

Comparative Primary Infection Characteristics of *Ustilago striiformis* and *Urocystis agropyri* on Cultivars of *Poa pratensis*

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ABSTRACT

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The ability of *Ustilago striiformis* and *Urocystis agropyri* teliospores to initiate primary infections via coleoptiles and axillary buds on several cultivars of *Poa pratensis* was examined. *Urocystis agropyri* initiated primary infections more aggressively than *U. striiformis*. Seed inoculations with *U. agropyri* reduced seed germination and resulted in greater numbers of coleoptile-infected plants than seed inoculations with *U. striiformis*. Axillary bud infection by *U. agropyri* occurred more rapidly, produced more infected shoots, and

resulted in greater proliferation of leaf-smutted shoots than did axillary bud infection by *U. striiformis*. Evaluations of individual cultivars of *P. pratensis* indicate that many are equal to or exceed Merion in their potential susceptibility to primary infection by *U. striiformis* and *U. agropyri*. Many of the newer cultivars also seem capable of greater proliferation of systemically infected shoots than Merion. Cultivars showed a considerable range of susceptibility to *U. striiformis* and *U. agropyri*.

Additional key words: epiphytology, etiology, leaf smuts, resistance.

Ustilago striiformis (West.) Niessl and *Urocystis agropyri* (Pruess.) Schroet. are responsible for leaf smuts of *Poa pratensis* L. throughout the northern United States. The epiphytology of *U. striiformis* on *P. pratensis* is reasonably well established (2, 9, 12, 16, 17). Soilborne teliospores are responsible for primary infection via coleoptiles and axillary buds on crowns and on nodes of rhizomes. The disease is perpetuated in mature stands of grass by subsequent primary infections of axillary buds and by systemic infection of shoots and rhizomes produced by infected plants (8, 10). Primary infection of *P. pratensis* by *U. agropyri* has not been studied; results of research with wheat, however, have established coleoptile infection as the predominant mode of primary infection (6, 13, 18). Shoot infection of wheat by *U. agropyri* is minor (6, 15, 18); it is probable, however, that shoot infection may be of greater significance in perennial grasses such as *P. pratensis*. Once established in *P. pratensis*, *U. agropyri* is systemic like *U. striiformis* (8).

Although *U. striiformis* and *U. agropyri* seem to possess similar primary infection characteristics, *U. striiformis* is the predominant cause of leaf smut of *P. pratensis*. The pathogens occur together occasionally (14), but *U. agropyri* alone is not nearly so common as *U. striiformis* and generally is classified as a minor pathogen of *P. pratensis* (1, 4). The reason for the predominance of *U. striiformis* over *U. agropyri* is not clear. The research reported herein was initiated to determine comparative rates of primary infection of various cultivars of *P. pratensis* by *U. striiformis* and *U. agropyri*, to evaluate the extent of systemic development of the pathogens in

infected plants of each cultivar, and to evaluate potential resistance of cultivars to both pathogens.

MATERIALS AND METHODS

Rates of primary infection via coleoptiles were evaluated on 20 cultivars of *P. pratensis*. Seeds of each cultivar were soaked 20-30 minutes in a 10% Clorox (5.25% sodium hypochlorite) solution, rinsed in distilled water, and placed in a steamed 2:1 loam-peat (2:1, v/v) soil mixture. Teliospores of *U. striiformis* and *U. agropyri* were collected from 200 g (fresh weight) of leaf tissue of *P. pratensis* 'Merion' plants infected by the respective pathogens. Infected leaves were chopped in a blender in 500 ml of distilled water, and the respective suspensions were passed through a 44- μ m sieve to remove most leaf tissue. Distilled water was added to the teliospore suspensions of each pathogen to increase the volume to 4,000 ml. One-hundred seeds of each cultivar were inoculated in groups of five each in compartmentalized (3.8 \times 6.0 \times 2.5 cm) plastic flats; each compartment received 10 ml of teliospore suspension, and all inoculated seeds were maintained in the greenhouse (18-24 C) and provided an 18-hour daylength with supplementary incandescent lights. All seeds were inoculated at the time of planting and again five days after planting. One-hundred noninoculated seeds of each cultivar were maintained as controls. All seeds were observed for percent germination and for production of leaf sori for 45 days after germination or until lateral shoots were produced by axillary buds (9). Plants that developed sori within this period were classified as having been infected via the coleoptile (9).

