was 7,100 g soil. At the same time seeds were also sown in an upland root-knot nematode infested nursery. The seedlings (30 days old) from this nursery were transplanted into the above semi-deep water field. The seedlings at planting had 6 adults with eggmasses, 15 adults and 19 other juvenile stages.

Observations at the maximum tillering stage revealed 12 eggmasses, 7 adults and 13 juveniles in direct sown rice and 24 eggmasses, 10 adults and 133 juveniles in transplanted rice plots. The low multiplication observed in direct seeded crop could be due to the low initial nematode population.

These results indicate that the root-knot nematode, *M. graminicola* can thrive and infect the rice crop even under semi-deep water conditions. Further, results also suggest that the water logging the infested field may not necessarily control the nematode.

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EFFECT OF LEAF CURL DISEASE ON SEED AND OIL QUALITY OF SESAME (*SESAMUM INDICUM* L)

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LEAF curl incited by tobacco leaf curl virus is a serious disease of sesame (*Sesamum indicum* L.)[1,2]. Since no information was available on the effect of this disease on host metabolism, preliminary investigations were conducted to see the quantitative and qualitative changes in the sesame seed and oil due to the virus infection.

When the crop matured, capsules were collected from healthy and severely infected plants of local sesame paired in terms of age, height, stand density, slope and soil type. Four samples with five pairs in each sample were collected. The seeds of five healthy and five diseased plants of each sample were mixed separately, weighed and the mean yield/plant was computed. Seeds of each sample were analysed for oil[3] and protein[4] contents. The oil obtained from the seeds of each sample was further analysed for fatty acids (oleic, palmitic and lauric acids) and for saponification and iodine values[4].

It is evident from table 1 that plant yield and oil content of seeds were greatly reduced due to the disease, however, protein content of seeds was increased. Analysis of the oil revealed that both saponification and iodine values were decreased and quantities of all the three fatty acids (oleic, palmitic and lauric acids) were increased as a result of infection.

Table 1 *Effect of leaf curl disease on quantity and quality of sesame seed and oil*

Source	Seed yield/ plant (g)	Seed analysis		Oil analysis				
		Protein (%)	Oil (%)	Oleic acid	Palmitic acid	Lauric acid	Saponification value	Iodine value
				(g/1000 g oil)				
Healthy plant	8.28	21.9	49.1	1.6	1.4	2.0	188.5	110
Leaf curl infected plant	3.64	26.0	46.3	2.4	2.3	2.2	133.2	105

Data represent mean of 4 values

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ROLE OF L-CYSTEINE ON AZIDE MUTAGENECITY IN AZOTOBACTER

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AZOTOBACTER is a free living nitrogen-fixing bacterium. Successful induced mutagenesis is not of common occurrence in certain strains of this organism[1]. Sodium azide which acts as a substrate[2] for enzyme nitrogenase involved in the process of biological nitrogen fixation, also acts as a potent mutagen[3]. L-cysteine is reported to inhibit azide mutagenicity in *Salmonella typhimurium*[4]. Certain mutations in cysteine biosynthetic pathway are also reported to reduce or abolish the mutagenic property of azide[5,6]. In the present communication we report the effect of L-cysteine on azide mutagenicity in a cysteine requiring mutant of *A. chroococcum.*

A cysteine requiring mutant[7] of *A. chroococcum* (N-4) was used in the present study. Burk's modified nitrogen-free medium[8] supplemented with 20 μg/ml ammonium sulphate (NA) was used for scoring cys^+ revertants. Complete medium was constituted by supplementing Burk's basal medium with 0.3% yeast extract (YE).

Cells were grown in YE medium to late log phase at 30°C in a shaking water bath. The cells (10 ml) were harvested by centrifugation and suspended in 1 ml of 0.01 M $MgSo_4$; 0.5 ml of this suspension was added to a tube containing 100 μg/ml azide in YE medium. Cell suspension (0.5 ml) was separately added to another tube containing azide (100 μg/ml) and L-cysteine (200 μg/ml) in YE medium. A zero sampling was done immediately after adding the cell suspension from both the tubes and the rest of the culture was allowed to incubate for 2 hr at 30°C under shaking condition.

The viable count was scored on complete medium and the cys^+ revertants were scored on NA medium. Cys^+ revertants from NA plates were further confirmed by spotting them individually on NA and complete medium.

The results (table 1) show that sodium azide alone is mutagenic to the cells of *A. chroococcum*. When azide treatment is given in the presence of L-cysteine, its mutagenicity is increased manyfold. This increase is however related with a high degree of killing. Azide mutagenicity is known to be exerted through the formation of a compound "azide metabolite"[9]. This metabolite is formed only in the growing cells[10] and L-cysteine stops this conversion making azide ineffective[5]. Our findings indicate that L-cysteine does not repress/feed back inhibit its biosynthetic enzymes as reported in *S. typhimurium*[11]. It seems that cysteine auxotrophy in the present case has not originated from a mutation in either cys E or cys K genes since this mutation does not inhibit the azide mutagenicity. These genes are reported to synthesize enzymes participating in cysteine biosynthesis required for conversion of azide into azide metabolite[5,6]. It is important to mention here that the mutant N-4 has been derived from a nitrogenase constitutive mutant and has the ability to grow in the presence of azide and ammonium

Table 1 *Cysteine interaction with azide on induced cys^+ reversion*

	Azide			Azide + L Cysteine		
Treatment time (hr)	Cell density	Survival (%)	Cys^+ revertants per 10^7 cells	Cell density	Survival (%)	Revertants per 10^7 cells
0	1.1×10^8	100	9.3	1.2×10^8	100	7.5
2	6.6×10^7	60	19.0	2.4×10^6	2.0	1150.0