

# RESISTANCE TO WILT IN CHICKPEA. I. INHERITANCE OF LATE-WILTING IN RESPONSE TO RACE 1<sup>1</sup>

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Received 27 July 1982

## INDEX WORDS

*Cicer arietinum* L., early-wilting, *Fusarium oxysporum* f.sp. *ciceris*, late-wilting, resistance.

## SUMMARY

Differences in time of wilting of chickpea (*Cicer arietinum* L.) in response to Race 1 of *Fusarium oxysporum* f.sp. *ciceris*, are confirmed. C-104 wilts later than JG-62 and the difference in time of wilting appears to be inherited as a single gene with early wilting partially dominant to late wilting. Considered in relation to earlier studies, the observations indicate that at least two genes are involved in the inheritance of resistance in chickpea to Race 1 and offer an explanation for previous difficulties in interpreting the inheritance of resistance.

## INTRODUCTION

Previous studies have indicated that in crosses involving the chickpea cultivar C-104 as the susceptible parent, resistance to wilt, caused by *Fusarium oxysporum* f.sp. *ciceris*, is controlled by the segregation of a single gene with susceptibility dominant to resistance (KUMAR & HAWARE, 1982). However, with the cultivar JG-62 as the susceptible parent, the numbers of susceptible segregants were too large to give good fits to the expected ratios.

Chickpea genotypes have been demonstrated to differ in times of wilting (HAWARE & NENE, 1980). Such differences may be indicative of different degrees of resistance and be reflected in differential behaviour in crosses. The present study was undertaken to determine whether JG-62 and C-104 differed in their reaction to Race 1 of *Fusarium oxysporum* and to investigate the inheritance mechanisms involved.

## MATERIALS AND METHODS

Two separate tests were conducted. In the first, disease reactions were assessed in 15 cm plastic pots in a glasshouse. The genotypes included: the cultivars JG-62 and C-104, both regarded to be susceptible to Race 1 (HAWARE & NENE, 1982); H-208 and K-850, which are classified as susceptible but wilt later than other cultivars; and F<sub>1</sub> and F<sub>2</sub> generations of the cross of JG-62 and C-104. For the four cultivars, seven seeds were sown in each of ten pots and thinned to leave five plants per pot. Five

<sup>1</sup> Approved ICRISAT journal article No. JA 243.

F<sub>1</sub> seeds were sown in each of two pots, and seven F<sub>2</sub> seeds in each of 60 pots. The pots were completely randomised and rearranged daily to reduce environmental effects. The mean maximum and minimum temperatures during the test were 30° and 17.8°C, respectively.

The second test was conducted in 37 cm plastic pots in a screenhouse. The genotypes included the parents and F<sub>1</sub>, F<sub>2</sub> and F<sub>3</sub> generations of the cross of JG-62 and C-104. Fifteen seeds were sown in each pot of which there were seven (JG-62), twelve (C-104), five (F<sub>1</sub>), 37 (F<sub>2</sub>) and two for each of 40 F<sub>3</sub> progenies.

The potting medium was prepared according to the method of HAWARE & NENE (1982). Inoculum was prepared from a single spore culture, multiplied for 14 days on 100 g of a 9:1 sand:chickpea meal in a 250 ml flask and mixed with autoclaved 1:1 soil (Vertisol): sand at the rate of 100 g inoculum to 2 kg soil and sand. The pots were washed in running water, treated with 5% copper sulphate solution and air dried before filling with the medium.

The number of days from sowing to initial symptoms of wilting was recorded for each plant. The means and variances were calculated for each cultivar and generation and the F<sub>2</sub> and F<sub>3</sub> generations classified as early or late-wilting for chi square tests of goodness of fit to expected ratios.

## RESULTS

In both tests, the mean number of days from sowing to the appearance of initial symptoms was much greater in C-104 (39.8 and 27.7) than in JG-62 (18.6 and 12.8), confirming that the two cultivars differed in time of wilting. In the first test K-850 (38.4 days) wilted around the same time as C-104 and H-208 (28.0) was intermediate.

The times to appearance of initial symptoms were much greater in the first, than in the second test, and may be related to the different conditions of the tests, both temperature (CHAUHAN, 1963) and inoculum level being known to affect the incidence of wilt.

The variances of time to initial symptoms were less in JG-62 (4.0 and 1.9) than in C-104 (22.1 and 47.8), K-850 (26.0) and H-208 (26.0), probably because of more pronounced effects of environment on the time of wilting of later wilting genotypes. Similar observations have been recorded for leaf rust of barley (PARLEVLIET, 1978).

The F<sub>1</sub> means were significantly greater than the means for JG-62 but were at the early wilting end of the range. The F<sub>2</sub> means lay between the F<sub>1</sub> means and mid parent values. The calculated degree of dominance for early wilting was 0.6 in both studies.