Primary infection via axillary buds was evaluated on 21 cultivars of *P. pratensis*. Seventy-five plants (all initiated from seed except cultivar A-20) of each cultivar were grown 90 days in 7.6-cm (3-in) diameter plastic pots (soil mix and greenhouse conditions as above); then 25 plants of each cultivar were inoculated with teliospores of each pathogen by pouring 10 ml of teliospore suspension on the surface of the soil in each pot. Teliospores were extracted as previously described, but the total volume of suspension prepared was increased to 5,500 ml. The inoculation procedure was repeated on the same plants at 30-day intervals for 360 days. Twenty-five noninoculated plants of each cultivar were maintained as controls. All inoculated plants were observed at 90-day intervals after the first inoculation for production of leaf sori in shoots produced from axillary buds (9). Plants were recorded as smutted when the first diseased shoot appeared; the comparative rates of infection by *U. striiformis* and *U. agropyri* and the total number of plants infected by the respective pathogens were recorded. Comparative development (from primary infection and systemic spread of pathogens) and persistence of smutted shoots infected by the respective pathogens on individual cultivars was determined after 360 days by expressing the mean number of smutted shoots on infected plants of each cultivar as a percentage of the total number of shoots produced by the infected plants.

RESULTS

Germination and primary infection via coleoptiles.—Total germination of seed of all cultivars inoculated with *U. striiformis* and *U. agropyri* was reduced 5.8% and 16%, respectively, below that of

controls (Table 1). Seed of some cultivars were not greatly affected by inoculation and germinated within $\pm 5\%$ of their controls. Eleven cultivars inoculated with *U. striiformis* (A-34, Bonnieblue, Delta, Fylking, Kenblue, Nugget, Olympris, Park, Pennstar, Prato, and Windsor) and three cultivars inoculated with *U. agropyri* (A-34, Adelphi, and Cougar) fell into this group (Table 1). Cultivars with germination reduced 6-15% included six inoculated with *U. striiformis* (Adelphi, Arista, Baron, Cougar, Merion, and Sydsport) and six inoculated with *U. agropyri* (Arista, Bonnieblue, Fylking, Nugget, Park, and Windsor). Reductions in seed germination greater than 15% included three (Arboretum, Newport, and Sodco) and 11 (Arboretum, Baron, Delta, Kenblue, Merion, Newport, Olympris, Pennstar, Prato, Sodco, and Sydsport) cultivars inoculated with *U. striiformis* and *U. agropyri*, respectively.

Coleoptile infections resulting in leaf-smutted seedlings occurred in small numbers for both pathogens. Leaf-smutted seedlings of all cultivars from *U. striiformis*- and *U. agropyri*-inoculated seed totaled six and 13 plants, respectively (Table 1). The number of seedlings of a specific cultivar smutted by the respective pathogens ranged from one to three; only Merion and Pennstar produced seedlings infected by both pathogens.

Primary infection via axillary buds.—Total number and rate of primary infection via axillary buds by *U. striiformis* and *U. agropyri* were dissimilar. The number of plants of all cultivars that produced leaf-smutted shoots after 360 days was 18.3% and 44.0%, respectively, of plants inoculated with *U. striiformis* and *U. agropyri* (Table 2). Cultivars A-20, A-34, Cougar, Nugget, and Prato were not infected by *U. striiformis*; only Arista remained free of infection by *U. agropyri*. Severely

TABLE 1. Percentage germination of seed of cultivars of *Poa pratensis* inoculated with teliospores of *Ustilago striiformis* and *Urocystis agropyri* and the number of leaf-smutted seedlings produced via coleoptile infection

Cultivars	Seed germination ^a (%)			Leaf-smutted plants (no.)	
	Control	<i>U. striiformis</i>	<i>U. agropyri</i>	<i>U. striiformis</i>	<i>U. agropyri</i>
A-34	63	65	59	0	1
Adelphi	62	51	61	0	0
Arboretum	56	40	31	0	0
Arista	78	63	70	0	0
Baron	63	57	31	0	1
Bonnieblue	60	55	49	0	0
Cougar	58	51	55	0	0
Delta	66	66	43	0	0
Fylking	58	60	43	1	0
Kenblue	61	61	34	0	0
Merion	62	47	39	2	1
Newport	59	36	42	0	1
Nugget	71	69	57	0	0
Olympris	59	61	43	2	0
Park	53	54	42	0	3
Pennstar	51	52	33	1	1
Prato	71	73	50	0	3
Sodco	72	53	51	0	0
Sydsport	59	48	37	0	0
Windsor	61	65	53	0	2
	Mean percent germination			Total no. smutted plants	
	62.2	56.4	46.2	6	13

^aBased on 100 seeds per treatment; leaf smut evaluations made 45 days after germination. Variability in germination of noninoculated seeds was due to different ages of seed lots.

infected cultivars (12 or more plants) occurred after inoculation by both pathogens, but predominated among cultivars inoculated with *U. agropyri* (Table 2).

