of its S-methyl isomer [Syn. isomalathion; O, S-dimethyl-S-(1,2-dicarboethoxy) ethyl phosphorodithioate] existing in various malathion products[1–4]. This subject has created widespread interest[3–6] and has been recently reviewed[7]. Subsequently, attention was only focussed on the solid malathion formulations. However, information on the isomalathion content of liquid preparations is lacking. It was therefore considered worthwhile to ascertain the effect of the common pesticide formulation solvents on the isomalathion content of the liquid products.

Technical malathion (96.68%) and its 50% W/W solutions in purified commercial grade aromex (a mixture of aliphatic and aromatic hydrocarbons), cyclohexanone and xylene [purified by eluting on silicagel column (30 g, MeOH washed and activated at 80–90°C, 2 hr) with cyclohexane containing 0.75% isopropanol; 50 ml in the case of aromex and 150 ml in the case of cyclohexanone and xylene; yield 92–97% of purer solvents] were incubated in stoppered glass bottles at 55 ± 1°C for 12 days. Samples (1 g malathion equiv.) taken at the start and after 1,3,6,9 and 12 days of incubation, were eluted through 5 g silica gel glass columns (i.d. 12 mm) with 70 ml cyclohexane containing 0.75% isopropanol and the eluate rejected. Subsequently, each column was washed with 30 ml isopropanol, the eluate collected, a pinch of anhydrous Na_2SO_4 added, filtered through Whatman No. 42 filter paper, isopropanol removed under reduced pressure at 50–55°C, the residue taken in isooctane containing 10% isopropanol (isomalathion recovery around 90%) and analyzed by HPLC [Spectra Physics 8000B, Lichosorb Si-100 (10 μm) stainless steel column (25 cm × 2.5 mm i.d.), loop injector and UV detector set at 225 nm] employing isooctane containing 5% isopropanol as mobile phase at 1.5 ml/min and sensitivity 0.16 aufs. The analysis parameters were adjusted based on various runs with reference to malathion and isomalathion and the isomalathion content of the samples was calculated employing a microprocessor-controlled data system.

The results revealed a maximum isomalathion content of 0.035% in different solutions. It was not only far below the permitted 1.8% level[4,5] but also for most part of the study, was below the level found in technical malathion (0.023–0.032%). The lower values of isomalathion obtained in this investigation suggest that it undergoes transformation to other products, as reported in earlier studies[6]. With the solid malathion formulations it has been reported that the employed carrier and the storage temperature and period etc influenced their isomalathion content[6]. These parameters played a subdued role in transforming malathion into isomalathion in solutions. Apparently, the liquid formulations offer a safer alternative to the solid malathion products.

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FREQUENCY AND EFFECTIVENESS OF *Lr13* IN CONFERRING WHEAT LEAF RUST RESISTANCE IN INDIA

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MANY leaf rust resistant wheats in Australia, Canada and Mexico are reported to carry *Lr13*, the gene which confers adult plant resistance to many leaf rust races[1,2]. In India, some *Lr13*-bearing wheats have been resistant to leaf rust since the last 15 years[3]. However, some wheats like WL711 and Inia-66 are highly susceptible while Manitou, HD2009, Swati, VL421 and HD2329 have shown

moderate to good resistance though all of these carry *Lr13*. A dominant complimentary necrosis gene *Ne2* and *Lr13* are tightly linked[4]. Consequently, crossing *Ne2–Lr13* carrying wheats to *Ne1* carrier wheats like Spica results in progressive hybrid necrosis, which can be used as a test for *Lr13*. Using this technique, we report here the presence of *Lr13* in 66 out of the 74 tested Indian and exotic wheats and its usefulness in conferring resistance to a highly virulent leaf rust race 77A.

Seventy-four randomly selected wheats were crossed to the cultivar Spica during 1984–85 to obtain at least 20 F_1 seeds. These F_1's along with the parents were sown in 3 m long rows on 2 December 1985. The presence or absence of necrosis in the F_1 seedlings/plants was critically observed at an interval of 3, 6 and 9 weeks after sowing. The parents were field-tested against the most frequent and virulent Indian leaf rust race 77A to test the leaf rust reaction of *Lr13*-bearing wheats. Four rows of susceptible cultivar Agra Local were sown around the experimental plot and two rows of Agra Local were also planted after every twenty experimental rows. All the experimental rows and the Agra Local were repeatedly inoculated with race 77A of leaf rust in soap water suspension. Rust reactions were recorded on 31 March 1986, using the standard method of scoring[5].

The observations on rust reactions of parents and the progressive hybrid necrosis in their F_1's with Spica are summarized in table 1. The F_1's from all crosses except those with eight wheats in group VII and VIII showed progressive hybrid necrosis indicating presence of genes *Ne2–Lr13*. Out of those wheats having *Ne2–Lr13*, thirteen wheats (group I) showed moderate to high susceptibility and rust severity on them varied between 40S and 80S and 29 wheats (groups II, III) showed rust reactions varying from ts to 20S. Twenty-four wheats (groups IV, V, VI) with *Ne2–Lr13*, showed rust reaction with infection types (IT's) varying from 'tr' to '20MR'. Of the eighteen wheats which did not show necrosis with Spica and do not have *Ne2–Lr13*, two wheats (group VII) had rust reaction up to 10S, six wheats (group VIII) showed rust severity varying between tr and 20MR, whereas Agra Local, the susceptible check had 80–100% of its leaf area covered with leaf rust.

