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## Chapter 1 : Potato Diseases: Rhizoctonia Stem Canker and Black Scurf (E) - MSU Extension

*Dates one gets = around 5 - 7 mya for when humans / chimps last shared a common ancestor (NOTE: This is time of "Ardi", Sahelanthropus, Orrorin). So what came before Lucy & Selam? Who was earliest ancestor?*

Received Feb 19; Accepted Aug  Abstract Spongospora subterranea is responsible for significant potato root and tuber disease globally. Study of this obligate non-culturable pathogen that infects below-ground plant parts is technically difficult. The capacity to measure the dynamics and patterns of root infections can greatly assist in determining the efficacy of control treatments on disease progression. This study used qPCR and histological analysis in time-course experiments to measure temporal patterns of pathogen multiplication and disease development in potato and tomato roots and tubers. Effects of delayed initiation of infection and fungicidal seed tuber and soil treatments were assessed. This study found roots at all plant developmental ages were susceptible to infection but that delaying infection significantly reduced pathogen content and resultant disease at final harvest. The pathogen was first detected in roots 15â€”20 days after inoculation DAI and the presence of zoosporangia noted 15â€”45 DAI. Following initial infection pathogen content in roots increased at a similar rate regardless of plant age at inoculation. All fungicide treatments except soil-applied mancozeb which had a variable response suppressed pathogen multiplication and root and tuber disease. In contrast to delayed inoculation, the fungicide treatments slowed disease progress rate rather than delaying onset of infection. Trials under suboptimal temperatures for disease expression provided valuable data on root infection rate, demonstrating the robustness of monitoring root infection. These results provide an early measure of the efficacy of control treatments and indicate two possible patterns of disease suppression by either delayed initiation of infection which then proceeds at a similar rate or diminished epidemic rate. Introduction Spongospora subterranea f. There are currently no sustainable and reliable controls for the pathogen [ 6 ]. The pathogen survives for prolonged periods in the soil and on seed tubers as conglomerates of resting spores sporosori from which motile primary zoospores are released. These actively swim toward host roots, encyst on the root surface and transfer the contents of the zoospore within root cells [ 1 , 6 ]. Root infection progresses with the formation of a plasmodium which becomes a sporangium and produces secondary zoospores that are released from the cell and initiate new infections within the root system and newly developing tubers in a polycyclic manner [ 1 ]. As the disease progresses there is a change from the zoosporangial to the sporogenic resting spore stage of the life cycle with formation of root and tuber galls where resting spores are produced within sporosori [ 1 , 7 ]. These are released into the soil providing new sources of inoculum. As an obligate pathogen with infections occurring beneath the soil, the study of disease epidemics is technically difficult. Recent advances in pathogen detection and quantitation using qPCR have enabled researchers to measure the pathogen during root infection and assessments of cultivar susceptibility made [ 8 ]. Previous research has identified that the critical period for tuber infection is shortly after tuber initiation where the host cells are susceptible to pathogen penetration [ 9 ]. In controlled experiments where application of S. However, these experiments examined tuber disease only and did not assess root infection and root galling. Until this present study it was unknown whether a similar defined period of susceptibility existed in potato roots and what affect a delay in inoculum application would have on the dynamics of root infection. In New Zealand and Europe a range of fungicides applied as to seed tubers or the soil at planting have successfully reduced the incidence of powdery scab increasing the yield of marketable tubers [ 12 , 13 ]. While, such fungicides may be an important part of an integrated management strategy for this disease [ 14 ], their impact on root infection and root disease has not been examined. Recent evidence suggests early associations between pathogen and the root are critical to subsequent disease expression [ 8 ]. Therefore, practices or strategies that can delay this interaction or slow subsequent disease progress may provide viable control options. The aims of this study are to 1 develop a system for measuring temporal patterns of pathogen replication and disease within potato roots, and use these data to 2 identify whether potato roots, like tubers, have distinct periods of susceptibility to S. We hypothesise that monitoring temporal root infection patterns can provide a reliable assessment of the ability of control strategies to mitigate root and tuber disease. Materials and Methods Ethics statement No