Rate of primary infection of all cultivars inoculated with *U. striiformis* was slow and progressive; the number of plants of the 96 plants infected by *U. striiformis* that showed leaf-smutted shoots at 90, 180, 270, and 360 days was 2.1%, 20.8%, 32.4%, and 42.7%, respectively (Table 2). Rate of infection by *U. agropyri* was rapid; of the 231 plants infected by *U. agropyri*, 9.5%, 48.5%, 23.8%, and 18.2%, respectively, were infected at the 90-, 180-, 270-, and 360-day observation periods (Table 2). Comparatively, maximum rate of appearance of plants infected by *U. striiformis* occurred between 270 and 360 days and, for *U. agropyri*, between 90 and 180 days.

Proportion of leaf-smutted shoots on infected plants.—The proliferation of leaf-smutted shoots resulting from axillary bud infection (and subsequently their systemically infected shoot progeny) differed for the respective pathogens. The percentage of leaf-smutted shoots of all shoots of all cultivars was 3.5 times greater among plants infected by *U. agropyri* than among those infected by *U. striiformis* (Table 2). With the exception of Olympris and Sydsport less than 5% of all shoots of all cultivars were infected by *U. striiformis*. Proliferation of *U. agropyri*-smutted shoots was extensive; except for Arista, Olympris, and Sydsport, all cultivars produced more shoots infected with *U. agropyri*

than with *U. striiformis* (Table 2). Many cultivars were heavily smutted by *U. agropyri*, but more than 20% of the shoots produced by Fylking, Kenblue, Merion, Park, and Prato were smutted.

DISCUSSION

Primary infection of coleoptiles and axillary buds of cultivars of *P. pratensis* by teliospores of *U. striiformis* and *U. agropyri* indicate that *U. agropyri* is the more aggressive of the two pathogens. Factors supporting this conclusion include greater reductions in seed germination, greater numbers of coleoptile-infected seedlings, a more rapid rate of axillary bud infection, greater numbers of leaf-smutted shoots from axillary bud infections, and greater proliferation of leaf-smutted shoots on plants inoculated with *U. agropyri* compared with those inoculated with *U. striiformis* (Tables 1, 2). These results establish that *U. agropyri* is the more efficient initiator of primary infections. It seems paradoxical that even though *U. agropyri* initiates primary infections more efficiently than *U. striiformis*, it remains only a minor pathogen of *P. pratensis*. A precise explanation cannot be provided except to suggest that the mortality rate of *U. agropyri*-infected plants is greater than that of plants infected by *U. striiformis*. A greater mortality rate of *U. agropyri*-infected plants would help explain the predominance of *U. striiformis* and the

TABLE 2. Progressive appearance of leaf-smutted plants of various cultivars of *Poa pratensis* infected via axillary crown buds by *Ustilago striiformis* and *Urocystis agropyri* and the percentage of leaf-smutted shoots on each cultivar after 360 days

Cultivars	Progressive appearance of leaf-smutted plants ^a										Percentage of smutted shoots ^b	
	<i>Ustilago striiformis</i> Days					<i>Urocystis agropyri</i> Days					<i>U. stri.</i> (%)	<i>U. agro.</i> (%)
	90 (no.)	180 (no.)	270 (no.)	360 (no.)	Total	90 (no.)	180 (no.)	270 (no.)	360 (no.)	Total		
A-20	0	0	0	0	0	1	3	3	2	9	0	7.9
A-34	0	0	0	0	0	0	1	2	2	5	0	0.5
Adelphi	0	0	1	3	4	0	0	3	0	3	0.2	1.8
Arboretum	0	1	2	1	4	3	3	2	8	16	3.3	17.2
Arista	0	0	3	2	5	0	0	0	0	0	1.5	0
Baron	0	0	2	3	5	0	4	4	9	17	0.9	6.6
Bonnieblue	0	1	2	2	5	1	2	0	0	3	0.1	9.4
Cougar	0	0	0	0	0	0	0	1	0	1	0	0.1
Delta	0	2	0	0	2	0	4	6	1	11	0.1	13.8
Fylking	0	0	6	6	12	1	20	4	0	25	0.8	52.1
Kenblue	0	0	1	0	1	3	8	6	3	20	0.1	32.6
Merion	0	1	1	3	5	8	17	0	0	25	2.6	76.5
Newport	0	2	3	8	13	1	5	1	0	7	4.4	4.7
Nugget	0	0	0	0	0	0	1	0	0	1	0	0.7
Olympris	2	10	4	7	23	0	0	1	0	1	49.4	0.3
Park	0	0	2	0	2	1	3	4	10	18	0.1	22.4
Pennstar	0	0	2	4	6	1	9	7	3	20	0.2	10.1
Prato	0	0	0	0	0	1	17	4	3	25	0	49.5
Sodco	0	0	1	1	2	0	8	3	0	11	0.2	15.8
Sydsport	0	3	1	0	4	0	3	3	0	6	10.6	2.4
Windsor	0	0	2	1	3	1	4	1	1	7	2.6	11.4
Totals and (last two values) mean percent	2	20	33	41	96	22	112	55	42	231	4.8	16.8

