Preliminary Evidence for Introduction and Spread of TRV under Center Pivot Irrigation[1,2]

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[2] *Names are necessary to report factually on available data; however, the USDA neither guarantees nor warrants the standard of the product, and the use of the name by USDA implies no approval of the product to the exclusion of others that may also be suitable.*

The Problem

A corky ringspot disease epidemic has been underway in the potato fields of the Columbia Basin since the late 1980's. The soils on more than 5000 acres in Washington are now contaminated with the causal virus, and new areas of contamination are found each year. An unusual aspect of the epidemic in the Columbia Basin is that the virus is found dispersed across the entire area of newly contaminated circles in the first year of its appearance. In the typical case, within-field virus spread from points of initial introduction is restricted to the dispersal rate of its nematode vectors in the soil, a few meters per year. Two questions critical to the resolution of the problem in the Columbia Basin remain unresolved. How is the virus being introduced into Columbia Basin fields, and why is it dispersed across entire fields in the first year of detection? This talk will address these questions in light of new experimental evidence 1) that TRV could be introduced into new soils by growing infected potato seed, a finding contrary to previous reports, and 2) that TRV may be introduced on center pivot access roads and then distributed around the circle on the wheels of the center pivot.

Understanding Corky Ringspot in the Columbia Basin

The first step in control is to understand the basic biology of the disease. Corky ringspot disease of potato is caused by tobacco rattle virus (TRV). Typically, the disease causes distinctive dark rings and arcs of dead tissue in tubers and renders them unmarketable. Tuber symptoms in the Columbia Basin are commonly not the typical rings and arcs but, rather, internal flecks and spots similar to those caused by internal brown spot (IBS). The virus has an extensive host range (Robinson and Harrison, 1989; Cooper and Harrison, 1973). that includes many common weeds and many of the crops commonly grown in rotation with potato. We have found that corn and small grains; Shepherds Purse, Lambsquarter, Chick Weed, Pigweed and Prickly lettuce are infected in the Columbia Basin. Infection of chickweed and Shepherds purse is of grave concern because seed transmission has been demonstrated in these hosts (Harrison and Robinson, 1978).

TRV may be spread in the soil by several species of stubby root nematodes (*Paratrichodorus* and *Trichodorus* spp.) (Brown et. al. 1989; Van Hoof, 1968) but spread of the virus in the Columbia Basin appears to depend on a single, indigenous species, *Trichodorus allius* (Santo, et. al. 1997), that propagates well on many weeds and crops grown there. The nematode vectors acquire and transmit the virus while feeding (Taylor and Robertson, 1970) on root cells of their host plants. They can survive in the soil without host plants up to 3 years and still retain an ability to transmit the virus (Van Hoof, 1971). Some chemical treatments reduce nematode numbers and provide temporary but not long-term control of the disease. R. E. Ingham (Ingham, 1993) tested a wide variety of chemicals in Oregon. Only fumigation with Telone II or treatment with Temik alone and in various combinations provided good control. More recently, Santo et.al.(Santo et. al. 1998) found that Temik treatments applied under new regulations does not provide adequate temporary control. Thus, only Telone II is available to provide good chemical control of the disease.

Historically, contamination of soil with TRV took the land out of potato production. Long-term control was not possible in the past since neither the causal virus or the nematodes that spread it could be totally eliminated from contaminated soils. More recently, we have evidence that cultivation of **weed-free** alfalfa can eliminate the virus, but not its nematode vector, from contaminated soils (Thomas, 1998; Thomas et.al., 1999). Many growers have now tried this approach and report encouraging results.

Both the nematode vector and the virus are required to introduce TRV into new soils. Although the nematode vector is endemic in the Columbia Basin, the virus is not, and the vector it is not uniformly present in all fields. The virus apparently was not present until recent years. We reported the first case of corky ringspot in Washington state in 1975 (Thomas, 1977). Growers were advised then and again in 1990 (Thomas and Santo, 1990) on methods to prevent or reduce spread of the virus to new soils, and the disease initially appeared to remain localized to a few locations. From the first report in 1975 until 1989, only two new cases were diagnosed in our lab. However, after use of Temik was eliminated in 1989, the disease began to appear in new locations that had no previous history of corky ringspot (Thomas, et.al., 1993). By repressing numbers of nematode vectors, the widespread use of Temik may have masked dissemination of the virus during these years.

Although, TRV is commonly found dispersed across the entire area of a newly contaminated circle in the first year of its appearance, it is important to understand the virus may have been introduced in an earlier crop and then spread undetected in the highly susceptible crops that are grown in rotation with potato in the Columbia Basin. Major rotation crops, particularly corn, wheat and other small grains are much better hosts of TRV and its vector than potato. Thus, within-field spread and build-up of the virus may be most rampant in the rotation crops.

Potential for Introduction of TRV in Potato Seed Stocks.

