# Studies on the reversal of lead induced mitostatic effect in Allium Cepa root tip cells with myrobalan (fruit of Terminalia Chebula, Retz, Combretaceae)

H Rathore, R Punyasi, P Joshi, D Rathore, D Bhatnagar

**Citation**

H Rathore, R Punyasi, P Joshi, D Rathore, D Bhatnagar. *Studies on the reversal of lead induced mitostatic effect in Allium Cepa root tip cells with myrobalan (fruit of Terminalia Chebula, Retz, Combretaceae)*. The Internet Journal of Alternative Medicine. 2006 Volume 4 Number 1.

**Abstract**

The present study was undertaken to find the protective role of myrobalan against lead (Pb) induced cytogenetic effects on mitosis in Allium-cepa root tip cells. Onions were initially cultivated in deionized water for 3 days and were then exposed at 100, 1000, 3000 and 10,000 ppm of lead nitrate solution for 1 hr. After exposure to lead, the onions were allowed to recover naturally or in myrobalan suspension (0.01 mg/ml) for 72 hrs. The root color, mean root length (MRL) and mitolic index (MI) were recorded and the metaphases and anaphases were scored for chromosomal aberrations. During the natural recovery (NR), roots did not grow following 1000, 3000 and 10,000 ppm Pb exposure but myrobalan treated drug recovery (DR) showed root growth following 1000 and 3000 ppm Pb exposure. The root growth was observed in both NR and DR, more in DR in 100 ppm Pb exposure. Among the control, root growth during these periods and MI did not change throughout the experimentation. Pb exposure at all concentrations, lowered MI. NR was ineffective in Pb treated root tip cells as these were seen in interphase with hypertrophied nucleoli showing mitostatic effect. DR could not revert the mitostatic effect in root tip cells exposed at 10,000 ppm Pb, however, drug could do so in 100, 1000 ppm and 3000 ppm Pb exposure. Both, NR and DR reverted mitostatic effect after 100 ppm Pb exposure and the effect was observed earlier in DR. No chromosomal aberrations could be seen at 1 hr, the cells failed to show typical metaphase arrangement. The effect appeared dose dependent. DR reverted mitostatic effect from 1 hr onwards and completed at 72 hrs in 1000 and 3000 ppm Pb exposure. At 100 ppm Pb exposure, mitostatic effect disappeared at 48 hr in NR while in DR, it reverted after 24 hr. Control root tip cells showed no mitostatic effect.

## INTRODUCTION

Lead is known to be toxic, mutagenic and carcinogenic in human beings [1]. The modern system of medicine uses chelation therapy to cure Pb toxicity [2]. However, Ayurveda (Indian herbal medicine system) in Pb toxicity suggest the use of myrobalan (fruit of Terminalia chebula) [3]. Our earlier study, showed the protective role of myrobalan towards Pb toxicity in mice [4]. The present study, was undertaken to find out whether myrobalan can lower the cytogenetic effects of inorganic Pb in Allium test. The short term Pb exposure for 1 hr was used as Pb is known to disturb mitosis in Allium root tip cells [5,6,7,8].

## MATERIALS AND METHODS

### ALLUM CEPA

Dry healthy onion bulbs 1.5-2.0 cm in diameter were obtained from the local market.

### DRUG

Myrobalan, dried young nuts of Terminalia chebula were procured locally, gently baked for few minutes and cooled. The swollen nuts were grinded to a fine powder. The recommended dose of myrobalan for adults is 3-9 gm/day [3]. However, low dose (0.01 mg/ml) of myrobalan was used in the present study to test the recovery of the mitostatic effects of Pb in Allium test.

Lead nitrate (Hi Media A.R) was dissolved in deionized water to prepare solutions of 100, 1000, 3000 and 10,000 ppm concentrations.

