Pathogenicity of Puccinia coronata from Buckthorn and from Oats Adjacent to and Distant from Buckthorn

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ABSTRACT

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The pathogenicity of *Puccinia coronata* isolates from Texas was compared with the pathogenicity of isolates from oats and from buckthorn (*Rhamnus catharticus*) in a nursery in Minnesota in which the fungus had cycled for many years between the buckthorn and the oats planted adjacent to it. Pathogenicity was tested on 24 differential cultivars and lines of oats (*Avena sativa*) known or assumed to have single genes for reaction to this pathogen. In 1975, a collection of 107 uredial isolates from Texas had 14 virulence patterns, and included virulence on 17 of the 24 differentials. Collections of 17 aecial and 51 uredial isolates from the Minnesota nursery had 15 and 39 virulence patterns, respectively, and both sets of material included virulence on 19 differentials. In 1976, a collection of 73 uredial isolates from Texas had 16 virulence patterns and virulence on 16 differentials. Collections of 49 aecial and 138 uredial isolates from the Minnesota nursery had 22 and 73 virulence patterns, respectively, and virulence on 18 and 21 differentials, respectively. In 1975 and 1976, the Texas material averaged 8.9 and 9.0 virulence genes per isolate, respectively. Corresponding values for the Minnesota uredial isolates were 9.2 and 9.4.

Additional key words: pathogenic specialization.

Pathologists generally believe that growth of the crown rust fungus (*Puccinia coronata* Cda. var. *avenae* Fraser and Led.) of oats (*Avena sativa* L.) on its alternate host, buckthorn (*Rhamnus catharticus* L.) is a potential source of new pathogenic variation (9). This belief is logical because hybridization and recombination occur in the pathogen on buckthorn, and the recombination of existing genes for pathogenicity could lead to new patterns of pathogenicity. Experimental work, however, has not clearly substantiated this view and actually has led to some confusion about the role of the alternate host in the origin and maintenance of pathogenic variation in the fungus.

Murphy (9) noted that many rare races of *P. coronata* were collected only from buckthorn and, in Canada, Fleischmann (4) reported that the proportion of isolates representing relatively avirulent races was far greater among aecial than among uredial cultures. Also, the relative number of races on oats ordinarily is greater in a given number of isolates from buckthorn than in the same number of isolates collected from oats (3,10). When large samples are studied, however, aecial and uredial collections do not always differ significantly. Thus, Fleischmann (4) found that new races occurred no more frequently, and variation in pathogenicity of the races represented was no greater among a large sample of aecial isolates than among a large sample of uredial isolates. Over many years, only minor differences in pathogenicity on oats were noted between uredial and aecial isolates. Similarly, Schwartz and Moore (11) found little difference in pathogenicity of *P. coronata* from buckthorn in Minnesota and that from oats in the north central USA. Michel and Simons (8) observed shifts in pathogenicity of *P. coronata* on buckthorn that tended to follow, rather than precede, shifts in pathogenicity of the general crown rust population.

Saari and Moore (10) and Schwartz and Moore (11) described the establishment of a nursery in Minnesota in which oats and buckthorn were juxtaposed and the crown rust fungus was allowed to cycle between them for many years. They suggested that such a system would enable measurement of the pathogenic potential of the fungus and that the potential value of sources of crown rust resistance could be tested more efficiently in such a nursery than by conventional procedures. The objectives of our study were to compare the pathogenicity of *P. coronata* collected: (i) from buckthorn in the Minnesota nursery, (ii) from oats in this nursery at different times during the season, and (iii) in Texas, which is remote from buckthorn areas.