The fact that TRV typically occurs spread across entire circles in the first year of its appearance in the Columbia Basin suggests that the virus may be introduced across the entire circle. In that case, within-field spread of the virus would not be required. Introduction of the virus in irrigation water could achieve widespread distribution in a field, but we reject this possibility because many circles in an area would become contaminated simultaneously from a common source of water. TRV may also be introduced broadly in fields that already contain the nematode vector in infected, vegetatively propagated plants, such as tulip, narcissus, daffodil, and other flower bulbs and roots and in nematodes in soils associated with transplants (Harrison and Robinson, 1978). Based on cultural practices in the Columbia Basin, only potato itself could serve as the source of virus in propagation stocks.

The potential for introduction of TRV into new soils in seed potatoes has generally been rejected in the past. This rejection was based largely on a report from Holland (Engspro, 1976) that the virus was not introduced into virus-free soil after growing TRV-infected seed for 7 years on the same soil. The fact that potato is generally a very poor host of TRV may have given credence to this early report that may have inhibited subsequent testing of the earlier results. Depending on the potato cultivar and the virus strain, TRV is often confined to the symptomatic regions of infected tubers and does not move from seed pieces to roots or foliage of the potato. In those instances when it does move, the virus that moves is usually a naked RNA form that is not transmissible by nematodes and is not mechanically transmissible.

Contrary to the earlier reports, we (Crosslin and Thomas, 1998) have found that the roots of about 2% of the plants produced from infected seed of our major cultivars are systemically infected with the form of TRV that is transmissible by nematodes. Our studies show that both symptomatic and asymptomatic tubers harvested from TRV-contaminated soils may produce potato plants that are systemically infected with the form of the TRV that is transmissible by nematodes. Furthermore, we have actually transmitted TRV from infected potato roots to tobacco plants. Thus, potato roots do sometimes become infected with the nematode-transmissible form of TRV from the seed pieces in the Columbia Basin, and the infected potato roots certainly can serve as a source of TRV for infection of other plants. Thus, it seem clear that Northwest strains of TRV may be introduced into virus-free soils through infected seed potatoes, although a clear case where this has occurred in a commercial planting has never been proven. The movement of the form of TRV that is transmissible by nematodes from seed pieces to roots of new plants has been verified (Xenophontos et.al. 1999), but transmission of TRV from those roots to other plants has not been verified.

At the 2% rate of root infection we have demonstrated with the form of TRV that is transmissible by nematodes, a 1% infection rate in the seed potatoes could produce four infection centers per acre. With the rapid increase in virus that would occur in the highly susceptible rotation crops (corn and wheat) grown in the Columbia Basin, this level of introduction could produce the field-wide distribution of TRV contamination observed in the Columbia Basin in but one cycle of the rotation.

Columbia Basin sources of seed potatoes are not actually tested for TRV. The fact that the symptoms of TRV in the Columbia Basin often resemble IBS may have allowed the disease to escape visual detection in the seed potato in the past. It seems clear that seed lots should be examined for internal symptoms, and seed lots expressing IBS symptoms should be tested for TRV. A test was not available to detect the virus in tubers in previous years, but we (Crosslin and Thomas, 1995) have now developed a highly reliable PCR procedure that detects TRV in tubers.

Potential for introduction and spread of TRV in infected nematodes:

Both the TRV and its vector may be introduced simultaneously in infective nematodes in soil clinging to machinery, boots, or the hooves or feet of animals. These modes of introduction would normally result in localized spots of contamination in the circle. This would mandate a highly efficient mode of within-field disbursement from the localized spots of contamination to account for the wide-spread occurrence of the virus across entire circles observed in the Columbia Basin. Pasturing animals on circles with localized areas of contamination could result in a relatively rapid disbursement of the virus across a circle, but this is not a common practice on potato circles of the Columbia Basin.

We have observed only one mode of field disbursement that could play a role in rapid, within-field disbursement of TRV under the cultural practices of the Columbia Basin. Our hypothesis stipulates that the virus in its nematode vector may be deposited on the center pivot access road. The infective nematodes are then picked up from the access road and carried around the circle on the wheels of the center pivot system. It is a relatively short distance then for normal movement of the nematodes to fill in the distance between the wheel tracks.

This hypothesis is based on a TRV disbursement pattern we observed associated with the center pivot system in a field in Colorado. We were able to observe this distribution pattern only because the field is contaminated with an apparent new strain of the virus that invades potato foliage and causes distinctive foliage symptoms. Systemic foliage infection of potato with TRV is rare and has never been reported to occur routinely. However, we observed areas of TRV infection in a half-circle field in Colorado where plant emergence was much delayed as compared with emergence in non-diseased areas of the half-circle, and plants were severely stunted after emergence (See Figure 1A and 1B). In aerial photographs (Figure 2), these disease symptoms provided a striking visual assessment of TRV distribution in the half-circle that previously was never possible. Symptomatic areas are slightly darker in the photograph and have a darker border.

