PEST ASSESSMENT: *Puccinia tritici*, (Wheat leaf Rust)

USDA-APHIS-PPQ-CPHST-PERAL/ NCSU

Section A: Species Information

Scientific name:
Puccinia triticana Erikss. & Henn
Order:, Family:
Common Name: Wheat leaf Rust, Source:
Synonyms: *Puccinia recondite*

Figure 1. (a)

Description:

On wheat, the disease is recognized by small to relatively large yellowish-brown to cinnamon-brown pustules scattered on the upper leaf surface and leaf sheath (CPC, 2003). Small secondary pustules may develop in a circle around older pustules on susceptible host cultivars. On resistant cultivars, pustules may be small or appear only as necrotic spots which do not develop spores. A halo of pale green or yellow appears around the uredinium when host resistance is incomplete (McIntosh et al., 1995). When the temperature increases, some pustules turn black due to the production of teliospores. Telia remain covered by the host epidermis and are blackish-brown in colour.

On Thalictrum, pycnia are clustered in small groups on slightly swollen yellowish to reddish-brown areas on the upper leaf surface (CPC, 2003). Aecia are usually cupulate in clusters on gall-like areas on the undersurface of the leaf.

Life History:
Primary hosts: Triticum (wheat).

Secondary and alternate hosts: Anchusa (Bugloss), Clematis, Thalictrum (meadow rue).

Wild hosts: Elymus repens (couch grass), Triticale.

Epidemics in areas where alternate hosts are functional, occur when the overwintered telia from wheat produce basidiospores and infect the young leaves of Thalictrum. Pycnia bearing the spermatia (pycniospores) and receptive hyphae are produced on the upper surface of Thalictrum leaves in 7-10 days. These gametes are heterothallic and are carried between pycnia when insects visit in search of the nectar. Fertilization takes place when compatible spermatia come in contact with receptive hyphae; this is followed by the appearance of aecia on the lower side of the infected leaves. The binucleate aeciospores are capable of infecting nearby wheat plants and producing viable urediniospores.

When the source of inoculum to initiate epidemics is not aeciospores from alternate hosts, but urediniospores produced on infected wheat leaves, the inoculum may be either local or exogenous in nature. Aerial dispersal occurs as urediniospores are passively released from uredinia on wheat leaves. The urediniospores become airborne and may be transported up to several hundred kilometres in an air mass before being deposited by gravity (Hirst and Hurst, 1967) or washed out of the atmosphere by rain (Rowell and Romig, 1966). The annual movement northward of urediniospores from northern Mexico and southern Texas, USA, and the resulting development of leaf rust across the Great Plains of the USA into Canada in the spring serves as an excellent example of the effects of exogenous inoculum. However, infections may also occur from urediniospores that survive between wheat crops or during the dormancy stage of winter wheat on volunteer wheat or native grasses until a fresh crop of wheat is available (Chester, 1946; Eversmeyer and Kramer, 2000). Mehta (1940) suggested that these green bridges occurring in the cooler mountain areas of India and elsewhere serve as an over-summering source. Urediniospores are spread from these areas by wind to cause annual recurrence of the disease in areas a few hundred kilometres away.

Once primary infection centres become established in an area, subsequent inter- and intra-field spread is through wind-dispersed urediniospores (Chester, 1946; Joshi et al., 1977; Eversmeyer and Kramer, 2000). Survival of the pathogen between wheat crops or during dormanacy of the winter wheat crop may be critical to the development of severe epidemics in some of the major wheat-producing regions. After completing the infection process, the pathogen may remain latent for up to 60 days if temperatures are below 10°C. Under favourable weather conditions, one spore-uredinia-spore cycle may be completed in 7-8 days on a susceptible host, and a minimum of 7-9 cycles are needed, in a continuing 'compound interest' growth rate, to cause an epidemic in most areas.

