STUDIES ON INDUCED DESYNAPSIS IN *CAPSICUM ANNUUM* L.

M. VENKATRAJAM AND K. SUBHASH
Botany Department, Kakatiya University,
Warangal 506 009, India.

DESYNAPSIS is a condition where post-pachytene separation of paired chromosomes results in the formation of univalents, probably due to the failure of crossing over. Based on the univalent frequency amongst meiocytes, desynapsis can be grouped as weak, medium strong and complete desynapsis[1]. Desynapsis can reduce male and female fertility but only in extreme cases it imparts complete sterility[2]. Praaken[1] reported a variation of male fertility from 1–59% in rye. Even 80% of the pollen sterility was noticed in desynaptic mutant of Safflower by Sahu *et al*[3]. In this paper author describe the meiotic behaviour and inheritance pattern of x-rays and ethyl methane sulphonate (EMS) induced desynaptic mutants in *Capsicum*.

During the course of a cytological investigation of a population of *C. annuum* cultivar CA 960 treated with x-rays and EMS, desynapsis of chromosomes resulting in meiotic alterations was observed at 5 kR and 0.1% (24 hr) respectively in two plants. The growth habit and the morphology of these plants resembled those of the normal plants, except that the mutants exhibited 78% pollen sterility with a poor seed set.

Cytological examination of the mutants in meiotic system disclosed various types of chromosomal alterations. Maximum number of bivalents were observed in 2% PMCs only, while $0^{II} + 24^{I}$ and $1^{II} + 22^{I}$ were noticed in 62% of the PMCs (figure 1). These desynaptic mutants were classified as a medium strong type. Anaphase I separations were highly irregular and such unequal distribution of chromosomes at AI would lead to the formation of aneuploid gametes, which in turn would produce aneuploid plants. Besides lack of chiasma formation and the presence of univalents, these mutants revealed several other irregularities such as large number of laggards at AI and AII (figure 2) and micronuclei at TII (figure 3). Micronuclei were abundant and they resulted in polyspory (figure 4). The multipolar segregations at AII were also observed.

The plants from these seeds retained the abnormality in further generations indicating the true breeding nature of the mutant. To elucidate the inheritance further, these mutants were crossed with normal plants. The hybrids were normal indicating dominance of normal condition. The F_2 progeny comprised of 70 normal and 18 desynaptic mutants which closely fitted the 3:1 ratio ($=0.969$, $P=0.50-0.30$). This indicated that the mutant was recessive and was controlled by a single gene.

Consequent to the univalent formation in the early meiotic stages, a high degree of irregularities in the following stages resulted in the production of non-functioning germ cells. Similarly, it is presumed that the desynaptic behaviour of the chromosomes in these plants must be one of the primary causes of sterility prevalent. According to Praaken[1], desynapsis may be due to i) gene action ii) loss of chromosome pair iii) apomixis and iv) structural or numerical changes of chromosomes. Desynapsis appears, at diakinesis is a timing mistake (Anticipation of chromosome separation in bivalents) and it is generally attributed either to partial failure of synapsis or to the inability to exchange DNA strands after synapsis. In both cases, a lack of chiasma formation is supposed. Consequently, the diakinesis metaphase I transition is anticipated and an irregular chromosome congression to the equatorial plate and early separation of chromosomes in bivalents is final result[4].