MATERIALS AND METHODS

The 24 cultivars and lines of oats that were used included the 10 standard crown rust differential cultivars (12) and 14 lines with potentially useful crown rust-resistance genes (Table 1). These 24 cultivars and lines hereafter will be referred to as "differentials." Differential Ascençao was introduced from Brazil, and the other 13 nonstandard differentials carry genes from different strains of *Avena sterilis* L. from Mediterranean countries (13). The three differentials designated "Iowa ..." are from the oat multiline development program of the Iowa Agriculture and Home Economics Experiment Station (2), and each carries a single major gene for crown rust reaction. The differentials prefixed "Pc" are part of a series of lines developed in Canada (5) that are isogenic except for different crown rust resistance genes. Differential TAM-0-301 was developed by the Texas Agricultural Experiment Station, and C-234 was developed by a private seed company. The differentials prefixed "H" were developed by the first and third authors.

Puccinia coronata was collected from the Minnesota nursery mentioned above in which oats had been grown adjacent to buckthorn for many years (10,11). Aecium-bearing leaves were collected from buckthorn in the spring of 1975 and 1976, and infected leaves of the universally susceptible oat cultivar Markton were collected near the end of the season in 1975 and on 10 July, 29 July, and 4 September 1976. Most of the Texas isolates were collected from susceptible cultivars near the end of the season in

1975 and 1976 by M. E. McDaniel, College Station, Texas.

A single-pustule isolate from each collection was increased to provide inoculum for six or eight seedling plants of each of the 24 differentials. Plants were inoculated and according to the standard methods of Murphy (9) the isolates were rated as virulent or avirulent.

Terminology used in this paper follows that of Loegering in which "pathogenicity" is a character or trait of the fungus (1,7). In an individual fungus isolate this character may have a phenotype of either "virulence" or "avirulence" in relation to a specific host cultivar. Virulence or avirulence toward individual members of a group of cultivars constitutes a "virulence pattern". Space limitations preclude description of each virulence pattern; instead virulence patterns on the 24 differentials used in this study will be designated by a race number that denotes a virulence pattern on the standard differential cultivars (differentials 1-10 in Table 1) (12), followed by the numbers of lines 11-24 (Table 1) on which the fungus isolate was virulent. For example, the virulence pattern of an isolate virulent on lines 1 through 9 of the 10 standard differential cultivars and on line 13, and avirulent on the other differentials shown in Table 1, would be 264-13.

RESULTS

Comparison of isolates from aecia and uredia in the buckthorn-oat nursery. In 1975, we tested 17 aecial and 51 uredial isolates for pathogenicity (Table 2). Five differentials were resistant to all the aecial collections and the same five also were resistant to all the uredial collections (Table 1). The percentages of isolates virulent on the other differentials were remarkably similar in the two sets of collections, with the exception of virulence on two lines; about 30% of the aecial isolates were virulent on Pc-50 while only about 5% were virulent on Pc-46. The reverse was true for the uredial isolates. With regard to virulence patterns on all 24 differentials, nine of the 15 patterns found in the aecial isolates did not appear among the 39 patterns detected in the uredial isolates. However, there was no indication of a decrease in diversity from the aecial to the uredial generation.

In 1976, 49 aecial and 138 uredial isolates were tested; the uredial isolates were collected on three dates during the summer. The 49 aecial isolates were virulent on 18 differentials and had 22 virulence patterns; eight of these 22 did not appear among the 73 patterns found in the uredial isolates.

One of the aecial virulence patterns (263-14) was found in nine isolates. This pattern also was the most common in the uredial isolates collected on the first date, but it was found only once in each of the uredial collections made on 19 July and on 4 September. Virulence pattern 263-13 was found in eight of the aecial isolates, but in only two of the uredial isolates first collected, and in none of the second or third collections.

Virulence patterns 325-0 and 263-0 were, next to 263-14, the most common in the 10 July uredial material. Virulence pattern 325-0 was not found in the 29 July collection and was identified only once in the 4 September collection. Likewise, virulence pattern 263-0 did not appear in the 29 July material, but was observed in two isolates in the 4 September collection.

