THE EFFECT OF TEMPERATURE DURING THREE DEVELOPMENTAL
STAGES OF THE WHEAT LEAF RUST SYSTEM

by

ANDREW D. NICKELL

B. S., University of Illinois, 1986

A MASTER'S THESIS

submitted in partial fulfillment of the

requirements for the degree

MASTER OF SCIENCE

Department of Plant Pathology

KANSAS STATE UNIVERSITY
Manhattan, Kansas

1988

Approved by:

Signature
Co-Major Professor

Signature
Co-Major Professor

Table of Contents

List of Tables

ACKNOWLEDGEMENTS

I wish to thank and express my gratitude to the co-major professors of this work, Dr. L. E. Browder and Dr. F. W. Schwenk. This thesis research was funded by the USDA-ARS, and I want to extend my appreciation to that agency. A special thanks to Dr. M. G. Eversmeyer, C. Elburn Parker and Cathy Bolte for all of their invaluable assistance. This thesis would not have been possible without the support of my parents, who have always been there when they were most needed.

The Effect of Temperature During Three Developmental

Stages of the Wheat Leaf Rust System

INTRODUCTION

Race-specific resistance to the cereal rusts and some other dis-
eases result from the function of corresponding gene pairs in the host
and parasite. Corresponding gene pairs are comprised of genes for
resistance in the host and complementary genes for avirulence in the
parasite. When both genes are present, the disease process is inter-
rupted. If, however, the gene for resistance or avirulence is absent,
the disease process continues uninterrupted (6). The phenotype express-
ed when a parasite and a host interact is a result of the genotype of
both entities functioning in the extant environment. Loegering (33)
called this genetic interaction an aegricorpus. An aegricorpus acts as
a third entity, deriving its phenotype from the genotypes of the host
and parasite and the influence that environment has on all three.

Several studies have shown the influence of environment on the
genetic mechanisms which interrupt the association between parasites
and their hosts. Waterhouse reported (54) that some cereal rust infec-
tion types were altered by temperature changes. Environmental influ-
ences are not limited to temperature effects; light also plays an impor-
tant role in disease development. Light is important in determining
the infection type of poplar rust (15,52), stem rust of wheat (1,17,
28), stem rust of oats (24), and corn rust (53). Chemicals also play

1

a role (30); DDT was shown to influence infection types of stem rust of wheat. DDT, applied to control white flies and other greenhouse insects on the wheat variety Khapli, resulted in high infection types instead of the normally low infection types. Many gene pairs appear to be specific to the temperature at which the parasite and host associate, in several parasite:host systems (2-4, 6-11, 13-14, 16-23, 25-27, 29, 31-32, 34-40, 42-53, 55-56). All corresponding gene pairs in the wheat leaf rust system are temperature specific (6). This phenomenon is not restricted to the wheat leaf rust system, but is universally applicable to all parasite:host systems (6).

Most of the studies have reported the effect of postinoculation environments on expression of resistance. Sharp (47,48) and Mohamed (41) have reported that preinoculation temperatures influenced final infection types. It has been demonstrated that _Lp1_/_Lr1_, _Lp16_/_Lr16_, and _Lp17_/_Lr17_ do not function at a 5 C postinoculation temperature (10). An experiment designed to determine if a low preinoculation temperature and moist period temperature also influenced the corresponding gene pairs is reported in this paper.

MATERIALS AND METHODS

Four lines of wheat (_Triticum aestivum_ L.) were inoculated with two cultures of _Puccinia recondita_ f. sp. _tritici_ Rob. ex Desm. and exposed to eight temperature-regime environments.

The wheat cultivar Thatcher, CI 10003, (abbreviated as TC) and three backcross-derived, near-isogenic lines of TC that vary in the presence or absence of the genes for low reaction _Lr1_, _Lr16_, and _Lr17_, were used to test the effect of preinoculation, moist period, and post inoculation temperatures on infection types. The lines used were LR1(TC), RL6003; LR16(TC), RL6005; and LR17(TC), RL6008; these lines were developed by P. L. Dyck and D. J. Samborski of the Agriculture Canada Research Station, Winnipeg, Manitoba (10). Thatcher and the three backcross lines have the following known genotypes: TC—Hr1Hr16Hr17, LR1(TC)—Lr1Hr16Hr17, LR16(TC)—Hr1Lr16Hr17, and LR17(TC)—Hr1Hr16Lr17. The abbreviation _Hr_ refers to the absence of the _Lr_ gene (10). The recurrent parent Thatcher was included as a control since it had none of the three genes for low reaction under consideration.