A temperature of about 20°C and 4 hours of leaf wetness are conducive to infection by urediniospores. However, they can cause infection at temperatures between 2 and 32°C with free moisture. Once the leaf has become infected, temperature dictates the incubation period (Hogg et al., 1966; Eversmeyer et al., 1980). Uredinia may be converted into telia at about 35°C. Teliospores form towards the ripening stage of wheat and are not capable of causing autoinfection

Prediction of risk in the United States if Introduced:

Prediction Model:

Interactive templates are a tool to create models 'on the fly.' A combination of templates are used to model wheat rust including the Generic Infection Template and the generic disease template. These templates are described in appendix 2. The risk of wheat rust is influenced by weather conditions occurring during i) dispersal of inoculum; ii) overwintering; and iii) epidemic development during the growing season.

Other projects are examining conditions for dispersal of rust spores, so this will not be discussed in this document (Main et al. 2003). The fungus is an obligate parasite so it requires a living host to survive. In this analysis, we will use the maps developed for soybean rust to describe frost-free overwintering areas based upon the data of Pivonia and Yang (2003). The authors of the soybean study concluded that if the rust is introduced into the US during winter it is likely to be restricted to Florida in frost-free areas or areas where the fungus could overcome short periods of freezing temperatures. Consequently, occurrence of soybean rust epidemics in the US would depend on uredospores dispersal from south to north. The epidemic development during the growing season primarily depends upon sufficient moisture and favorable temperature conditions for infection, sporulation and dispersal. The infection process is often the key component for disease risk forecasting (Madden and Ellis 1988). This is due to the fact that infection requires moisture which is often limiting in most terrestrial environments.

The environmental conditions required for wheat rust infection have been well defined in temperature/leaf wetness combination studies (CABI 2003; Vallavieille Pope et al., 1995). The optimal temperature for infection is about 20-25 C and requires at least 4-5 hours of leaf wetness. Infection occurs between 3 and 30-32 C. The incubation period is about 7-9 days and the latent period is about 7 to 8 days.

In creating the wheat rust model, we assumed that the distribution of the organism can be related to the frequency of infection periods. The influence of weather on other biological factors such as overwintering, sporulation and dispersal were ignored. However, the conditions for sporulation are similar to that for infection. There was adequate data in the literature to construct the model. Data from other species however can be used for validation purposes.

Results

Cold stress

As a simple measure of cold stress we used the probability of freezing temperatures ($T \leq 0$ C). For computational purposes, we selected the first week in January as an indicator of these conditions based on 30-year climatic averages. Maps created for February were similar. The map shows the frost free zones are restricted to Southern regions of Florida and Arizona, Southeastern Texas and Coastal California.

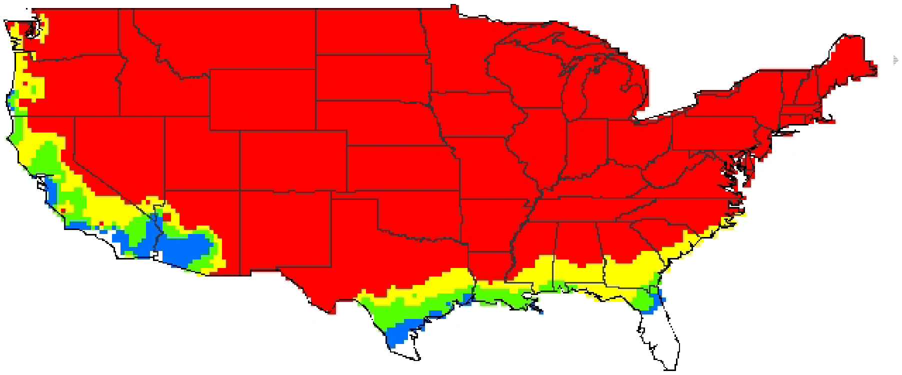

Figure 3. Probability map of freezing temperature occurrence ($T \leq 0$ C) during the period of January 1-7. Map based upon 30-year climatic averages.

Although the fungus may not overwinter in areas where freezing temperatures occur, the rust is easily dispersed large distances by winds.

Infection parameters

The recommendations for infection model parameters are shown in the table below. Values for *Puccinia striiformis* was also included as the model may also be helpful for validation purposes and for illustrating the differences between the species.

Table 1 Parameters obtained from the literature for Puccinia species pathogenic on wheat.

