Relationship between Silver Scurf Levels on Seed and Progeny Tubers from Successive Generations of Potato Seed

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ABSTRACT

The level of silver scurf on potato seed tubers on successive generations of potato seed tubers and their progeny tubers was investigated during 3 years in the field. The objective was to determine the importance of seed-borne inoculum on silver scurf development on the subsequent progeny tubers. Silver scurf incidence and severity increased with each generation. Coefficients of determination for disease levels among generations were significant and ranged from 0.89 to 0.97, indicating that seed tuber source accounted for a large proportion of silver scurf on progeny seed tubers. Silver scurf incidence and severity also increased with decreasing time periods between potato crops in the field. In a field near Paterson, WA, where potatoes had not been previously grown, the severity of silver scurf increased on progeny tubers of cvs Russet Norkotah, Ranger Russet, and Shepody as disease severity increased on seed tubers of successive generations. Disease severity index significantly increased as disease incidence increased. The relationship between the two was best described using a curvilinear regression model.

INTRODUCTION

Silver scurf, caused by Helminthosporium solani Durieu & Montagne, is an increasingly important potato disease in many potato production areas because of the occurrence of fungicide-resistant strains, increased disease severity, and increased financial losses from blemished tubers (Bain et al. 1996; Hide and Hall 1993; Kawchuk et al. 1994; Merida and Loria 1994). Blemishes produced by Helminthosporium solani consist of buff or brown lesions that develop on tuber periderm and become silvery in appearance when wet. Severe infections obscure periderm pigmentation of red- or white-colored cultivars (Jellis and Taylor 1977). Increased water loss through infected areas can result in up to 13% less yield of marketable potatoes, which affect grower returns whether or
not the tubers are sold to processors or fresh markets (Jellis and Taylor 1977; Lennard 1980; Read and Hide 1984). Helminthosporium solani lesions are particularly important on potato tubers sold on the fresh market because of low market tolerance for cosmetic blemishes (Goth and Webb 1983; Jellis and Taylor 1977; Merida and Loria 1994; Rodriguez et al. 1995).

Silver scurf is seed-tuber-borne, but the importance of this inoculum source in disease development in the field is not fully understood and is often overlooked. Evidence that seed tubers are an important source of inoculum comes from experiments showing the effectiveness of seed tuber fungicides in reducing silver scurf (Cayley et al. 1983; Denner et al. 1998; Frazier et al. 1998) and from the location of lesions on progeny tubers taken directly from the soil. Initial infections occur at the stem end of the progeny tuber, in the region around the stolon but not always adjacent to it (Jellis and Taylor 1977; Zimmerman-Gries and Blodgett 1974). Disease-free seed tubers typically produce disease-free progeny tubers (Zimmerman-Gries and Blodgett 1974).

Relationships between silver scurf severity on the seed and disease severity on the progeny are not always consistent (Jellis and Taylor 1977; Kawchuk et al. 1994; Mooi 1959). Seed tubers of cvs King Edward and Ulster Sceptre that were severely, moderately, and slightly infected with silver scurf were tested to determine the resulting levels of infection of progeny tubers (Read and Hide 1984). Progeny tubers produced from severely infected seed tubers had less infection than tubers from moderately to slightly infected seed. Severely infected seed tubers had older lesions, which produced fewer spores than the younger lesions on the moderate or slightly infected seed (Jellis and Taylor 1977; Read and Hide 1984). Consequently, progeny tubers from moderately infected seed had the highest infection (Read and Hide 1984). Others have reported no significant differences in disease severity on progeny tubers from slightly, moderately, and severely infected seed tubers (Zimmerman-Gries and Blodgett 1974). Such conflicting results suggest that the relationship between the amounts of H. solani on seed- and progeny tubers is complex.

Soil-borne inoculum of H. solani was not considered a viable source of initial infection until recently (Bain et al. 1996; Firman and Allen 1995; Jellis and Taylor 1977), when Merida and Loria (1994) observed that H. solani survived saprophytically on many crops grown in rotation with potatoes. To avoid infection from seed tubers, previous studies have produced seed tubers from tissue culture plantlets or stem cuttings, thus eliminating the possibility of contamination from the seed tuber (Jellis and Taylor 1977; Merida and Loria 1994). These findings support and help to explain how fields where potatoes had never been grown, or were grown 1, 2, 3, or 4 years previously could produce potato crops with significant incidences of silver scurf even when the seed or stem cuttings used were free of disease (Bain et al. 1996).

Efforts to manage silver scurf are hampered because the method of spread of the pathogen and the relationship between silver scurf on seed and progeny tubers is unclear. Cultural practices, including planting pathogen-free seedpieces, may be important in minimizing disease severity, but these practices need to be commercially practical. The purposes of this study were to determine the relative importance of tuber-borne inoculum of H. solani on the development of silver scurf on progeny tubers and to quantify the relationship between disease incidence and disease severity indices for potato silver scurf.

MATERIALS AND METHODS

Tuber Assay for Silver Scurf

Seed tubers were evaluated for silver scurf prior to planting, as were the progeny tubers following harvest and after storage. Tubers were first washed with running water and soaked for 10 min in 0.05% sodium hypochlorite (NaOCl) at 70 C. Tubers were then assessed for the percentage of silver scurf and divided into one of six disease severity classes: 0 = tubers with no silver scurf symptoms; 1 = 1%-5%; 2 = 6%-25%; 3 = 26%-50%; 4 = 51%-75%; and 5 = 76%-100% surface area covered with visible lesions. These data were then used to calculate a disease severity index from the formula: DSI = Σ (disease severity class x number of tubers in that class) x 100 / total number of tubers x 5 (Sherwood and Hagedorn 1958). DSI varies from 0 to 100, and a disease index of 0 means that all tubers were disease free, 100 means that all tubers were in the most severe class. Disease incidence was determined as the percentage of all tubers inspected that showed some incidence of silver scurf.

Levels of Silver Scurf in Successive Generations

Incidence and severity of silver scurf in successive generations (nuclear through generation 3) seed tubers were determined based on samples collected from four growers in Montana. All growers indicated that previous generations of