

## Race 2.5, a new race of *Cladosporium fulvum* (*Fulvia fulva*) on tomato

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### Introduction

*Cladosporium fulvum*, the causal organism of leaf mold of tomato, is a versatile parasite. Since the beginning of resistance breeding against this disease, the fungus has regularly overcome resistance. Fortunately many non-exploited resistance genes have always been, and are still, available (Kanwar et al., 1980; Kanwar, 1981). Hubbeling (1978) has published about the most advanced race found in practice up till now, i.e. race 2.3.4.5. Boukema (1981), however, announced a new race which appeared during experimental work. This race, 2.4.5.9, would be detrimental to many of the most advanced resistant tomato lines available these days, but it has never been encountered in commercial tomato growing.

In the meantime Laterrot and Clerjeau (1979) have shown that races of the fungus with virulence genes corresponding to resistance genes not yet in use in France, do occur in that country. Clearly new races are a constant menace to tomato growers and breeders.

### Experimental work

In Bulgaria, Stamova (1982) described the appearance of race 5 on cultivars resistant to race 2.4 such as 'Angela' (resistance gene Cf5). Results of our experiments in 1983, however, aroused the suspicion of the presence of race 2.5, but the cultures got lost. From a sample of 'Angela' with leaf mold which we received from Bulgaria in 1983 at the Research Institute for Plant Protection (IPO), Wageningen, we obtained three isolates. Inoculation of a differential series of tomato plants led to the conclusion that two isolates were race 5, but one was 2.5 (Table 1). Monosporous isolates proved that these isolates were uniformly so. In France, Laterrot and Clerjeau (1979) reported to have found race 5 in 1977 in Bretagne and the Val de Loire on the susceptible cultivar Rustrel. In March 1984 we proved an isolate from the cultivar Prisca (resistance gene Cf5), originating from the same region as the race 5 isolated in Bretagne in 1977, to be race 2.5. In test inoculations we found that this isolate did not attack germplasm with genes Cf4 or one of the new genes Cf6 or Cf9, which are now most commonly used by breeders (Table 1).

Table 1. Reactions of some races of *Cladosporium fulvum* on differential cultivars/hybrids.

Cultivars/hybrids	Genes resistance	Races					
		2 <sup>1</sup>	4 <sup>1</sup>	5 <sup>1</sup>	2.4 <sup>1</sup>	2.5 <sup>2</sup>	2.4.5 <sup>1</sup>
Moneymaker	cf	S	S	S	S	S	S
Vetomold	Cf 2	S	R	R	S	S	S
Purdue 135	Cf 4	R	S	R	S	R	S
Vagabond	Cf 2, Cf 4	R	R	R	S	R	S
PI 187002-1	Cf5	R	R	S	R	S	S
F1 Vetomold x PI 187002-1	Cf 2, Cf 5	R	R	R	R	S	S
F1 Vagabond x PI 187002-1	Cf 2, Cf 4, Cf 5	R	R	R	R	R	S
F 77-38 (= ONT 7818)	Cf 6	R	R	R	R	R	R
ONT 7719	Cf 9	R	R	R	R	R	R

<sup>1</sup> Standard races included in the experiments for comparison.

<sup>2</sup> Virulence as found for isolates from 'Angela' (Bulgaria) and 'Prisca' (France).

## Discussion

Durability of resistance ranks high among the aims of plant breeders. Van der Plank (1968) postulated stabilising selection, which would result in disappearance of unnecessary genes for virulence in the pathogen population. The detection of genes for virulence, matching resistance genes not yet introduced, as is the case for race 5 in France in 1977, is the most solid proof of absence of stabilising selection in this particular plant-pathogen interaction. Parlevliet (1981) analysed several comparable situations, most of them for cereal rusts. He also mentioned *Bremia lactucae*, which in England generally has many more virulence genes than needed. Trimboli and Crute (1983) have shown the high frequency of virulence genes 5 and 10 of *B. lactucae* in Australia, whereas cultivars with resistance genes 5 and 10 are uncommon. Notteghem et al. (1980) cite Bidaux who had shown the presence of virulence genes in the population of *Pyricularia oryzae* in Africa before the introduction of rice cultivars with the corresponding resistance genes.

It is remarkable that both in France and Bulgaria race 2.5 has been found on varieties where race 5 would also have been able to attack. In both countries race 2 has never been found. Race 2.4 is common, but race 4.5 has not (yet) been isolated. The occurrence of some specific combinations of virulence genes must have a relation to fitness, whatever the exact mechanism. Van der Plank (1982) has discussed reasonable explanations for the occurrence of some and the absence of other combinations of virulence genes.

For breeding purposes a large inventory of existing races might help in selecting those genes for resistance, against which no matching genes pre-exist in the wild pathogen population (Laterrot, 1981). This increases the chances of stabilising selection to operate in respect to these genes. Another approach to increase the durability of resistance is the incorporation of two new genes at a time, as advocated by E.A. Kerr (unpublished communication at a meeting on *Cladosporium fulvum*, Wageningen, Febr. 1980), Laterrot (1981) and Hubbeling (1977). This method has credit, especially

with mutable fungi like *C. fulvum*, notwithstanding the enormous extra effort for the breeder.

### Acknowledgement

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### Samenvatting

#### *Fysio 2.5, een nieuw fysio van Cladosporium fulvum op tomaat*

Van *Cladosporium fulvum*, de veroorzaker van bladvlekkenziekte van tomaat, zijn talrijke fysio's bekend. Van tomaterrassen met resistentiegen Cf5 werd, zowel bij materiaal uit Bulgarije als uit Frankrijk, een nieuw fysio, 2.5, geïsoleerd. Dit bezit naast het noodzakelijke virulentiegen 5 ook het overbodige gen 2. Dit is wéér een voorbeeld van afwezigheid van 'stabilising selection'.

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