surface of the plectochymatic culture mat. In the genus *Fusarium* macroconidia are borne in sporodochia. Further, in order to be identified as a *Fusarium* sp., these macroconidia should be long, slender, rather pointed at both end, dorso-ventrally curved, sickle-shaped, septated, and posses a basal foot cell (that is, the basal cell of the septated spore has a slight notch on the dorsal side near the attachment point to the conidiophore). The macroconidia are phialospores, that is to say they are produced in a phialide, which is a small opening at the tip of the conidiophore from which the spores emerge one by one, appearing apex end first, all initially attached to the conidiophore. They are produced in moisture, often in a small droplet, and, whether produced in culture or in nature, these conidia continue to be carried by water. Typically they do not survive as conidia in soil for long periods. This sums up the morphological features which define the fungus as a *Fusarium*.

Besides the particular macroconidia that define the genus, there are other spore types that *Fusarium* species may or may not bear, and their presence or absence often serves to distinguish between the species of the genus. Microconidia may be formed. Typically they are present on the aerial mycelium of the culture growth, appearing as small, usually one-celled spores, and oval-shaped, although in some species they may be apiculate, tear-drop or pear shaped and sometimes even spherical. Microconidia may be phialospores or they may also be blastospores, which are dry spores produced by budding at the tip of the conidiophore. These sporogenesis features are also used by taxonomists to distinguish species, however they are often difficult to ascertain and in some isolates production of both spore types occur. Sometimes microconidia from phialides remain attached to each other in sequence to form chains. This is also a character used in taxonomy. Microconidia are usually moisture-borne, but they can be air-borne, usually for relatively short distances. Blastospores are more likely to be carried further in air currents. Microconidia are cited as involved in the internal transport of the wilt pathogens within the vessels of the vascular system of the host plants, where microconidia (or at least microconidia-like bodies) arise by budding from hyphae that penetrate the pits in the walls between adjoining vessel cells. Thereafter the microconidia formed proceed upward with the transpiration stream within the vessel until they meet another cell wall, whereupon they germinate to form hyphae and to repeat the process. This process is important to the movement of the pathogen in the progress of disease within the host plant.

Resting spores, chlamydospores, exist in some, but not all *Fusarium* spp. Such spores are more or less sphaerical, approximately 7-16 μ in diameter. They occur often singly, but sometimes they are doubles or are even in chains or in big clumps in some species. They have thick, double, often very rough cell walls, and their cytoplasm contains a great deal of nutrients, as is evident by oily globules therein. Microscopically the walls appear as light yellowish in color, but when viewed *en masse* macroscopically they are brown. Thus large clumps in culture may appear as brown clumps, sometimes below the agar surface. They form in conidia or in hyphae, either terminally or intercalary, and appear usually when the available nutrients are becoming depleted and the culture is already old. They can also form when some conidia are placed in a tube of distilled water and left in a dark place for about one week. They will be at the bottom of the tube, where the conidia have settled. As they form the content of the hyphae or conidia which bear them becomes empty, and eventually lyse. These resting spores are usually the ultimate soil-borne propagule of the true soil inhabitant members of the genus and of all such Fusaria that are

capable of producing them. In soil, chlamydospores nearly always embedded in or on bits of plant debris, remnants of previous crops. So it seems that even these spores with their rough heavy walls need protection from all of the antagonistic organisms and their antibiotic bi-products existing in the soil and along with that, the absence of sufficient nutrients. Further significant in soil, it is only with application of fresh nutrient will they come out of the resting state, germinate, to produce fresh hyphae and grow.

Some Fusaria are capable of producing a sexual stage. Perithecia bearing ascospores may appear in nature and in culture under certain specific conditions, such as proper lighting, temperature and moisture. Most mycologists recognize at least four different ascomycete genera, all in the order Hypocreales--- *Gibberella*, *Nectria*, *Calonectria* and *Micronectriella*. Thus, when you encounter these names, they all may well have a *Fusarium* as their imperfect stage. There are often other synonyms used by mycologists for these ascomycete stages. Ascospores may be carried great distances and are therefore the most distinctly air-borne of all the spore types that are encountered among fungi that are identified as *Fusarium* in the imperfect form. Ascospores do not usually survive for long periods in the soil. The sexual stages may be either homothallic or heterothallic. If homothallic, the cultures can produce perithecia without the presence of another mating type and, therefore, they may be produced in a pure culture. Heterothallic fungi require that two mating types be present to form perithecia, one acting as male the other as female. This implies that there is much more likely to be variability in the subsequent generations from the ascospore progeny, albeit that matings are likely to be less frequent.

Gibberella produce perithecia that are blue-black in color. Macroscopically they may appear as black, but their dark blue color can be seen when viewed under transmitted light, such as that from a microscope. The eight ascospores per ascus are smooth-walled, fusiform, slightly curved, with blunted ends, hyaline and usually three-septate. *Nectria* perithecia are usually red to red-brown. Their asci usually contain eight ascospores that are hyaline to tan, ellipsoidal to obovate, not curved, usually one-septated and striated. *Calonectria* species have bright yellow to orange colored perithecia and possess asci containing 4-8 ascospores, whose shape and septation resemble that of *Gibberella*. In *Micronectriella* the light brown-colored perithecia remain immersed within the plant tissue or substrate with only a papillate ostiole protruding. Ascospores, usually eight per ascus, are hyaline and, like *Nectria*, ellipsoidal with a central septum.

Fusarium taxonomy has always been and continues to be a controversial subject. This paper, however, principally concerns those soil-borne members of the genus, about whose classification there is more apt to be some agreement, *Fusarium oxysporum* and *Fusarium solani*. Among other soil-borne species, of which the pathogenic forms are those of cereal crops, the matter will be dealt with as such fungi are mentioned, i.e. soil-borne members of *Fusarium roseum*, *Fusarium tricinctum*, *Fusarium episphaeria*, etc. (all sensu Snyder and Hansen). A complete taxonomic discussion is beyond the scope of this paper, but may be obtained by consulting the books of Wollenweber and Reinking [149], Bilai [15], Booth [17, 18], Joffe [63, 64], Nelson, Toussoun *et al.* [88, 91, 92, 133], Gerlach and Nirenburg [44], the collective papers of Snyder and Hansen [114, 115, 116], Huang, Sun *et al.* [51, 52, 53], Gordon [45], referenced here, and others.

THE SPECIES *FUSARIUM OXYSPORUM* DESCRIBED

The members of this species produce both macro- and microconidia. The macroconidia are typically 25-35 μ long ×3-5 μ wide, dorsi-ventrally curved, sickle- shaped 3-5 septated, with thin walls and tapering toward the ends which are quite pointed (in fact the term "oxysporum" roughly translates to "pointed spores"). The widest part of the spore tends to be about a third of the distance from the apex to the base. The basal cell possesses a marked foot or sometimes an appendage. In most cultures macroconidia are borne in definite sporodochia, however in some cultures, namely those of the 'Orthoceras' group (Subsection "Orthoceras" in Wollenweber's Section "Elegans" [149], which to most of us nowadays all belong to the species *F. oxysporum*) sporodochia are hard to find in culture. Indeed, these *F. oxysporum* varients produce macroconidia rather sparsely, and the cultures are usually white mycelial masses bearing mostly microconidia; there are some well-known pathogens of this type. *F. oxysporum* also produces microconidia abundantly, which tend to be 5-12 ×3-5 μ in size, oval to ellipsoidal, occurring singly or in aggregates as false heads held together by moisture, which dries out to become a powdery substance dispersed in the mycelium. *F. oxysporum* is light in color or takes on tints of the stroma coloring, commonly of purple-red, blueish-grey or yellowish-tan. The stroma is plectochymatic and may erupt with blue-black or brown sclerotia. In addition masses of sporodochial macroconidia, pionnotes, can be seen macroscopically as small yellow to orange upraised mats borne on the tops of sclerotia in a culture. In *F. oxysporum* chlamydospores are also formed, sometimes in great abundance. They are intercalary or terminal in stroma hyphae or in conidia. No perfect stage is known in this species. Figure 1 contains drawings of some typical *F.*

oxysporum spores made over the years from isolates grown on typical laboratory media. They, by no means describe the whole range of spores that may be seen of various isolates on different media and conditions, but are merely examples to accompany the foregoing discussion.