These lines were inoculated with _Puccinia recondita_ f. sp. _tritici_ cultures PRTUS3 and PRTUS21. Culture PRTUS3 had corresponding genes for low pathogenicity to _Lr1_, _Lr16_, and _Lr17_, when grown at continuous 20 C (10). Culture PRTUS21 had no identified genes for low pathogenicity corresponding to any genes for low reaction in the wheat lines utilized in the study (10).

3

Eight environments were created by varying sequences of 5 C and 20 C at each of three different phases of infection: preinoculation (PR), moist period (MP), and postinoculation (PT). A random design was used to determine the temperature each set of plants received during each phase.

Ten to twelve seeds of each of the four wheat lines were planted separately in one of the four positions in each four-inch diameter plastic pot. Order of host lines within pots, plants to be inoculated with the different cultures, and pots to be placed in the different environments were randomly selected. The experiment was performed three times with four replications of each parasite:host:environment treatments in each run. The seedlings were grown in a soil mix consisting of three parts loam:one part sand:one part peat moss. No supplemental nutrients were added throughout the experiment; water was added as needed. Each pot was marked to establish the first relative plant position in that pot. Plants to be placed in 5 C preinoculation treatment were seeded 25 days early, to compensate for slow seedling growth at the lower temperature. The remaining pots were seeded 10 days before inoculation. The staggered planting dates permitted a uniform inoculation.

Inoculation was accomplished by spraying fully expanded primary leaves with urediniospores suspended in Soltrol 170 oil. The inoculated seedlings were then placed inside black plastic bags, misted with tap water, sealed, and transferred to the appropriate moist period. The seedlings were removed from the bags after 16 hours and then transferred to the various postinoculation treatments.

4

Three types of data were recorded. First, the date of first sporulation was recorded and used to ascertain the length of the latent period. First sporulation was considered to occur when the majority of pustules were producing spores. Latent period was recorded as the time from inoculation to the date of first sporulation. If no sporulation was observed then the latent period was recorded as the length of the observation period. Next, estimates of the relative number of infection sites were recorded. Three categories were used: low (approximately 1 to 10 pustules per leaf), moderate (approximately 11 to 20), and heavy (21 and above). Finally, the amount of sporulation observed for each near-isogenic line was coded at the end of the observation period, utilizing the system proposed by Browder (5) and Browder and Young (12), during the postinfection period. Color slide photographs were made, after the observation period, of representative observations of the materials to illustrate differences within the results and determine consistency of the three experimental runs.

Results from combining four host lines to two P. recondita cultures are summarized in Tables 1 through 4. Table 1 summarizes the statistical analysis of the experiment. Differences in the relative amount of sporulation, relative number of infection sites, and latent periods were consistent among replications and trials when compared to Thatcher.

All four lines, regardless of environmental treatment, were observed with high infection types when associated with PRIUS21. These results indicate that PRIUS21 has a genotype of Hp1Hp16Hp17. The only discernible difference in the associations between the three near-isogenic lines with PRIUS21 occurred when the moist period temperature was 5 C. Numbers of infection sites were greatly reduced, from the heavy infection obtained by the other treatments to as little as a total of five pustules on all of the plants in one position (Table 3). This phenomenon was consistent on all four lines, except for LR1(TC) in the 20 C postinoculation environment. The total amount of spores produced per treatment by the pustules remained constant even though the relative number of infection sites had decreased (Table 2). Latent periods were lengthened with increasing exposure to 5 C (Table 4). Plants held constantly at 20 C for the preinoculation, moist period, and postinoculation periods had aegricorpi that were the first to sporulate, while the plants treated with constant 5 C had aegricorpi that sporulated last. Only LR1(TC) had a latent period longer than in

6

Table 1. Statistical analysis of $ln1/lr1$, $lp16/lr16$, and $lp17/lr17$ interactions involving latent period, relative mean sporulation, and relative number of infection sites