### CULTIVATION OF ONIONS

Descaled healthy onions (Allium cepa L.) bulbs (90) were grown initially in deionized water for 3 days. Mean root length (MRL) was taken as baseline and were divided into 5 groups of 18 bulbs in each group. The onion bulbs with growing roots were exposed to Pb solution at 100, 1000,

3000 and 10,000 ppm (group 2-5) and in deionized water as control (group 1) for 1 hr. After Pb exposure few of the root tips from each group were fixed for cytogenetic study.

After Pb exposure to onion bulbs for 1 hr, each group was subdivided into two and was allowed to recover naturally (NR) or in the presence of the drug (DR). Roots were exposed to myrobalan at a concentration of 0.01 mg/ml. The recovery in NR and DR was allowed for the next 72 hr. MRL was recorded at 24, 48 and 72 hr of myrobalan treatment. Five root tips from each onion were cut and fixed in acetoalcohol (1:3 v/v) stained in N-HCl-acetocarmine (1:9 v/v) and squashed in 45% acetic acid. The squashed preperations were stored in a refrigerator. The mitotic index and the movement and arrangement of chromosomes at metaphase and anaphase after 24, 48 and 72 hr was observed.

The statistical analysis was performed by student t-test and values less than 5% were considered significant.

## RESULTS

No morphological i.e., shape and color changes were noticed in the tips of roots of any group of bulbs. The root tips under NR for 72 hr did not grow following 1000, 3000 and 10,000 ppm Pb exposure for 1 hr (Table 1). DR for 72 hr also remained ineffective following 1 hr Pb exposure at 10,000 ppm, however, roots grew following Pb exposure at 1000 and 3000 ppm. The roots grew in both, NR and DR, more in DR following 1 hr Pb exposure at 100 ppm. The control roots grew during the corresponding recovery period.

Among the control root tips, MI did not change throughout the experiment (Table 2). Pb exposure for 1 hr at 1000, 3000 and 10,000 ppm, lowered MI. NR for 72 hr remained ineffective after 1000, 3000 and 10,000 ppm Pb exposure as cells of root tips appeared in interphase with hypertrophied nucleoli and did not resume mitotic course. DR could not revert mitostatic effect of lead at 10,000 ppm Pb exposure, however it was able to do so in roots expose to 1000 and 3000 ppm Pb during the 72 hr period of recovery. Following, 100 ppm Pb exposure, both NR and DR could overcome the mitostatic effect, more earlier in DR. Control root tips revealed no any mitostatic effect.

The observation of metaphases and anaphases after 1 hr exposure to growing roots indicates probable interference during transition of cells from prophase into metaphase (Table 3). The effect appeared dose dependent as following 100, 1000, 3000 and 10,000 ppm Pb exposure, 51%, 55%, 67% and 88% of the cells failed to show typical metaphase arrangement. No aberrations were noticed. Control root tips did not revealed any disturbed pro-metaphase transition.

### Figure 1

Table 1: Mean root length (MRL) of onion bulbs grown following lead exposure at various concentrations

| S. No. | Parameters | Groups | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | 1 | 2 | | 3 | | 4 | | 5 | |
| | | Control | NR | DR | NR | DR | NR | DR | NR | DR |
| 1 | MRL at 24 hr | 54.15[a] ± 0.58 | 42.67[a] ± 0.23 | 49.78[a] ± 0.54 | 42.56 ± 0.54 | 46.48[a] ± 0.13 | 42.32 ± 0.40 | 44.70[a] ± 0.19 | 42.33 ± 0.76 | 42.37 ± 0.60 |
| | (% change) | 31.24% | 3.44% | 20.67% | – | 12.67% | – | 8.36% | – | – |
| 2 | MRL at 48 hr | 58.37[b,d] ± 0.60 | 44.89[b,d] ± 0.21 | 55.22[b,d] ± 0.45 | 43.00 ± 0.85 | 51.01[b,d] ± 0.22 | 42.33 ± 0.42 | 47.13[b,d] ± 10.26 | 42.34 ± 0.88 | 42.39 ± 0.63 |
| | (% change) | 41.50% | 8.82% | 33.86% | – | 23.66% | – | 14.25% | – | – |
| 3 | MRL at 72 hr | 63.27[ce] ± 0.63 | 46.55[ce] ± 0.19 | 59.37[ce] ± 0.50 | 43.40 ± 0.99 | 56.31[ce] ± 0.41 | 42.35 ± 0.45 | 54.10[ce] ± 0.36 | 42.36 ± 0.66 | 42.50 ± 0.66 |
| | (% change) | 53.38% | 12.84% | 43.92% | – | 36.50% | – | 31.15% | – | – |