The species *Fusarium oxysporum* in its many pathogenic forms is arguably the most damaging species of the genus from the standpoint of plant diseases. Furthermore, this species is usually the most abundant *Fusarium* in the plow layer in consideration of the number of propagules per gram of soil from highly cultivated field soils. The numbers of *F. oxysporum* to be found in an ordinary cultivated field are so high that often when we gaze at a 40 acre field, we are looking down at soils containing several buckets-full of chlamydospores of *F. oxysporum* therein. However, fortunately, the majority of these soil propagules are not those of the highly pathogenic wilt forms, but rather they are soil saprobes, or better stated they are the secondary invaders of lesions caused by other fungi, or inhabitants of other soil organic

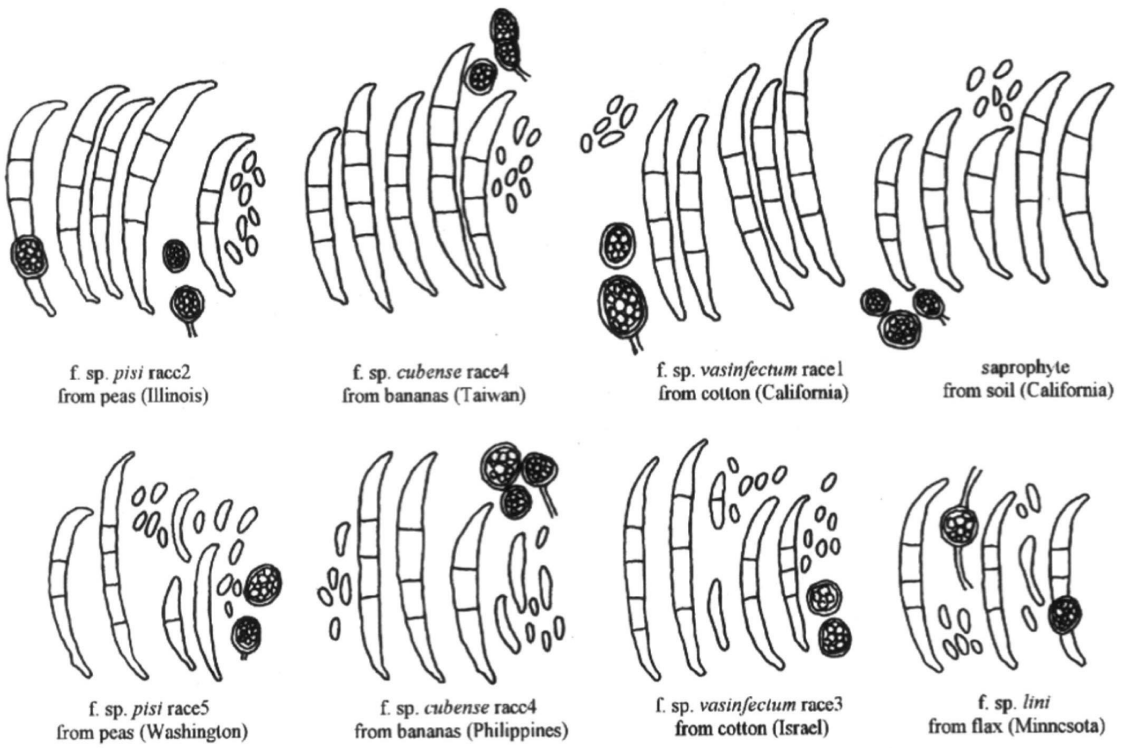

Fig. 1. Outline sketches represent certain *Fusarium oxysporum* forms from PDA cultures. (Approx. 250X) Top row shows 'Oxysporum' types. Bottom row shows 'Orthoceras' types.

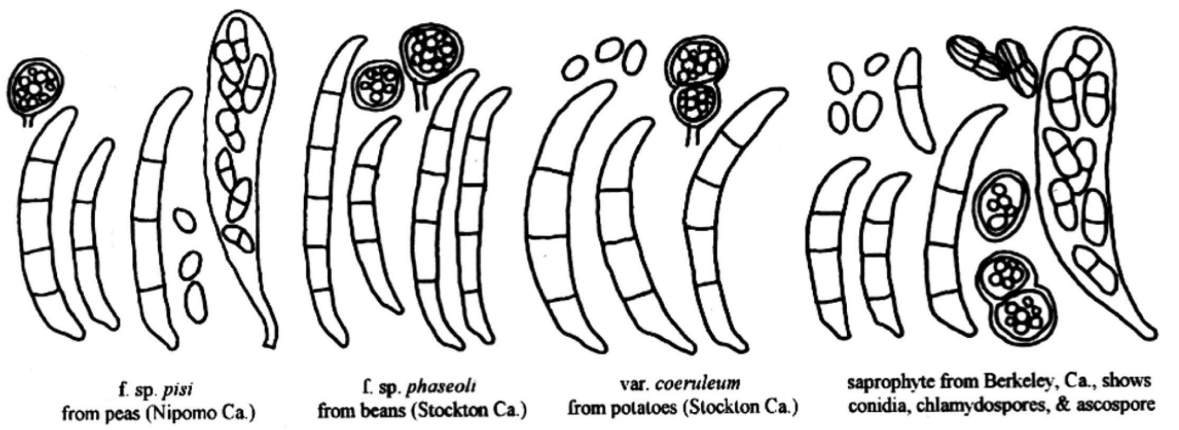

Fig. 2. Outline sketches showing *Fusarium solani* isolates grown on PDA. (Approx. 250X)

matter, or at most, parasitic invaders of the cortex of plant roots, causing no lesions or obvious damage. The damage that they may cause comes only in extenuating circumstances. They are probably doing more good than harm in breaking down old crop residues, and in their aggressive nature may well lessen the crop damage from virulent fungal and bacterial pathogens by successfully competing with them for nutrients and domain.

However, there are to be found a few cases where a cortical rot rather than a vascular wilt is caused by a pathogenic member of *F. oxysporum*; one such rot is tomato crown rot, caused by a specific fungus that has been named *F. oxysporum* f. sp. *radicis-lycopersici*[61]. This disease can be very damaging, but it occurs most often in fields where tomato culture has become particularly intensive and also in hot house grown tomatoes. Further to this there are vascular wilt fusaria, such as some of those that attack bulb plants, and also with wilts caused by *F. oxysporum* f. sp. pisi race 2 , wherein a significant rot of below-ground tissues accompanies the vascular invasion.

THE PATHOGENS, "FORMAE SPECIALES"

Morphologically, the virulent wilt pathogens that cause havoc in the production of so many crops are not easily distinguishable in culture from common soil-borne *F. oxysporum* propagules that populate all agricultural fields, yet they represent an overall small minority among total propagule numbers of this species present in soil. These propagules are unevenly distributed in field soil[135], populations varying from one area of a field to another, which is reflected in the spotty appearence of disease in the field. This depends not only on the place of introduction of the pathogen, but also on its ability to establish at the site. Important to establishment of the pathogen is that the immediate environment favors the fungus, such as soil moisture and temperature, a dearth of antagonistic and competitive microflora, and above all, a conducive soil type[113]; these factors may be interrelated. Sometimes the first observation of an occurrence of a *Fusarium*-wilt infestation is seen as just a small area in the field affecting only a few plants, but if the field conditions are conducive to the fungus and when the susceptible crop is planted year after year, this spot can be seen to expand and other spots are observed, until after some years these spots coalesce, rendering a large part of the field nonproductive. Moreover, pathogen propagules can renew themselves without causing vascular symptoms by superficially invading root cortical tissue of many kinds of plants[2] or, also similarly to the saprobes albeit less aggressively, they secondarily enter lesions caused by other organisms[88]. Consequently, once a wilt pathogen is established in the soil, it remains present for a very long time[107, 110, 113, 123, 144] perhaps forever, whether or not the susceptible crop is raised.

Fusarium wilt pathogens are definitely host specific and, denoting this, the term "forma specialis" followed by another name designating the host, is added to the binomial "*Fusarium oxysporum*." Thus the pathogen wilting garden peas is called *Fusarium oxysporum* forma specialis pisi (*F. oxysporum* f. sp. *pisi*), that of tomato is *F. oxysporum* f. sp. *lycopersici*, etc. This same type of nomenclature is used in host specific forms among many plant pathogenic fungal species. Wilt fusaria have many different hosts among a widely differing plants. Booth[17] in his 1971 monograph lists 82 formae speciales of *F. oxysporum*, and more have since been described, including *F. oxysporum* f. sp. *mordicae*, described in 1983 by Sun & Huang[128] on bitter gourd in Taiwan. There are likely to be more yet to be found.

RACES ENUMERATED.

Within the formae speciales there have been races designated, based on more detailed host range suceptibilities to all of the different clonal types of pathogenic isolates in a form. This came about because susceptibility/resistance vary within different cultivars of a given crop[140], as well as between different isolates of a wilt pathogen form. For example, historically 'Bonny Best' tomatoes were found to be susceptible to *F. oxysporum* f. sp. *lycopersici* (race 1). Then 'Panamerican' tomatoes were developed, using crosses between *Lycopersicon esculentum* and a wild plant relative, *L. pimpinellifolium* to obtain a gene, the I-gene[102] for complete resistance to the pathogen. However when pathogenicity again occurred after race 2 was discovered a new gene was sought, the I-2 gene[1], which was incorporated into the 'Walter' tomato, resistant to both race 1 and race 2 of *F. oxysporum* f. sp. *lycopersici*[118]. The type of resistance just described, where a single gene is replaced for a resistant characteristic is called "Vertical Resistance". Previous to its development there existed "Horizontal Resistance", wherein plants tolerant to the particular wilt were selected from among those left standing in a field where serious wilting had occurred. In such plants multiple genes are likely to be involved in the resistance and as such resistance is not absolute; some disease was inclined to occur under conditions environmentally unfavorable for the host. On the other hand, there is less chance of the entire crop being affected by a rather sudden appearance of a new race of the fungus, as can occur in single gene resistance[140, 141]. Some tomato lines developed by this type of search for resistance such as 'Marglobe' and 'Rutgers' were grown for many years and are still planted in certain areas. In culture the

members of *F. oxysporum* f. sp. *lycopersici* produce typical sporodochia bearing sickle-shaped macroconidia. A feature distinctive to these cultures is that the sclerotia that form are white, rather than the typical dark blue-black or brown in most *F. oxysporum* cultures.