Source	Latent Period				Relative Sporulation				Infection Sites			
	DF	Type III SS	F Value	Pr>F	DF	Type III SS	F Value	Pr>F	DF	Type III SS	F Value	Pr>F
CULT	1	378.58	15.60	0.0001	1	673.48	759.14	0.0001	1	1.06	2.14	0.14
LINE	3	910.82	12.51	0.0001	3	318.05	119.50	0.0001	3	26.73	17.92	0.0001
CULT*LINE	3	799.97	10.99	0.0001	3	314.43	118.14	0.0001	3	40.79	27.36	0.0001
CULT*IR	1	4.79	0.20	0.67	1	0.30	0.34	0.56	1	3.27	6.59	0.01
CULT*IP	1	128.91	5.31	0.02	1	0.29	0.33	0.57	1	37.81	76.08	0.0001
CULT*IT	1	169.89	7.00	0.01	1	444.55	501.10	0.0001	1	14.43	29.03	0.0001
LINE*IR	3	14.05	0.19	0.90	3	0.06	0.02	1.00	3	1.03	0.69	0.57
LINE*IP	3	7.03	0.10	0.96	3	3.07	1.15	0.33	3	0.61	0.41	0.75
LINE*IT	3	356.83	4.90	0.0022	3	304.22	114.31	0.0001	3	25.35	17.00	0.0001
CULT*LINE*IR	3	88.49	1.22	0.30	3	2.05	0.77	0.51	3	2.92	1.96	0.12
CULT*LINE*IP	3	50.35	0.69	0.56	3	1.13	0.43	0.74	3	0.23	0.19	0.91
CULT*LINE*IT	3	683.66	9.39	0.0001	3	292.07	109.74	0.0001	3	21.61	14.49	0.0001
CULT*LINE*IR*IP*IT	32	1006.98	1.30	0.13	32	27.40	0.97	0.52	32	17.40	1.00	0.33
IR	1	757.06	31.24	0.0001	1	7.65	8.62	0.0034	1	39.54	79.55	0.0001
IP	1	966.41	39.83	0.0001	1	23.81	26.84	0.0001	1	92.58	186.23	0.0001
IT	1	81232.67	3471.93	0.0001	1	889.26	1002.48	0.0001	1	1.58	3.18	0.03
REP	3	16.18	0.22	0.88	3	2.19	0.82	0.48	3	1.93	1.33	0.26

Table 2. The effect of preinoculation, moist period, and postinoculation temperatures on mean sporulation[a] on two cultures and four lines

Treatments	Mean sporulation with Culture x Line interaction							
	PRIUS3				PRIUS21			
(PR-MP-PT)[b]	IR1	IR16	IR17	TC	IR1	IR16	IR17	TC
5- 5- 5	7.7	7.5	7.2	7.5	8.0	7.9	8.0	8.0
20- 5- 5	7.7	7.9	7.8	7.6	7.9	7.9	7.9	7.9
5-20- 5	7.6	7.6	7.7	7.9	8.2	8.1	8.1	8.0
20-20- 5	7.9	8.3	7.7	8.0	8.4	8.5	8.3	8.5
20-20-20	0.4	5.5	3.0	7.6	7.9	7.9	7.9	7.9
5-20-20	0.0	5.0	3.6	7.3	7.6	7.4	7.5	7.2
20- 5-20	0.3	4.3	2.5	7.4	7.2	7.0	7.6	7.5
5- 5-20	0.0	5.0	2.1	7.3	7.1	7.0	6.6	7.6

[a]Mean sporulation on a 0-9 scale with 0 = no sporulation and 9 = largest amount of sporulation.

[b]PR = preinoculation temperature in Celsius; MP = moist period temperature; PT = postinoculation temperature.

Table 3. The effect of preinoculation, moist period, and postinoculation temperatures on the relative number of infection sites[a] on two cultures and four lines

Treatments (PR-MP-PT)[b]	Infection sites with Culture x Line interaction							
	PRTUS3				PRTUS21			
	LR1	LR16	LR17	TC	C			
5- 5- 5	7.7	7.5	7.2	7.5	8.0	7.9	8.0	8.0
20- 5- 5	7.7	7.9	7.8	7.6	7.9	7.9	7.9	7.9
5-20- 5	7.6	7.6	7.7	7.9	8.2	8.1	8.1	8.0
20-20- 5	7.9	8.3	7.7	8.0	8.4	8.5	8.3	8.5
20-20-20	0.4	5.5	3.0	7.6	7.9	7.9	7.9	7.9
5-20-20	0.0	5.0	3.6	7.3	7.6	7.4	7.5	7.2
20- 5-20	0.3	4.3	2.5	7.4	7.2	7.0	7.6	7.5
5- 5-20	0.0	5.0	2.1	7.3	7.1	7.0	6.6	7.6

[a]Mean sporulation on a 0-9 scale with 0 = no sporulation and 9 = largest amount of sporulation.