Root tips were initially grown in deionized water for 3 days and were then exposed at 0, 100, 1000, 3000 and 10,000 ppm lead nitrate for 1 hr. After lead exposure the root tips were washed with deionized water and were allowed to recover naturally (NR) or in presence of drug (DR) for 24, 48 and 72 hr. MRL is given in mm.

Values are shown as mean ± SEM of 20 roots in each group.

$p > 0.05$ [a, b, c.] MRL at 24, 48 and 72 hr vs MRL at 3 days of initial growth. [d, e.] MRL at 48 and 72 hr vs 24 hr. (—) is nil.

**Figure 2**

Table 2: Mitotic Index (MI) of onion roots following lead exposure at various concentrations

| S. No. | Parameters | Groups | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | 1 | 2 | | 3 | | 4 | | 5 | |
| 1 | MI (at 1 hr) | 48.56 ± 1.56 | 23.44[a] ± 0.68 | | 22.97[a] ± 0.68 | | 21.44[a] ± 1.10 | | 19.14[a] ± 1.10 | |
| | % change | 0.83% | 52.13% | | 53.09% | | 56.21% | | 60.91% | |
| 72 hr recovery period after 1 hr lead exposure | | | | | | | | | | |
| | | | NR | DR | NR | DR | NR | DR | NR | DR |
| 2 | MI (at 24 hr) | 48.41 ± 1.40 | 25.08 ± 1.45 | 26.20[b] ± 1.65 | 0.00 | 23.46 ± 0.22 | 0.00 | 22.77 ± 0.78 | 0.00 | 0.00 |
| | (% change) | 0.30% | 6.99% | 11.77% | — | 2.13% | — | 3.87% | — | — |
| 3 | MI (at 48 hr) | 48.42 ± 1.26 | 27.36[e] ± 0.82 | 29.36[e] ± 0.87 | 0.00 | 24.45 ± 0.20 | 0.00 | 23.93 ± 0.83 | 0.00 | 0.00 |
| | (% change) | 1.42% | 16.72% | 25.25% | — | 6.44% | — | 11.61% | — | — |
| 4 | MI (at 72 hr) | 48.44 ± 1.01 | 32.10[d] ± 1.33 | 36.01[de] ± 1.25 | 0.00 | 26.02[d] ± 0.68 | 0.00 | 24.23[d] ± 0.69 | 0.00 | 0.00 |
| | (% change) | 0.24% | 36.94% | 53.62% | — | 13.27% | — | 13.01% | — | — |
| 5 | % change in MI compared with control | | 33.73%[f] | 25.66%[f] | — | 46.28%[f] | — | 49.97%[f] | — | — |

After 3 days of initial growth, MI was 48.97 ± 1.15.

$p > 0.05$. [a.] MI after 1 hr of Pb exposure vs MI after 3 days of initial growth. [b,c,d.] MI after 24, 48 and 72 hr of recovery vs MI after 1 hr Pb exposure.

[e.] DR vs NR at 24 hr, 48 and 72 hr

[f.] MI at 72 hr vs MI after 3 days of initial growth.