Similar to that in tomatoes, in peas the cultivars 'Perfection' and 'Alaska' were selected early on for their tolerance to *F oxysporum* f. sp. *pisi* race 1. Later these and other lines succumbed to wilt when race 2 of *F. oxysporum* f. sp. *pisi*, the so-named "near wilt" pathogen appeared. An interesting feature of this disease is that the host symptoms are slightly different, alluded to above. In near wilt there is wilting and yellowing, but also the plants develop a more significant root rot than with race 1 infection, and the vascular symptoms may remain confined to the lower above-ground nodes of the plant. Moreover, the race 2 causative fungus is culturally very different from that of race 1 isolates, in that it is a typical 'Oxysporum', bearing plenty of sporodochia with sickle-shaped macroconidia and dark-colored sclerotia. On the other hand the race 1 isolates are definitely 'Orthoceras' types; in culture sporodochia are obscure, small and.have developed in separate sites long ago. Often, however, a new race emerges within a field in which another race of the same pathogen forma exists, and so resembles that race as to be indistinguishable from it culturally, as in blackeye pathogens *F. oxysporum* f. sp. *tracheiphilum* races 2, 3, and 4. Such is also the case in *F. oxysporum* f. sp. *pisi* races 5 and 6 which appear to have been derived from race 1 (races 3 and 4 were each reported to occur one time at one site each, and no cultures have been made available for either of these claimed races). Thus a new race may well arise among the field population of the existing pathogen and in response to the currently grown line of the crop, developed for resistance, and such pathogens probably differ by only a few genes from the original pathogen that was well established in the field. It is of particular interest that DNA RAPD research has also concluded that race 2 differs genetically from the other races in the pea form[46].

The numbering of races within pathogenic forms is a convenient way to refer to groups of isolates, differing in host range from one another. They vary by few or many genes. Sometimes different race numbers within a forma speciales are assigned to groups of isolates whose host ranges include different species, even that of different plant genera; thus "race 1" of *F. oxysporum* f. sp. *cubense* was assigned to the pathogens, causing Panama disease in many of the triploid banana cultivars of *Musa acuminata* (AAA), such as 'Gros Michel'; race 2, on the other hand, is virulent on some of the cooking type bananas that have gene sets of *Musa balbisiana* in their genome[98, 99, 125, 139], such as the cultivar 'Bluggoe' (ABB). Race 3 causes a wilt of *Heliconia* spp. (*H. caribaea*) and is only mildly parasitic in bananas[138]. Race 4, as virulent pathogens of 'Cavendish' cultivars, an AAA group here-to-fore considered PD resistant, has been known only since l967 in the Canary Islands[124] and in Taiwan[126, 127] and in 1974 in the Davao area of Mindanao of the Philippines, where damaging disease was seen on 4 plantations, Lapanay, Hijo, Farmington and Evergreen (J. Silva, personal communication). Although previously there had been noted a few small sporadic Panama disease observations in 'Cavendish' plantings in Central America and elsewhere, the disease was confined to few plants and did not spread. Poor growing or environmental conditions were assessed as being responsible for some race 1 infections on this usually resistant crop. When Panama Disease was found on 'Cavendish' lines in Taiwan and the Canary Islands, it was believed that these subtropical sites were marginal for banana production, being too cold or grown on poorly drained land, but when the disease appeared on Cavendish lines, especially on the Lapanay plantation in the Philippines where the soils are deep and rich, and since all southern Mindanao disease sites have a good climate for banana growth, the marginal growing conditions argument did not apply. It was of some interest that according to Philippine Packing personnel, who were responsible for production of the plantations, these sites had all been abaca plantations in the past and Reinking had identified the pathogen on abaca in the Philippines as *F. oxysporum* f. sp. *Cubense*.

Kinds of 'Horm Plantain' or 'Platanos' AAB (*Musa paradisiacal* L. and *M. corniculata* Lour.) are very important staple crops in much of the world, including Latin America, but are resistant to all known PD races. However another AAB type, the popular dessert banana 'Silk' and 'Latundan' are very susceptible to *Foc* race1. The 'Latundan' of the Philippines is also susceptible to race 2[139].

The most usual means of spread of Panama disease in bananas, a vegetatively produced crop, is though the movement of infected corms from place to place. After race 4 became recognized in the Philippines on several of the large 'Cavendish' plantations, there was an accusation that one of the large companies had introduced the pathogen from Taiwan by bringing in infected planting stock. However, subsequent investigations showed that the *Foc* race 4 from all of the heavily diseased areas then isolated in Taiwan produced sporodochial cultures. Moreover they all appeared to be of the same clonal type, all bearing rather large sclerotia and a pionnotal mass of macrocodia at the center of the single-spored isolates. On the other hand, all of the isolates from the four large

plantations in the Philippines, where the disease was first observed, also all closely resembled each other, but were of the 'Orthoceras' type, the fluffy white colonies produced few sporodochia and thereby few macroconidia and only very small blackish sclerotia, borne on the stroma surface (See Fig. 3). Furthermore, the isolates from the Philippines had a special biotin requirement for growth initiation beyond that of the Taiwan isolates. The Taiwan isolates, on the other hand, are typical sporodochial *F. oxysporum*, with many large stromatal sclerotia and sporodochia, and on PDA agar single-spored isolates bear a distinctive large central sclerotium topped with a mass of light yellow macroconidia (S. N Smith, unpublished data). Since these dates in the 1960s and early 1970s race 4has been found elsewhere, such as in Australia, South Africa, Central America and in Florida[100].

Since the advent of more modern fungal research techniques, such as DNA fingerprinting and defining vegetative compatibility group (VCG) studies[101], fungal groupings have been shown to be more diverse and complex than previously known and when Australian workers, Benley *et al.*[14] studied over 300 isolates of a world-collection, they found that in the 9 different DNA groups (DFG), I to III contained all of the race 4 isolates (appear to be similar to those we had isolated in Taiwan being in DFG III and those found in Davao of the Philippines in DFG II), DFG IV to VIII contained almost all of the races 1 and 2 isolates and DFG IX contained *Heliconia* isolates, and oddly a few isolates from Western Australia from 'Cavendish' bananas It appears that like the host plants, the Fusaria that attack bananas and their close relatives are also geneticallycomplex.

Because the bananas of commerce are triploids, breeding for resistance to plant diseases is very difficult.

There were some attempts to make tetraploid plants and cross them, but it was hard to get an agronomically satisfactory crop plant. Cultural practices, including adding soil amendments to favor the plant over the soil-borne pathogens have been used with, at best, limited success. Even altering the environment by long term flooding was tried with only a short term advantage to the subsequent crop, after which Panama Disease returned with a vengeance-hardly cost effective. Latterly, the combination of tissue techniques along with radiation of small plantlets, etc. has been used with some success. Fruit produced at the Taiwan Banana Research Institute, by cultivars 'Tai Chiao No. 1' and 'Formosana', resistant to *F. oxysporum* f. sp. *cubense* race 4 and derived through somoclonal variation, have been well received by customers in Taiwan and the Japan[55]. Extensive reviews of Panama Disease are available in earlier books of Wardlaw[144] and by Stover[123] and later that edited by Ploetz[98].

Another *F. oxyxporum* special form whose race nomenclature crosses species and even genera lines is the well-known cause of a cotton wilt, *F. oxyxporum* f. sp. *vasinfectum* (Fov), the very first vascular wilt pathogen

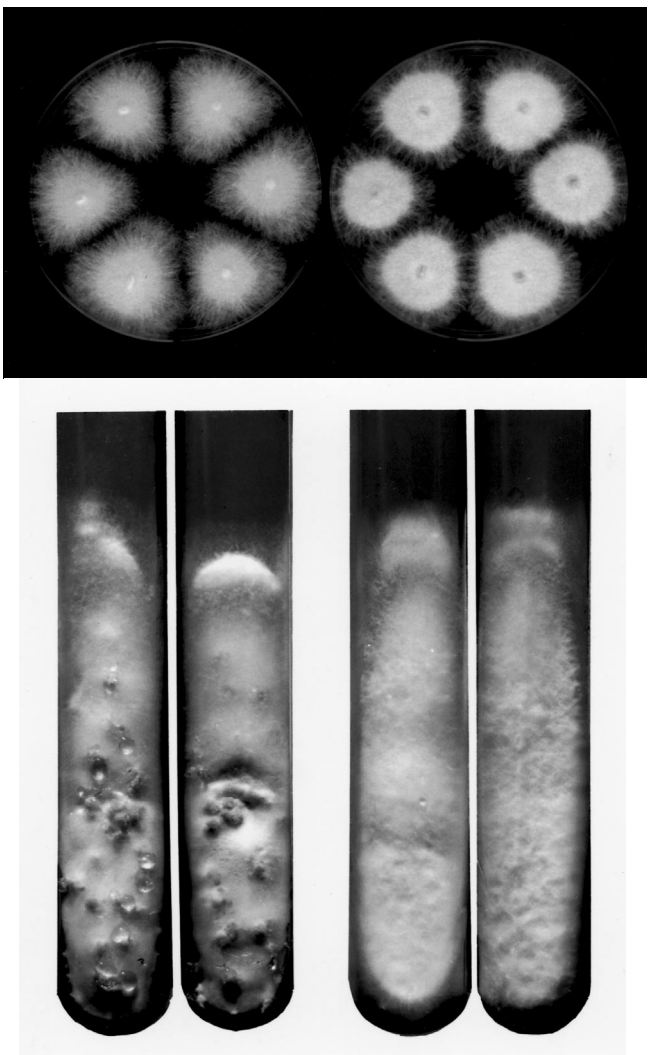

Fig. 3. Cultures of *Fusarium oxysporum* f. sp. *cubense* race 4 from Davao Philippines (left) and Taiwan (right). The PDA plates shown on the top feature single-spored cultures from 6 different isolates, 6 days old. Even at this early age cultural differences are noticeable between isolates from the two locations.
The tubes shown below the plates contain 6 week old single-spored cultures. Those from Davao appear to be typical of cultures which from sporodochia only sparsely, appearing mycelial macroscopically, whereas the Taiwan isolates produced ample sporodochia and pinnotes, throughout and profusely in the central area near to the point where the initial sporling had been transferred. Note that patch mutations, often observed in cultures of this age, are present here, particularly noticeable in the left central area of the left hand Taiwan culture tube.

ever described [7]. Furthermore, similar to the banana wilt pathogens, the genetics of members of this forma specialis, whose races have also been analyzed by VCG and RAPD methods [6, 34], is also complex like that of its hosts.

