Epidemiology and Spread of Soybean Rust

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Introduction

Soybean rust is caused by two fungal species, *Phakopsora meibomiae* and *P. pachyrhizi*. The Asian soybean rust pathogen is known as *P. pachyrhizi* and is the species of greater concern since it is the more aggressive species and it has been identified in new geographical locations beyond Asia. *Phakopsora meibomiae*, the less virulent species, has only been found in the Western hemisphere, and it is not known to cause severe yield losses in soybean. The focus of almost all the literature (Hartman et al., 1992) and this paper is on *P. pachyrhizi.*

Soybean rust is one of the major diseases of soybean in many Asian countries (Sinclair and Hartman, 1999) and now in Brazil, since its recent introduction in 2001 (Yorinori, 2003). Significant yield losses due to rust have been reported in most soybean-producing countries throughout Asia, where the disease is endemic. In some locations where rust is endemic, it may be a limiting factor in soybean production. The continental U.S. is one of the last major soybean production areas without soybean rust.

Geographical Distribution

P. pachyrhizi. The first report of soybean rust in the literature was from Japan in 1902. By 1934 the pathogen had been reported throughout much of Asia and Australia as well as on many of the islands between them (Fig. 1A). In tropical Southeast Asia, the disease is endemic and occurs throughout most of the year. The disease has been reported as far north as Siberia, Manchuria, North Korea, and northern Japan where it occurs infrequently. Since spores are moved by wind from the south into the northern soybean production areas, the disease is usually found late in the season.

On the African continent, the distribution of soybean rust was not well known before 1996. There was a report in 1982 of soybean rust in Togo (Mawuena, 1982), but since then a more expanded view of soybean rust in Africa has been reported indicating that it was found in 1996 in Uganda, Kenya, and Rwanda, in Zimbabwe and Zambia during 1998, Nigeria in 1999, Mozambique in 2000, and South Africa in 2001 (Levy, 2003).

Soybean rust was first reported in the U.S. in Hawaii in 1994, where the pathogen was found on soybean on Oahu, Kakaha, Kauai, and Hilo islands (Killgore, 1995). The pathogen is still present in Hawaii and precautions have been outlined by the Hawaiian Department of Agriculture including recommendations for seed movement.

The first detection of *P. pachyrhizi* in South America was in Paraguay in the 2000-2001 growing season (Yorinori et al., 2003). The disease was found on soybeans grown in the Parana River basin on the eastern border with Brazil in a limited number of fields. The presence of the disease was confirmed in the 2001-2002 season in both Paraguay and Brazil, with reports of severe disease in some fields. Argentina confirmed the occurrence of soybean rust in early 2002 (Rossi, 2003). During the 2003 growing season the pathogen was found in most of the soybean growing regions of Brazil with the first report in Bolivia late in the season (Yorinori et al., 2003). In 2002, the pathogen spread from an estimated 10% of the soybean production area near the Parana River bordering Brazil to over 90% of the total production area of Brazil.

Potential movement to North America. The North American continent has not had confirmed reports of soybean rust. A recent article (Miles et al., 2003) asked the question whether the U.S. soybean crop is at risk. One of the possible pathways that the disease may reach the soybean growing areas in North America is through the land bridge that extends from South America through Central America and into the southern U.S. Soybean and other susceptible hosts are found throughout this land bridge (Fig. 1B). Another possible spore pathway may occur on winds from Africa to the Caribbean or to the U.S. mainland. Also, the pathogen may enter the U.S. mainland through debris in seed produced in South America or as a contaminant on clothes, boots, etc., of people moving from infested regions back to the U.S.

Regardless of how the pathogen moves, another concern is that the fungus infects many other hosts and it is likely that these hosts may play a role in its spread. The fungus is known to infect 95 species of plants from 42 genera (Ono et al., 1992). These bridge hosts can play an important role in movement and survival of the pathogen whether soybean is present or not. For example, the common exotic weed Kudzu that covers approximately 8 million acres in the U.S. may serve as not only a bridge host on which the fungus may over-winter in the southern-most parts of the U.S., but also may provide a springboard, by increasing spore numbers early in the season for transport to soybean fields.

