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Plant pathology (review)

Fusarium wilt of peas (a review)

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Summary – Pea wilt was first described in 1925 and the pathogen identified as *Fusarium oxysporum* f sp *pisi* race 1 in 1929. Three additional races have been described and characterized as races 2, 5 and 6. Resistance to all 4 races is governed by separate, single dominant genes in the host. Current classification of pathogen races is based on host response using differential cultivars and defined, reproducible, inoculation procedures. Recent molecular techniques, using RFLPs and RAPDs, have detailed the genetic similarities and differences among races of this pathogen. Vegetative compatibility groupings of pathogen isolates have also characterized pathogen isolates based on fungal genetics rather than host–pathogen interactions. This paper also describes the host–parasite interactions and briefly discusses what little is known about seed transmission of the pea wilt pathogen.

Résumé – Fusariose du pois. Revue. *La fusariose du pois, causée par* Fusarium oxysporum *Schlecht f sp* pisi *(Van Hall) Snyd et Hans, a été signalée dans tous les pays où le pois est cultivé commercialement (Haglund, 1984). La maladie est souvent sévère là où on pratique des rotations courtes avec d'autres cultures. Dans ces conditions, quand le pathogène a développé des quantités suffisantes d'inoculum et qu'on plante un cultivar sensible, il en résulte des pertes sévères. Les symptômes sur la plante consistent en folioles chlorotiques, qui s'enroulent vers la base et deviennent flasques. Les plantes finissent par se flétrir et deviennent jaune brun. Souvent le système vasculaire aérien et souterrain devient jaune clair à rouge brique et la partie souterraine de la tige devient plus grosse que la normale.*

INTRODUCTION

Wilt of peas (*Pisum sativum* L), caused by *Fusarium oxysporum* Schlecht f sp *pisi* (Van Hall) Snyd and Hans, has been reported in every country where peas are grown commercially (Haglund, 1984). The disease is often severe where short rotations with other crops are practiced. Under these conditions, when the pathogen has built up sufficient inoculum numbers, and a susceptible cultivar is planted, severe crop losses result. Plant symptoms consist of chlorotic leaflets, which curl downward and become flaccid. The plant eventually wilts and turns a yellowish-brown color. Often, the above- and below-ground vascular system turns a light yellow to brick-red color and the lower subterranean portion of the stem becomes larger than normal.

DISEASE DESCRIPTION AND HISTORY

This disease was first described and distinguished from *Fusarium* root rot by Jones and Linford (1925), who named it "an undescribed wilt disease". At that time, the disease was found in 50 fields in Wisconsin, and caused greater losses in some areas than those reported for root rot. The causal organism was named *F othoceras* App and Wr var *pisi* in 1928 (Linford, 1928). The pathogen was later named race 1 of *F oxysporum* Schl f sp *pisi* (van Hall) Snyd and Hans in 1935 (Goth and Webb, 1981). Resistance was quickly found and the introduction of wilt resistant varieties gave complete control. Wade (1929) determined that resistance to race 1 was inherited as a single dominant gene. Race 1 was not an economic problem in the United States again until 1972 (Kraft *et al*, 1974). Race 1 has not been eliminated but the disease is under control through the growing of resistant cultivars.

In 1933, Snyder described a new race of *F oxysporum* that was capable of wilting plants resistant to race 1, and they labeled the pathogen race 2. The disease was called 'near-wilt' because it appeared later in the growing season, often only at full pod development. Near wilt is as widespread as race 1 (Haglund, 1984). Plants infected with race 2 are most often scattered throughout the field rather than being concentrated in specific areas as with race 1. In addition, race 2 is most prevalent in coarser textured soils when soil temperatures are near 25°C. Because symptoms caused by the race 2 pathogen do not usually occur until plant maturity, the likelihood of seed transmission is greatly increased.

Hare *et al* (1949) determined that resistance to race 2 was also controlled by a single, separate, dominant gene not linked with the race 1 resistance gene. Delwiche Commando was the first cultivar developed possessing resistance genes for both races 1 and 2 (Goth and Webb, 1981).