The F<sub>2</sub> and F<sub>3</sub> generations did not segregate into completely discreet categories so that accurate classification of early and late wilting plants was not possible. Nevertheless, in the first test (Fig. 1) the distributions of JG-62 and C-104 did not overlap and the point of separation coincided with a discontinuity in the F<sub>2</sub> distribution, around 30 days after sowing. In the second test, a few C-104 plants wilted as early as JG-62 (Fig. 2) but again there was discontinuity in the F<sub>2</sub> distribution (17–19 days) coinciding with the point of overlap. The F<sub>3</sub> progenies could similarly be categorised as early or late wilting or segregating and individuals in the segregating progenies classified as early or late wilting, relative to the times of wilting of JG-62 and C-104 (Fig. 2).

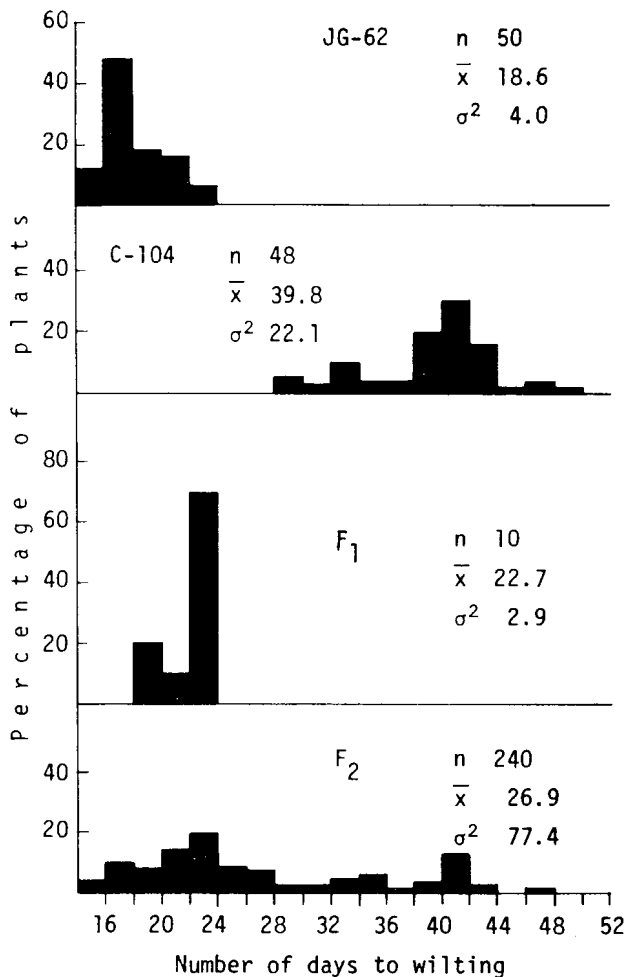


Fig. 1. Percentage frequencies for numbers of days from sowing to appearance of initial symptoms of wilting caused by *Fusarium oxysporum* f.sp. *ciceris* in the chickpea cultivars JG-62 and C-104, and in their F<sub>1</sub> and F<sub>2</sub>, first test.

Although some individuals would have undoubtedly been included in the wrong categories, this would be expected to occur in both directions so that distortion would be slight, and the numbers of early and late wilting plants in the F<sub>2</sub> generations and segregating F<sub>3</sub> progenies fitted extremely well to the 3:1 ratios expected from the segregation of a single gene (Table 1). Similarly, of the F<sub>3</sub> progenies 11 wilted uniformly early, 22 segregated and 7 wilted uniformly late which gave a good fit ( $\chi^2 = 1.2$ ,  $P > 0.50$ ) to the expected 1:2:1 ratio.

#### DISCUSSION

The results presented here confirm the existence of differences among chickpea genotypes in time to appearance of initial symptoms of *Fusarium* wilt. In particular, C-104 wilts much later than JG-62 and the difference appears to be controlled by the segregation of a single gene with early wilting partially dominant to late wilting.