Thirteen of the 66 wheats which have *Lr13* (group I) were susceptible to leaf rust. Earlier reports also state that wheats with *Lr13* alone are not resistant to leaf rust in India[6]. Since *Lr13* does not confer seedling or adult plant resistance against race 77A of leaf rust[7], the resistant IT's (tr and MR) seen on 24 *Lr13*-bearing resistant wheats (groups IV, V, VI) must be due to some additional resistance gene(s) effective against race 77A. The 29 wheats with *Ne2–Lr13* which have susceptible IT's on adult plants (groups II, III) do not appear to carry any effective resistance gene against race 77A though their disease severities were very low in comparison to Agra Local. This may be due to the interaction of

Table 1 *Rust reaction of 74 wheat cultivars to race 77A and progressive hybrid necrosis in their F_1's with Spica*

Group	Stock/Cultivar	Field reaction	Hybrid necrosis
I	CPAN 2029, NO166, HP1102, HP1469, HP1479, HP1488, VL421, BR322, K7410, K8001, K8020, K8027, Lok-1	40S–80S	P
II	CPAN 1798, VL609, CPAN 1884, CPAN 2025, CPAN 2034, HI1011, HD2327, HD2329, Swati, H991, HP1487, HB501, BR298, BR319, K8021, K8028, NI8280, NI8305, NI8306 BW35, BW36, HD2009, Manitou	10S–20S	P
III	Raj 1972, HD2190, CPAN 2024, Janak, NI8188, CPAN 1285,	ts–5S	P
IV	CPAN 1828, Nainari 60, CPAN 1883	20MR	P
V	NP 846, Carazinho, WG138, Frontana(I), Teznos Precos Pintos Frontana(C), CPAN 1909, CPAN 1910, CPAN 1905	5MR–10MR	P
VI	CPAN 2026, CPAN 1885, CPAN 1993, CPAN 1869, CPAN 1959 HD2315, CPAN 1886, HW517, CPAN 1995, HW921, HB618, CPAN 1955	Free/tr	P
VII	CPAN 2018, MP504	10S	A
VIII	HW840, CPAN 1956, CPAN 1951, HW888, HW922, VW110	tr-20MR	A

P, present, A, absent.

Lr13 with other unknown gene(s) present in these wheats. Such interaction between *Lr13* and other major genes like *Lr16* and *Lr30* to give enhanced resistance to Canadian leaf rust races virulent on *Lr13* as well as these two genes has been reported[8].

Since all the resistant wheats tested here which have *Ne2–Lr13* are susceptible to race 77A at seedling stage[3], some additional and as yet unknown adult plant resistance genes must be present in these wheats which are currently being identified.

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INDUCED MALE STERILE MUTANT IN GREEN GRAM (*VIGNA RADIATA* (L) WILCZEK)

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GREEN gram is strictly a self-pollinated crop. The male sterile lines have proved quite worthy and as a quick breeding tool for incorporating superior traits in desirable genotypes. Reports in the past have indicated that gamma-ray treatments have effectively induced sterile mutants in green gram[1] and allied species[2]. The present report deals with a male sterile mutant that may eliminate tedious work during emasculation while making crosses for its improvement.

The cultivar T_{44} (origin number, 44-4-1, a selection from cross between, $T_1 \times T_{49}$ evolved at Kanpur) of green gram was taken as the experimental material in the present investigation. Two hundred dry (moisture, 9%), healthy and uniform size seeds of the cultivar were exposed to gamma rays (^{60}Co source) at 5, 10, 20, 30, and 40 kR doses at National Botanical Research Institute, Lucknow. The R_2 lines were raised from R_1 individuals following plant to progeny method. Out of a number of mutants in R_2 generation, a male sterile mutant (tested in 2% acetocarmine solution) was scored from 40 kR gamma-treated population. The segregation behaviour of the mutant was calculated by using χ^2 method. The data for the mean performance of days to flowering, plant height, number of branches per plant, number of racemes per plant, pollen sterility, number of pods per plant and grain yield per plant of the parent and the mutant obtained from 10 random selected plants from each of the 4 replications at R_3 generation were compared by student *t* test.

The male sterile mutant could easily be isolated from the mutagenic population by characterizing as narrow and compact projection of its leaves, branches and peduncles in contrast to semispreading nature of the parent. The days to flowering (38.25 ± 1.26) and the number of racemes per plant (13.44 ± 1.06) of the mutant differed significantly from the parent which had 35.00 ± 1.18 days to flowering and 10.50 ± 0.85 being the number of racemes per plant. Increased bearing of buds or flowers of the mutant can rapidly be utilized in hybridization. The segregation behaviour of the mutant in R_2 generation (104 normal, 32 mutant; $\chi^2 \pm 0.157$) clearly reveals that the mutant genotype is controlled by a single recessive gene. But it also exhibited other morphological variations. It can be inferred that either this gene has a pleiotropic effect or that more than one closely linked genes is involved causing such variations[1-3].

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