With the concern that *P. pachyrhizi* may enter into the soybean belt in the U.S., there has been increased interest in studying aerial dispersal of rust spores and developing models to forecast the spread of the pathogen. At the Soybean Rust Workshop in 1995, Yang (1995) provided an update on assessment and management of the risk of soybean rust. His overview provided a review of the rust model and risk management. More recently, Yang (2003), outlined the establishment and dispersal potential of the disease in the U.S. soybean production region. One new project under development involves a global network for reporting soybean rust using a high-resolution internet mapping for recording spatial and temporal dimensions of infestations. Along with this, a system to generate daily forecasts of the risk of aerial dispersal of *P. pachyrhizi* spores to U.S. soybean fields is being developed. Although it may be difficult to estimate the exact number of spores released into the air, estimates of spore release indicate that 10^{12} spores per hectare per day could be release during the height of an epidemic (S. A. Isard, pers. comm.). When dealing with predictive models, it is important to understand how an epidemic develops. Such models need to consider aerial transport of spores (Isard and Gage, 2001) as part of a bigger picture examining all aspects of spores, including production, escape from the canopy, turbulent movement and dilution in the atmosphere, survival while airborne, deposition, and conditions relating to germination (Fig. 2).

Components of an Epidemic

In 1978 the United States Department of Agriculture (USDA) and the Asian Vegetable Research and Development Center (AVRDC) started a cooperative project to study the epidemiology of soybean rust. This cooperative project continued until 1982 (Tschanz, 1982) and the research on soybean rust continued at AVRDC until 1992 (Hartman, 1995). This initial research at AVRDC/USDA helped to establish what we now know about soybean rust epidemiology. After soybean rust was found in Hawaii in 1994 (Killgore, 1995), the United Soybean Board sponsored a workshop on soybean rust in order to develop recommendations for action to deal with the possible introduction of soybean rust into the continental U.S. (Sinclair and Hartman, 1996). Since soybean rust is no longer confined to the Eastern Hemisphere any longer, additional studies on epidemiology and spread of rust have been initiated.

There are many factors to consider when studying epidemics. The classic disease triangle of host, pathogen and environment overlaid with time is important. For example, the infection of soybean by *P. pachyrhizi* is conditioned by an interaction of temperature and moisture. Although there are many studies related to each of the parameters in the triangle, the following just briefly outlines what may be the most important components.

Host. Soybean can be infected by *P. pachyrhizi* at all growth stages. In the field, the rate of rust development is related to the physiological age of the plant. Rapid increases in lesion numbers are observed with the onset of flowering. Early maturing cultivars develop rust earlier and at a more rapid rate than later maturing cultivars. Therefore, when evaluating rust severities among cultivars or treatments, the effect of host maturity on rust severity needs to be considered. A correction factor using the relative lifetime (RLT), a time element from 0 to 100, helps to correct for differences in host maturity (Tschanz, 1982). This interaction between host development and rust increase occurs in all field experiments and confounds rust ratings between lines.

The pathogen. This is the missing link so far in the soybean production belt of North America; although once it arrives questions related to survival will be important. So far, only a few studies have reported on survival of urediniospores. When stored in plastic bags, spores were viable for up to 68 days, adding or removing moisture from infected leaves reduced spore viability (Misman and Purwati, 1985). Additional data indicates a survival of up to 55 days when kept dried at 15 to 20 °C, but freezing or near freezing temperatures reduce spore viability (Patil et al., 1997). Few studies have examined the intensity and exposure time to sunlight or UV light, which could be a major degradation factor of spores moving long distances.

In addition to a having a broad host range as mentioned earlier, the pathogen also has a complex virulence pattern. Yeh (1983) identified three races among 50 single uredial cultures using a set of five differential cultivars/lines. Other isolates from Taiwan were reported to cause rust on all known or suspected sources of specific resistance, including PI 200492, PI 230970, PI 339871, PI 459025, and PI 462312 (Tschanz et al., 1986). The presence of multiple virulence genes in the pathogen population and the absence of multiple specific resistance genes in the host could make techniques like gene rotation and pyramiding of specific resistance genes ineffective.