Virulence patterns in relation to overwintering. Because the telial material produced in the Minnesota nursery, which was represented by the uredial isolates studied, provided the inoculum for infection of the buckthorn, a comparison of virulence patterns in 1975 uredial isolates and 1976 aecial isolates should indicate whether certain patterns overwinter better than others. The 51 uredial isolates collected in 1975 were extremely diverse in pathogenicity and comprised 39 virulence patterns. The most common of these (264B-0) was found in only three isolates. The 49 aecial isolates collected in 1976 comprised only 22 virulence patterns. Virulence pattern 263-14 was found in nine isolates, and pattern 263-13 in eight isolates. The greater concentration of virulence patterns in the 1976 aecial isolates than in the uredial isolates from the preceding season means that certain virulence patterns may be associated with fitness for winter survival.

No clearly discernible pattern was noted in the differences in virulence patterns between the 1975 uredial and 1976 aecial isolates. Many of the differences were in the reactions of the 10 standard differential cultivars. Some changes were in the direction of greater virulence, and some were toward lesser virulence. Some uredial isolates parasitized the diploid cultivar Saia, which was resistant to all aecial isolates. In contrast, any aecial isolate that parasitized cultivar Landhafer also parasitized Santa Fe, whereas some uredial isolates parasitized Landhafer, but not Santa Fe.

TABLE 1. Pathogenicity on 24 oat differentials of *Puccinia coronata* isolates collected in 1975 and 1976 from three sources

Differential		Percentage of virulent isolates								
		Minn. aecia		Minnesota uredia					Texas uredia	
		1975	1976	1975	1976				1975	1976
No.	Name				10 July	29 July	4 Aug	Mean		
1	Anthony	100	100	100	100	100	100	100	100	100
2	Victoria	43	32	47	50	45	68	54	82	74
3	Appler	100	100	96	98	100	100	99	100	100
4	Bond	100	100	100	100	100	100	100	100	100
5	Landhafer	100	100	100	96	100	100	99	99	99
6	Santa Fe	100	100	96	98	100	100	99	99	99
7	Ukraine	54	26	53	16	36	37	30	67	68
8	Trispernia	64	100	75	88	90	100	93	96	99
9	Bondvic	68	100	80	90	92	100	94	96	99
10	Saia	8	0	9	0	4	0	1	0	1
11	Ascençao	11	2	18	4	10	2	5	0	0
12	Iowa X-421	4	6	4	0	6	0	2	2	4
13	Iowa X-434	16	43	15	22	17	8	16	7	33
14	Iowa X-475	48	52	50	48	50	64	54	14	24
15	H-382	25	24	33	8	40	68	39	2	0
16	H-441	0	4	0	0	0	2	1	0	0
17	H-555	0	0	0	0	0	0	0	0	0
18	Pc-38	0	0	0	0	2	2	1	1	0
19	Pc-39	4	6	4	0	6	0	2	3	3
20	Pc-45	4	8	9	4	10	8	7	14	14
21	Pc-46	4	24	34	26	37	40	34	14	17
22	Pc-50	27	0	6	8	18	12	13	0	0
23	C-234	0	0	0	0	0	0	0	0	0
24	TAM-0-301	0	0	0	0	0	0	0	0	0

Comparison of 1975 and 1976 aecial isolates. We found no virulence pattern common to both the 1975 and 1976 aecial isolates. On the basis of number of virulence patterns in relation to number of collections, the 1975 isolates appeared more variable than those collected in 1976; the 17 isolates collected in 1975 comprised 15 virulence patterns, whereas the 49 isolates collected in 1976 comprised only 22 virulence patterns.

Comparison of uredial isolates from the Minnesota nursery and from Texas. In 1975, the 107 uredial isolates from Texas comprised only 14 virulence patterns. In contrast, the 51 uredial isolates from oats in the Minnesota buckthorn nursery comprised 39 virulence patterns. The findings were similar in 1976; 73 isolates from Texas comprised 16 virulence patterns and 138 isolates from Minnesota comprised 73. Pathogenic variation in terms of the number of virulence patterns per uredial isolate tested, obviously was much greater in the isolates from the Minnesota buckthorn nursery.