Races 3 and 4 were described in the Netherlands and Canada, respectively (Schreuder, 1951; Bolton *et al*, 1966). However, it seems likely that races 3 and 4 are more virulent cultures of race 2 (Huebbeling, 1974). The genetic basis for resistance to races 3 and 4 was not determined. In 1970, race 5 was described in northwestern Washington (Haglund and Kraft, 1970), where all commercial cultivars resistant to races 1 and 2 were susceptible. Because of the short crop rotations and favorable climate in that area for wilt development, race 5 spread rapidly and currently affects *ca* 4 000 to 12 000 ha planted to peas each year. Resistance to race 5 was also attributed to a single dominant gene factor in the host.

In 1979, a new race of wilt was again described from western Washington, which was pathogenic on cultivars and breeding lines resistant to races 1, 2 and 5, and was named race 6 (Haglund and Kraft, 1979). The pathogen was first observed in 1971, and by 1977 was recovered from 175 of 640 fields examined. The pathogen was widespread and pathogenicity tests using differential cultivars possessing single dominant gene resistance to races 1, 2 and 5 were susceptible. In genetic studies of inheritance, resistance was again attributed to a single dominant gene.

The pathogenicity of races 1, 2, 5 and 6 of *F oxysporum* f sp *pisi* can be distinguished by their reaction on the differential varieties listed in table I. The disease reaction of these differentials is based on a resistant response (no observable disease) and a susceptible reaction (dead or severely stunted, chlorotic plants). The use of differential cultivars to differentiate races of *F oxysporum* f sp *pisi* is essential (Haglund, 1974). It is recommended that these differentials be maintained as selections of the variety and not be obtained commercially to ensure their predicted disease reactions.

Classification of isolates of *F oxysporum* f sp *pisi* based on host–pathogen interactions is governed by the genetic makeup of both the host and pathogen. The availability of cultivars to differentiate strains of the pathogen is also of concern. To further complicate matters, virulence tests are subjective because they are influenced by temperature, host age, method of inoculation, *etc.* Work by Puhalla (1985), as summarized by Correll (1991), has classified strains of *F oxysporum* based on vegetative compatibility groupings (VGC). Isolates of *F oxysporum* can be tested for vegetative compatibility by pairing nitrate nonutilizing (*nit*) mutants that are generated on a 1.5–4.0% potassium chlorate medium. Strains, races or subspecific groups of *F oxysporum* may possibly be further characterized based on fungus genetics along with host–pathogen interactions

Within *F oxysporum* f sp *pisi*, 4 VGCs have been reported. Isolates of races 1 and 6 are both reported to be in a single VGC, race 5 is in a second VGC, and race 2 isolates occur in at least 2 additional VGCs. The pattern of genetic diversity identified within a given *forma specialis* may have

Table I. Suggested pea lines to differentiate races of the Fusarium wilt fungus.

Pea line	*Wilt Reaction*				*Source* [a]
	R1	*R2*	*R5*	*R6*	
M410	S	S	S	S	Brotherton Seed Co
Vantage	R	S	S	S	Brotherton Seed Co
Mini	S	R	S	S	Asgrow Seed Co
Mini 93	R	R	S	S	Asgrow Seed Co
Sundance II	R	S	R	S	Pure Line Seed Co
Grant	R	S	S	R	Brotherton Seed Co
WSU 23	R	R	R	S	WA Haglund
WSU 28	R	S	R	R	WA Haglund
74SN5	R	R	R	R	JM Kraft

[a] Brotherton Seed Co Inc, Moses Lake, WA, USA; Asgrow Seed Co, Twin Falls, ID, USA; Pure Line Seeds Inc, Moscow, ID, USA; WA Haglund, NW Washington Research and Extension Unit, Washington State University, Vernon, WA, USA.

direct bearing on breeding for resistance. For example, race 2 belongs to at least 2 different VGC groups, which indicates possible genetic differences in virulence, survival, *etc.* Elucidation of genetic diversity of *formae speciales* in virulence interactions with particular host genotypes requires further study. This technique, coupled with molecular techniques (Coddington *et al*, 1987; Kistler *et al*, 1991), should aid our understanding of the population biology, pathology, and race (strain) relationships of this fungus.