F. oxyxporum f. sp. *vasinfectum* races 1and 2, sometimes called race "A" [6],attack Upland cotton (*Gossypium hisutum*) lines. The pathogens are typical sporododochial cultures. Many of them, similarly to some of the banana with Fusaria produce a particularly fragrant volatile substance in culture. Often it is so powerful that if just a few such agar culture tubes are present they can be detected the minute one walks into the lab. Field selections in upland cotton for resistance to this disease have been made by Orton since 1900 [93]. Thus so many of the common cotton cultivars in use in U.S.A.. have some inherited tolerance to the pathogen. It is also significant that this wilt disease occurs in sandy and sandy loam and is complexed with root knot (RK) nematode infection [67, 108]. In fact, clearing out the nematode infections usually prevents wilt disease, no matter how high the soil *Fov* population may measure after fumigation [38, 66]. The disease is manifested by stunted plants, epinasty of the petioles, followed by inter-veinal yellowing; later, leaves fall off leaving nearly bare sympodia and the plant dies, often starting from the top. As in most severe wilt diseases, cutting through the plant reveals a dark brown stele extending its entire length. Race 1 of this pathogen is distributed throughout upland cotton growing areas in the U. S. A., Mexico, Africa, Russia, China, and South America. It is a well known disease, although it was not found in California, until the late 1950s [39]. Race 1, but not races 3 or 4 [47] may also cause a serious wilt of okra (*Abelmoschus esculentus*). Indeed, agricultural scientists often grow okra on a site destined to becoming a cotton wilt research plot because the crop is so efficient in building up both *Fov* propagules and RK nematodes, *Fov* has long been known to be carried on a low percentage of seeds [30]. However it enters by growing into seed coats via boll contamination in the field rather than penetrations through vascular connections in the seed pedicel. Race 2, which is reported to be similar to race 1, but can also affect certain soybean and tobacco cultivars [3] is more obscure. In addition an *Fov* particularly virulent to upland cotton is found in Brazil and designated as race 6 [5].

F. oxyxporum f.sp. *vasinfectum* race 3 attacks the so-called Egyptian cottons, the lines of *Gossypium barbadense*, whose lint consists of longer fibers than those of *G. hirsutum*. This race has also been known for a long time, having been first described by Fahmy [33] in the Nile Valley of Egypt in 1927. Race 3 was known to occur only in Egypt and the Sudan for many years, but is now known in Israel [26], in Uzbekistan, China [50] and elsewhere. During much of the twentieth century through agreements with the government of Egypt, the growing of Egyptian cottons were very restricted in USA, only allowed in Pima County Arizona, where there had been a long held precedence in growing a line of *G. barbadense* called 'Pima'. Perhaps elsewhere similar restrictions applied. This restriction probably prevented a wider distribution of *Fov* race 3.

Unlike the disease in upland cotton, wilt on *G. barbadense* ismore likely to occur in deeper, heavier soil types and is not associated with RK, but may occur with reniform nematode infections [29, 31]. The causative fungal cultures are also different. They appear to belong among 'Orthoceras' *Fov*, rather than with the strongly sporodochial cultures as do those of race 1. Similar to race 3, there has been described in the Sudan a race 5 [56], which has a broader host range than race 3, infecting both 'Sakel' and 'Ashmouni' cotton cultivars.

Both host cottons *G. barbadense* and *G. hirsutum* crops had their origins in prehistoric times in America, respectively in Peru and Mexico (even though *G. barbadense* has been grown in North Africa for centuries). They also are both allotetraploids, that is they have four sets of each kind of chromosome. The "Indian" cotton crops, on the other hand are diploids. They are infected by yet another *Fov*, namely race 4. Historically this race has long been known in India where crops of *G. arboreum* and *G. herbaceum* have been grown for thousands of years and recently has been implicated to occur in a limited area of China and Central Asia [75], but it was known as avirulent on the commonly grown Egyptian cultivars [4, 28]. However at present, now with the many cotton variety restrictions lifted along with increased globalization isolates bearing genetic relationship to race 4 have been found elsewhere. It is because of DNA relationships that race 4 is now believed to cause disease on 'Pima' cotton, today widely grown in California's San Joaquin Valley. Unlike other Egyptian cotton, 'Pima' seems to have some resistance to race 3 *Fov*, (37, and Mike Davis, personal communication). Is this an example that *Fusarium oxyxporum* pathogens do not necessarily follow the rules and act according to how to we humans assign race numbers to them, being capable of much wider mutation? To answer these questions it seems probable that more attention should be given to the relationships between genetics and cultural characteristics and how these factors bear on host susceptibility. Moreover, in todays climate it seems that the term "race" takes on different meanings to different scientists. In the case of *Fov*, the original race 4 was described as having a narrow host range attacking, only diploid cottons [4, 5]. Now it is described going to a large variety tetraploid cottons, based on DNA likenesses. It seems that a word other than "race" should be used for

defining DNA types, leaving "race" to continue denoting host range rather than DNA likeness groups.

Fov race 7 and 8 are found in China [21]. For a long time the status of cotton wilt China eas not known in the western world and I doubt that it had even been well studied in China during the years of the cultural revolution. Now we known that it not only exists there, but their races also appear to be spreading throughout the world. These observations along with the fact that a particularly virulent *Fov* strain, first appearing in Australia in 1994 [76], which they report as being genetically close to Brazilian race 6, lead to the conclusion that this wilt is still very much a threat to the world cotton crops. The Australian strain may have a distant relationship to race 1, but it apparently prevails in a great variety of soil types and without the RK influence. Given the inherent complexity of the genetics in both cotton plants and of their *Fusarium* wilt pathogens and also the fact that with globalization comes the increased movement of planting seed (legally and illegally) that may contain low levels of these occasional seed-borne pathogens, it is no wonder the problem increases.

On occasion a group of two or more similar wilt Fusaria, virulent on similar host plants, had originally been described and named as separate formae speciales, but later other scientists have deemed them as races of the same pathogenic forma. There usually exists in them a low degree of pathogenicity across various host lines. A case in point is that of *F. oxysporum* f. sp. *brassicae*, originally known only as a pathogenic form on cabbage and cauliflower, but f. sp. *raphani*, which goes to radishes and f. sp. *mathioli* which wilts stock also existed. Now the latter two forms are generally embraced as merely races of *F. oxysporum* f. sp. *brassicae*. The same may be said for the pathogens of watermelon and cantaloup, although most people still like *F. oxyxporum* f. spp. *niveum* and *melonis*; nevertheless the fungi are similar and there can be a low degree of cross infections among these hosts and pathogens, that is, in trials some watermelons may show very mild disease symptoms when infected with the cantaloup pathogen and visa versa, especially in the seedling stage. It can be reiterated that *races* and perhaps all taxonomic designations exist so that scientists can communicate, rather than they're being edicts handed down by God.

HOW DO PATHOGENIC FUSARIA PERSIST IN SOILS THROUGHOUT ADVERSE ENVIRONMENTAL CONDITIONS AND AMID COMPETING SAPROBES AND ANTAGONISTIC MICROFLORA?

Because soil-borne pathogens do not have an opportunity to spread as rapidly as do the above-ground organisms, it takes a longer time to get started at a site, Therefore they need "staying power" in their environment, the soil. They also need some special advantages in order to make it to the plant rhizoplane where they may initiate an infection. However, if they do and are able to colonize the stele of a living plant, they have a most distinct advantage over all the other flora of that environment. Inside this plant tissue there may well arise the next generation of inoculum for subsequent susceptible crops. This occurs as the plant dies or senesces, the occupying vascular fungal hyphae move out into the heavier-walled sclerenchyma and parenchyma tissue and form their chlamydospores. When refuse from this crop is returned to the soil, much of it will rot away, but the resting spores of the pathogen lie protected in this resistant tissue for long periods.

The fact that chlamydospores are the survival structures of many Fusaria existing under rather harsh conditions has been known for a long time. Wollenweber and Reinking mentioned this function in their 1937 monograph [149]. It was later learned that when present in field soils, chlamydospores germinate only in response to nutrients secreted by the plant roots. Studies on this response began is the late 1950s, early reports by Richard Jackson concerned the germination of conidia and chlamydospores and hyphal growth of fusaria (including *F. oxyspourm* f. sp. *pisi*) and other common soil fungi in the rhizospheres of pea and other seedling roots [57, 58, 59]. Hyphae produced by these spore germlings grew toward these roots where they presumably might sometimes be able to penetrate. Although Jackson's work was initiated mostly to study the roles of microbial antagonism versus competition, it stimulated the rest of us toward further investigations of activities of pathogenic fusaria in a hosts rhizosphere.

