putrescine counteracted the encystation-promoting activity of inhibitor; at 0.5 mM concentration putrescine almost abolished the action of $\alpha$MO. MGBG proved more effective in promoting encystation and gave maximum encystation at 0.5 mM concentration. Polyamines themselves were not very effective in enhancing encystation. Putrescine was completely inactive while spermidine and spermine enhanced encystation to some extent at 1 mM concentration. Increase or decrease in polyamine concentration did not further enhance encystation; higher concentrations of polyamines resulted in lysis of amoebae. The presence of $Mg^{++}$ was also required for encystation inducing effect of MGBG, and the removal of $Mg^{++}$ lowered encystation.

The present results establish a rapid drop in polyamine levels during encystation of *A. culbertsoni*. Enhancement of encystation in the presence of $\alpha$ MO and MGBG, suggests that inhibition of polyamine biosynthesis and decrease in polyamine levels may signal cessation of growth and activate encystation related genes. Inhibitors of DNA biosynthesis[18] and mitochondrial function[19] also trigger encystation. Polyamine metabolism may also be a target for encystment-inducing action of magnesium and some organic effectors. Putrescine, on the other hand, promotes considerable encystment of *A. culbertsoni*,[20] supporting a key regulatory role of polyamine metabolism in amoebic differentiation.

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## TRANSPLACENTAL INDUCTION OF MICRONUCLEI FOLLOWING MATERNAL ADMINISTRATION OF MERCURIC CHLORIDE

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MERCURIAL compounds are known to be clastogenic, teratogenic and embryotoxic. However the foeto-maternal barrier is more effective against inorganic forms of mercury[1-3]. The present investigation was undertaken to observe the foeto-toxic effects of inorganic mercuric chloride with the help of the micronucleus test, which indicates a damage at the chromosome level. Its presence in foetal liver cells can be taken as an index of clastogenicity in the foetal tissue[4-9].

Three subtoxic doses of mercuric chloride ($LD_{50}$ = 37 mg/kg body wt.)[10] were administered orally to an inbred laboratory strain of pregnant female rats *Rattus norvegicus* on day 7 of gestation. The doses used were 1/20th $LD_{50}$ (1.85 mg/kg body wt.), 1/15th $LD_{50}$ (2.47 mg/kg body wt) and 1/10th $LD_{50}$ (3.70 mg/kg body wt.). The animals were sacrificed on day 18. All foetuses were examined and a portion of the liver of two from each set were dissected out, minced finely and fixed in 1:3 acetic ethanol (three changes). Slides were

**Table 1** *Micronuclei induced in foetal liver cells following maternal administration of $HgCl_2$ in rats*

| Sets studied | Total aberrant cells (mean ± SD) | Type of cells affected | |
|---|---|---|---|
| | | Large | Small |
| Control | 0.36 ± 0.51 | 0.21 ± 0.32 | 0.14 ± 0.19 |
| *Treatment* 1 1/20th $LD_{50}$ (1.85 mg/kg body weight) | 0.26 ± 0.28 | 0.16 ± 0.22 | 0.15 ± 0.21 |
| *Treatment* 2 1/15th $LD_{50}$ (2.47 mg/kg body weight) | 1.13 ± 0.67 | 0.68 ± 0.24* | 0.56 ± 0.56 |
| *Treatment* 3 1/10th $LD_{50}$ (3.70 mg/kg body weight) | 1.14 ± 0.55* | 0.65 ± 0.23* | 0.64 ± 0.62 |

df = 8; $^{*}P < 0.05$

prepared following the usual fixative-flame drying schedule and stained in Giemsa.

500 cells were scanned from each set. The liver cells were categorized into two types, large and small according to their diameters.

The percentage of micronuclei recorded in the liver cells of the foetuses obtained 11 days after the administration of $HgCl_2$ to the pregnant mothers ranged from 0.21 in control to 1.14 with the maximum dose (1/10th $LD_{50}$). The difference was statistically significant indicating that $HgCl_2$ in higher doses induces chromosomal breaks resulting in micronuclei. The breaks were more frequent in the larger cells (table 1).

Several external agents have been shown to cause transplacental micronuclei which may be either detected from foetal bone marrow or liver[4-6, 10-13].

The induction of micronuclei in the foetal liver cells following the administration of $HgCl_2$ to pregnant rats even after eleven days of recovery indicates the placental permeability of the salt and its toxicity even at low doses to the foetal organs.

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## INFLUENCE OF GROWTH REGULATORS ON POLLEN VIABILITY AND POLLEN TUBE GROWTH IN TWO SPECIES OF *CLEOME*

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ATTEMPTS have been made to overcome the lack of pollen tube growth with the use of various chemicals by a number of workers[1]. While much information on the effect of plant growth regulators in plant systems is available, the effect on pollen viability and pollen tube growth has received inadequate attention. Considerable knowledge has accumulated on the constituents and exogenous chemicals affecting pollen viability and tube growth[2]. The present study was undertaken to assess the effect of growth regulators, individually or in combination, on the pattern of pollen tube growth *in vitro* in two members of Capparidaceae (now, Cleomaceae) namely, *Cleome gynandra* Linn and *Cleome viscosa* Linn.