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POTATO EARLY DYING—A SERIOUS THREAT TO THE POTATO INDUSTRY

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Abstract

Potato early dying (PED) is characterized by a loss of plant vigor during mid to late summer followed by senescence and death of the crop a few weeks prior to normal maturity. This disease is of serious importance in areas of long-term or intensive potato production and is largely uncontrolled. Symptoms of PED, which are difficult to distinguish from normal senescence, especially in early-maturing cultivars, are uneven chlorosis and necrosis of vines and tan discoloration of vascular tissues. The basic cause of PED is the soil fungus *Verticillium*. Two species are involved—*V. albo-atrum* predominating in cooler areas and *V. dahliae* in warmer areas. Both fungi commonly occur in cultivated soils and persist as melanized hyphae or microsclerotia, respectively. Infection occurs through roots followed by colonization of the vascular system. Contamination of uninfested fields can occur by wind or mechanical movement of soil-borne propagules or introduction of infested seed stock. Although *Verticillium* is the primary pathogen in potato early dying, other soil organisms are involved, resulting in a "disease complex." Research is under way in many areas to further our understanding of these pathogenic interactions and to exploit this knowledge for use in new systems of prediction and control.

Resumen

La muerte prematura de la papa (MPP) se caracteriza por pérdida de vigor de la planta entre mediados y finales del verano, seguida de senescencia y muerte del cultivo pocas semanas antes de su madurez normal. Esta enfermedad es importante en áreas de producción intensiva de papa y con frecuencia se deja sin control. Los síntomas de la MPP, que son difíciles de distinguir de la senescencia normal, especialmente en cultivares de maduración precoz, son clorosis desigual, y necrosis del rastrojo y descoloración de los tejidos vasculares. La causa de la MPP es el hongo del suelo *Verticillium,* y hay dos especies involucradas: *V. albo-atrum,* que predomina en áreas frías; y *V. dahliae,* en áreas cálidas. Ambos hongos se encuentran comúnmente en suelos cultivados y persisten respectivamente como hifas melanizadas y como microsclerocios. La infección ocurre a través de las raíces y continúa

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con la colonización del tejido vascular. La contaminación de suelos antes libres su puede deber al viento, o al movimiento mecánico de inóculo del suelo o a la introducción de semilla infestada. Aunque el *Verticillium* es el patógeno primario de la MPP están involucrados otros organismos del suelo, dando lugar a un 'complejo'. Hay investigación em marcha, en diversos campos, para entender mejor estas interacciones patogénicas y para utilizar este conocimiento en nuevos sistemas de predicción y control.

In many areas, growers have experienced problems with declining potato yields after fields have been in production for a number of years. Plants often fail to maintain vigor during mid to late summer and the crop matures and dies a few weeks early. This syndrome, called "early maturity wilt" or "potato early dying" (PED), is a serious problem in many areas of long-term or intensive potato production (2, 3, 6, 10, 12, 17).

In long-established potato production areas in the United States, especially in the mid-western states of Ohio, Michigan and Wisconsin, the early dying pattern has developed slowly over many years. In some cases, growers do not even realize that their yield expectations are low and have come to consider early maturity a "normal" situation on their land.

A factor further complicating grower recognition of this disease in established production areas has been the trend toward increasing yields in the last 10 to 20 years due to improved cultivars, increased fertility, irrigation and improved pest control. In many cases, the disease actually may not have caused yield reductions but has limited yield increases that could have been expected from other investments made in cultural improvements.

Although PED has not been as serious a problem in the northern production areas of Maine, North Dakota, and Minnesota, it has very quickly come to the forefront in newly-irrigated desert production areas of the Pacific Northwest (10, 12). After high initial yields on these "virgin soils," the early dying pattern has often developed quickly with subsequent croppings. Growers with large investments in land development and irrigation systems are quite alarmed and often recognize the problem more quickly than those who have dealt with it on a chronic basis for many years.

Symptoms of PED are difficult to distinguish from normal senescence. Initial symptoms are only uneven chlorosis of lower leaves on occasional plants. Later, some wilting of leaflets may occur, but more typical is uneven death of lower leaflets. Leaf yellowing and death proceed up the stems which usually remain erect. A light brown or greyish, vascular discoloration is usually visible at the stem base when sliced. Vascular discoloration at the stem end of tubers is also typical of PED but may be due to other causes. Advanced symptoms do not usually occur until after flowering and may consist of decline of isolated plants or, in severe cases, early maturity of an entire crop (3, 6, 7, 12, 17).

Yield decline due to potato early dying is highly variable. Losses of 6-10 metric T/ha have been documented in Idaho (3), New York (15) and Ohio (13). In an Oregon study (10), two comparable fields under center-pivot irrigation were compared; one new to potato production and one with three previous potato crops. Yield in the latter field, where PED was severe, was approximately 50% that of the former. In spite of these observations, yield decline is not always consistantly associated with foliar symptom development (14). This is probably a result of environmental conditions affecting the rate of vascular infection and of the compounding effects of temperature and/or moisture stress during tuberization.