**Figure 3**

Table 3: Metaphases, anaphases and chromosomal aberrations following 1 hr lead exposures

| Groups | Treatments | Mitotic Index | Microscopic Effect in Percentage | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Metaphase | | Anaphase | | Sticky Chromosomes | C-Mitosis | Vagrant (Lagging) Chromosomes | Multi-polar Anaphase | Bridges | Fragments | MNC Micronucleated cells | Polykaryocytes |
| | | | N. | Ab. | N. | Ab. | | | | | | | | |
| 1 | Control | | | | | | | | | | | | | |
| | 72 hr grown | 48.97 ± 1.51 | 100% | – | 100% | – | – | – | – | – | – | – | – | – |
| | 1 hr | 48.56 ± 1.56 | 100% | – | 100% | – | – | – | – | – | – | – | – | – |
| | Recovery | | | | | | | | | | | | | |
| | 24 hr | 48.41 ± 1.40 | 100% | – | 100% | – | – | – | – | – | – | – | – | – |
| | 48 hr | 48.42 ± 1.26 | 100% | – | 100% | – | – | – | – | – | – | – | – | – |
| | 72 hr | 48.44 ± 1.01 | 100% | – | 100% | – | – | – | – | – | – | – | – | – |
| 2 | 1-hr-Pb (100 ppm) | 23.44[c] ±0.68 | 48.59% | 51.41% | 100% | – | – | – | – | – | – | – | – | – |
| | NR - 24 hr | 25.08 ± 1.45 | 86.20% | 13.80% | 100% | – | – | – | – | – | – | – | – | – |
| | 48 hr | 27.36 ± 0.82 | 96.42% | 3.58% | 100% | – | – | – | – | – | – | – | – | – |
| | 72 hr | 32.10 ± 1.33[f] | 100% | – | 100% | – | – | – | – | – | – | – | – | – |
| | DR - 24 hr | 26.20 ± 1.05[b] | 100% | – | 100% | – | – | – | – | – | – | – | – | – |
| | 48 hr | 29.36 ± 0.87[e] | 100% | – | 100% | – | – | – | – | – | – | – | – | – |
| | 72 hr | 36.01 ±1.25[d] | 100% | – | 100% | – | – | – | – | – | – | – | – | – |
| 3 | 1-hr-Pb (1000ppm) | 22.97[a] ±0.68 | 44.53% | 55.46% | 100% | – | – | – | – | – | – | – | – | – |
| | NR - 24 hr | – | – | – | – | – | – | – | – | – | – | – | – | – |
| | 48 hr | – | – | – | – | – | – | – | – | – | – | – | – | – |
| | 72 hr | – | – | – | – | – | – | – | – | – | – | – | – | – |
| | DR - 24 hr | 25.46 ± 0.22 | 96.77% | 3.23% | 100% | – | – | – | – | – | – | – | – | – |
| | 48 hr | 24.45 ± 0.20 | 97.22% | 2.77% | 100% | – | – | – | – | – | – | – | – | – |
| | 72 hr | 26.02 ± 0.68[f] | 100% | – | 100% | – | – | – | – | – | – | – | – | – |
| 4 | 1-hr-Pb (3000 ppm) | 21.44[a] ±1.10 | 33% | 67% | | – | – | – | – | – | – | – | – | – |
| | NR - 24 hr | – | – | – | – | – | – | – | – | – | – | – | – | – |
| | 48 hr | – | – | – | – | – | – | – | – | – | – | – | – | – |
| | 72 hr | – | – | – | – | – | – | – | – | – | – | – | – | – |
| | DR - 24 hr | 22.27 ± 0.78 | 95.83% | 4.17% | 100% | – | – | – | – | – | – | – | – | – |
| | 48 hr | 23.93 ± 0.83 | 97.14% | 2.86% | 100% | – | – | – | – | – | – | – | – | – |
| | 72 hr | 24.23 ± 0.69[f] | 100% | – | 100% | – | – | – | – | – | – | – | – | – |
| 5 | 1-hr-Pb (10000 ppm) | 19.14[a] ±1.10 | 12% | 88% | | – | – | – | – | – | – | – | – | – |
| | NR - 24 hr | – | – | – | – | – | – | – | – | – | – | – | – | – |
| | 48 hr | – | – | – | – | – | – | – | – | – | – | – | – | – |
| | 72 hr | – | – | – | – | – | – | – | – | – | – | – | – | – |
| | DR - 24 hr | – | – | – | – | – | – | – | – | – | – | – | – | – |
| | 48 hr | – | – | – | – | – | – | – | – | – | – | – | – | – |
| | 72 hr | – | – | – | – | – | – | – | – | – | – | – | – | – |