During the middle of the last century soil microbiologists of Britain and the Commonwealth Nations were often the philosophical leaders in the ecology of soil-born fungi. David Park [94, 95] introduced us to the term soil inhabitant as "an ecological saprophyte able to continue its existence and activities in soil, colonize using dead substrata and maintain itself ... without the necessity of an imposed parasitic phase". S. D. Garrett [40, 41, 42] defined competitive saprophytic ability as characteristics that allow the soil organism to colonize dead organic matter and thereby succeed to exist in plant refuse in soil.In 1953 Dobbs and Hinson [28] described "the widespread fungistasis in soils which causes most fungi to live most of their lives in soil in a dormant state. Harry Katznelson, a Canadian, made observations on the rhizosphere effect, [71,

[72] the influences of the plant root on microflora, the influence of rhizosphere organisms on the plant, and which kinds of micro-organisms are stimulated to grow there. Jack Warcup, and Australian [143] studied which kinds of fungi were inclined to appear in plate counts and which kinds were missed even when present. Further he observed that *Fusarium* spp. frequently developed colonies from humus particles in soil and that such propagules were less apt to be killed by desiccation than were naked spores of the species in soil. Making more investigations along this line, we in Berkeley [87] demonstrated that naturally occurring soil humus propagules of wilt *F. oxysproum* all contained a chlamydospore or group of chlamydospores buried deep insde decay-resistant plant tissue. Referring to the earlier discussion, in the case of *F. oxysporum* wilt propagules, no doubt much of such soil residue came from parenchyma/sclerenchyma tissue of the original host attacked by the pathogen.

Groundwork on the pre-mentioned ecological studies had been laid on studies of antibiosis and antagonism by soil micro-organisms earlier in the last century and some of this work was done by Americans. Briefly mentioning relevant research, Waksman [137] had studied microbial antagonism and antibiotic production, often involving actinomycetes, since 1916. Weindling [146] and others discussed the antagonistic effects associated with *Trichoderma* spp. and similar soil fungi since the 1930s.

In the 1950s the U.S. government greatly increased funding to research the effects of crop residues on plant diseases, including those caused by *Fusarium* spp.. The projects were divided regionally within the country, grants going to agricultural colleges in each region, and supporting many graduate students. During the first year or so our progress at U. C. Berkeley was rather slow. Many of us plant pathologists were not sufficiently familiar with all the works of the soil microbiologists (such as some of that cited above) and students with chances to earn a degree elsewhere left for greener pastures. In time with the leadership of Bill Snyder and by cooperating with each other in Berkeley and elsewhere [87, 117, 135], we got the hang of it, learning new things about soil-borne fusaria, host plants and crop residues [32, 96, 97].

Soon following came wider communications between scientists. In the west Bill Snyder, together with Bill Kreutzer and Bill Rader, both from Shell Chemical in Modesto CA and Kenneth Baker, then of U. C. L. A., started a group, open to all that who were interested in informally discussing the control of soil-borne pathogenic fungi. This Soil *Fungus Conference*, now after over 50 years, still continues to meet and talk informally on controlling soil fungi. Mostly, as an outgrowth of these meetings, important international symposia were held in Berkeley CA in 1963 and next in London in 1968 (in conjunction with the first International Congress of Plant Pathology); proceedings were published [9, 132]. These meetings and those since brought together plant pathologists and microbiologists from all over the world. Such communications stimulated much research including that leading to some of the knowledge mentioned here, concerning how the large spectrum of *Fusarium* pathogens exist, survive in nature and infect their varying hosts.

Depending on the particular *Fusarium*, the chlamydospores of forms are more likely to germinate and produce growing hyphae in some soils than in others. Soils which allow such germination and growth are considered to be conducive to the fungus, and if it is a vascular invader, disease is more likely to occur in such soil, all other things being equal. Soils less likely to allow chlamydospore germination and growth are said to be suppressive to the fungus and hence disease that it may cause. (We may still assume, however, that all soils are naturally quite suppressive to fungal growth, and these terms denote only degrees of differences between them.) Usually the lighter sandy loam soils with a low pH are more conducive to the common sporodochial *Fusarium oxysporum* formae, but the type of clay mineral in their make up [122] and soil nutritional factors are also involved. As may be expected, the common soil-borne saprobes of the species are more inclined to possess chlamydorpores that germinated and grow more prolifically than do those of pathogens [112]. In an earlier concept of this same phenomenon, Panama disease researchers in Central America, notably Knutson, Volk and Reinking in the 1920s and '30s found some soils to be more "resistant" than others to infestation of the with fungus, as Stover reported [123], in abstracting old Untied Fruit Co. data. Wardlaw and McGuire [145] reported soil ytpe influences in Caribbean Island banana wilt. Whether a soil was considered short-lived or long-lived depended upon how long 'Gros Michel' bananas might be grown before succumbing to Panama disease. The entire subject of soil suppressiveness is extensively reviewed elsewhere [8, 80].

INFECTION PROCESSES.

Fusarium hyphae tend to grow toward the roots of plants, in response to rhizosphere nutrients exuded by plants and they often proliferate on root surfaces before they can enter the cortical tissue. Small rifts in the root cuticle or a root injury may allow a fungus this entrance. For *Fusarium* wilt pathogens it is necessary for the hyphae to penetrate through the cortex and get inside the vessels of the host in order to cause disease. Considering the normal defenses of living plants and the resistance of the tissues involved, this is a formidable task for any

prospective invader. A comprehensive review of our knowledge of how wilt pathogens invade, colonize and eventually destroy the functioning of plant suscepts is clearly presented in Beckman's 1987 monograph [11], "The Nature of Wilt Diseases of Plants" and also in an earlier volume edited by Mace, Bell and Beckman [81]. Here is a brief resume of strategies used by fungi in colonizing a host and the defenses that plants call into play in response to the invasion. Inside the root cortex the pathogen may grow through the intercellular spaces. They can also recognize plant cell components, such as celluloses, and produce cellulases in response, thereby attacking the walls to enter cells. They tend to grow toward pits in the walls. They recognize and adhere to surface polymers of vessels. Plants, on the other hand, respond early to fungal invasions by manufacturing callose and depositing it around paravascular parenchyma pits and vessel walls where the fungus may attempt to enter. Secondary metabolites, harmful to the pathogen, increase and phytoalexins and lignification appears.

Assuming that the fungus has grown through the cortex, upon reaching the stele, entrance into the vessels is not easy. It is greeted by a succession of structural barriers formed by the plant in response to the fungal invasion, as described by Talboys [130]. These defenses consist of the production of papillae and cell wall apposition layers which lignify to become very resistant to fungal enzyme actions. It is only near root tips, where these barriers have not yet formed, that the fungus may enter vascular tissue. Kawamura and Hurano [73] observed penetration of *F. oxysporum* f. sp. *lycopersici* into tomato roots in pockets of wounded meristem tissue destroyed previously after the entrance of RK nematodes, and suggested this is the site whereby the fungus gets into the plants. However, other workers failed to recognize this pattern because of the time differential between nematode infection and fungal entrance. Furthermore, this explanation does not reveal why RK infection is important for some pathogen forms and not others. Hepple [49] found that *F. oxysporum* f. sp. *pisi* infections occurred through cotyledonary bundles, usually as they decayed and became infested with bacteria.

Many *F. oxysporum* members can and do gain entrance into plant vessels, but whether the invader becomes a true pathogen is determined by whether it continues to colonize the vascular system throughout the plant or merely remains localized near its entrance site. The speed at which a pathogen can advance through the vascular tissue is important to its virulence and depends on the individual characteristics of each pathogen and each host. Once inside the vessels, pathogens produce, in addition to hyphae, microconidia and they travel upwards in the transpiration stream. Upon reaching and end wall they germinate at a pit, and a tiny germ tube squeezes into the next vessel cell. Considering the length of individual vessel cells, some being several cm long, a good pathogen that is capable of escaping and avoiding plant barriers can make rapid headway and colonize the entire vascular system by this unique method [12].

In order to thwart the advance of the invader, the plant early on is able to synthesize stress metabolites [13, 23, 24, 82], such as phenolics and phytoalexins, produced by enzymatic hydrolysis of normal plant phenolic glycosides. In resistant plants they are elicited copiously during the first few days of infction, but production soon subsides along with the challenge of the pathogen. In susceptible plants less of these products form early on, but the hosts long continue making them [85]. Phenols convert to lignins to form the afore-mentioned fungal barriers. Phenols also infuse plant cell walls and gels for further inhibition. Also to be found are many phytoalexins, other inhibitors of plant origin. In this category are chlorogenic acids, ipomeamaronne, scopolin, pisatin, phaseolllin, trifolirhizin, flavonoids, tomatine (an alkaloid stearoid), and rishitin (a terpinoid). Vessel occlusions [144] are promoted when gels and gums, infused with phenolics, serve to cut off the transpiration stream and immobilize embedded spores. So important in root vascular tissue, gels arise from pit membranes and perforation plates. Tyloses formation are yet another important type of plant-produced barrier for localizing infections. They develop by extension of paraparenchyma cells into the vessel lumins until they become a type of cross wall. Proper function in walling off disease means the plant must recognize the invader early and contain the infection promptly. It is only the most efficient pathogens that are successful in their role of colonizing the entire plant.

Other substances made by plants in defense of infection are hormonal, indole acetic acid (IAA), ethylene, etc. Such substances stimulate the barrier formation discussed above, and ethylene also can swell pits. Furthermore, they can instigate root and shoot proliferstions and increase plant vascularization. This helps the plant to maintain its transpiration and respiration in vessels having occusions and tyloses formed in response to the invasion. Inaddition plant abscissic acid (ABA) is formed for closing stomates and causing leaf abscissions to prevent further water losses.