Environmental conditions. Spores germinate 1 to 4 hours after deposition on a leaf under ideal conditions. Germination is affected by temperature, with 18 to 25 °C considered optimum. Direct penetration of the leaf surface occurs within 6 hours at temperatures of 20 to 25 °C. Maximum infection rates occur with a dew period of 10 to 12 hours. As temperatures decrease the length of the dew period needed for minimum infection increases. Interruption of the dew period also produces lower infection rates.

Some of the more precise data from field experiments have been derived from studies done in Taiwan at AVRDC (Tschanz 1982; Wang and Hartman, 1992). Field studies indicated that low and high temperatures, below 15 and above 28 °C, reduced lesion numbers and prevented lesion development in the field. In a 2-year study in Taiwan from 1980 to 1981, plants that were grown in the spring and fall seasons had more disease than plants grown in the summer season. This was primarily due to temperature and rainfall patterns. In that part of Taiwan, the spring and fall seasons are much like the summer conditions in the soybean belt. It was noted that rust development was affected by precipitation patterns, with the most rapid rust development occurring when precipitation was evenly distributed throughout the growing season. Furthermore, soybean rust severity was positively correlated with rainfall and the number or rainy days during pod-fill (Tschanz 1982).

Rust Development and Yield Losses

Disease progress curves. Monitoring rust epidemics has provided important information on how the disease develops. Various schemes for rating rust have included using a three-digit notation (Yang, 1977), Horsfall-Barratt scale (Tschanz and Wang, 1980), and percentage leaf area infected (Hartman et al., 1991).

In addition to leaf ratings, defoliation also needs to be considered. This is usually done by counting leaves on plants in fungicide control plots versus those in rust infected plots. Several reports have used healthy tissue or green leaf area to compare treatments that compensate for the loss of leaves (Hartman et al., 1991; Yang et al., 1991b). Assessments must be done over time during the growing season to be sufficient to analyze disease progress data. Most reports have used the disease severity ratings or green leaf area progress data to develop areas under the curve to make statistical comparisons of treatments, whether they are fungicide applications or tests of different cultivars/lines. Based primarily on dew period and temperature, a simulation model for assessing soybean rust epidemics was developed (Yang et al., 1991a).

Yield losses. Yield losses of up to 80% have been reported from experimental trials in many countries throughout Asia and in Australia. Heavily infected plants have fewer pods and lighter seed (Hartman et al., 1991; Yang et al., 1991b). Marketable yields are even less because of poor seed quality (Tschanz and Wang, 1980).

Disease parameters to yield components. Several publications have addressed the issue of quantifying disease parameters like leaf area infected, defoliation, pustule counts, and area under curves to yield components. The leaf area infected, defoliation, and total number of pustules per plant, per leaf, and at node 7 on 12 soybean lines inoculated with *P. pachyrhizii* were compared

as well as yields and seed weights (Hartman, 1995). Data such as this is useful for developing predictive models for yield losses as well as for understanding how fast the pathogen spreads within the soybean canopy. Yang et al., (1991b) regressed the relative area under the disease progress curve to seed growth rates, seed growth periods from R4 to R7 growth stage, and yield. Hartman et al., (1991) regressed leaf area infected at growth stage R6 and the area under the disease progress curve to percentage yield of fungicide-protected plots. In these reports and in others, quantifying disease parameters to yield was effective and with additional information, this data has provided the basis for disease forecasting and yield loss models.