Another measure of pathogenic variation, the total number of differentials parasitized, gave a different picture. In 1975, the Texas isolates parasitized 17 differentials and the Minnesota isolates parasitized only 19. The difference was greater in 1976, with 16 and 21 differentials being parasitized by Texas and Minnesota isolates, respectively. The percentages of Minnesota isolates that parasitized the additional lines varied from line to line. Virulence on differential 16 was found in only one isolate, and on differential 18 in only two Minnesota isolates in 1976. Virulence on line 22 was found in 6% of the Minnesota isolates in 1975 and in 13% in 1976. Virulence on line 11 was found in 18% of the Minnesota isolates in 1975 and in 5% in 1976. Certain genes for virulence (for example, those conditioning virulence on Victoria and Ukraine) were more common in the Texas than in the Minnesota isolates. There was, however, no specific virulence in the Texas isolates that was not found in at least some of the Minnesota isolates.

We also compared the average number of virulence genes per isolate in the Minnesota and Texas material. In making this comparison, we assumed that the 24 differentials each had a single gene for resistance and that there were corresponding genes for virulence in the fungus (1,6). Although the numbers of genes for resistance and their relationships have not been determined for some of the differentials, and the one-to-one relationship may not hold strictly for others, this assumption allowed a reasonable approximation for the purposes of our comparison. An unexpected similarity between the two sets of isolates was evident. In 1975, the Texas isolates averaged 8.9 virulence genes, and the Minnesota isolates 9.2. In 1976, the Texas isolates averaged 9.0 virulence genes, and the Minnesota isolates 9.4. Thus, in spite of the obviously greater variation in the Minnesota material, a host plant attacked by *P. coronata* would be subject to infection by isolates representing about the same number of virulence genes (on the average) whether it was growing in Texas or Minnesota.

TABLE 2. Number and pathogenic variation of *Puccinia coronata* isolates collected from three sources in 1975 and 1976

Year	Source	Number of isolates	Pathogenic variation	
			Differentials parasitized (no.)	Virulence patterns (no.)
1975	Minnesota aecia	17	19	15
	Minnesota uredia	51	19	39
	Texas uredia	107	17	14
1976	Minnesota aecia	49	18	22
	Minnesota uredia			
	10 June	50	16	28
	29 July	48	20	36
	4 September	40	18	26
	Total	138	21	73
	Texas uredia	73	16	16

DISCUSSION

The main reason for establishing the Minnesota buckthorn nursery was to maximize variation in *P. coronata*, and provide inoculum most nearly representing the pathogenic potential of the fungus for testing promising sources of crown rust resistance (10,11). The objective was only partly realized. *Puccinia coronata* in the Minnesota nursery did show much greater variation in virulence patterns than *P. coronata* collected in Texas. But, most of the additional variation was derived from the same genes for virulence being distributed into more combinations. Therefore, testing sources of resistance in the Minnesota nursery seems to provide no advantage over exposing them to natural inoculum in Texas. This basic difference in number of virulence patterns does, however, suggest other questions for investigation such as the relative effects of differing *P. coronata* populations on multiline cultivars, or on pure line cultivars known to carry more than one gene for resistance to *P. coronata*.

Aside from purely virulence pattern differences, however, there is good reason to think that *P. coronata* from the Minnesota nursery did contain certain virulence genes not present in the Texas isolates. Quantitatively, there were perhaps 10-20% more virulence genes in the Minnesota nursery isolates than in the Texas isolates (Table 2). Because the number of identifiable virulence genes is limited by the number of host resistance genes used, we may assume that *P. coronata* in the Minnesota nursery contained 10-20% more of the potentially large number of virulence genes that may exist. Under this assumption, the advantage of testing breeding material in the Minnesota nursery becomes more evident.

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