Coddington *et al* (1987) studied several race 2 and 6 isolations of *F oxysporum* f sp *pisi*. Total DNA was purified, digested separately with restriction endonucleases, and DNA fragments separated by gel electrophoresis. The patterns obtained from the race 2 isolates were identical for a given enzyme as were the patterns for the race 6 isolates. Interestingly, the classification of isolates of *F oxysporum* f sp *pisi* into races by restriction enzyme digestion patterns correlated well with the pathogenicity–host response patterns of each race studied.

Recently, randomly amplified polymorphic DNA typing (RAPDs) has been applied to fungal genetic studies (Williams *et al*, 1990). Work at Washington State University (M Grajal, personal communication) has related RAPDs to the genetic diversity among the 4 races of *F oxysporum* f sp *pisi*. The relatedness and possible phylogeny of different races was also studied. It appears that races 1, 5 and 6 are closely related and that race 2 is distinct. All isolates of race 2 exhibited a highly conserved banding pattern whereas isolates of the other races exhibited more variability with the primers used.

INOCULATION AND SCREENING PROCEDURES

There has been considerable disagreement in the literature on the race classification scheme of *F oxysporum* f sp *pisi* (Armstrong and Armstrong, 1974; Haglund, 1974; Kraft and Haglund, 1978). The only sure way to determine the race classification of any isolate is the host–pathogen response. Inoculation procedures, genetic purity of the host and pathogen, environmental conditions, and inoculum levels all directly affect the host–pathogen response. It is important to standardize all these variables so that repeatable results are the norm (Huebbeling, 1974; Kraft and Haglund, 1978). Techniques to screen peas for resistance to wilt include: 1) pruning roots while submersed in a conidial suspension (Wells *et al*, 1949; Haglund, 1989); 2) pouring mycelial fragments and conidia into a trough adjacent to seedling roots growing in sand (Armstrong and Armstrong, 1974); and 3) pouring conidia and hyphal fragments into holes punched in soil with a pointed rod to wound roots (Doling, 1963).

In Washington state, resistance of numerous breeding lines to Fusarium wilt caused by *F oxysporum* f sp *pisi* race 1 is determined under field conditions at Pullman, WA, in cooperation with F Muehlbauer, USDA/ARS, who has established a race 1 field nursery. As described by Wade (1929), elimination of the susceptible controls by race 1 wilt is complete, when inoculum levels are high, which greatly reduces or eliminates the chances for escapes. This is indeed the case at the field nursery at Pullman, WA. Resistance to race 1 as well as races 2, 5 and 6 is also determined in pure culture under greenhouse conditions. Care is taken to screen for resistance to race 2 when ambient greenhouse temperatures are 20–24°C (Wells *et al*, 1949). Screening tests for races 1, 5 and 6 are conducted in the winter months when ambient greenhouse temperatures range from 15–21°C. To ensure pure cultures of the pathogen, nodal tissue from above the fourth node can be surface disinfested and plated on Nash and Snyder's PCNB (1962) or Komada medium (1975).

Wild-type cultures, used as primary inoculum, are derived from single spores on 2% water agar, increased on fresh PDA under fluorescent light with a 12-h photoperiod, and stored in autoclaved soil tubes (Toussoun and Nelson, 1976). Primary inoculum of a test isolate is produced by dispersing a small amount of infested soil on a PCNB plate (Nash and Snyder, 1962) and a resulting colony is selected, which is representative of the wild type for each race (Haglund, 1974). A small agar plug from the colony margin, after a 5-d incubation period under constant fluorescent illumination, is cut and removed with a No 6 cork borer, and placed in 50 ml of liquid medium (Kerr, 1963). Inoculated flasks are incubated for 5 d on a rotary shaker (120 cycles/min) with constant fluorescent light on a laboratory bench. At that time, spore concentrations for each isolate are determined with a haemocytometer. We usually combine 3 isolates of the same race for any wilt screening test so that the final combined spore concentration is 1×10^6 conidia/ml.