The distribution of symptoms in the half-circle is clearly associated with the access road, which traverses the entire circle, and the wheel tracts of the center pivot system (Figure 2). In addition to the symptomatic areas associated with the wheel tracts and access road, there are several circular, symptomatic spots that occur randomly in the half-circle. The grower pastured cattle on the circle for a short time two years earlier. Movement of the virus on the hooves of the cattle during that period may account for these random areas of infection.

This half-circle also grew potatoes in the year previous to the current crop. The grower reports that there were no corky ringspot symptoms in the tubers harvested that year, and he noticed no areas that expressed the symptoms obvious in the current season. This information may give some indication concerning rate of virus spread in the half-circle.

Growers may be able to guard against introduction of TRV on access roads by thorough cleaning of vehicles prior to using the roads. If this is not a practical option, it may be possible to prevent spread of nematodes from the access road by periodic treatment of the road with nematicide.

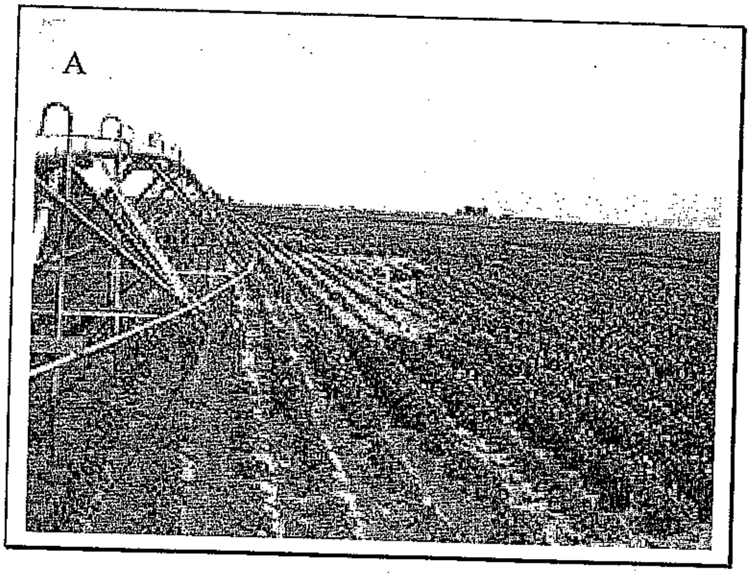
Literature cited:

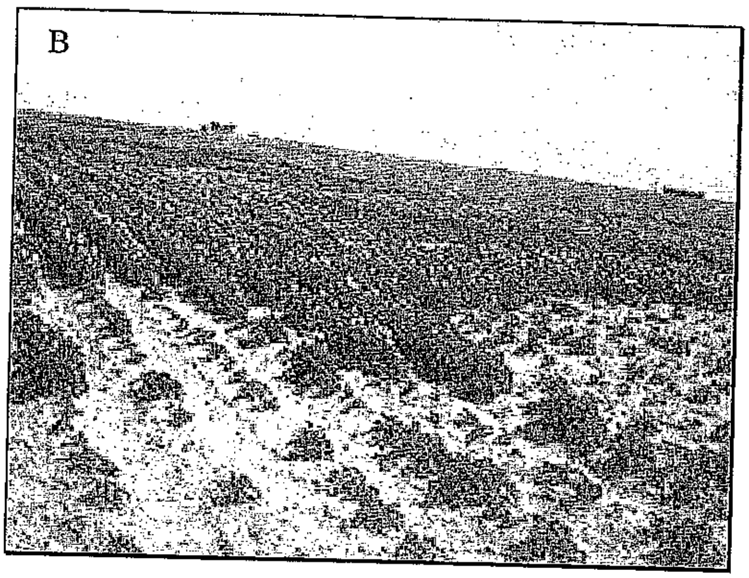
1. Brown, D.J.F., A.T. Ploeg, and D.J. Robinson. 1989. A review of reported associations between *Trichodorus* and *Paratrichodorus* species (Nematoda: Trichodoridae) and tobraviruses, with a description of laboratory methods for examining virus transmission by trichodorids. Revue de Nematologie 12:235-241.
2. Cooper, J.I., and B.D. Harritson. 1973. The role of weed hosts and activity of vector nematodes in the ecology of tobacco rattle virus. Annals of Appl. Biol. 73:53-66.
3. Crosslin, J. M. and Thomas, P. E. 1995. Detection of tobacco rattle virus in potato tubers by use of PCR. Am. Pot. J. 72:605-609.
4. Crosslin, J.M., P.E. Thomas, and C.R. Brown. 1999. Distribution of tobacco rattle virus in tubers of resistant and susceptible potatoes and systemic movement of virus into daughter plants. Amer. J of Potato Res. 76:191-197.
5. Engsbro, B. 1976. Undersogelser og forsog vedrorende jordbarne vira I. Rattlevirus fortsatte undersogelser I kartofler. Tidsskrift for Planteavl 80:405-410.
6. Harrison, B.D. and D.J. Robinson. 1978. The Tobraviruses. Adv. in Virus Res. 23:25-77.
7. Ingham, Proc. of the 32nd Annual Washington State Potato Conf. and Trade Fair. 1993
8. Robinson, D.J. 1992. Detection of tobacco rattle virus by reverse transcription of polymerase chain reaction. J. of Virol. Methods 40:57-66.
9. Robinson, D.J. and B.D. Harrison. 1989. Tobacco rattle virus. *In* AAB Descriptions of Plant Viruses. 346:In press
10. Santo, G.S.Proc. of the Annual Washington State Potato Conf. and Trade Fair 1998
11. Santo, G.S., Mojtahedi, H., Brown, C.R., Thomas, P.E. and Crosslin, J.M. 1997.Host range of Paratrichodorus allius and resistance of potato to the corky ringspot disease. Journal of Nematology 29:604.
12. Taylor, C.E. and W.M. Robertson. 1970. Location of tobacco rattle virus in the nematode vector *Trichodorus pachydermus* Seinhorst. J. of Gen. Vir. 6:179-182.
13. Thomas, P. E. 1977. Corky ringspot disease in Washington. Spud Topics 20:25.
14. Thomas, P.E., Mojtahedi, H., Crosslin, J.M., and Santo, G.S. 1999. Eradication of tobacco rattle virus from soils by growing weed-free alfalfa,

