Caucasians have a large C-band on chromosome 9. The sum of the C-bands on chromosomes 1, 9 and 16 in different racial groups were 16.57% (Oriental), 17.07% (Caucasian), 16.30% (Filipino), 15.84% (Polynesian), 16.30% (other)[25].

Further, we report the location of h regions since they have a tendency for pericentric inversion. We have proposed a classification of such heteromorphisms into 5 categories. The frequencies of the 5 categories found in 100 East Indians are recorded in the table and data are also compared with Caucasians and Blacks. For chromosomes 1 and 9, East Indians and Blacks have similar frequencies of inversion heteromorphisms while Caucasians have a significantly ($p < 0.01$) lower incidence of this type of heteromorphisms. Inversion of the h region in chromosome 16 is very rare and was not found in Black and Caucasian populations while 1.5% of chromosome 16 in the East Indian population showed inversion.

The biological and clinical implications of such heteromorphisms are poorly understood. There are preliminary indications that certain of the rare heteromorphisms may carry an increased risk, like mental retardation, fetal wastage, infertility, etc.[2]. Racial differences may be of anthropological interest and are of great value in linkage and population studies. Other important applications of heteromorphisms are to study the mechanism of mosaicism, paternity testing, maternal cell contamination during amniocentesis, and identifying the transmission from one of the parents of a specific chromosome that may be carrying a deleterious gene. The present study provides base line data in a normal population for comparing size and inversion heteromorphisms with an abnormal population.

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The effect of L-cysteine on presoaked barley seeds treated with methyl methanesulfonate

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Summary. Experiments were performed to analyse the effect of L-cysteine on barley seeds treated with methyl methanesulfonate (MMS) at a critical stage of the cellular cycle. Contrary to expectation, L-cysteine did not protect the barley seeds against MMS damage except for a slight protection at high doses (0.5, 0.7%).

It has been reported that applying mutagenic treatments at the beginning of DNA synthesis (S) in barley seeds increases mutation frequency and reduces physiological damage[2–4]. Cysteine has been used with success as a protector against the toxicity of alkylating agents in higher plants[5–8], and it is known to be capable of acting at all stages of the cellular cycle[9]. Methyl methanesulfonate, together with L-cysteine, was previously shown to exhibit a synergistic effect on lethality rather than conferring protection in bacteria[10]. This experiment was designed to determine the action of L-cysteine at a critical stage of barley seed germination (end of G_1 and onset of S) while subjected to methyl methanesulfonate (MMS) treatment.

Batches of 130–210 barley seeds (variety 'comun') were presoaked for 15 h in demineralized water at 20 °C in order to reach the end of G_1 and the onset of S[4,11]. The seeds were then treated for 1 h at 20 °C in 0.2, 0.5 and 0.7% w/v aqueous solutions of MMS (Merck) and then washed for 5 min in tap water. Each seed lot treated with MMS was then post-treated during 1 h in a 0.01 M aqueous L-cysteine (Merck) solution also at 20 °C. Both MMS and L-cysteine solutions were prepared with demineralized water and used fresh. Since L-cysteine is easily oxidized to cystine, the L-cysteine solution was prepared with boiled water in order to remove dissolved oxygen. To test for oxidation to cystine, TLC was performed according to the method of Slaten et al.[12] at 0, 2, 4, and 18 h. No oxidation to cystine was detected at 0 and 2 h, but at 4 and 18 h a progressive transformation of L-cysteine to cystine was noted. Because our post-treatment period was only 1 h long, no precautions were deemed necessary.