After 3 days of initial growth, MI was 48.07 ± 1.51.
[a.] MI after 1 hr of Pb exposure vs MI after 3 days of Initial growth.
[b, c, d.] MI after 24, 48 and 72 hr of recovery vs MI after 1 hr of Pb exposure.
(—) Nil.
N and Ab is normal and abnormal.

After 3 days of initial growth, MI was 48.07 ± 1.51.

[a.] MI after 1 hr of Pb exposure vs MI after 3 days of Initial growth.

[b, c, d.] MI after 24, 48 and 72 hr of recovery vs MI after 1 hr of Pb exposure.

(—) Nil.

N and Ab is normal and abnormal.

## DISCUSSION

In Allium test, the important effect exhibited by Pb at a lower concentration was mitodepression and with higher concentrations, Pb exerted inhibition of prophase and finally

resulted in the disappearance of mitosis. The mitostatic effect did not revert to normal condition i.e., cells did not reentered into mitosis from G1 during NR except among 100 ppm Pb exposed roots, however, recovery with DR could remove the mitostatic effect following 1000 and 3000 ppm Pb exposed roots. The effect of 10,000 ppm Pb exposure was not reverted during drug or in natural recovery process.

The earlier workers observed inhibition of root growth accompanied with spindle disturbing effect along with chromosomal aberrations following Pb exposures [5,6,7,8,9,10,11,12,13]. In the present study, no chromosomal effects could be noticed. This may be due to the short Pb exposure time of 1 hr in the present study, while it was 24 hr or more in other studies. However, 1 hr exposure time was enough to find out early effects of Pb as within 1 hr Pb gets accumulated in the onion root cells [5]. A temporary effect of Pb i.e., incompletely organised metaphase were seen after 1 hr Pb exposure, however, no abnormal anaphase was observed.

Pb has been shown to have varied effects. Pb may inhibit DNA repair processes [14,15], denature proteins [16] and disturb the transcription process [17]. Pb has been shown to induce lipid peroxidation [18], alter membrane permeability [19] and bind to SH-groups of tubulins in cell, disturbing typical arrangement of chromosomes at metaphase [20]. Pb inhibits cell elongation, irrespective of mitotic activity due to changes in cell wall characteristics [21]. Pb has been shown to compete with calcium ions, altering certain signal transduction processes [22]. During the late G1 phase, restriction point gate opens in the presence of complex molecules at promoters for essential cell cycle genes [23]. It appears that Pb may interfere with some of these processes, resulting in the mitostatic effect in onion root top cells and myrobalan may be able to counteract some of these activities.

Pb is removed from the cytoplasm of the cells in the process of detoxification [5]. Root cell cortex wall is main storage site of lead [24]. The ability of plants to bind heavy metals in cell wall and external polysaccharides has been shown [25]. The detoxication takes place in the cytoplasm and cell wall within 12-24 hr, which may be responsible for the mitotic activity in root tips after low Pb intoxication.

Myrobalan may bind with the root tips as color changes were noticed from very pale to dark brown at higher concentration. It is suggested that myrobalan bind to Pb, making it unavailable, thereby reducing Pb toxicity. It is likely that Pb induced peroxidative damage may result in the mitostatic effect. Myrobalan has been shown to exert antioxidant and free radical scavenging activities [26,27], which may counteract the mitostatic effect of Pb.

