sionally one quadrivalent was observed. Root tip cells showed 40 chromosomes ranging in length from 2.83 μ to 5.33 μ (figure 2). The karyotype consists of six pairs of M-type, 12 pairs of m-type and two pairs of sm-type of chromosomes. The karyotype asymmetry is found to be[6] 2A. The plant showed about 80% pollen fertility and good seed set and it is propagated by seeds as well as by suckers.

The basic number of *Coix* is $x = 5$ and it is interesting to note that most of the previous reports are tetraploids with $2n = 20$ chromosomes[7,8]. The somatic number $2n = 40$ recorded now for the first time in this species indicates that it is an octoploid. Regular meiosis with a high per cent of pollen fertility and seed set shows that it is a stabilized species.

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DISEASE REACTION OF CHICKPEA GRAFT-HYBRIDS TO *FUSARIUM OXYSPORUM* F SP *CICERI* RACE 1

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CHICKPEA wilt caused by *Fusarium oxysporum* f sp *ciceri* is a serious soil-borne fungal disease in many chickpea-producing countries. The pathogen multiplies in the vascular bundles, following successful invasion from the soil and blocks the water conducting tissue. Consequent to the combined effect of water deficit and phytotoxic substances produced by the fungus, a susceptible host cultivar shows typical drooping of shoot tips eventually leading to its death. However, there are genotypes that resist the growth and multiplication of the pathogen. Although the nature of the defense mechanism is not exactly known, genetic studies have revealed that two recessive genes impart resistance in cultivars WR 315 and CPS 1 while a dominant gene delays wilting in H 208[1]. Since, the cost-effective solution of problems caused by soil-borne disease is by breeding resistant cultivars, it is useful to understand the mechanism of resistance in view of its transfer to susceptible but otherwise high yielding cultivars. To that end an experiment was conducted in which resistant and susceptible genotypes were reciprocally grafted.

The grafting was done as follows. One week-old seedlings were cut to a 10 mm wedge 2 cm above the soil surface. Growing tips with a single developing terminal leaf were cut from one week-old plants. The epidermis and phloem region at the end of the scion was scraped with a razor blade to fit the wedge. Stock and scion were secured tightly with a cotton thread and covered with a polythene bag.

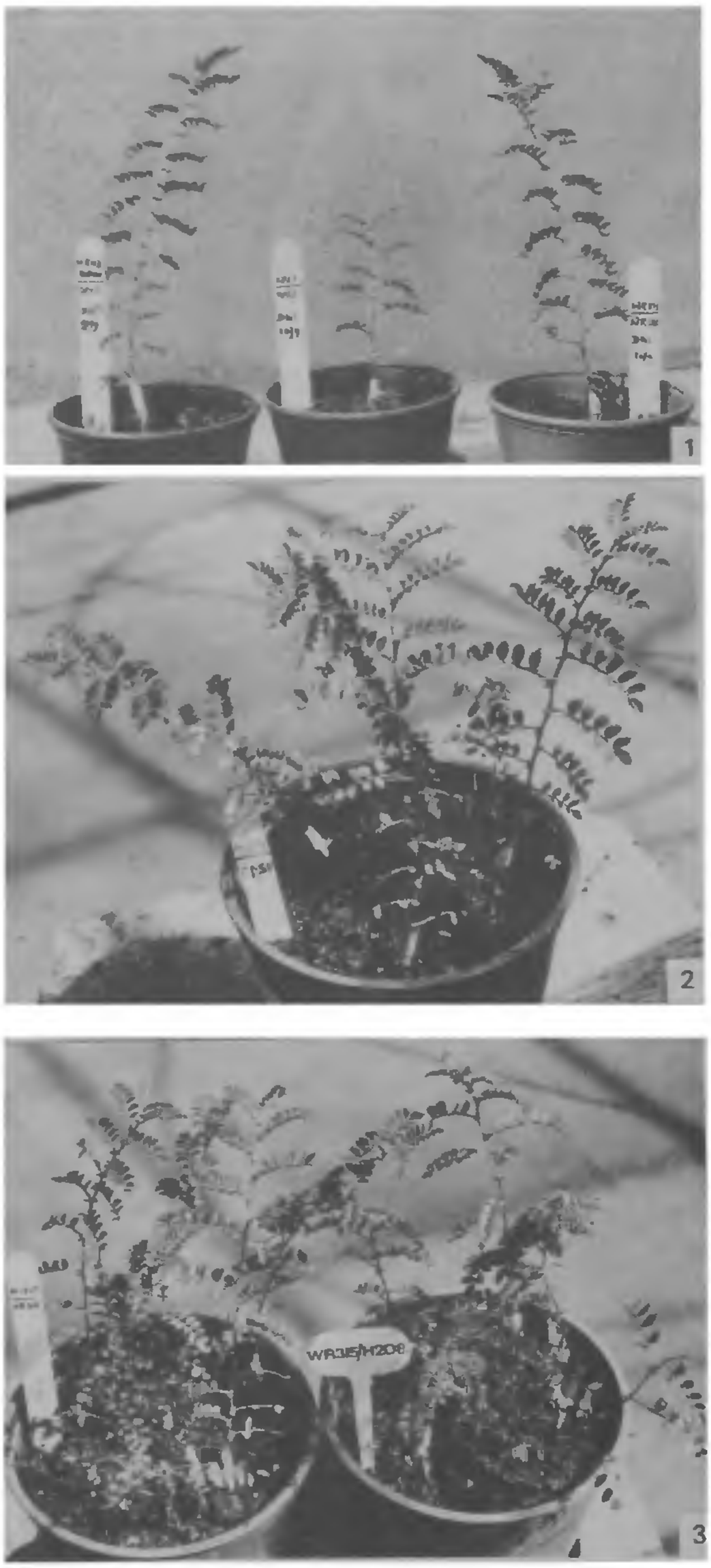
One week after the grafting the graft combinations were carefully transferred to 15 cm plastic pots containing wilt-infested soil. The wilt-infested soil was prepared by adding 15-day-old single spore cultures of *F. oxysporum* f sp *ciceri* race 1 on sand-chickpea meal to a (1 : 1) mixture of black soil and sand at the rate of 100 g of inoculum to 2 kg of soil mixture. Ten grafts were tested for each of the following graft combinations for their disease reaction