For homogeneity, the MMS treatments were carried out in

The effect of L-cysteine (0.01 M) applied as post-treatment on reduction in length of coleoptiles and seedlings induced by MMS in barley seeds presoaked for 15 h

MMS treatment (1 h) dose (%)	Post-treatment 1 h	Coleoptile length[a] Xmm	± SE	% Damage[b]	Seedling length[a] Xmm	± SE	% Damage[b]
0.0	H_2O	53.98	0.10	–	201.20	1.96	–
0.0	L-Cysteine	54.52	0.10	–	195.30	2.41	–
0.2	H_2O	29.75	0.04	44.88	152.95	1.56	23.98
0.2	L-Cysteine	29.80	0.05	44.79	153.00	1.33	23.95
0.5	H_2O	20.84	0.04	61.30	60.80	0.69	69.78
0.5	L-Cysteine	21.26	0.05	60.61	66.70[c]	0.76	66.84
0.7	H_2O	20.54	0.05	61.94	42.12	1.19	79.06
0.7	L-Cysteine	25.40[c]	0.03	52.94	54.46[c]	1.09	72.93

[a] Average of 3 replications. [b] Percentage of reduction of coleoptiles and seedlings with reference to the H_2O control. [c] Differ significantly from the H_2O control ($p < 0.05$) according to Z-test.

a constantly agitating and bubbling system[13], but the post-treatments were performed in closed flasks with only slight agitation. Afterwards, the seeds were washed for 5 min in running tap water and immediately sown in plastic boxes provided with a layer of cotton and filter paper moistened with distilled water. Germination was carried out in a controlled temperature chamber at 20 °C in darkness. Determinations of the effects on coleoptile and seedling length in the treated seeds were taken 5 and 15 days after sowing. The percentage of damaged coleoptiles and seedlings was calculated by the method of Conger et al.[14]. The MMS doses used herein have previously been shown to be active[15]. Post-treatments, 1 h long, with several L-cysteine doses (0.01, 0.05, 0.1 and 0.2 M) alone were tested for their toxicity to the barley seeds. Although no effects on any of the parameters studied were detected in the 1st 2 concentrations, a slight reduction in coleoptile and seedling length was apparent at higher doses (0.1 and 0.2 M). For this reason L-cysteine at 0.01 M dose was selected for use in this work. Furthermore, this dose has been reported to confer optimal protection against the action of alkylating agents in higher plants[6,8].

A variance analysis of our data showed that L-cysteine does not exert a protective effect against the damage induced by MMS in coleoptiles and seedlings. When the means were analyzed individually (table) however, a slight protection at MMS doses of 0.5% in seedlings and 0.7% in coleoptiles and seedlings was detected. In other words, at higher MMS doses, our results are in agreement with those of Bhojwani and Kaul[8] who treated pea seeds with ethyleneimine (EI) and found the greatest protection to occur at the highest EI dose post-treated with L-cysteine.

Narayanan and Konzak[6] reported protection of barley seeds against ethylmethanesulfonate (EMS) with cysteine post-treatments, but this protection was poor when compared with other thiosulfates. These results agree with ours to a certain degree (table) and the differences are probably due to the different reaction mechanisms of EMS and MMS, the first being an S_N1/S_N2 mixture[16] while MMS is S_N2 exclusively[17]. Moreover, MMS is a simpler molecule with a higher substrate constant (s) value than EMS[17]. As a result, EMS reacts more slowly than MMS with proteins or important enzymes which allow the remnant mutagen to be scavenged by L-cysteine. Methyl methanesulfonate, on the other hand, apparently acts more rapidly and specifically on the proteins and does not permit the L-cysteine to exert protection. When the MMS dose is increased, more of the mutagen remained available in the cells for interaction with L-cysteine to give some protection (table). Most of our results disagree with those reported by the authors previously mentioned[5–8], but their use of different kinds of alkylating agents could explain the differences observed. Furthermore, some of these authors observed a preferential protection in treatments consisting of a mixture of the mutagen and L-cysteine[5,7]. Thus the protector appeared to react with the mutagen before it entered the cell thereby neutralizing it and thus conferring some protection against physiological damage. The reported protection of L-cysteine against EI damage[7,8] is more difficult to reconcile with our results because EI and MMS are very similar in that both react via an S_N2 mechanisms[16–18]. In addition both possess similar s-values and are simple molecules. However EI requires a preliminary step of opening the ring in its structure before alkylation[18] and hence is slower than MMS which acts more rapidly on the proteins and causes the toxicity observed. Moreover it has been shown[19] that at pH 7.0 EI is in the immonium cation form and because of its charge cannot penetrate the cell, thus facilitating its reaction with L-cysteine and resulting in a protective effect.

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