Further promoting colonization, wilt fusaria are capable of producing carbohydrases to break down gels, retard tyloses formation and some pathogens can even produce enzymes for breaking down stress metabolites. The enzymes induced in fusaria, which may hydrolyze the gels that occlude vessels to impede the pathogen's movement, are polygalacturonases (PG), cellulose, α-and

β-galactosidases, α-arabinofuron oxidase and β-xylosidases. Not only can gels be broken down, but also there is cell wall maceration, involving an array of fungal produced PG and pectin esterase enzymes too. Moreover they appear in a systematic orderly sequence [10, 11]. The resulting destruction of vessel walls further hampers gel formation. There is an enormous amount literature covering this whole subject, but suffice it to say here that just as plants vary in their complex structural polysaccharides, the various pathogenic forms of fusaria also differ in the amounts and classes of enzymes they are capable of inducing. These varying abilities among the species may account, in part, for their dramatic specificities and surely aid special forms in colonizing hosts. An efficient pathogen must be able to induce formation of these enzymes during the first few days of infection. Otherwise it may not move through the plant fast enough to escape being trapped by tyloses formed. Some of plants own stress metabolites, formed in response to the fungal invasion, namely dinitrophenol and rishitin, may in fact inhibit tyloses formation. There are pathogens that can also enzymatically cleave stress metabolites, such as those in certain *F. oxysporum* f. sp. *lycopersici* strains that break down tomatine [35].

Still another response of plants to infection is to increase their rate of respiration, and as this is important to all the other developments, the faster this occurs (perhaps within the first four days), the more likely it is that the plant will resist the pathogen. If the pathogen becomes contained, the respiration rate returns to normal [48]. However, again there are pathogens capable of repressing plant respiration.

In reflecting on all the fore-mention activities that engage plants in their fight against fungal invasions, it becomes evident that most of the disease symptoms expressed are brought on by the plant itself. The wilting due to the plugging of vessels with gels and gums and the tyloses barriers, the vascular browning caused by the plant phenolics, the epinasty, caused mostly from the plants emission of ethylene, the abscission of leaves caused by the plant-produced ABA, the stunting, the adventitious roots and shoots, etc. are all mainly due to plant reactions to the advance of the invader, as suggested by Beckman [11]. Continued development of symptoms signifies that the plant is losing the battle. If the invasion is sealed off early there are essentially no outward signs that it had occurred and the plant appears healthy. However, even in healthy plants close examination of root tissue may reveal small segments of root vascular discoloration, remnants of a failed fungal attack.

For their part fungi have been reputed to be prodigious toxin producers, but many such substances, so prevalent in culture, are merely staling products and are formed too late to be of much use in the infection process. The most important of such substances is fusaric acid (FA) [43], which is present in most infections [23, 74]. Beckman believes that it can inhibit gel formation in plants. Its presence is linked to early establishment of a pathogen and its production has been inhibited in resistant tomatoes, such as 'Red Currant'. Generally speaking, it is fungal production of hydrolytic enzymes, acting on plant structural components, the ability of pathogens to withstand plant metabolites, etc. and to progress rapidly through the vascular system, which leads to disease, but it is the plants own efforts in protection that may lead to its final demise.

CONTROL.

Pathogenic forms of fusaria do not arise *de novo* in field soils. They are either introduced from contaminated soil or trash brought into a site with seed, equipment, etc., blown in by the wind with dust, or, in the case of vegetatively produced crops, by the use of infected planting stock from another location. Therefore, it is important to avoid such introductions of pathogens into clean sites, if possible, and thereby avoid the need to deal with control measures. Another way that a new pathogen may appear is through mutation from a race of the same form that is already present.

It is most difficult to fusarium wilts. Long rotation periods are rather fruitless because not only dose the fungus persist very long in the soil, but even should its population level drop in the intervening years, it usually reinstates itself again, often during the first season of growing the susceptible crop. Many farmers solve the problem simply by continuing to grow non- susceptible crops in heavily infested soil sites. It is fortunate that so many soils contain sufficient disease-suppressive factors to allow the growth of wilt- susceptible crops, considering the continuing wide distribution of the pathogens. Complete soil fumigation is not only costly, but nearly impossible to do. Even with a very tiny *missed* spot, the whole field soon becomes re-contaminated, because the lack of antagonists there give the pathogens no resistance to spreading. Marois & Mitchell [83, 84] however, found by adding back microbial antagonistic communities and use of proper soil amendments the practice becomes more successful. Soil amendments alone, in the absence of fumigation have been found to be useful in lessening disease incidence, and Sun & Huang [129] have patents on substances used in Taiwan. Especially useful are those amendments that raise soil pH, make soil more friable and support the native competitiflora. Soil nutrition is also involved. At pH near 7, phosphorus, iron and zinc become

more available, so lime may be part of an amendment. Nitrate-nitrogen is less favorable to all soil-borne fusaria than is ammonium nitrogen (See Fig. 4).

Ordinarily incorporation of antagonistic organisms into field soil without adding amendments doesn't work well because cultured organisms applied directly into soil, already containing all of the competitive flora. do not stand a chance of adapting and surviving well there, let alone protecting a host from a resident pathogen However, embedding antagonists in a proper substrate and placement of the material near a root has had some success [19].

Katan *et al.* [70] and others have shown soil solarization to be a successful control method against many soil-borne diseases including fusarium wilts of tomato and cotton. Pullman *et al.* [103] demonstrated that proper and timely irrigations enhance the chances of destroying soil-borne pathogens. Briefly the technique involves securing a 1-3mm thick, clear plastic tarp as a mulch over the moist, fallow field soil, devoid of large clumps, and allowing the sun's power to heat the soil to partially sterilize it. However, to be effective a sufficient number of warm and sunny days are required during the 4-6 week solarization period. The method is effective not only in lowering the population level of several kinds of pathogens, but it also removes much RK [120] and many weed seeds as well, but leaves intact mycorhizae, and such pathogen antagonists as *Trichoderma* spp. and actinomycetes. Therefore, fungal pathogens may be slow to re-establish again after treatment. As in Israel, in the sunny Central Valley in California there are excellent conditions for solarization during the late spring and summer. In a Kern County field, heavily infested with *F. oxysporm* f. sp. *vasinfectum*, after 5 weeks of tarping the soil populations of the pathogen, as well as that of most of the rest of the fusaria present, was reduced by more than 95% in all solarized samples taken from the top foot soil layer [111]. Even two feet down, where all fusarium propagules became sparse, the numbers dropped to almost none. Of interest to me was that while most of the fusaria present died off at about the same rate, *F. solani* numbers remained relatively high. During solarization temperatures below the plastic rose to as high as 48℃ in the top inches of soil. The maximum air temperature during the period was recorded at 40℃. The solarizaion subject has been studied and reviewed over the years by Katan *et al.* [68, 69], and by De Vay and other U. C. personnel, past and present, [25, 36, 105, 120], who held an interest in it, and have reported their work.

There has been some success in controlling wilts by

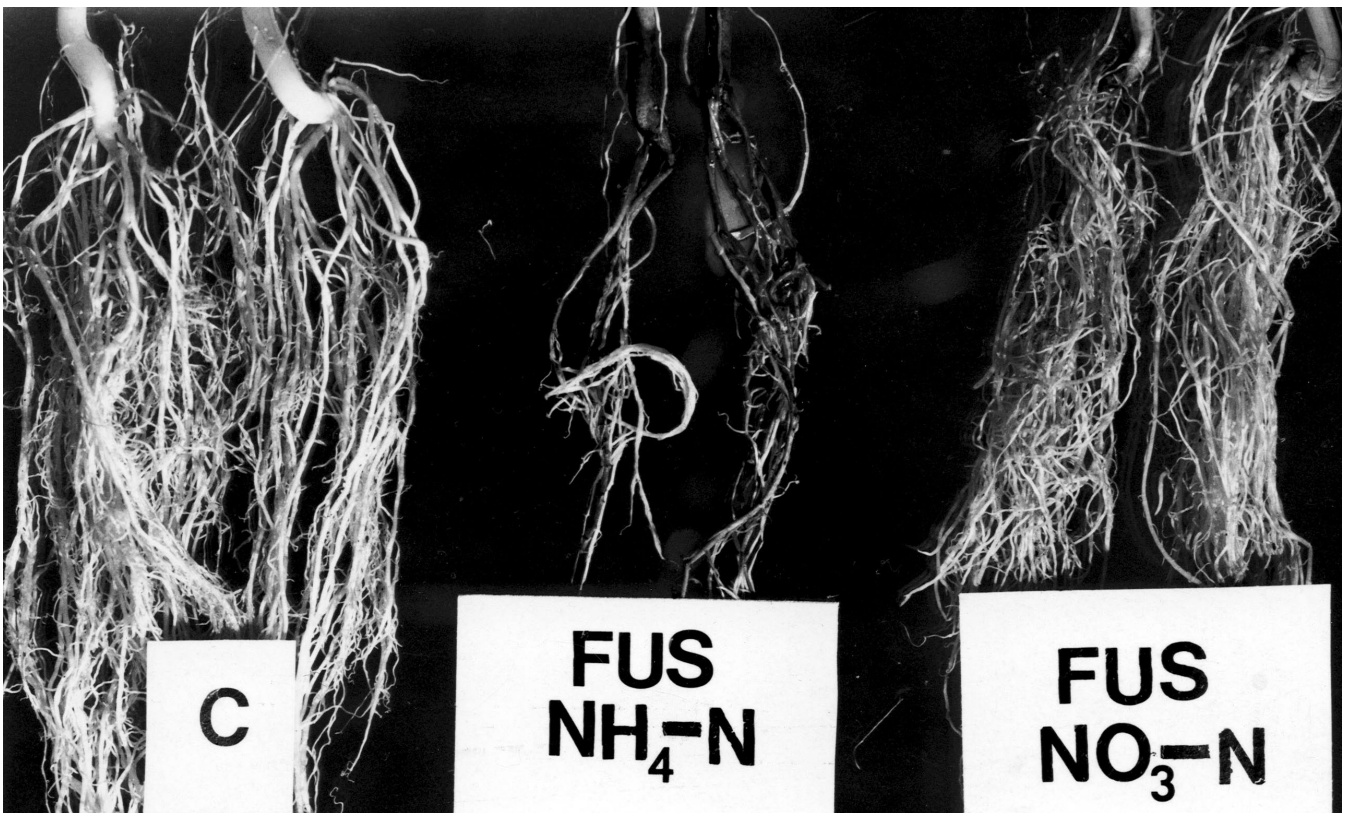

Fig. 4. Common bean seedling grown in pot soils treated as follows: C= uninoculated control fertilized with greenhouse Hoagland solution, FUS= pot soils heavily infested with conidia of *F. solani* f. sp. *phaseoli*, and fertilized with ammonium sulfate = NH_4-N or potassium nitrate = NO_3-N, shows how different fertilizers influence disease expression.

grafting a susceptible host on to resistant root stock. In that way a particularly desirable plant may be grown in infested soil. This method has been used in Taiwan, particularly by the *No-You* Seed Company in watermelon breeding projects and also in field-grown specialty cucurbit crop plants[78]. Although the method is quite labor intensive, it is useful where the value of the harvested crop warrants it.