[b]PR = preinoculation temperature in Celsius; MP = moist period temperature; PT = postinoculation temperature.

Table 4. The effect of preinoculation, moist period, and postinoculation temperatures on latent period (in days) on two cultures and four lines

Treatments (PR-MP-PT)[b]	Latent period with Culture x Line interaction							
	PRIUS3				PRIUS21			
	IR1	IR16	IR17	TC	IR1	IR16	IR17	TC
5- 5- 5	34.4	34.0	35.4	36.6	37.6	35.7	33.1	35.1
20- 5- 5	32.8	31.4	32.0	33.1	34.9	34.9	34.4	30.0
5-20- 5	36.6	32.8	35.4	33.0	31.1	31.8	31.4	32.7
20-20- 5	30.6	28.1	28.9	28.3	27.7	27.9	28.2	28.9
20-20-20	17.9[a]	8.6	8.8	8.1	8.0	8.0	8.0	8.1
5-20-20	17.2[a]	9.5	9.7	8.4	8.1	8.1	8.1	8.1
20- 5-20	18.3[a]	9.8	10.3	8.7	8.9	8.8	8.7	9.2
5- 5-20	19.3[a]	9.3	11.5	9.0	10.2	12.0	10.0	12.3

[a]When no sporulation was observed a value of 21 (length of observation) was given for the latent period.

[b]Preinoculation temperature in Celsius; MP = moist period temperature; PT = postinoculation temperature.

the 5-5-5, and that occurred within the 5-20-5 treatment. Corresponding gene pairs $Hp1/Lr1$ and $Hp17/Lp17$ performed in a manner identical to that of $Hp1/Hr1$, $Hp17/Hr17$, and $Hp16/Hr16$ in all treatments; gene pair $Hp16/Lp16$ did not perform the same as $Hp16/Hr16$ (Table 4). At a constant temperature of 20 C for all three periods, PRIUS21: LR16(TC) were observed with similar sporulation amounts, latent period, and relative number of infection sites as compared to the other associations, but a chlorotic ring surrounded all of the pustules. This chlorotic ring was associated only with the constant 20 C treatment and did not seem to alter any of the parameters being measured, yet it was clearly detectable.

Variations in sporulation were clearly visible with the associations of all three isolines and PRIUS3. Browder and Eversmeyer (1986) found that temperatures at which the three near-isogenic lines and the parasite were placed regulated aegricorpus phenotype. The PRIUS3: LR1(TC) association was interrupted when the environment was maintained at a constant 20 C. By lowering the temperature, the association could be reinitiated. Only when the postinoculaton temperature was at 20 C was the interaction interrupted. Both the preinoculation and moist period temperatures could be at 20 C, but when the postinoculation temperature was 5 C, a high infection type would result. The opposite also held true; i. e., the preinoculation and moist periods could be set at 5 C, but a low infection type would result from a 20 C postinoculation temperature (Table 2). Relative sporulation amounts of the PRIUS3:LR1(TC) association with a 20 C postinoculation treatment were a

.4 or lower on the inoculated leaves. However, a 5 C postinoculation treatment had a relative sporulation rating of between 7.5 and 8 (Table 2). Regardless of the previous temperature treatments, the postinoculation treatment determined the amount of relative sporulation. This holds true for both IR1(TC) and IR17(TC) associating with PRIUS3. PRIUS3:IR16(TC) associations did not result in the same low relative amounts of sporulation as did IR1(TC) or IR17(TC). Instead, a rating of 5 was recorded when the association was interrupted at the 20 C temperature. At a postinoculation temperature of 5 C, the relative amount of sporulation was the same as for Thatcher and PRIUS3 at all temperatures, but the latent period was increased and the relative number of infection sites decreased. PRIUS3:IR17(TC) had an average sporulation rating of around 3 at 20 C (postinoculation). Once again, the parasite:host association was interrupted with a postinoculation temperature of 20 C, but was successful at 5 C. At a postinoculation temperature of 5 C, all three corresponding gene pairs produced similar aegricorpus phenotypes to that of Thatcher with PRIUS3 (Table 2). Utilizing infection types obtained at a 20 C postinoculation temperature for the three isolines with PRIUS3, the extrapolated parasite genotype for PRIUS3 is Ip1Ip16Ip17.