specific permissions were required for these pot and field trials. The studies did not involve endangered or protected species. Impact of delayed S. Tomato was used to enable experimental effects to be tested across more than one species as it is a known susceptible host of the pathogen and provides a convenient model plant system. Plastic pots 20 cm diameter, 4. In PT1 11 August â€"winter and PT2 8 January â€"summer pathogen-free mini-tubers of potato cultivars Russet Burbank and Desiree were planted at 10 cm depth one tuber per pot and in PT3 22 January â€"summer , 2-week-old healthy tomato seedlings of cv. Mortgage and Roma were transplanted into pots one plant per pot. Inoculum was prepared using a modification of previous methods [ 15 , 16 ]. PT1 included an additional inoculum treatment at 60 DAE. Plants from individual replicate pots were destructively harvested at 15 days intervals for up to five harvest periods after initial inoculum treatment and root and tuber tissues sampled for further analysis. In PT3, pots were amended with S. Plants were destructively harvested at 15 day intervals for up to seven harvest periods after initial inoculum treatment. For all PTs individual treatment combinations were replicated three times with pots arranged in a randomised complete block design and hand watered when required to maintain constant wet soil conditions. There were no pesticides or additional fertilizer applications. Impact of seed and soil applied fungicides on pathogen replication and disease Pot trials Two pot trials PT4, PT5 examined the impact of mancozeb, applied as a soil treatment, on potato plants grown in potting soil inundated with S. All trials were conducted in an ambient, outdoor environment in New Town, Tasmania. Treatments included mancozeb 7. A control treatment without S. Five tubers were planted at 10 cm depth 3 January in individual pots 20 cm diameter, 4. There were three replicates for each harvest. In PT5 two-week old disease-free tissue cultured plantlets of Russet Burbank and Desiree were used with inoculum prepared as for PT trials and applied to potting soil just prior to planting. Treatments included three rates of mancozeb equivalent to 3. A control with no inoculum or fungicide was included. Seedlings were transplanted 4 April into individual pots 20 cm diameter, 4. In both trials, each treatment was replicated three times one plant per pot with pots arranged in a randomised complete block design and hand watered when required to maintain constant wet soil conditions. There were no additional pesticide or fertilizer applications. In PT5 only root disease zoosporangial score was measured. Powdery scab had been recorded in recent potato crops at both sites. Analysis of soil sampled just prior to planting by qPCR [ 18 , 19 ] gave very low S. Seed and soil chemical treatments were applied at planting. Each treatment was replicated two times in each trial. Plots contained 15 seed tubers, spaced at 30 cm, with plots arranged in a randomized split-plot design. Fertiliser and irrigation scheduling followed standard commercial practice with no additional seed or soil pesticides applied. In both FTs the average emergence date was on 25th November with destructive sequential harvesting of one plant per plot made at 15, 30, 45, 60 and 75 DAE. All other plants 10 per plot were grown until senescence, tubers harvested and a random sample of 50 tubers per plot selected for disease assessment. This was to confirm the presence and quantity of pathogen in soil and tuber samples. DNA extraction from root tissue followed a modified technique with established quantification methodologies [ 8 , 21 ]. This was used as a positive internal control to confirm DNA quality, PCR amplification conditions and to normalise qPCR data for accurate quantification of the pathogen content. Ltd, Australia [ 8 ]. Disease assessment Root diseaseâ€"root infection and galling Root hair infection was assessed by microscopic examination using a method modified from Merz [ 17 ]. From each plant, three samples of root 2â€"5 cm long were cut at c. Specimens were mounted on a glass slide, stained with aqueous 0. Fifteen fields of view were examined per slide with presence of zoosporangia rated: A root galling score was given per plant based on a visual rating scale modified from van de Graff et al. The percentage of tuber surface covered by lesions was then estimated by taking the mid values of these score ranges. The proportion of healthy tubers with no visible lesions was also recorded from which disease incidence was calculated [ 22 ]. Additionally, where different inoculation dates were utilised PT 1â€"3 a modified AUDPCi was also calculated measuring disease progress from the date of inoculum addition rather that the date of emergence. Data were truncated to ensure the same number of data points three was analysed for each curve. Where parameters were discrete, single measurements e. Data was only used where the assumptions of the general linear model could be met and where graphical diagnostics showed a normal distribution. Results Impact of delayed S. As expected, where application of inoculum was delayed there was a delay in the onset of infection

and a resultant decrease in the AUDPC.