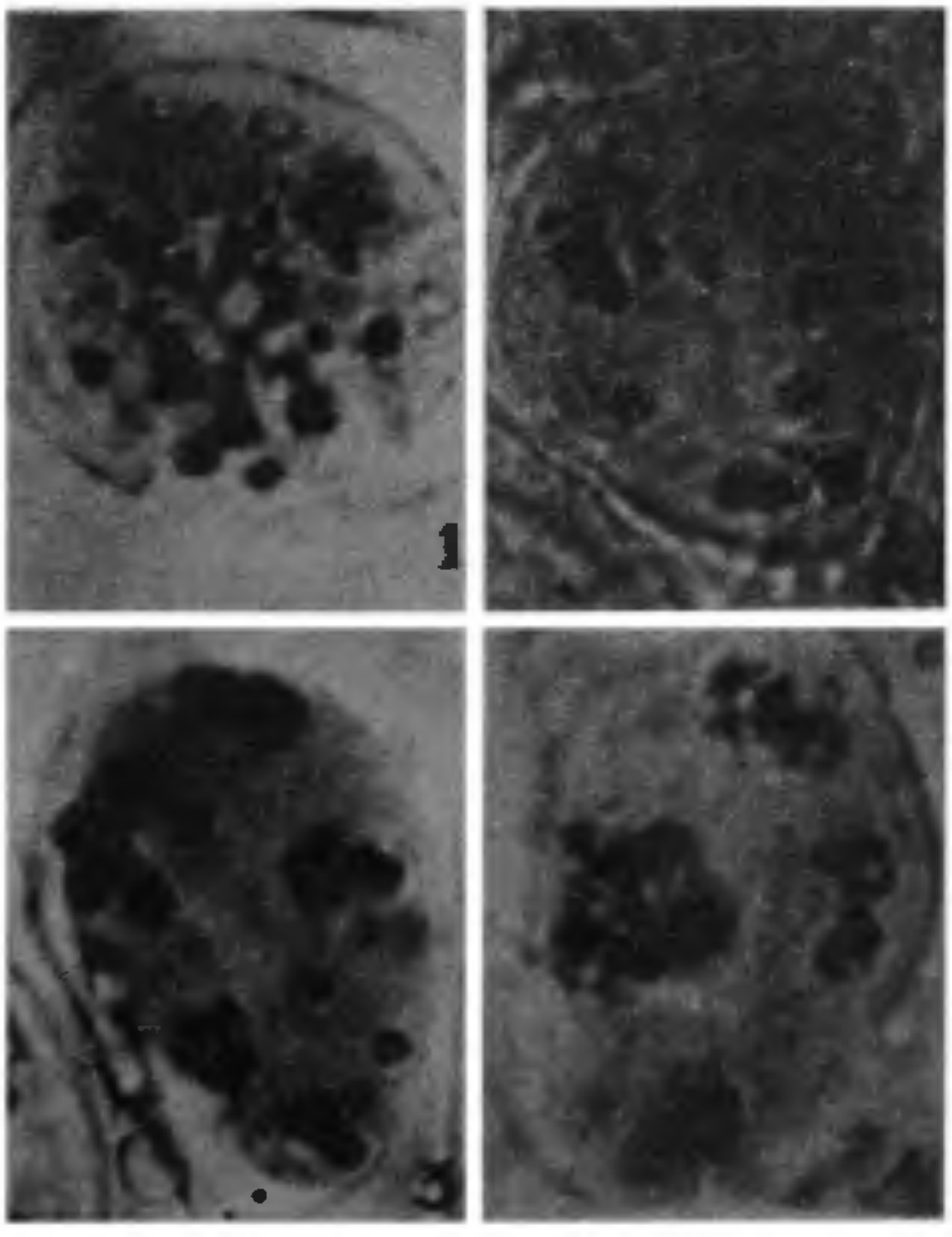

Figure 1–4 1. Univalents at diakinesis, 2. Lagging chromosomes at anaphase II, 3. Laggards and micronuclei at telophase II, 4. Polyspory.

The award of Post-Doctoral fellowship to MV by CSIR, New Delhi is gratefully acknowledged.

2 February 1983; Revised 26 July 1983.

1. Praaken, R., *Hereditas*, 1943, **29**, 475.
2. Gottschalk, W. and Baquar, S. R., *Can. J. Genet. Cytol.*, 1971, **13**, 138.
3. Sahu, G. R., Tewari, V. and Singh, R. B., *J. Cytol. Genet.*, 1981, **16**, 71.
4. Bozzini, A. and Martini, G., *Caryologia*, 1971, **24**, 307.

ENZYME ADAPTATION IN THE KIDNEY OF COBALT FED RATS.

S. V. S. RANA, V. P. AGRAWAL AND R. PRAKASH
School of Environmental Contamination and Toxicology, Department of Zoology,
D. A. V. College, Muzaffarnagar 251 001, India.

THE phenomenon of enzyme adaptation has been studied in the kidney of cobalt fed rats through histochemical parameters. Disappearance of alkaline phosphatase, acid phosphatase, 5-nucleotidase and cholinesterase from kidney after a longer exposure to cobalt and then appearance after still longer treatment has been discussed in terms of adaptation to the changed environment of cell and its organelle.

Enzyme regulation, denaturation, turnover, cycling, modification by drugs, sex, nutrition and hormonal status are well known events in cellular biochemistry[1]. In addition, influences of circadian rhythms on enzyme levels have also been recorded[2]. Most interesting and important are the spontaneous processes involved in enzyme reversal after cell injury[3]. Occurrence of such a phenomenon warrant confirmation after diverse experiments in biology and medicine. Since heavy metals are enzyme poisons, it was found appropriate to gather more information on such mechanisms during investigations on heavy metal toxicity in the laboratory animals. Present communication describes reversal of a few important enzymes *viz.* alkaline phosphatase, 5-nucleotidase and cholinesterase in the kidney of rats (*Rattus rattus* albino) fed on cobalt for considerably longer periods.

Cobalt as cobalt acetate (BDH, mol. wt. 145) was fed for 30 days by gavage (50 mg/kg body weight) each day to a group of 10 laboratory bred, 3 months old (100 ± 10 g) rats maintained on laboratory chow (Hindustan Lever Ltd., Bombay) and tap water *ad lib* under standard laboratory conditions after making primary toxicological observations[4] like LD_{60}. Another group of ten rats was subjected to the same treatment but for 60 days. Whereas twenty rats, ten with each group, provided with a cobalt free diet but same environment served as controls.

Ten rats were sacrificed by decapitation on 30 days and the rest ten after 60 days. Kidneys were carefully removed and fixed frozen in 10% neutral formaline. Paraffin and frozen sections were processed for histochemical assay of alkaline phosphatase[5], acid phosphatase[6], 5-nucleotidase[7] and cholinesterase[8]. Histopathological observations were made after staining sections with hematoxylin and eosin. Suitable reactions controls were also run simultaneously.

Prolonged treatments with cobalt induced renal atrophy and extensive tubular necrosis (figures 1,2). It inhibited all the key enzymes in rat kidney after 30 days treatment (figures 3,4), whereas activities were found elevated in the kidney of rats fed on cobalt for 60 days (figures 5–8). However, no differences in the activities of these enzymes could be observed in the kidney of normal rats after 30 or 60 days. Changes in their distribution thus noted are presented by table 1.

TABLE 1

Enzyme activity in the kidney of cobalt fed rats.

Enzyme	Control				Treatment							
					30 days				60 days			
	A	B	C	D	A	B	C	D	A	B	C	D
Alkaline phosphatase	+	+	±	−	±	−	−	−	++	+	−	−
Acid phosphatase	+	++	−	−	−	±	−	−	+	+	−	−
5-Nucleotidase	++	+	−	−	$+^n$	$+^n$	−	−	++	+	±	−
Cholinesterase	−	−	+	−	−	−	−	−	±	±	+	−

++, strong activity; +, moderate; ±, dull; −, no acitivity; $+^n$, moderate activity only in nucleus. A = Proximal convoluted tubules, B = Distal convoluted tubules, C = Glomeruli; D = Medulla.