Pathogen	T_{min}	T_{max}	T_{opt}	W_{min}	**Reference**
Puccinia tritici	3	30	25	5	Vallavieille Pope *et al.*, 1995, Angus *et al.*, 1981.
Puccinia striiformis	3	18	9	6	Angus *et al.*, 1981, Dennis, 1987; Vallavieille Pope *et al.*, 1995

The Angus et al. (1981) study was used to determine the value for T_{min}, since this parameter was not adequately defined in the other studies.

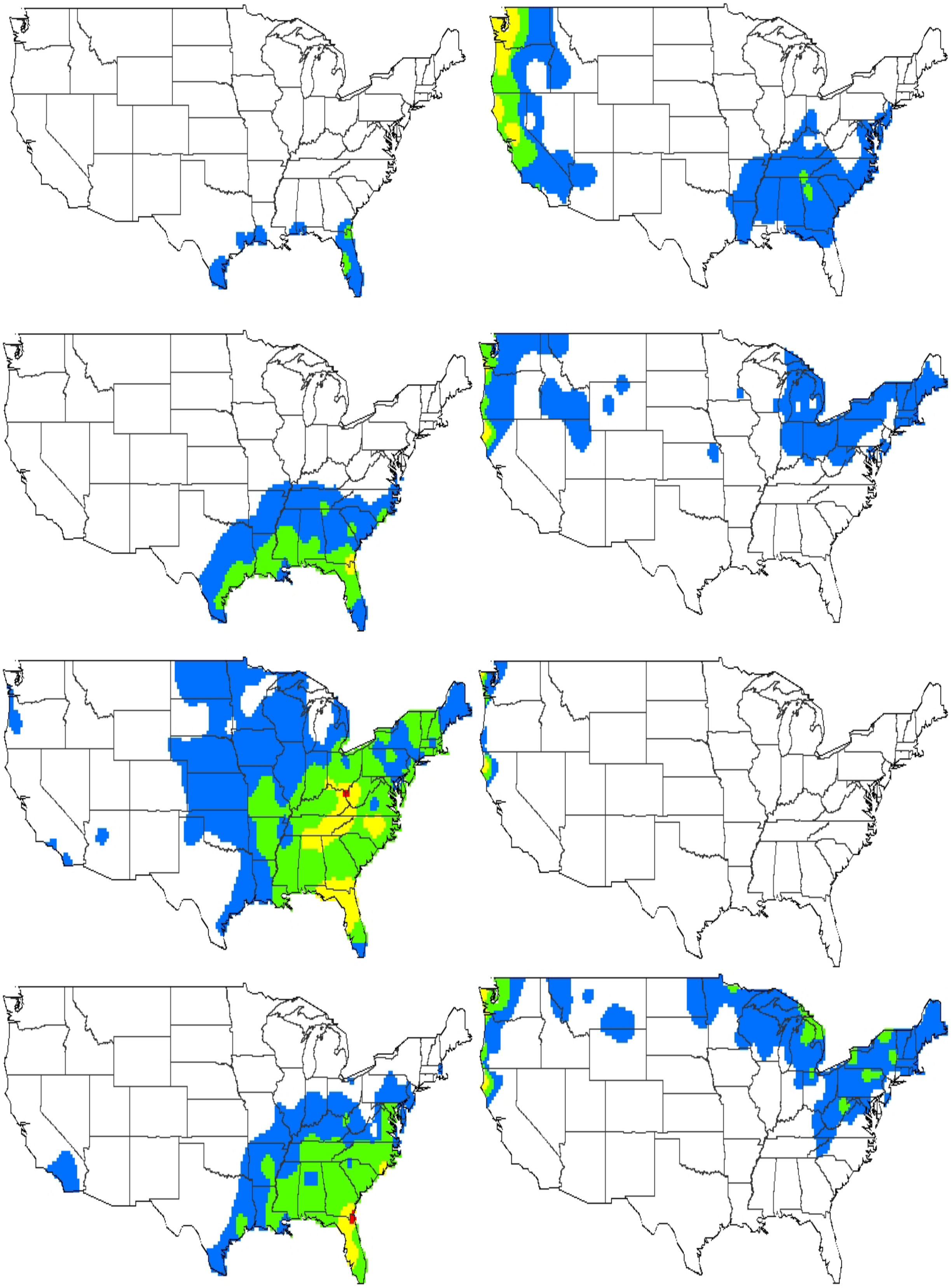

Figure 3. Probability maps of *P.* tritici (left) and *P. striiformis* (right) infection during the periods of March 1, May 1, July 1 and October 1 (top to bottom), respectively. Maps based upon 30-year climatic averages.