^aTwenty-five plants each were inoculated with each pathogen. Plants were recorded as smutted with the appearance of the first diseased shoots.

^bThe percentage represents the mean proportion of leaf-smutted shoots on the plants of each cultivar infected by the respective pathogens.

greater aggressiveness of *U. agropyri* teliospores in initiating primary infection; i.e., a higher mortality rate among host plants would require a higher efficiency at initiating new infections for the survival of the pathogen.

The interpretation of susceptibility or resistance of cultivars of *P. pratensis* to *U. striiformis* and *U. agropyri* must be approached with caution because teliospores of both organisms possess different degrees of dormancy (3, 6, 18), and primary infections are limited to coleoptiles and axillary buds (2, 9, 12, 17). Under these circumstances, coleoptile infection represents the least desirable type of primary infection for evaluation of cultivar resistance because the combination of teliospore dormancy and the short-lived presence of coleoptiles provides few infected plants (Table 1). Cultivar resistance is best evaluated on the basis of axillary bud infection and subsequent systemic proliferation of the pathogen in the shoots (Table 2). This implies long-term evaluation (12 months or longer) and necessitates repeated infestation of soil with teliospores during the test period. This form of inoculation increases the potential for primary infections; the teliospore population is progressively increased, and the plant continually produces new axillary buds on crowns and rhizomes. Proliferation of leaf-smutted shoots provides an estimate of how successfully a given cultivar can support systemic proliferation of the respective pathogens in diseased plants (Table 2).

Several studies (5, 7, 11) have established that Merion is the cultivar of *P. pratensis* most susceptible to *U. striiformis*; as such, it has become the standard against which the susceptibilities of other cultivars are evaluated. In the present study, only five of the 25 Merion plants produced smutted shoots (Table 2), and after 360 days, the smutted shoots of the infected plants represented 2.6% of all shoots produced (Table 2). By comparison, several other cultivars produced four to six *U. striiformis*-infected plants (Table 2), and of these, Arboretum and Sydsport had 3.3% and 10.6% of their shoots smutted (Table 2). Other cultivars; e.g., Adelphi, Arista, Baron, Bonnieblue, and Pennstar, with four to six infected plants had a smaller percentage of smutted shoots than did Merion. These reactions indicate that successful primary infection of a cultivar by *U. striiformis* does not necessarily result in successful systemic proliferations of the pathogen. This is illustrated by Fylking; 12 of 25 plants became smutted (Table 2), but after 360 days, only 0.8% of all shoots on infected plants were smutted (Table 2). Fylking is very susceptible to primary infections, but is incapable of producing infected shoots; this probably indicates that many infected shoots die. Some cultivars are very susceptible to primary infection and also support heavy production of systemically infected shoots. This type of reaction is illustrated best by Olympris and to a lesser extent by Newport (Table 2). Olympris was severely infected by *U. striiformis* and perhaps should replace Merion as a susceptible check cultivar in future tests.

This work represents the first evaluations of *P. pratensis* cultivars to infection by *U. agropyri* and, on the basis of the evaluation characteristics used, shows that Merion is the most susceptible cultivar to this pathogen (Table 2). In general, a greater percentage of smutted shoots occurred on all cultivars infected by *U. agropyri* than on those infected by *U. striiformis* (Table 2). This

