

PHYTOPATHOLOGICAL NOTES

A Third Infection Type on the Flax Rust Differential Variety Cass

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ABSTRACT

A collection of flax rust, *Melampsora lini* (Ehrenb.) Lév., was made in North Dakota in 1968 to which the flax rust differential Cass, monogenic for resistance, had an intermediate reaction (infection type 2). Cass had been either immune (infection type 0) or highly susceptible (infection type 3-4) to all cultures of the flax rust fungus with which it had been tested. The existence of a race inducing a third infection type on a variety monogenic for resistance suggests that genes for pathogenicity, heretofore considered as nonallelic, may occur as multiple alleles. *Phytopathology* 60:1691-1692.

The flax rust differential variety Cass, C.I. No. 1182, *Linum usitatissimum* L., carries the M^3 gene for rust reaction. For 20 years prior to 1968, the M^3 gene conditioned immunity, infection type 0 (1), from all North American races of *Melampsora lini* (Ehrenb.) Lév. When lightly inoculated, the leaves show no evidence of infection. When heavily inoculated (Fig. 1-A), the leaves may be slightly distorted or stunted, but evince no macroscopic chlorosis or necrosis.

Cass has been susceptible to races of South American origin and to approximately one-fourth of the F_2 cultures of crosses of North American with South American races (3). When lightly inoculated with a virulent race, large compound pustules are formed with little or no leaf distortion, chlorosis, or necrosis. When heavily inoculated, numerous vigorous pustules, without chlorosis or necrosis, are produced (Fig. 1-C).

In 1968, a culture of flax rust to which Cass has a moderately resistant reaction (Fig. 1-B), was collected in a North Dakota flax field. On the differentials, it keyed out as race 352, virulent on Dakota, Akmolinsk, Williston Brown, and Bison. It has been designated race 352-A because of the reaction of Cass. Pustules that developed on the youngest leaves, those just unfolding in the terminal bud at the time of inoculation, are vigorous and productive for several days after they rupture the epidermis. Sufficient urediospores are produced on Cass to maintain the culture without difficulty in the greenhouse. The older leaves become increasingly necrotic, with minute uredia (infection type 2) which are surrounded by necrotic tissue. No pustules have been observed on the stem. After a few days, a necrotic area commences to surround the

vigorous pustules on the younger leaves and teliospores begin to form in the uredial pustules. The heavily inoculated leaf portions become necrotic and die prematurely.

The flax rust differentials presumably are monogenic for resistance (2). Most have had but two reaction types to the cultures of *M. lini* with which they have been tested. They have been either immune (no uredia) or highly resistant (minute to small uredia formed in chlorotic to necrotic areas of the leaves) or highly susceptible (no evidence of incompatibility). The genes for resistance occur as dominant alleles and lie in five loci (3). The Cass differential had been backcrossed to Bison 15 times, and probably is near-isogenic to Bison except for the M^3 gene. Bison has been highly susceptible to all North American races of the flax rust fungus.

In *M. lini*, avirulence is dominant and nonallelic (3). On Cass, the A_{M^3} gene has conditioned infection type 0 (immune) in all North American races except 352-A. The relation of the pathogenicity gene in race 352-A that conditions infection type 2 on Cass to gene A_{M^3} has not been determined. When an F_1 urediospore culture of *M. lini*, heterozygous for a number of pathogenicity genes, was subjected to X-rays (LD_{50} and LD_{90}), the A_{M^3} gene showed the highest frequency of mutation to virulence (4). But no intermediate infection types, such as that of race 352-A, were obtained. All cultures mutating to virulence on Cass were highly virulent, as would be expected of a deletion of the dominant allele or a mutation to the recessive.

The new gene that conditions the intermediate reaction on Cass (infection type 2) could be a mutation of A_{M^3} . It would lie in the same locus and be designated A_{M^3r} . Since greater avirulence is epistatic to lesser avirulence in the flax-flax rust interaction, A_{M^3r} probably would be dominant to a_{M^3} and recessive to A_{M^3} . This does not conflict with the gene-for-gene hypothesis (3) that has been generally confirmed in studies involving highly specific resistance to plant diseases. It does assume that some genes for pathogenicity may occur as multiple alleles as do genes for resistance in the host.

The reactions of the differentials that serve to identify the physiologic races of the cereal rusts are much more complicated than those for flax rust. Stakman et al. (6) report that of the 12 wheat stem rust differentials, 10 display all six infection types (0, 1, 2, 3, 4, and X) when inoculated with the various races of *Puccinia graminis* Pers. f. sp. *tritici* Eriks. & E. Henn. Loegering & Powers (5) suggest that this multiplicity of infection types results from the interactions of genes at two or more corresponding loci in host and pathogen. This hypothesis was confirmed in a study by Williams et al. (7). However, if pathogenicity genes occur as multiple alleles, as indicated by the culture of race 352-A which produces a type-2 infection on Cass, the determination of the genetic control of the numerous intermediate infection types in the rust fungi becomes more complicated.