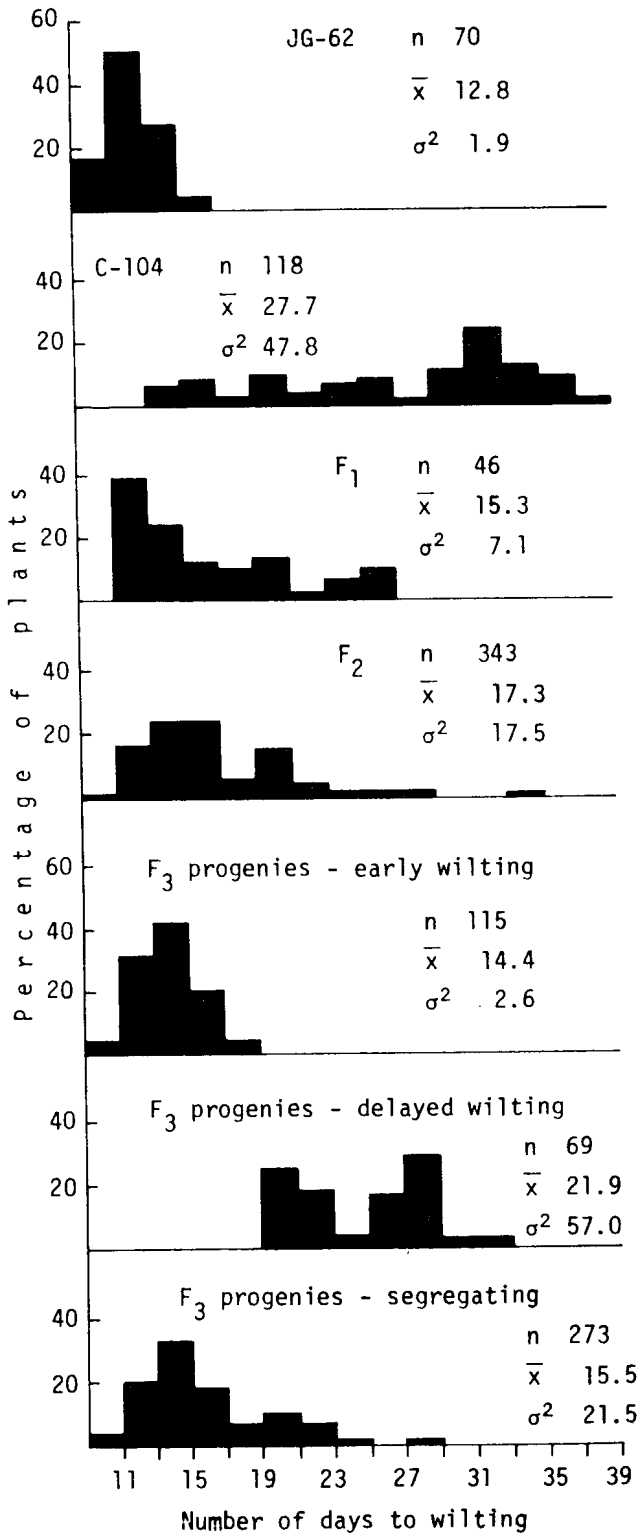


Fig. 2. Percentage frequencies for numbers of days from sowing to appearance of initial symptoms of wilting caused by *Fusarium oxysporum* f.sp. *ciceris* in the chickpea cultivars JG-62 and C-104, and in their F<sub>1</sub>, F<sub>2</sub> and F<sub>3</sub>, second test.

Table 1. The numbers of early and late wilting chickpea plants and chi-square tests and probabilities for goodness of fit to the expected 3: 1 ratios in F<sub>2</sub>s and segregating F<sub>3</sub> progenies of the cross of JG-62 and C-104.

Generation	Time of wilting		$\chi^2$	P
	early	late		
F <sub>2</sub> -first study	175	65	0.55	0.30-0.50
F <sub>2</sub> -second study	246	97	1.97	0.10-0.20
F <sub>3</sub> progenies	203	70	0.06	0.80-0.90
Total	624	232	2.02	0.10-0.20
Heterogeneity			0.56	0.70-0.80

The situation is analagous to slow rusting in cereals (WILCOXSON, 1981) and differences in time of appearance of symptoms of *Verticillium* wilt in cotton (HIJATOVA, 1978), where both monogenic and polygenic inheritance have been reported. Such delay is important in reducing yield loss by delaying disease development and this has been demonstrated for *Fusarium* wilt in chickpea (HAWARE & NENE, 1980).

Furthermore, the mechanism offers an explanation for the earlier observation (KUMAR & HAWARE, 1982) that while resistance to *Fusarium* wilt is inherited as a simple recessive in crosses involving C-104, where JG-62 is used as the susceptible parent there is a marked excess of susceptible segregants.

This situation may result from the segregation of two genes, both of which must be present in homozygous recessive form for complete resistance and separately result in late or reduced wilting. C-104 must be homozygous recessive at one of the loci so that, in crosses with resistant parents, segregation patterns for late wilting and resistance would be expected to be consistent with the segregation of a single gene. In contrast, in crosses of JG-62, which will be expected to carry both genes in homozygous dominant form, and resistant parents, the numbers of early wilting, late wilting and resistant plants should be consistent with digenic segregation.

In the earlier study, the numbers of susceptible plants were insufficient to fit a 15: 1 ratio (KUMAR & HAWARE, personal communication), but the times to wilting were not recorded and the segregation ratios might have been distorted by differences in environmental conditions or by discontinuation of the tests prior to the wilting of all susceptible plants.

Further support for the involvement of more than a single gene is the occurrence of resistant segregants in crosses of susceptible parents (KUMAR, personal communication).

Clearly the inheritance of resistance to *Fusarium* wilt in chickpea is not simple. Evidence has been presented for the existence of physiologic races of the pathogen (HAWARE & NENE, 1982). The studies discussed here indicate that at least two genes control resistance to Race 1. Further evidence is presented in part II of the paper. The elucidation of the inheritance mechanisms is crucial to the conduct of effective breeding programs.

#### ACKNOWLEDGEMENTS

The technical assistance of Mr J. Narayan Rao, in preparation of inoculum, and Mr K. J. Reddy who made the crosses and assisted in tests is gratefully acknowledged.

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