reflects, in part, the more rapid rate of primary infection by *U. agropyri* and, subsequently, a longer period for systemic proliferation of the pathogen. In that *U. agropyri* is a relatively minor pathogen, the large percentages of smutted shoots may be artifacts that exist under near-optimal conditions of light, water, and fertility (i.e., the greenhouse conditions under which the study was conducted), or they may be representative of the potential ability of *U. agropyri* to be supported by many of the newer cultivars of *P. pratensis*. There is a considerable range of susceptibility and resistance among cultivars to *U. agropyri*, but most cultivars with 11 or more infected plants also had the greatest percentage of infected shoots (Table 2), suggesting that the cultivars most readily infected by *U. agropyri* also potentially were capable of supporting systemic proliferation of the pathogen in diseased plants. It should be noted, however, that several cultivars (A-34, Arista, Cougar, Nugget, Olympris) showed relatively low levels of primary infection and production of diseased shoots (Table 2). In this group of cultivars, Olympris is of special interest as a test cultivar because it is relatively resistant to *U. agropyri* and highly susceptible to *U. striiformis* (Table 2). Of the cultivars tested in this study, only A-34, Cougar, and Nugget seemed to show resistance to both pathogens on the basis of the combined characteristics of susceptibility to primary infections and proliferation of smutted shoots (Table 2).

LITERATURE CITED

1. ALLISON, J. L., and D. W. CHAMBERLAIN. 1946. Distinguishing characteristics of some forage-grass diseases prevalent in the north central states. U. S. Dep. Agric. Circ. 747. 16 p.
2. CLINTON, G. P. 1900. Leaf smut of timothy, redtop, and bluegrass. Ill. Agric. Exp. Stn. Bull. 57:348-349.
3. FISCHER, G. W., and C. S. HOLTON. 1957. Biology and control of smut fungi. Ronald Press. New York. 622 p.
4. GASKIN, T. A. 1965. Varietal resistance to flag smut in Kentucky bluegrass. Plant Dis. Rep. 49:1017.
5. GASKIN, T. A. 1966. Evidence for physiological races of stripe smut (*Ustilago striiformis*) attacking Kentucky bluegrass. Plant Dis. Rep. 50:430-431.
6. GRIFFITHS, M. A. 1924. Experiments with flag smut of wheat and the causal fungus, *Urocystis tritici* Kcke. J. Agric. Res. 27:425-449.
7. HALISKY, P. M., C. R. FUNK, and S. BACHELDER. 1966. Stripe smut of turf and forage grasses—its prevalence, pathogenicity, and response to management practices. Plant Dis. Rep. 50:294-298.
8. HODGES, C. F. 1970. Comparative morphology and development of *Poa pratensis* infected by *Ustilago striiformis* and *Urocystis agropyri*. Phytopathology 60:1784-1797.
9. HODGES, C. F., and M. P. BRITTON. 1969. Infection of Merion bluegrass, *Poa pratensis*, by stripe smut, *Ustilago striiformis*. Phytopathology 59:301-304.
10. HODGES, C. F., and M. P. BRITTON. 1970. Directional growth and the perennial characteristic of *Ustilago striiformis* in *Poa pratensis*. Phytopathology 60:849-851.
11. KREITLOW, K. W., and F. V. JUSKA. 1959. Susceptibility of Merion and other Kentucky bluegrass varieties to stripe smut (*Ustilago striiformis*). Agron. J. 51:596-597.
12. LEACH, J. G., C. V. LOWTHER, and M. A. RYAN. 1946. Stripe smut (*Ustilago striiformis*) in relation to bluegrass improvement. Phytopathology 36:57-72.
13. MC ALPINE, D. 1910. The smuts of Australia. Australia

- Department of Agriculture. Melbourne. 288 p.
14. NIEHAUS, M. H. 1968. Relative amounts of stripe and flag smut on Merion Kentucky bluegrass. *Plant Dis. Rep.* 52:633-634.
 15. NOBLE, R. J. 1924. Studies on the parasitism of *Urocystis tritici* Koern., the organism causing flag smut of wheat. *J. Agric. Res.* 27:451-490.
 16. PAMMEL, L. H., J. B. WEEMS, and F. L. LAMSON-SCRIBNER. 1900. Fungus diseases of grasses. Page 308 in *Grasses of Iowa*. Iowa Agric. Exp. Stn. Bull. 54:68-344.
 17. THIRUMALACHAR, M. J., and J. G. DICKSON. 1953. Spore germination, cultural characters and cytology of varieties of *Ustilago striiformis* and the reaction of hosts. *Phytopathology* 43:527-535.
 18. VERWOERD, L. 1929. The biology, parasitism and control of *Urocystis tritici* Koern., the causal organism of flag smut in wheat (*Triticum* spp.). [and recording the occurrence of *Urocystis occulta* (Wallr.) Rab., in South Africa as the cause of "stem smut" in rye.] *Union S. Afr. Sci. Bull.* 76. 59 p.