Climate and other environmental factors are important influences in determining Fusarium wilt development. It is usual for wilts to be more damaging in warmer soils. Thus, in many northern areas cabbages susceptible to yellows may be grown during cooler periods, even in pathogen-infected soils[131, 142]. Similarly, in the cool Castroville area of coastal California, at a site where radishes are a year-around crop and where the soil is wilt-infested, susceptible lines such as 'Comet' are usually grown. It is only during the few warmer periods that it has been necessary for growers to plant resistant lines, such as 'Red Prince' for a successful harvest. Fusarium wilt of tomato is especially restricted by temperature considerations. Early on Clayton[22] reported 28℃ an optimum soil temperature for occurrence of this disease and temperatures below 21℃ or above 33℃ were definitely restrictive to disease development. More recently Jeffers[62] found that Fusaium wilt development of Upland cotton grown in California's San Joaquin Valley was affected by relatively small climate changes. In a 3-year study it was determined that by varying the date of planting in three different plantings, each 3 weeks apart, disease measurements and subsequent crop yields in the plots significantly differed, the late March planting being more severely diseased than those of early and late April. All of these observation, and many others not mentioned here suggest that, where it is at all feasible, alterations of planting schedules may benefit a grower whose land is contaminated with wilt Fusaria.

Perhaps the most effective way of disease control over the years has been through obtaining resistant host plants, and it seems evident that, whether consciously or not, mankind has been seeking the healthiest plants in the field for future propagation since his earliest attempts at agriculture. It also seems very probable that many of our soil-borne pathogens have been present, though unrecognized, since prehistoric times. Regrettably their survival propagules have not yet been seriously sought after among all the archaeological agricultural plant materials dug up. On the other hand, during the previous century the search for crop plants that are resistant to the various fusarium wilt scourges has occupied the time and attention of many plant breeders, plant pathologists and agronomists and reports on their findings go back to the first years of the 1900s[16, 65, 79, 93, 141], resistance often being defined in plant lines shortly after the cause itself of the disease became known, as in cotton and flax wilts. As previously mentioned, the first crop plants deemd as "wilt resistant" came from field selections and most often possessed multigenic resistance[140] and being a heterozygous mix, were likely to segregate. Thus in the same field both very resistant and susceptible plants could be found. Supplying a plant line with a single resistant gene is perhaps a more exact science. First the gene needs to be identified in a plant source. Frequently, it is found to exist in a wild and weedy land plant. Thus, may crosses and selections need to be made, leading to something desirable to harvest and agronomically feasible to grow. That is to say, having the characteristics of the original crop plant minus the disease susceptibility, and after all of that intense work, it may be that sometime following production of this resistant line, a fungal mutant appears that causes the same old disease in the field again. This same problem can occur in "transgenic" plants and although the techniques are intriguing and the results exciting, this whole line of gene transfer carries some other baggage of it own, such as some decreases in resistance may develop to otherwise avirulent problems, or often, a lack of public acceptance of the product. Nevertheless, the various means of obtaining resistance to disease have been on the whole most successful over time.

The health and growing conditions of the crop is most important in fighting disease. In general rapidly growing plants with deep root systems, growing in a proper climatic setting, in well aerated, fertile soil and without too much or too little moisture supplied are far less likely to become infected by pathogenic forms of *F. oxysporum* than are slow-growing plantings in shallow, acid soils, perhaps under conditions that are too hot or even too cold, and especially plants stunted by nematode infections or other problems. Often the soils that are "suppressive" to wilt are the deep, soils with an adequate clay component, especially monimorillinites, where the structure of the clay lattice may enhance its water-holding capacity, its retention of nutrients and the calcium ions that adjust the pH buffering effect. The clay lattice may also serve as a matrix to harbor microflora antagonistic to the pathogens and aid their survival. The role of the clay fraction and also humus paricles in soil, especially in the host rhizosphere, including that of absorbing and then slowly eluting antibiotics, and harboring antagonistic bacteria, actinomycetes, etc., has been reviewed by Jackson[60] and discussed by Baker and Cook[8] and by others. However, the exact effect of each component of soil and rhizosphere has on the soil pathogens, including those in *F. oxysporum* has yet to be clearly elucidated. Nevertheless, it may be

concluded that all in all a vigorously growing, healthy host plant is in better condition to produce the necessary components to fight off an invading pathogen.

FUSARIUM SOLANI

Another soil-borne species in the genus that possesses both plant pathogenic and saprophytic members is *Fusarium solani*. Similarly to *F. oxysporum* this species also produces both macro- and microconidia and chlamydospores (Fig. 2) and occurs ubiquitously in agricultural soils world-wide, as a major component in the plow layer. However, thereafter the similarity of the two species lessens. The macroconidia of *F. solani* tend to be cylindrical in the central area, the walls appearing to be parallel and are comparatively heavy and strong looking. Macroconidia are not often strongly curved; some individuals being nearly straight. They are borne on comparatively long phialides, produced in sporodochia and sometimes are so numerous in cultures that they merge to form a large mat on the surface. These spores have blunted rather than sharply pointed ends, although foot cells are usually quite evident. Very often the macroconidia contain an insoluble blue, green or yellowish pigment, which appears to be firmly attached to the inside of the conidial wall. Isolates from Brazil, pathogenic to okra, contain spores so dark blue that *en mass* they appear to be black (Robb, personal communication and cultures sent to S. N. Smith for identification). Cultures appear as blue, green or creamy, depending on spore color. There are also quinine-related pigments, and as in *F. oxyspourm*, many of which are water soluble and color the medium. Often carotenes also occur.

Microconidia appear either numerously or sparsely in culture, and the same is true of chlamydospores. In any case, usually there are less of these two spore types than one finds in *F. oxysporum* isolates. Microconidia are oval in shape, but some are quite wide, almost approaching a sphaerical form. Sclerotia are also sometimes present.

Some members of this species are air-borne and produce ascospores borne in asci of reddish to red-brown colored perithecia. Many of such isolates are homothallic, meaning that a single isolate can produce sexual spores, without the need of fertilization by another mating type. Often they are found in woody plant branches and may have invaded wounds or cankers produced by other fungi. Ascospores of this species are two-celled, eight per ascus and they often bear faintly visible striations (see Fig. 2). There are also heterothallic members in the species and, among the primary pathogens, those capable of forming perithecia are heterothallic. It is of especial interest that matings in these pathogens have only been observed in the laboratory because the different mating types that have been found are distributed in different geographical areas, and thus their perithecia only have been observed as products of laboratory crossings.

The formae speciales of *F. solani* pathogenic to herbaceous plants cause rots of root hypocotyl tissues. They ordinarily penetrate through thin areas or rifts in the cuticle or in hypocotyls through stomata and pass through the epidermis and parenchyma in the intercellular spaces, break down the middle lamella and eventually cell walls. Details of the invasion progress in *F. solani* f. sp. *phaseoli* in bean roots and hypocotyls was studied by Christou [20]. Infection causes large brick red lesions, which coalesce and sometimes cover all of the underground plant parts When root rots are severe the hosts essentially stop growing, become unthrifty and the crop yields are low.

Similarly to *F. oxysporum*, the soil survival structures in *F. solani* are also chlamydospores, usually embedded in, or clinging to surfaces of plant residues. These chlamydospores also need a source of organic nitrogen and sugar to germinate such as the nutrients exuded from the underground parts of the subsequently invaded host plants. The germling is able to grow only a short distance in soil before it is stopped by antibiosis and competition for nutrients by the rest of the soil microflora, at which point it must reach a growing plant or the hypha will lyse and if there is not sufficient storage energy in the chlamydospore, it too may perish. If it does contact a host plant the hyphae may grow on its surface below-ground until it finds a suitable point of entry. Once in the plant hyphae ramify though the cortical tissue until the plant succumbs to disease, or more often, dies following an early senescence. By that time the next generation of chlamydospores are formed and ready to return to the soil in the crop residue.