The number of infection sites decreased with increasing lengths of time during which the aegricorpus was being established at the lower temperature. PRIUS3:host associations had more infection sites at the lower temperatures than did the PRIUS21:host associations. Although the frequency was recorded as "low" on the constant 5 C treatments and

12

5 C moist periods, infection sites could be observed on most of the plants inoculated with PRIUS3, but PRIUS21 associations often were observed on only a few leaves of each individual host. Apparently, the relative number of infection sites was determined by the temperature of the moist period. Plants grown in 20 C moist periods produced the highest number of infections while associations in 5 C produced the fewest (Table 3). As with the associations involving PRIUS21, a constant 5 C treatment had the fewest number of infections, but the constant 20 C treatment had the highest number.

Length of the latent period was directly related to the duration of exposure of the associations to 5 C (Table 4). All results were compared to the results observed from the 20-20-20 environment. Preinoculation temperatures of 20 C had the shortest latent periods. Moist periods of 20 C also shortened latent periods, but did not have as much impact as the higher preinoculation temperatures. Treatments of 5 C increased latent periods, with postinoculation having the largest effect, moist period, and preinoculation the least.

The results of this experiment support the concept that aegri-
corpus genotypes are expressed in specific environments and are depen-
dent upon the parasite and host genotypes. They also indicate that the
aegricorpus phenotype observed is dependent upon the postinoculation
temperature and not preinoculation temperature for the Ip1/Lr1, Ip16/
Lr16, and Ip17/Lr17 corresponding gene pairs in the Puccinia recondita:
Triticum aestivum system.

Although this experiment demonstrated that, for these three gene
pairs, postinoculation temperature was the determining factor, in the
Puccinia recondita:Triticum aestivum system, preinoculation temperature
could alter aegricorpus' phenotypes. Sharp has detailed the impact of
preinoculation temperatures on infection types associated with Puccinia
striiformis and Triticum aestivum. In his 1962 paper, he describes how
a previously undescribed race of P. striiformis produced different
infection types with various wheat varieties. When the parasite and
host were allowed to associate in a 15 C preinoculation environment,
some associations, such as with Omar and Idaed, produced a "suscep-
tible" reaction, but a "resistant" reaction resulted when the assoc-
iation occurred in a 24 C environment. However, the opposite reaction
was observed on other varieties. Webster and Holzapfels associations
were labeled as "resistant" in a 15 C preinoculation temperature, but
"susceptible" in a 24 C environment. A third group of varieties
produced no change in infection type, regardless the preinoculation

temperature. Sharp also notes that all of the varieties investigated were "resistant" with a postinoculation temperature of continuous 24 C, but by varying the length of exposure in the postinoculation period to 24 C, the interactions became variable. Mohamed (41) wrote of the sensitivity of the association between Race 139 of _Puccinia graminis_ var. _tritici_ and the wheat variety Lerma 52, when exposed to various preinoculation, moist period, and postinoculation temperatures. Warmer preinoculation temperatures increased the number of infection sites and increased the infection type. The same phenomenon occurred with moist and postinoculation periods.

The results from this experiment support both Mohamed and Sharp's assertions that temperatures influence disease systems. Although preinoculation and moist period temperatures did not alter the final aegricorpus phenotype (relative mean sporulation), they did influence the length of the latent period and the relative number of infection sites. All of the corresponding gene pair and Thatcher associations were effected equally by the lower preinoculation and moist period temperatures. The corresponding gene pairs produced reactions not significantly different from nonfunctional gene pairs (Table 1). From these results it can be concluded that temperature effects not only the aegricorpus, but the host and parasite separately. There are two levels at which temperature influences a disease system. First, temperature effects the host and parasite separately, before they can interact with each other. Tables 3 and 4 demonstrate that a low preinoculation and moist period temperature lengthened latent period and lowered the relative number of infection sites of all parasite:host

15

associations. If only the corresponding gene pairs had been affected, then the PRTUS21:LINE associations would not have demonstrated the same effect. However, all of the PRTUS21:LINE associations did perform the same as the corresponding gene pairs. The second point of influence is on the aegricorpus itself. At a postinoculation temperature of 20 C, the corresponding gene pairs interrupted the association. When the postinoculation temperature was lowered to 5 C the association was not interrupted. Evidence of this point of influence is given by examining the mean sporulation of all eight temperature regimes and noting that the PRTUS3 associations had mean sporulation values that did not vary significantly from PRTUS3:Thatcher and PRTUS21:LINE associations at 5 C PT (Table 1). When the postinoculation temperature was at 20 C, there was a significant difference in the resulting mean sporulation values for Lp1/Lr1, Lp16/Lr16, and Lp17/Lr17 when comparing them to PRTUS3:Thatcher and all PRTUS21 associations (Table 1).