Conclusions

The epidemiology of soybean rust has once again become a major focus of research because of the recent movement of *P. pachyrhizi* to so many new locations and its inevitable entry into the continental U.S. The research of over 20 years completed at AVRDC has provided a number of important discoveries related to the interaction of soybeans, *P. pachyrhizi*, and the environment. However, perplexing questions remain unanswered. For example, where does the initial inoculum originate? Precisely what environmental parameters cause the most severe rust epidemics? How far can spores travel from field to field or in the upper atmosphere and still be infective? Can remote sensing be used to monitor soybean rust over large areas? Will predominate races or populations cause variations in epidemics? Considerable more data needs to be collected to fully develop and utilize forecasting models or systems that will provide the information necessary for soybean growers to cost effectively manage soybean rust.

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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 the USDA implies no approval of the product to the exclusion of others that may also be suitable.

References

Hartman, G.L., E.M. Saadaoui, and A.T. Tschanz. Scientific Editors. 1992. Annotated Bibliography of Soybean Rust (*Phakopsora pachyrhizi* Sydow), AVRDC Library Bibliography Series 4-1, Tropical Vegetable Information Service. Taipei: Asian Vegetable Research and Development Center.

Hartman, G.L. 1995. Highlights of soybean rust research at the Asian Vegetable Research and Development Center, pp. 19-28 In J. B. Sinclair and G. L. Hartman, eds. Proceedings of the Soybean Rust Workshop, 9-11 August 1995, Vol. 1. College of Agricultural, Consumer and Environmental Sciences, National Soybean Research Laboratory, Urbana, IL.

Hartman, G.L., T.C. Wang, and A.T. Tschanz. 1991. Soybean rust development and the quantitative relationship between rust severity and soybean yield. Plant Dis. 75:596-600.

Killgore, E. 1995. Field notes on the detection of soybean rust, initial surveys and the current status of the disease in Hawaii, pp. 38-45, In J. B. Sinclair and G. L. Hartman, eds. Proceedings of the Soybean Rust Workshop, 9-11 August 1995, Vol. 1. College of Agricultural, Consumer and Environmental Sciences, National Soybean Research Laboratory, Urbana, IL.

Isard, S. A. and S.H. Gage. 2001. Flow of life in the atmosphere: an airscape approach to understanding invasive organisms. Michigan State University Press, East Landsing, MI 239 pp.

Levy, C. 2003. Measures to control soybean rust in Southern Africa and an initial investigation of the meteorological factors that favor its development. (Abstr.) Phytopathology 93:S103.

Mawuena, G. 1982. Preliminary observations on the soybean rust incidence in Togo. Soybean Rust Newsl. 5:20-21.

Miles, M. R., R.D. Frederick, and G.L. Hartman. 2003. Soybean rust: Is the U. S. soybean crop at risk? APSnet Feature Article, http://www.apsnet.org/online/feature/rust.

Misman, R. and E.S. Purwati. 1985. A study on host plant types of soybean rust fungi (*Phakopsora pachyrhizi* Syd.) on various types of legumes. Bull. Ilmaih Unsoed 11:68-82.

Ono, Y., P. Buritica, and J.F. Hennen. 1992. Delimitation of *Phakopsora*, *Physopella* and *Cerotelium* and their species on Leguminosae. Mycol. Res. 96:825-850.

Patil, V.S., R.V. Wuike, C.S. Thakare, and B.B. Chirame. 1997. Viability of uredospores of *Phakopsora pachyrhizi* Syd. at different storage conditions. J. Maharashtra Agric. Univ. 22:26-261.

Rossi, R.L. 2003. First report of *Phakopsora pachyrhizi*, the causal organism of soybean rust in the province of Misiones, Argentina. Plant Dis. 87:102.

Sinclair, J.B., and G.L. Hartman, (eds.) 1996. Soybean Rust Workshop, pp. 1-68. College of Agricultural, Consumer, and Environmental Sciences, National Soybean Research Laboratory Publication Number 1, Urbana, Illinois.

Sinclair, J.B., and G.L. Hartman. 1999. Rust, pp. 25-26, In G. L. Hartman, J.B. Sinclair, and J.R. Rupe, eds. Compendium of Soybean Diseases, 4th Ed. American Phytopathological Society, St. Paul.

Tschanz, A.T. 1982. Soybean Rust Epidemiology. Final Report. Asain Vegetable Research and Development Center, Shanhua, Tainan, Taiwan. 157 p.

