similar to the well established behaviour at Gubbio[1-6]. Since most sites have been confined to European and American continents, the presence of this anomaly in the eastern Tethys, where no observations have been made so far, support the global nature of the iridium enrichment. The fallout flux of iridium at Um Sohryngkew river site is estimated to be 40 ng/cm^2. This yields a chondritic fallout of 60 mg/cm^2. These values are similar to fallout at Gubbio but a factor of 2–4 lower than the values observed at Stevns Klint and Carvaca.

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EVALUATION OF GENOTOXIC EFFECTS OF TURMERIC IN MICE

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HUMAN populations are not only exposed to environmental mutagens but also ingest thousands of chemicals as food components. Some of them may have genotoxic potentiality. Turmeric powder (commonly known as Haldi) is obtained from the root of *Curcuma longa*. It has an aromatic pepper-like bitter taste and is widely used as colouring and flavouring agent in food. It is known to cause chromosomal aberrations in plants[1] and cultured mammalian cells[2]. The present study was undertaken to determine the clastogenic potential *in vivo* in mammalian system.

Extraction of turmeric

Turmeric powder (50 g) was boiled in 500 ml methanol at 70°C for 4 hr followed by filtration and evaporation under reduced pressure on rotatory vacuum evaporator. Out of 50 g turmeric powder 4.15 g dry powder was obtained.

Cytogenetic test

The extracted powder was dissolved in dimethylsulphoxide (DMSO). Male(ddy), 12-week-old mice were divided in different groups. Each group comprised of 4 animals. Animals were injected i.p., with mitomycin (2 mg/kg b.w.)—positive control group, with different doses of turmeric-treated group and maximum equivalent amount of DMSO—control group. The highest dose was determined after conducting the toxicity experiment. After 22 hr of treatment colchicine (0.2 mg/kg b.w.) was injected. After 2 hr, the animals were sacrificed by cervical dislocation. Bone marrow of one femur was flushed out with foetal bovine serum (FBS)—for micronucleus assay, and of another one with medium (minimum essential medium + 10% FBS)—for chromosomal assay. Smears for micronucleus assay were prepared after one centrifugation at 800 rpm and then resuspending the cells in optimum volume of FBS. For chromosomal study, the centrifuged cells were resuspended in 0.075 M KCl followed by incubation at 37°C for 20 min and then fixation in acetic acid:methanol (1:3). After washing once with fixative, the cells were stored at −10°C overnight and again washed. Slides were prepared by flame dry method. Giemsa-stained slides were scored blindly.

It is apparent from the table that single acute dose treatment (500 mg/kg b.w.) could not significantly induce micronucleated polychromatic erythrocytes but caused considerably higher chromosomal aberrations (6.22%). It showed that micronucleus test does not reflect the exact index of clastogenecity of a chemical. Abraham and Kesavan[3] also did not observe significant increase in micronucleus frequency after administering orally 5 g/kg b.w. The most common abnormalities were of chromatid type—gap, break and fragments, while chromosomal types were observed occasionally (500 mg/kg b.w

†Deceased on 14 November 1986.

Table 1 *Cytogenetic effects of turmeric in mice*

			Turmeric treatment		
Dose mg/kg b.w.	Mitomycin 2 mg	DMSO control	100 mg	250 mg	500 mg
No. of animals	4	4	4	4	4
Micronucleus assay:					
No. of cells scored	8251	8523	7193	4707	4200
Polychromatic erythrocytes with micronucleus					
Mean ± SD	3.55 ± 0.82*	0.06 ± 0.12	0.29 ± 0.19	0.48 ± 0.24**	0.26 ± 0.21
Chromosomal aberration:					
No. of metaphses scored	164	199	200	173	193
No. of aberrant cells	21	1	4	3	12
Chromatid type					
Gap	2	1	1	2	6†
Break	5	–	–	–	2
Fragment	6	–	3	1	3
Exchange	1	–	–	–	–
Multiple	7	–	–	–	–
Chromosome type					
Gap	–	–	–	–	1
Break	–	–	–	–	1
% of aberrant cells	12.8	0.50	2.00	1.73	6.22

*Significant from control at 0.001 level *t* test; **Significant from control at 0.01 level *t* test; †One cell with chromatid and chromosome type gap.

treatment). Thus the present results are in contrast with the earlier findings[4]. The difference might either be due to difference in mode of treatment or due to elimination of targetted cell in long term treatment or variation in susceptibility of different species. It has been suggested that acute dose experiments are necessary for short term assays[3]. The active clastogenic component of turmeric may be Curcumin which has been found to be clastogenic in cultured mammalian cells[5]. Curcumin is generally found in turmeric at an average level[6] of 3%. It means, the presence of 0.003 g/kg b.w. Curcumin may be potentially genotoxic. Further, the possibility that the genotoxic effects of Curcumin may be enhanced or diminished by the promoting or antagonistic action of other dietary factors cannot be ruled out. Such complex combinational studies are needed further to achieve concrete results.

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