The discovery of a third infection type involving a

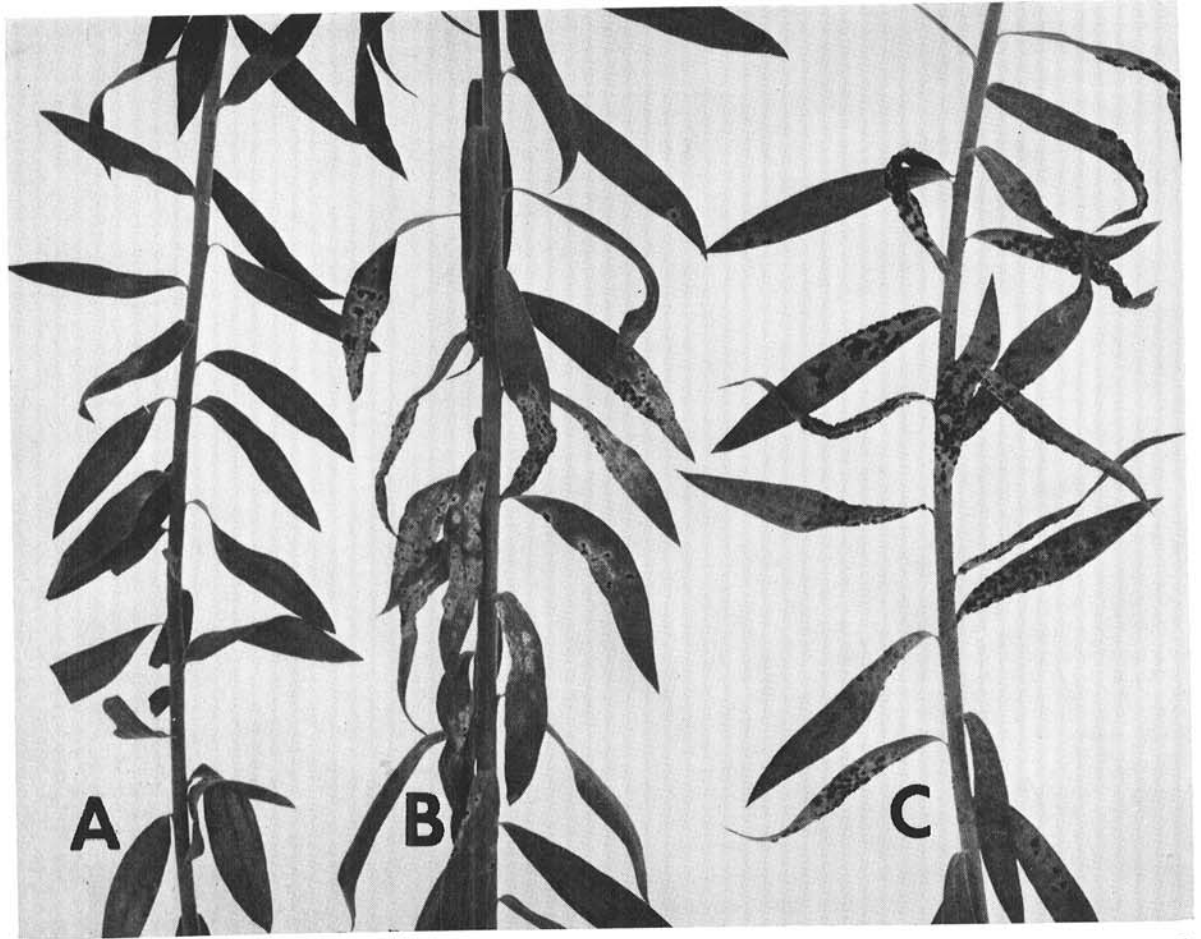


Fig. 1. Five-week-old plants of the flax rust differential variety Cass heavily inoculated with 1-50 urediospore-talc mixtures of **A)** Race 1, infection type 0 (immune). **B)** Race 352-A, infection type 2 (resistant) and **C)** Race 121, infection type 3-4 (susceptible).

single resistance gene may help to explain the complexity of infection types in systems less simple than that of flax and flax rust. Also, studies on the physiology of resistance may be facilitated by the use of races that induce intermediate as well as immune and susceptible reactions on a variety with a single resistance gene.

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