In conclusion, the results from one experiment examining a small subsection of all of the corresponding gene pairs of a parasite:host system should not be used to extrapolate to the remaining corresponding gene pairs within that system or other systems. One conclusion that can be seen is the influence that environment exercises over parasite: host systems. Environment not only affects corresponding gene pairs, but also the parasite, the host, and the other genotypes of the aegricorpus other than corresponding gene pairs. While infection types are determined by gene pairs, the length of latent period and the number of infection sites can be influenced by the environment without the presence of corresponding gene pairs.

16

REFERENCES

1. Bever, W. M.   1934.   Effect of light on the development of the uredial stage of <u>Puccinia glumarum</u>.   Phytopathology 24:507-516.

2. Bromfield, K. R.   1954.   Effect of temperature on the reaction of wheat seedlings to <u>Puccinia graminis tritici</u>.   Phytopathology 44:482 (abs.).

3. Bromfield, K. R.   1961a. Effect of variations in temperature on the reaction of temperature-sensitive wheat varieties to wheat stem rust. Phytopathology 51:794-797.

4. Bromfield, K. R.   1961b.   The Effect of postinoculation temperature on seedling reaction of selected wheat varieties to stem rust. Phytopathology 51:590-593.

5. Browder, L. E.   1971.   A proposed system for coding infection types of the cereal rusts. Plant Dis. Reptr. 55:319-322.

6. Browder, L. E.   1985. Parasite:host:environment specificity in the cereal rusts. Ann. Rev. Phyto. 23:201-222.

7. Browder, L. E., and M. G. Eversmeyer.   1983a. The probable occurrence of two <u>Lr</u> genes in Maris Fundin wheat and their expression at different temperature.   Phytopathology 73:825 (abs.).

8. Browder, L. E., and M. G. Eversmeyer. 1983b. Influence of low temperature on the expression of some <u>Puccinia recondita</u>:<u>Triticum</u> corresponding gene pairs. Phytopathology 73:816 (abs.).

9. Browder, L. E., and M. G. Eversmeyer. 1984. The influence of temperature on "slow-leaf-rusting" in <u>Triticum aestivum</u> 'Suwon 85.' Phytopathology 74:877 (abs.).

10. Browder, L. E., and M. G. Eversmeyer. 1986. Interactions of temperature and time with some <u>Puccinia recondita</u>: <u>Triticum</u> corresponding gene pairs. Phytopathology 76:1286-1288.

11. Browder, L. E., and M. G. Eversmeyer.   1987.   Influence of temperature on development of <u>Puccinia recondita</u> with <u>Triticum aestivum</u> 'Suwon 85'. Phytopathology 77:423-425.

12. Browder, L. E., and H. C. Young, Jr. 1975. Further development of an infection-type coding system for the cereal rusts. Plant Dis. Rep. 59:964-965.

13. Cassell, R. C. 1939. Effect of temperature on infection and development of eight physiologic races of *Puccinia graminis tritici* on wheat seedlings. Phytopathology 29:4 (abs.).

14. Chandrashekar, M., and W. A. Heather. 1981. Temperature sensitivity of reactions of *Populus* spp. to races of *Melampsora larici-populina*. Phytopathology 71:421-424.

15. Chandrashekar, M., and W. A. Heather. 1982. Temperature-light effects on resistance of poplar cultivars to races of *Melampsora larici-populina*. Phytopathology 72:327-330.

16. Emge, R. G., and K. R. Bromfield. 1958. The use of controlled temperature and selected hosts to differentiate between two cultures of wheat stem rust. Pl. Dis. Reptr. 42:360-362.

17. Forsyth, F. R. 1956. Interaction of temperature and light on the seedling reaction of McMurachy wheat to race 15B of stem rust. Can. J. Bot. 34:745-749.

18. Gerechter-Amitai, A. K., E. L. Sharp, and M. Reinhold. 1981. Temperature sensitive genes for stripe rust resistance in *Triticum dicoccoides* indigenous to Israel. Phytopathology 71:218 (abs.).

