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The gene for gene relationship in flax rust.

In recent years Flor's[1,2,3] researches on flax rust, caused by Melampsora lini, have reached a point where they represent the most fully worked out example of the interaction of genetic systems in host and parasite of any plant disease. This essay will discuss the evidence for the hypothesis that for every gene for resistance to flax rust in the host there is a corresponding gene in the parasite determining whether the outcome of interaction between them will be host susceptibility or host resistance.

This hypothesis is of great importance in developing a concept of the nature of physiologic specialisation in pathogenic fungi. It has been applied with much less supporting evidence than is the case for flax rust, to Phytophthora infestans, Puccinia graminis and Cladosporium fulvum

First of all it is necessary to examine Flor's methods. Manipulation of the host is straightforward. Flax is an inbreeding diploid so that control of host genotype is very simple. The rust however presents certain difficulties. It is not possible when handling crosses to ascertain that each pycnium is the result of infection by a single sporidium and not two of the same mating type. Since the aecia are handled as though the aecial spores all belong to the same dicaryotic clone & avirulent strains (contaminating the clone) would tend to be missed in inoculation studies.

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increase the proportion of
This might ~~lead to an undue preponderance of~~
virulent cultures in F_2 progenies. A similar source of
error ~~of similar effect~~ arises from the spread in aecia
maturation. The aeciospores are shed as soon as the
leaf epidermis covering the pustule ruptures and may
lead to contamination of other aecia.

In connection with this aspect of the work
there is no data concerning the infective abilities
of sporidia. Since Melampsora overwinters by means
of teliospores this would be of some significance for the
establishment of infection on resistant varieties.

The inoculation of differential host varieties
is carried out by ~~means of~~ brushing uredospores
onto the leaves. A single plants can be tested
with many different races in this way. Flor recognises
5 reaction types but for the purpose of race classification
groups these into two classes: resistant and susceptible.
This means that most of Flor's data which may be
examined for evidence of the gene for gene relationship
will only show it at this level. That is the difference
between resistance and susceptibility or virulence and
avirulence will be of significance for determining races
and the difference between immunity and resistance or
between grades of avirulence will not.

While an acceptance of the gene for gene
relationship has become implicit in Flor's latest papers
he nowhere specifically cites the only convincing examples from his data.
In the following table data from two papers are presented.

The interrelation of genes for resistance in the host with genes for pathogenicity in the parasite after Flor [102].

	Ottawa 770B	Bombay	F_1	F_2
Race 22	S	R	R	153 R : 41 S
Race 24	R	S	R	142 R : 52 S
F_1	R	R	—	—
F_2	101 R : 32 S	105 R : 28 S	—	—

S = susceptible R = resistant — = no data.

The F_2 segregation ratios show that interaction of host and pathogen is controlled by a single gene in each and that, in these instances, avirulence is dominant in the pathogen and resistance is dominant in the host.

Flor has published data concerning 21 resistance genes and over 200 races. Are there exceptions to the scheme outlined above? There are two possible exceptions to the gene for gene hypothesis

1. Virulence on a monogenic resistant host requiring the presence of the correct alleles at two or more loci in the pathogen.

or 2. Virulence on a host carrying two or more distinct resistance genes conditioned by a single gene in the pathogen.

In practice both of these examples would be difficult to discriminate from examples of two or more

tightly linked genes apparently segregating as a single gene. In Flor (1946) there are several examples which might be described as exception type 2.

Thus in the F_2 of crosses between race 22 and race 24 and between 6 and 22 reaction on the varieties Kenya and Pale Blue Crimped was inherited as a unit. In these crosses the three parental rust races each showed the same reaction on the two varieties. Thus both were susceptible to 22 but both were resistant to 6 and 24. The evidence for the fact that these two varieties do in fact contain different genes lies in their use to differentiate other races. Flor concludes that the two pathogenicity genes are tightly linked. It is of interest in this connection to note that Flor regards the resistance genes in these two varieties to be either linked or allelomorphic. While in the rust it has not been possible to recombine the corresponding pathogenicity genes in experimental crosses one is forced to conclude an allelomorphic relation between them because of the natural occurrence of recombinant types.

In the same paper is a similar example involving the unit inheritance of reaction on the three monogenic resistant varieties Akmolinsk, Abyssinian and Leona in the F_2 of the cross race 22 x race 24. In the cross of race 6 x race 22 race 6 was found to be heterozygous for virulence on Akmolinsk. However in two cases where the F_1 was heterozygous for ~~resistance or~~ virulence on Akmolinsk the F_2 segregation on the three varieties showed unit inheritance. Again Flor concludes that

three tightly linked pathogenicity genes are involved rather than a single gene on the basis of the differential reactions of Abyssinian and Leona to other races. The three resistance genes are tightly linked or allelomorphic.

While it is possible to presose that such cases of unit inheritance are governed by 'master' genes which combine the properties of other genes it is simpler to regard these examples as cases of tight linkage. It would be of great interest to see to what extent this linkage hampers recombination in the rust and whether it represents a step towards the synthesis of a super gene whereby once the right combination of virulence alleles occurs it is maintained as a unit in subsequent crosses without breaking down. It may be noted that the linkage between pathogenicity genes in the rust extends beyond the linkage groups for resistance in the host.

There ~~would seem~~ is even less evidence which might be interpreted as exception type 1. In every case where more than one pathogenicity gene segregates in relation to a resistant variety such a variety to can be shown to have a corresponding number of resistance genes in crosses with susceptible varieties.

Flor's[3] most recent approach to the problem of physiologic specialisation is through the synthesis of monogenic resistant varieties. His latest publication lists the reactions of flax rust races on 18 lines, 17 of them bearing a single gene for resistance. The

selection of these genes was designed to ~~enable the~~ facilitate differentiation of the most significant North American rust races.

At the present time the evidence for a gene-for-gene relationship in flax rust is limited in extent. It does however form a useful working hypothesis. ~~If exceptions appear from both work this may be~~

References.

1. H.H. Flor (1946) Genetics of pathogenicity in Melampsora lini
J. Ag. Res. 73 : 335-357

2. " (1947) Inheritance of reaction to rust in flax
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3. " (1954) Identification of races of flax rust by lines with single rust conditioning genes.
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