The unrepaired DNA does not allow cells to go beyond G1 stage [23]. The Pb induced DNA damage may be corrected by myrobalan as it has been shown to exert antimutagenic activity in Salmonella typhimurium against direct acting mutagens such as sodium azide and 4 nitro-o-diphenylenediamine [28]. The antimutagenic activity of myrobalan was suggested to be due to hydrolysable tannins [29]. The various plant components present in the extracts such as sulphahydryl and flavonoid compounds, gallic acid, ellagic acid, mucic acid, citric acid, reducing sugars and tannins etc. can modulate effects of environmental genotoxicants [30]. As myrobalan possess many of these compounds, it may be able to reduce the cytotoxic effect of Pb.

## ACKNOWLEDGEMENT

The authors thank the Head of the Department for providing the facilities.

## CORRESPONDENCE TO

Tel: 91-734-2512658 Fax: 91-734-2511645 E-mail: hemant2005hemant@sancharnet.in

### References

1. Anonymous, Toxicological Profiles for Lead, U.S. Department of Health and Public Services, Agency for Toxic Substances and disease registry prepared by Research Triangle Institute (1997) 13-283.
2. A.R. Carol. Chelation therapies for metal intoxication, In: Toxicology of metals, Ed. L.W. Chang, CRC Press, USA (1996), 487-504.
3. Bramhaverchasva. Jadi butyon dwara swasthya sanrakshan, 9th Edition. Yug Nirmana Yojna, Gaytri Tapobhumi, Mathura (1997): 73-80.
4. H.S. Rathore, G.S. Kelwa, P. Mewara, D.M. Kumawat, R. Prasad, D. Bhatnagar, Influence of Myrobalan (fruit of Terminalia Chebula) on lead-induced toxicity in mice, Indian J. Occup. Hlt. 44 (2001), 169-174.
5. M. Wierzbicka, Mitotic disturbances induced by low doses of inorganic lead, Caryologia. 41 (1988) 143-160.
6. M. Wierzbicka, Disturbances in cytokinesis caused by inorganic lead, Environ. Exp. Bot. 29 (1989) 12-13.
7. A.I. Dovhalynk T.B. Kalynyak Ya.B. Blyum, The toxic effect of metal ions on the root growth and mitotic activity of onion (Allium cepta L.) Cells. Dopovidi Natsional noyi-Akademiyl Nauk, Ukrayiny 6 (1998) 173-178.
8. N.V. Reutora, V.A. Shevchenko, Mutagenic effect of two different lead compounds, Genetika 27 (1991) 1275-1279.
9. R.K. Sengupta, P. Ghosh, Comparative assessment of the effects of lead nitrate through in-vitro and in-vivo studies,