Tschanz, A. T., and T.C. Wang. 1980. Soybean rust development and apparent infection rates at five locations in Taiwan. Prot. Ecol. 2:247-250.

Tschanz, A. T., T.C. Wang, and B.Y. Tsai. 1986. Recent advances in soybean rust research at AVRDC, pp. 23-245, In S. Shanmugasundaram and E. W. Sulzberger, eds., Soybeans in tropical and subtropical cropping system. ed., AVRDC, Shanhua, Tainan, Taiwan. p.237-245.

Wang, T.C., and G.L. Hartman. 1992. Epidemiology of soybean rust and breeding for host resistance. Pl. Prot. Bull. 34:109-124.

Yang, C. Y. 1977. The IWGSR rust rating system. Soybean Rust Newsl. 1:4-6.

Yang, X.B. 1995. Assessment and management of the risk of soybean rust, pp. 52-63, In J. B. Sinclair and G. L. Hartman, eds. Proceedings of the Soybean Rust Workshop, 9-11 August 1995, Vol. 1. College of Agricultural, Consumer and Environmental Sciences, National Soybean Research Laboratory, Urbana, IL.

Yang, X. B., W.M. Dowler, and A.T. Tschanz. 1991a. A simulation model for assessing soybean rust epidemics. J. Phytopathology 133:187-200.

Yang, X.B. and S. Pivonia. 2003. Risk assessment of soybean rust - establishment and dispersal potential of the disease in the U.S. soybean production region. (Abstr.) Phytopathology 93:S103.

Yang, X. B., A.T. Tschanz, W.M. Dowler, and T.C. Wang. 1991b. Development of yield loss models in relationships of components of soybean infected with *Phakopsora pachyrhizi*. Phytopathology 81:1420-1426.

Yeh, C. C. 1983. Physiological races of *Phakopsora pachyrhizi* in Taiwan. Jour. Agric. Res. China 32:69-74.

Yorinori, J.T., W.M. Paiva, R.D. Frederick, L.M. Costamilan, P.F. Bertagnolli, G.L. Hartman, C.V. Godoy, and J.J. Nunes. 2003. Epidemics of soybean rust (*Phakopsora pachyrhizi*) in Brazil and Paraguay from 2001 to 2003. Phytopathology 93:S103.

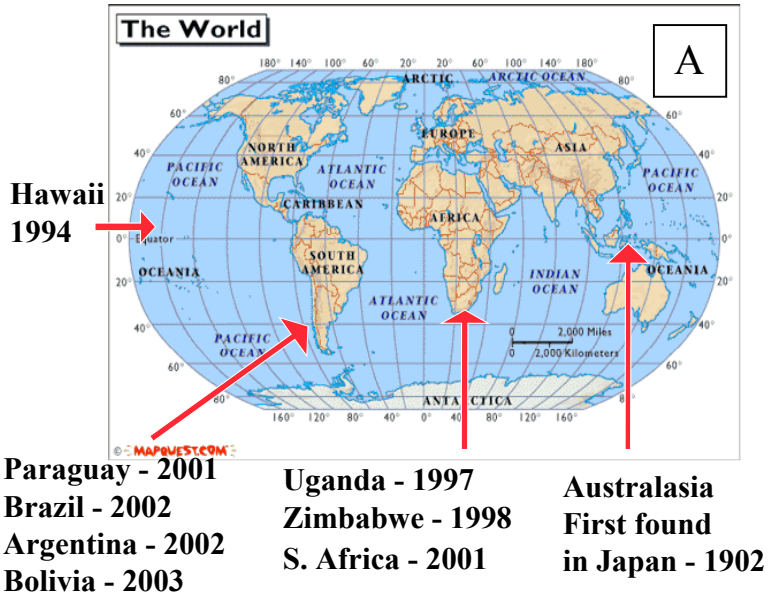

Fig. 1A. Distribution of soybean rust, caused by *Phakopsora pachyrhizi* and (B) potential spread of rust to North America.