The basic cause of PED is the soil fungus *Verticillium* (1, 6, 7, 8, 10). This organism causes Verticillium wilt in a wide range of crops including vegetables such as potatoes and tomatoes; fruits such as grapes and raspberries; field crops such as cotton and alfalfa, and even many ornamentals and shade trees. In potatoes, two species of *Verticillium* are involved, *V. albo-atrum* Reinke and Berthold and *V. dahliae* Kleb. The two are separated on the ability of *V. dahliae* to form true microsclerotia as survival structures, while *V. albo-atrum* forms only melanized hyphae within infected tissues. They also differ in temperature sensitivity, in that *V. albo-atrum* grows optimally at temperatures up to 24 C, while *V. dahliae* grows well up to 27 C. *Verticillium albo-atrum* is the primary cause of PED in cooler production areas where summer temperatures do not normally exceed an average of 21-24 C. *Verticillium dahliae* is more widespread and predominates where average temperatures are often much higher. Prior to the early 1970's, both species were considered forms of *V. albo-atrum,* but most authorities now recognize them as separate species (6, 7).

Both fungi are extremely common and can be found in many cultivated soils. Contamination of uninfested fields with *Verticillium* can occur by wind or mechanical movement of soil particles containing viable propagules, but a primary method is introduction on infected seed stock (6, 7). As a vascular pathogen, *Verticillium* can colonize tubers through stolons and remain present as dormant mycelium in the vascular tissues.

In spite of the obvious potential for introduction on seed stocks, studies have shown that *Verticillium* may not have to be introduced into new lands, but in many cases, may be there naturally on roots of native vegetation (7). In these cases, serious outbreaks of PED may well result from large increases in the populations of propagules already present in the soil due to intensive cultivation of highly susceptible host plants such as potatoes.

Verticillium can survive in soil for long periods in a dormant state as microsclerotia or melanized hyphae free or embedded in organic debris (Figure 1). Because of a wide host range, the fungus can also survive at low levels on roots of many crop and weed species (7). These dormant propa-

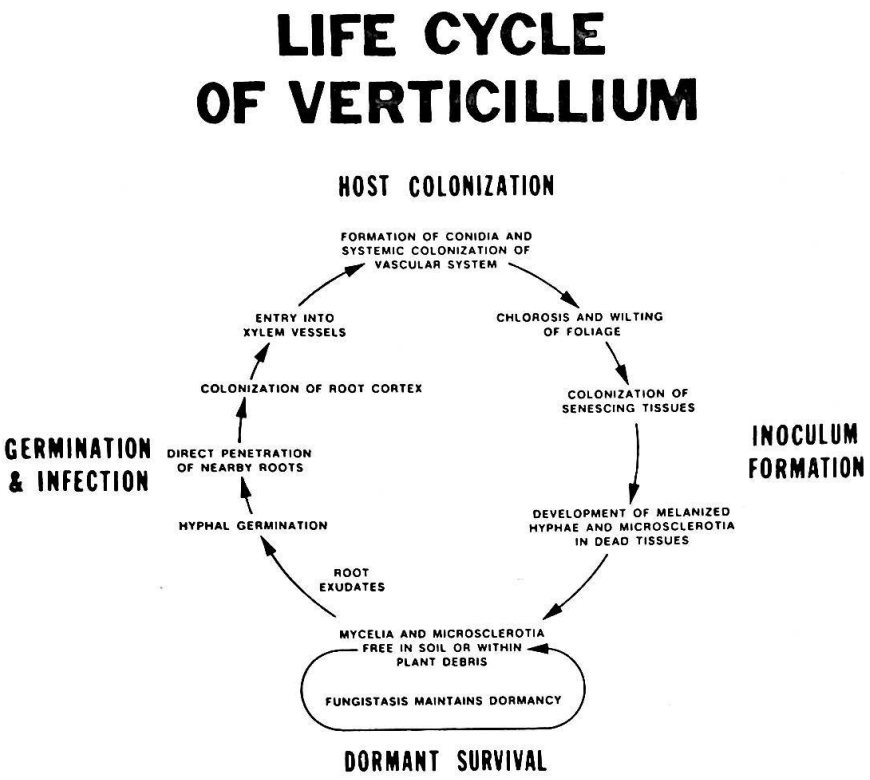

FIG. 1. Life cycle of the soil fungus *Verticillium*.

gules are stimulated to germinate by nutrients exuded from plant roots and infection occurs by direct penetration. The fungus then penetrates cortical tissues and enters into the root xylem. Systemic colonization of the plant then occurs by growth within the xylem vessels and by formation of conidia which are transported with the xylem fluid and germinate to form new infections at remote sites. Following systemic colonization, symptom development occurs as a result of toxin production and vascular dysfunction. As the host plant dies, the entire plant is colonized and new resting structures are formed within the dying tissues. These are eventually incorporated onto soil in fragments of plant debris, raising the level of inoculum for succeeding crops (7).

Although *Verticillium* is the primary pathogen in potato early dying, the situation is not that simple. Other organisms are also involved, resulting in what is called a "disease complex." Interestingly, the cause of PED may not be the same in all locations because interacting factors may vary geographically. Studies in Ohio (14) and Israel (5) have demonstrated involvement of root-lesion nematodes (*Pratylenchus* spp.) in PED. In Oregon (4) and Wisconsin (11), a connection with blackleg (*Erwinia carotovora*) has

been established and there has been some evidence for involvement of other fungi (9, 16).

The following speakers in this symposium will elaborate on these interactions, as well as discuss approaches to control of this complex disease.

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