19. Gerechter-Amitai, A. K., E. L. Sharp, and M. Reinhold. 1984. Temperature-sensitive genes for resistance to *Puccinia striiformis* in *Triticum dicoccoides*. Euphytica 33:665-672.

20. Gousseau, H. E., B. J. Deverall, and R. A. McIntosh. 1985. Temperature-sensitivity of the expression of resistance to *Puccinia graminis* conferred by the *Sr15*, *Sr9b*, and *Sr14* genes in wheat. Physiol. Plant Pathol. 27:335-344.

21. Green, G. J., and T. Johnson. 1955. Specificity in the effect of high temperature on the adult plant reaction of wheat varieties to races of stem rust. Can. J. Bot. 33:197-201.

22. Harrington, J. B. 1931. The effect of temperature on the expression of factors governing rust reaction *Triticum vulgare*. Can. J. Res. 5:200-207.

23. Hart, H., and K. Zaleski. 1935. The effect of light intensity and temperature on infection of Hope wheat by *Puccinia graminis tritici*. Phytopathology 25:1041-1066.

24. Hingorani, M. K. 1951. The effect of temperature and light on the mesothetic reaction of Jostrain oats to stem rust. Indian Phytopathology 4:85-87.

25. Hyde, P. M. 1982. Temperature-sensitive resistance of the wheat cultivar Maris Fundin to _Puccinia recondita_. Pl. Pathol. 31:25-30.

26. Johnson, D. A. 1980. Effect of low temperature on the latent period of slow and fast rusting winter wheat genotypes. Plant Dis. 64:1006-1008.

27. Johnson, L. B., and J. F. Schafer. 1965. Identification of wheat leaf rust resistance combinations by differential temperature effects. Pl. Dis. Reptr. 49:222-224.

28. Johnson, T., and M. Newton. 1940. The influence of light and certain other environmental factors on the mature plant resistance of Hope wheat to stem rust. Can. J. Res. Sect. C. 18:357-371.

29. Johnson, T., and M. Newton. 1941. The effect of high temperature on the stem rust resistance of wheat varieties. Can. J. Res. C. 19:438-445.

30. Johnson, T. 1946. The effect of DDT on the stem rust reaction of Khapli wheat. Can. J. Res. Sect. C. 24:23-25.

31. Knott, D. R. 1981. The effects of genotype and temperature on the resistance to _Puccinia graminis tritici_ controlled by the gene _Sr6_ in _Triticum aestivum_. Can. J. Genet. Cytol. 23:183-190.

32. Line, R. F. 1972. Relationship of stripe rust infection types on wheat seedlings at controlled temperatures to resistance in the field. Phytopathology 62:772.

33. Loegering, W. Q. 1966. The relationship between host and pathogen in stem rust of wheat. Hereditas Suppl. 2:167-177.

34. Luig, N. H., and S. Rajaram. 1972. The effect of temperature and genetic background on host gene expression and interaction to _Puccinia graminis tritici_. Phytopathology 62:1171-1174.

35. Martens, J. W., R. I. H. McKenzie, and G. J. Green. 1967. Thermal stability of stem rust resistance in oat seedlings. Can. J. Bot. 45:451-458.

36. Milus, E. A., and R. F. Line. 1983. Inheritance of adult-plant, temperature-sensitive resistance to stripe rust in Pacific Northwest wheats. Phytopathology 73:817 (abs.).

37. Milus, G., and R. F. Line. 1984. Heritability of durable, adult-plant, temperature-sensitive resistance to _Puccinia striiformis_ in Pacific Northwest wheats. Phytopathology 74:794 (abs.).

38. Milus, E. A., and R. F. Line. 1986a. Number of genes controlling high-temperature, adult-plant resistance to stripe rust in wheat. Phytopathology 76:93-96.

39. Milus, E. A., and R. F. Line. 1986b. Gene action for inheritance of durable, high-temperature, adult-plant resistance to stripe rust in wheat. Phytopathology 76:435-441.

40. Mohamed, H. A. 1954. Temperature requirements for identification and differentiation of races 49 and 139 of _Puccinia graminis tritici_. Phytopathology 44:498 (abs.).

41. Mohamed, H. A. 1960. Predisposition of wheat seedlings to stem rust infection and development. Phytopathology 50:339-340.