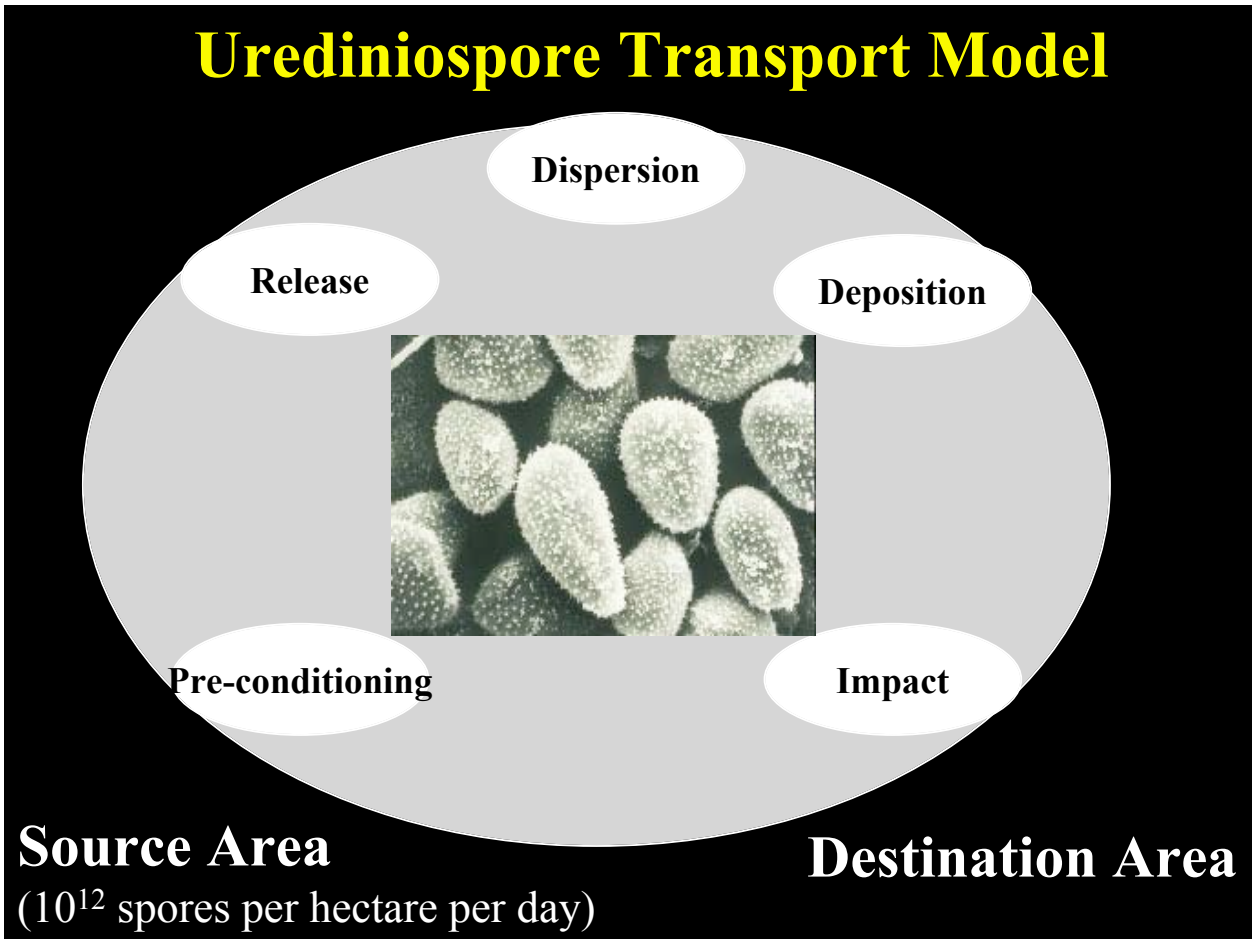

Fig. 2. Urediniospore transport model for *Phakopsora pachyrhizi* (from Isard and Gage, 2001)