Seed of each test line is surface-disinfested with a 10% chlorox solution for 1 min before planting in coarse, autoclaved perlite. All seedlings are inoculated in the third or fourth node stage by carefully removing each plant, dipping and excising one-half of the root system of each plant with a razor blade while immersed in a conidial suspension. Inoculated seedlings are transplanted back into the planting medium and incubated on a greenhouse bench (20–23°C) until wilt symptoms are evident and/or known susceptible inoculated controls are dead. Wilt symptoms consist of stunting, yellowing, dying of lower leaves, downward curling of leaf margins, and usually death of the plant (Kraft and Haglund, 1978). The pathogen is readily isolated from the above-ground stem of any susceptible, inoculated plant when whole plant symptoms are evident. New races or strains of the pea wilt fungus will probably appear, especially where peas are grown in short rotations or in monoculture.

Suggested pea lines to differentiate races of pea wilt are shown in table I. These lines are maintained by the author in a pure state.

HOST–PATHOGEN INTERACTIONS

Most *formae speciales* of *F oxysporum* exist as chlamydospores, which are dormant in decaying host tissue and soil until stimulated to germinate (Nelson, 1981). Root exudates are reported to be responsible for chlamydospore germination of several root rot and wilt pathogens (Huisman, 1982). No differences have been reported in germination of race 1 or 2 macroconidia, chlamydospore, or germtube growth in the spermosphere or rhizoplane of resistant or susceptible cultivars (Whalley and Taylor, 1976). However, it was reported that the total population of races 1 and 5 increased more in the rhizosphere of a susceptible rather than a resistant pea cultivar (Kraft, 1978; Charchar and Kraft, 1989).

It is apparent from earlier studies that *F oxysporum* is an efficient soil saprophyte and root colonizer, and may also infect epidermal and cortical cells of many nonhost or resistant plants which remain symptomless. Schippers and Voetberg (1969) found no differences in chlamydospore germination of *F oxysporum* f sp *pisi* race 1 on susceptible or resistant pea seedling roots. Likewise, Charchar and Kraft (1989) reported that races 1 and 5 colonized root-surface cells of both genotypes resistant to races 1 and 5 as well as resistant and susceptible pea plants. Root surfaces of susceptible pea plants, however, supported larger populations of *Fusarium* colonies.

Pathogenic forms of *F oxysporum* are reported to penetrate a host root either through wounds or directly through root apices (Nelson, 1981). The most common points of entry into the pea plant by the Fusarium wilt pathogen are the undifferentiated region of the root tips, the cotyledonary node, or wounded roots (Walker, 1935; Virgin and Walker, 1940; Doling, 1963; Nyvall and

Haglund, 1972). The infection of a susceptible pea plant can be inter- or intracellular until the xylem elements are invaded (Beckman, 1987). In advanced stages of disease development, the wilt fungus grows out of the vascular system and into the adjacent parenchyma producing conidia and chlamydospores. The life cycle is repeated when the chlamydospores germinate and growth occurs either saprophytically or by invasion of a suitable host plant.

In general, once *F oxysporum* has penetrated the host plant, the fungus moves into the vascular tissue. When juvenile roots are the site of infection, the fungus moves either intercellularly or intracellularly to the developing xylem vessel elements and invades them before they are fully mature. The pathogen is generally confined to the xylem parenchyma cells at an early stage of disease development. The pathogen is spread throughout the plant by means of mycelial growth or conidia, primarily microconidia, produced in infected xylem vessel elements. Following root penetration, the invading hyphae of all tracheal fungal pathogens are reported to grow through root tissue towards the stele. Once inside the vascular system, the fungus may change its mode of nutrition to adapt to the nutrient conditions in the vessel lumen. As disease development progresses, the fungus may invade tissues adjacent to the xylem.