42. Qayoum, A., and R. F. Line. 1985. High-temperature, adult-plant resistance to stripe rust of wheat. Phytopathology 75:1121-1125.

43. Roberts, B. J., and M. B. Moore. 1956. The effects of temperature on the resistance to oat stem rust conditioned by the BC genes. Phytopathology 46:584.

44. Saari, E. E., and M. B. Moore. 1962. The effect of temperature on crown rust reactions. Phytopathology 52:749-750.

45. Schein, R. D. 1961a. Temperature conversion of rust response of bean. Phytopathology 51:486-488.

46. Schein, R. D. 1961b. Some effects of temperature during the colonization period of bean rust. Phytopathology 51:674-680.

47. Sharp, E. F. 1962. Effects of preinoculation and postinoculation host temperatures on infection of wheat seedlings by _Puccinia striiformis_. Phytopathology 52:751-752 (abs.).

48. Sharp, E. L. 1965. Prepenetration and postpenetration environment and development of _Puccinia striiformis_ on wheat. Phytopathology 55:198-203.

49. Sharp, E. L. 1968. Interaction of minor host genes and environment in conditioning resistance to stripe rust. Proc. Cereal Rust Conference. Oeiras, Portugal. pp. 158-159.

50. Silverman, W. 1959. The effect of variations in temperature on the necrosis associated with infection type 2 uredia of the wheat stem rust fungus. Phytopathology 49:827-830.

51. Simons, M. D. 1954. The relationship of temperature and stage of growth to the crown rust reaction of certain varieties of oats. Phytopathology 44:221-223.

52. Singh, S. J., and W. A. Heather. 1983. Temperature-light effects
    on resistance of poplar cultivars to _Melampsora medusae_ Thum.
    Euphytica 32:111-120.

53. Syamananda, R., and J. G. Dickson. 1959. The influence of
    temperature and light of rust reaction of inbred lines of corn
    inoculated with specific lines of _Puccinia sorghi_. Phytopathology
    49:-102-106.

54. Waterhouse, W. L. 1929. Australian rust studies. I. Proc.
    Linnean Soc. N. S. W. 54:615-680.

55. Williams, E. Jr., and C. O. Johnston. 1965. Effect of certain
    temperatures on identification of physiologic races of _Puccinia
    recondita_ f. sp. _tritici_. Phytopathology 55:1317-1319.

56. Zimmer, D. E., and J. F. Schafer. 1961. Relation of temperature
    to reaction type of _Puccinia coronata_ on certain oat varieties.
    Phytopathology 51:202-203.

THE EFFECT OF TEMPERATURE DURING THREE DEVELOPMENTAL
STAGES OF THE WHEAT LEAF RUST SYSTEM

by

ANDREW D. NICKELL

B. S., University of Illinois, 1986

AN ABSTRACT OF A MASTER'S THESIS

submitted in partial fulfillment of the

requirements for the degree

MASTER OF SCIENCE

Department of Plant Pathology

Kansas State University
Manhattan, Kansas

1988

ABSTRACT

Experiments were designed to determine the effect of preinoculation temperatures on the $Lp1/Lr1$, $Lp16/Lr16$, and $Lp17/Lr17$ corresponding gene pairs of the wheat leaf rust system. Three near-isogenic lines of _Triticum aestivum_, with the $Lr1$, $Lr16$, or $Lr17$ genes, and their recurrent parent Thatcher, were inoculated with two cultures of _Puccinia recondita_ f. sp. _tritici_. From previous studies, it was determined that, at 20C, one culture (PRTUS3) is virulent on Thatcher and avirulent on the isolines, and that the second rust culture (PRTUS21) is virulent on all four lines. The materials were then exposed to eight different temperature combinations of preinoculation , moist (infection), and postinoculation periods. Two temperatures, 5C and 20C, were used in the various periods. Data obtained from the experiment indicate that, for all three corresponding gene pairs, postinoculation temperature determines the relative amount of spores produced, while the preinoculation temperature has no effect on the final spore numbers. The moist period temperature regulates the number of infection sites but not the infection type. Exposing the various associations to low temperatures during the moist period decreased the number of infection sites on the four lines. Latent period was lengthened by increasing the amount of time the materials were exposed to 5C. Low temperatures during the postinoculation period resulted in the failure of all three corresponding gene pairs to produce their characteristic low infection types when associating with PRTUS3. Temperature did not alter the infection type associated with PRTUS21 and the four lines.