The maps are above are selected to represent periods of the growing season when wheat is susceptible to infection. Since planting dates and crop phenologies are variable, these periods of susceptibility may differ by location. The picture is also complicated by the fact that wheat is both a spring and winter planted crop. The maps above show how seasonal infection risk can differ even for closely related pathogens.

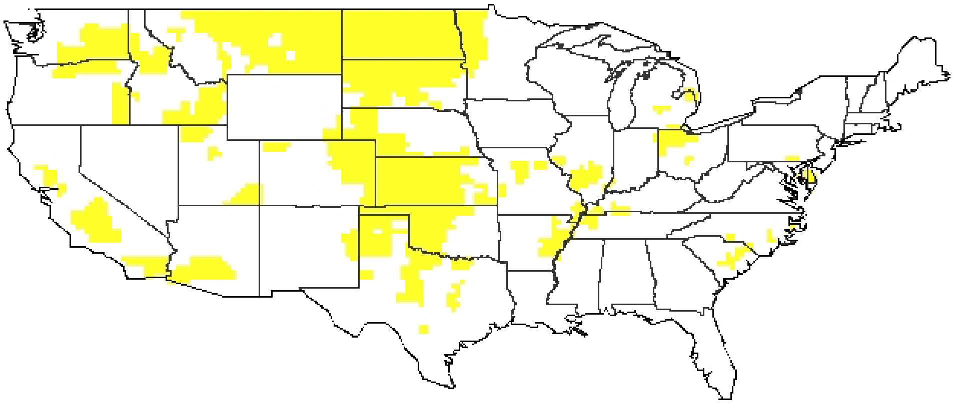

Figure 4. Counties in the United States where greater than 20,000 acres of wheat are grown. (Census of Agriculture 1997)

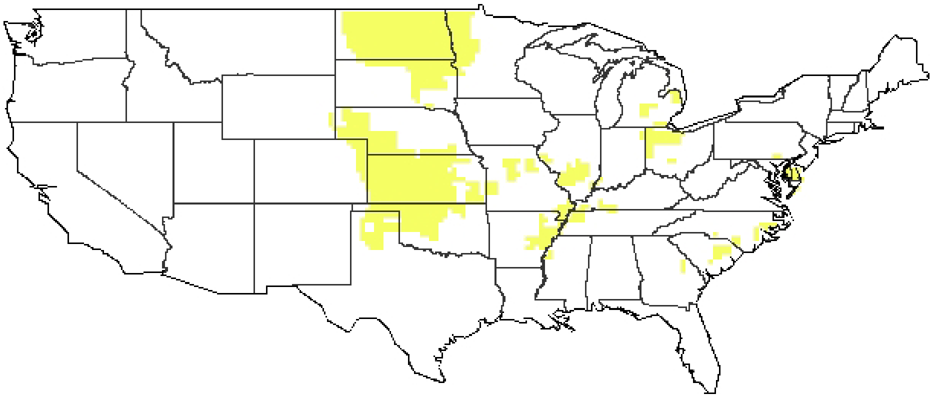

Figure 5. Regions of the United States where greater than 20,000 acres of wheat are grown and greater than two out of ten years with an infection period by *P. tritici* on July 1. Maps generated from 30 years of climactic data.

The final map (Fig. 5) is a demonstration of how the system can be used to show the overlap between areas where wheat is a major crop and where infection could be anticipated at a particular time of year. Interpretation of this map requires an agronomic knowledge of wheat production including planting dates and phenologies.

Acknowledgements:

Authors: Roger Magarey and Dan Borchert

Section D:

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