7th International Plant Virus Epidemiology Symposium, Aguadulce (Almeria) Spain, April 11-16.

15. Thomas, P. E. and G. S. Santo. 1990. Control of corky ringspot disease of potato in Washington. Spud Topics 36:10.
16. Thomas, P.E., G.S. Santo, and C.R. Brown. 1993. Corky Ringspot in the Columbia Basin. Spud Topics 38:24. 1993.
17. Thomas, P.E., Crosslin, J.M., and Richards, K. 1998. Control and eradication of corky ringspot disease. Proceedings of the Annual Washington State Potato Conference and Trade Fair 37:87-91.
18. Van Der Wilk, F., M. Korsman, and F. Zoon. 1994. Detection of tobacco rattle virus in nematodes by reverse transcription and polymerase chain reaction. European Jour. Of Plant Path. 100:109-122.
19. Van Hoof, H.A. 1968. Transmission of tobacco rattle virus by Trichodorus species. Nematologica 14:20-28.
20. Van Hoof, H.A. 1971. Onderzoek van virussen die samenhangen met de ground: tabaksratel-virus. Annual Report for 1970. Instituut voor Plantenziektenkundig Onderzoek, Wageningen, p 81.
21. Xenophontos, S., D.J. Robinson, M.F.B. Dale, and D.J.F. Brown. 1998. Evidence for persistent symptomless infection of some potato cultivars with tobacco rattle virus. Potato Res. 41:255-265

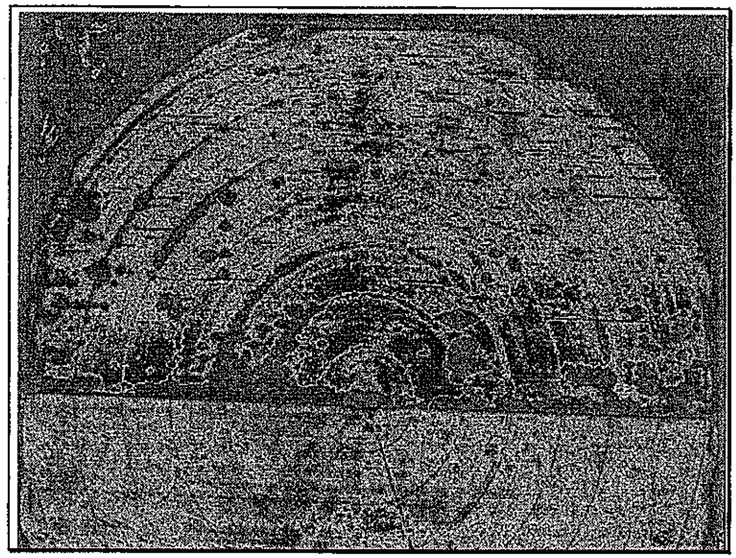
Figure 1.





Delayed plant emergence in a potato field caused by infection with TRV. TRV is restricted to the affected areas of the field.

Figure 2.



A half-circle (upper half) of potatoes with areas of delayed emergence caused by TRV. The TRV affected areas are associated with the access road and with the center pivot wheel tracks. The lower half of the circle is not relevant to this observation.