Fusarium wilt pathogens can survive in the absence of susceptible host plants by invasion and colonization of other plants that show few, if any, disease symptoms. When pea cultivars, differing by a single gene for resistance or susceptibility to race 1 or 5 of *F oxysporum* f sp *pisi*, were grown in soil infested with the appropriate pathogen, both races colonized root surface cells of either host (Charchar and Kraft, 1989). Charchar and Kraft further stated that each race could be isolated from apices of tap and lateral roots and from excised lateral root ends of the resistant cultivars, but were isolated more frequently from the susceptible cultivars. Lateral roots and stems of resistant cultivars were not invaded internally, in contrast to the equivalent plant parts of susceptible cultivars. Invasion of epicotyls of resistant cultivars was limited as of 20 d after planting. In comparison, epicotyls of susceptible cultivars were extensively infected by this time. Vascular plugging was found to seal off xylem elements of resistant but not susceptible pea varieties in the lateral roots, epicotyl, and above-ground stems. In contrast, xylem of the susceptible pea cultivars was extensively invaded by mycelium of the pathogen. Xylem fluids collected from excised stems of resistant cultivars inhibited germtube growth but stimulated conidial germination and germtube growth if from susceptible cultivars. Beckman (1987) has stated that vascular colonization by fungal wilt pathogens is extensive in wilt-susceptible plants but remains limited to the basal part of resistant plants. This localization at the base of the plant is also true with peas.

One of the earliest responses to infection by *F oxysporum* is the deposition within contact cells of additional wall callose material. Deposits in the form of papillae or more extensive apposition layers occur especially at sites of fungal contact (Beckman, 1987). The initiation of the response and the capacity to respond appears to be nonspecific in terms of the inducing organisms, which is inhibited in the resistant plant's response but not in the susceptible reaction. Callose deposition is a very general defense reaction. A strong deposition in infected cells is associated with pathogen failure to become established. Perhaps callose deposition is initiated by glycoproteins from the cell walls of the wilt pathogen.

Numerous theories have been advanced to explain the nature of resistance to wilt in crop plants. These theories include inhibitory root exudates (Buxton, 1957), chemical defenses in the host which cause growth inhibition of the pathogen (Hammerschlag and Mace, 1975; Bell and Mace, 1981; Danko and Corden, 1984), and physical barriers which retard or prevent vascular invasion (Elgersma *et al*, 1972; Stromberg and Corden, 1980; Beckman and Talboys, 1981; Baayen and Elgersma, 1985).

SEED TRANSMISSION OF THE FUSARIUM WILT PATHOGEN

Snyder (1932) reported that *F oxysporum* f sp *pisi* race 1 can be transmitted occasionally by seed when harvested from a wilt-infested field. Of 8 000 seeds tested only 4 transmitted the wilt pathogen as determined in a field grow-out test. Snyder stated that occasionally small soil particles can lodge in a seed surface indentation and the fungus could be contained therein. Snyder stated that the race 1 pathogen can be isolated from the stem node where the first pod develops on a wilted plant. Although no evidence was presented, it is possible that the pathogen could invade the pedicel and hence the pod and seed. Masheshwari *et al* (1982) isolated *F oxysporum* f sp *pisi* from surface disinfested seed of 6 vari-

eties grown in the Hoshiarpur district of Punjab, India, where pea root rot and wilt are a problem. The isolates of *F oxysporum* recovered were pathogenic but the authors did not classify these isolates to any race using differential varieties.

It is the author's opinion that true internal seed transmission of the pea wilt pathogen depends upon plant age when wilt symptoms are expressed. The probability of seed transmission of the race 2 pathogen, which attacks a pea plant at flowering to pod development, is much higher than for race 1, 5, or 6 which usually kill a susceptible plant before bloom.

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