In most *F. solani* representatives chlamydospores tend to live for long periods in nature, but this is not so in *F. solani* f. sp. *cucurbitae* [86]. This pathogen attacks squash plants and other cucurbits, principally at the crown, near the soil surface and produces copious quantities of macroconidia on the moist succulent tissue that it invades. Subsequently, these water-borne spores may spread through the field down the row or in circles, through irrigation or rain splash. This fungus may also invade the fruit lying on the ground and then grow into seed. It is one of the few pathogens of this species that may be internally seed-borne, rather than merely being passed along in field trash contaminations in the seed lot, as is the case with most members of this species and many of those of *F. oxysporum* as well. Although chlamydospores are readily produced in this form, they tend to be rather short-lived in soil. Thus it is quite safe to replant cucurbits after a disease outbreak with about a year or two hiatus from the crop. Most freshly isolated cultures of *F. solani* f. sp. *cucurbitae*

are capable of producing perithecia when paired with the proper mating partner. However the various clonal types of the form are widely separated geographically and thus perithecia have not been seen in nature. It is of interest to speculate as to why the organism maintains a complex sexual capability which it doesn't use. Nevertheless, since the isolates of each area exhibit different cultural characteristics, including (mating type, + vs -), sex (male vs female, different from compatibility), perithecial color (red vs white), ascospore color (tan vs white), members of this form have long been used in studies of fungal genetics and this may have more significance than their role as pathogens. Also known is a race 2 in *F. solani* f. sp. *cucurbitae* [134] which primarily attacks the fruit. It is heterothallic, but not interfertile with race l isolates and it seems to be better adapted than race l as a soil inhabitant. Nevertheless, it can cause a significant loss of crop in a field due to rot. The fungus enters the fruit where it rests on the soil.

The host range of certain individuals in formae speciales of *F. solani* can be larger than is usual among clones in *F. oxysporum* formae. *F. solani* f. sp. *cucurbitae* is known to attack not only a variety of squash, pumpkins and gourds, including some that are wild weeds, and these same fungal isolates may also cause disease in watermelons. Some squash cultivars, such as 'Hubbard', are resistant to all isolates.

F. solani f. sp. *pisi* in addition to peas also attacks garbanzo beans [77] and it has even been suggested by others that this form may cause cankers in woody plants. However, the field isolates from severely damaged garbanzos in greenhouse tests produced rather mild lesions compared with the damage done by those same isolates on peas, suggesting that in the field there may be other factors which increase the virulence of this pathogen. In a California coastal valley where garbanzos have occasionally been rotated with peas, an especially serious disease was noted [147]. Later observations in this same area revealed that there was not only extensive root rot, but also it was in a complex with a vascular wilt disease [109]. Two pathogens were isolated, *F. solani* f. sp. *pisi* from the roots and from the vascular tissue another fungus identified as *Phialophora gregata*. In greenhouse tests each pathogen on its own caused only mild disease symptoms, but an inoculation combining both fungi was potent. These results suggest that since similar pathogens (*F. solani* f. sp. *glycines* and *P. gregata*) can cause soybean diseases, it may be that together they may sometimes both be involved in the "sudden death" syndrome in that crop.

In addition to the afore mentioned forms that attack cucurbits, peas, beans and soybeans, there are pathogenic forms known in sweet potatoes: *F. solani* f. sp. *batatas*, potatoes: f. sp *eumartii* (although apparently this form has not been observed in many years), faba beans: f. sp. *fabae*, lupines: f. sp. *lupine*, piper: f. sp. *piperis*, mulberry: f. sp. *mori*, and other woody hosts such as robinia: f. sp. *robiniae*, passion fruit plants: f. sp. *passiflorae* and xanthoxylum: f. sp. *xanthoxyli*. Usually if this species occurs on woody plant, it produces perithecia, it may invade lesions produced by insects or other fungi and it may be homothallic *F. solani* diseases produced on these hosts may develop above the soil level and they too may cause severe damage to the crops.

The afore-mentioned eye infecting *Fusarium* spp. have most frequently been found to be *F. solani*, although other species are also capable of this invasion. We, at U. C. Berkeley, in identifying cultures sent from U. C. S. F. Medical Center found over 90% of such corneal isolates to be *F. solani*. Sometimes a forma specialis name has been designated to the culprit that causes corneal ulcers, *F. solani* f. sp. *keratitis*. However, since the isolates seem to be those present in all soils, appear to be the common saprobe type and that other soil-borne Fusaria may also play this role, the pathogen is likely to be a common opportunist rather than a specific forma. Nevertheless there is no doubt of the seriousness of the problems that it may cause in humans and animals, often leading to blindness. Many times *F. solani* eye infections are encountered by those whose work is closely associated with soli; thus its relatively high incidence among farm workers. The gritty nature of soil further tends to scratch the cornea surface, especially if the eye is rubbed. This encourages fungal entrance. The famous race horse, *Secretariat*, incurred an *F. solani* corneal ulcer, presumably induced by the soil and dust thrown up at his face at the track. More recently the Bauch and Lomb Co. was forced to recall a popular contact lens solution linked to corneal infections caused mostly by *F. solani*. Apparently the problem was not due to contamination inside the solution container, but rather by the method of usage. A container left open allowed the polymers in the solution to be exposed to the air, where they might dry out and form a film, easily contaminated by dirt and dust. This film can thus act as a growth medium for the introduced fungus. As a word of advise, it is not a good idea to dose an eye which may have such an infection with a cocktail of antibiotics. This fungus is resistant to almost all of them, but perhaps its competitors are not.

The widespread and persistent nature of *Fusarium* spp. perhaps is due to their abilities to break-down and sustain themselves on a great variety of complex carbohydrates and proteins (including keratin), to be able to withstand adverse climates and rather high levels of toxic substance, such as ammonia, many antibiotics and

fungicides, etc., that kill off other microbes. *F. solani* seems to embody some of the toughest members of the genus. This species can occur in the soil, in fresh water, in the sea, in the air and on a large number of animal and plant parts, living or dead, and their products.

Diseases caused by soil-borne Fusarium have had an influence on the characteristics of the food that we eat everyday. Selections and breeding programs lead to crops that may continue to be produced in the face of spread of destructive maladies. One may plant older cultivars of familiar crops in the home garden, such as the now popular *heirloom* tomatoes, and find that they produce tasty and interesting fruit, but alas, often when such plants are grown successively at the same location, they succumb to diseases, blights and wilts, so early on that the crop annually becomes smaller and less thrifty. Thus we owe the prolific food supply available today to the many small victories in plant protection research.

FINAL PERSONAL NOTE:

Originally, I did not intend this paper as a literature review nor to delve deeply into all aspects of this broad, diverse group of fungi, having been asked to write a brief summary of the effects that soil-borne Fusaria have had on agriculture in general and in our professional endeavors. However, in an attempt to discuss the biology of such a diverse group with the many representatives that there are among the soil-borne Fusaria, it became necessary to cite sources for our knowledge. In so doing, while writing it, the size of the paper grew somewhat out of hand, rather more than I'd imagined. Still trying to be brief, I have omitted much of the voluminous literature that would have been important to a full review of the subject. I apologize to the many worthy researchers for failing to mention a lot of the important discoveries. My hope is for students and also workers in related fields, for whom this paper is intended, to find the subject matter of enough interest to consult the many relevant monographs, reviews, symposia papers and other publications, some mentioned here, in order to get a deeper view of their area of interest within the scope of the subject. It is also hoped that those whose main interest lies in the molecular aspects of plant pathogens will endeavor to relate specific bands in DNA analyses more closely to specific enzymes involved in disease, survival of the fungus in nature, cultural characters and above all specific pathogenicity. At present there appears to be a lull of interest in soil-borne pathogens and the field work necessary to explore them, but meanwhile the pathogens continue to thrive and adapt. All problems cannot be solved in the lab using cultures supplied from elsewhere.

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Shirley Nash Smith

Shirley Nash Smith was born in Milwaukee, Wisconsin, U. S. A in l926, spending childhood with her family in Milwaukee and Waukesha County. After completing undergraduate and masters studies in bacteriology/microbiology at the University of Wisconsin, Madison and the University of Illinois, Urbana respectively, and a further 4 years working in private industry for a pesticide formulation firm in Racine and a Milwaukee barley malting company, she attended the University of California, Berkeley, obtaining the PhD in plant pathology under the direction of Professor William C. Snyder.

During her career Dr. Smith has been a researcher intermittently at U. C. Berkeley in the Plant Pathology Dept at U. C. Davis in the Plant Pathology and the Agronomy Depts., mostly working in soil-borne fusarium problems of cotton and legumes. She also has worked overseas in Zimbabwe on soybean and other crop diseases, in Algeria on bayoud disease of date palms and in England (Rothamsted Expt. Station) on cereal fusaria and has been a consultant in several other countries for briefer time periods. She has traveled to Taiwan 17 times during the past 35 years, investigating and observing fusarium diseases in vegetable crops, sugar cane, rice, bananas, flax, etc., accompanied by Taiwanese plant pathologists aned students.

Her interest in plant diseases continues in her retirement.

She is the widow of the late John Currie Smith, formerly a building inspector, whom she met and marired in Africa.

摘 要

鐮孢菌屬之眞菌廣泛存在於世界各地，也極具多樣性，其棲境包括土壤、活體植物、植物殘體、植物產物、活或死亡之動物體，可以說無所不在。本文從土壤傳播病原鐮孢菌之角度來探討此類眞菌之生態，並以*Fusarium oxysporum* 及 *Fusarium solani* 爲例子做說明，涵蓋病原菌分化型及生理小種與寄主之關係、於土壤中存活之機制、與其它腐生菌間之競爭及抗生作用，以及其對植物之侵染過程，最後藉由了解此類眞菌之生態，來選擇採取適當之防治方法。有關土壤傳播病原鐮孢菌之生態仍有許多待解決之問題，希望能以此文所提出之觀念，激發相關植物病理研究者做更深一步研究，並結合進代分子生物之技術，徹底釐清此類眞菌於自然界之生態謎團。

關鍵詞：*Fusarium oxysporum*, *Fusarium solani*, 土壤存活, 致病性, 防治