Indian J. Agric. Res. 30 (1996) 227-234.
10. A.I. Dovgalink, T.B. Kaliniak, Ia B. Blium, Assessment of Phyto-and-cytotoxic effects of heavy metals and aluminium compounds using onion apical root meristem. Trstol. Genet. 35 (2001) 3-9.
11. D. Liu, W. Jiang, W. Wang, F. Zhio, C. Lu, Effect of lead on root growth, cell division and nucleolus of Allium Cepa, Environ. Poll. 86 (1994) 1-4.
12. N.V. Obroucheva, E.I. Bystrova, V.B Iranov, O.V. Antipova, J.V. Sergegin, Root growth response to lead in young maize seedlings, Plant and soil. 2000 (1998) 55-61.
13. V.B. Ivanov, E.I. Bystrova, N.V. Ob Vouchera O.V Antipova, M. Sobtik, H. Bergmann, Growth response of barely roots as an indicator of lead toxic effects, Angewandte Botanik. 72 (1998) 140-143.
14. N.K. Roy, T.G. Rossman, Mutagenesis and comutagenesis by lead compounds, Mut. Res. 298 (1992) 97-103.
15. K. Steenland, B, Paolo, Lead and cancer in humans: Where are we now?, Amer. J. Indust. Med. 38 (2000) 295-299.
16. H.S. Rathore, H. Swarup, P.K. Sanghvi, Toxicity of cadmium chloride and lead nitrate to Chironomus tentans larvae, Environ. Pollut. London 18 (1979) 173-177.
17. J.S. Hanas, J.S. Rodgers, J.A. Bantle, Y.G. Chang, Lead inhibition of DNA - binding mechanism of Cys(s) His(2) zinc finger proteins, Mol. Pharmacol. 56 (1999) 982-988.
18. K.L. Stefanov, K.A. Seizova, S.D. Pandey, N.V. Yanishlieva, E.M. Marinova, L.A. Tyankova, L.L. Kuleva, S.S. Popov, Effect of lead ions on lipid peroxidation and antioxidant complex activity of Capsicum annuum leaves, pericarp and seeds, J.Sci. of Food & Agric. 67 (1995) 259-266.
19. N. Kwartirnikov, J. Necher, N. Lavchieva, G. Nacheva, N. Nikolova, K. Seizova, M. Kwartirnikov, V. Lavchiev, S. Popov, Lipids and sterols in Musca domestica L. (Diptera, Muscidue): Changes after treatment with sucrose and lead, Mol. Biol. 131 (2002) 543-550.
20. M. Wallin, F. Bo, M. Biller, Studies of the interaction of chemicals with microtubule assembly in-vitro can be used as an assay for detection of cytotoxic chemicals and possible inducers of aneuploidy, Mut. Res. 201(1988) 303-311.
21. S.D. Lane, E.S. Martin, J.F. Garrod, Lead toxicity effects on indolyl 3-acetic acid induced cell elongation, Planta 144 (1978) 79-84.
22. T. Andesirk, C. Pedersen, G. Andesirk, M. Kern, Low levels of inorganic lead noncompetitively inhibit mu-calpin, Toxicology 16 (1998) 167-174.
23. T.D. Pollard, C.E. Williams, The G1 phase and the regulation of cell proliferation, In - Cell Biology, Saundaurs, U.S.A. (2002) pp 687-696.
24. M. Ksiazek, A. Wozny, Lead movement in popla adventititious roots, Biologia Plantarum 32 (1990) 54-57.
25. J.R. Cummings, G.J. Taylor, Mechanism of metal tolerance in plants: physiological adaptations for exclusion of metal ions from the cytoplasm In: Eds R.G. Alscher, J.R. Cummings, Stress responses in plants: adaptation and acclimation mechanism. Wiley-Liss, NY (1990) 329-359.
26. F. Naiwu, Q. Lanping, L.H. Zhang, Antioxidant activity of extracts of Terminalia chebula and its preventive effect on DNA breakage in human white cells induced by TPA. Chinese Trad. and Herbal Med. 23 (1992) 26-29.
27. C.H. Yew, L. Tachen, Y.K. Tang. C. Min L. Chunchiang, H.Y. Cheng, Antioxidant and free radical scavenging activities of Terminalia chebula, Biol. Pharmaceut Bull. 26 (2003) 1331-1335.
28. I.S. Grover, S. Bala, Antimitogenic activity of Terminalia chebula (myrobalan) in Salmonella typhimurium, Ind.J.Exp.Biol. 30 (1992) 339-341.
29. S. Kaur, I.S. Grover, Antimitogenicity of hydrolyzable tannins from Terminalia chebula in Salmonella typhimurium, Mut.Res. 41 (1998) 169-179.
30. D. Sarkar, A. Sharma, Plant extracts as modulators of genotoxic effects, Bot. Rev. 62 (1996) 275-300.

## Author Information

**H. S. Rathore, Ph.D.**
Cell Biology Unit, School of Studies in Zoology, Vikram University

**Ranjana Punyasi, MPhil**
Cell Biology Unit, School of Studies in Zoology, Vikram University

**Pooja Joshi, MPhil**
Cell Biology Unit, School of Studies in Zoology, Vikram University

**Deepika Rathore, MPhil**
Cell Biology Unit, School of Studies in Zoology, Vikram University

**D. Bhatnagar, PhD**
School of Biochemistry, D.A. University