Root Stock	Scion
WR 315	H 208
CPS 1	H 208
H 208	WR 315
H 208	CPS 1

WR 315 and CPS 1 = wilt resistant
H 208 = wilt susceptible

All the attempted grafts were successful without any visible adverse effect on the development of root stock or the scion (figure 1). Therefore, this simple technique of grafting was adopted for studying the disease reaction of reciprocal grafts to

Fusarium wilt. The susceptible cultivar H 208 normally shows wilt symptoms 28–34 days after sowing in wilt-infested soil. However, in the case of the scion/root stock combination H 208/WR 315 and H 208/CPS 1, the H 208 scion did not show the disease symptoms even after 60 days (figures 2 and 3). Similar observations on grafts involving resistant root stock and susceptible scion were reported for tomato[2]. Interestingly, in the reciprocal graft WR 315/H 208, wilt symptoms were not seen until pod setting. The symptoms however were restricted to the lower leaves which turned greyish and later dried. Seeds were harvested from all the graft combinations. The successful establishment of resistant WR 315 on susceptible H 208 (figure 3) indicates that the effect of the pathogen is reduced by the resistant genotype. The occurrence of fungitoxic susbstances at levels that suppressed the *F. oxysporum* has been reported in tomato[3]. Our preliminary observations show that similar antifungal substances might impart resistance in chickpea-resistant cultivars to *F. oxysporum* f sp *ciceri* race 1.



Figures 1–3. **1.** Chickpea grafts in disease-free soil. **2.** Wilt susceptible H 208 on resistant CPS 1 in wilt-infested soil along with susceptible check JG 62 (wilting). **3.** Reciprocal grafts in wilt-infested soil along with susceptible check JG 62 (wilting).

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HOST RANGE OF BLACKEYE COWPEA MOSAIC VIRUS

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BLACKEYE cowpea mosaic virus (BlCMV) infecting cowpea (*vigna unguiculata*) in nature was reported in India[1]. In host range studies 61 plant species belonging to 16 families, were inoculated with the virus by leaf rub method. Of these the following 11 plant species belonging to leguminosae showed systemic mosaic symptoms, *Cajanus cajan, Crotalaria juncea, Cyamopsis tetragonoloba, Phaseolus vulgaris, P. lunatus, Vigna mungo, V. radiata, V. umbellata, V. unguiculata, V. sesquipedalis* and *V. catjang*. Hypersensitive reaction was observed in two species of leguminosae viz *Dolichos biflorus,*