GENETICAL AND TRANSFORMATION STUDIES IN RICE, *Oryza sativa* Linn.

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C E R T I F I C A T E

Certified that this thesis entitled "**GENETICAL AND TRANSFORMATION STUDIES IN RICE, ORYZA SATIVA Linn.**" embodies the results of a piece of bona fide research work carried out as part fulfilment for requirements of the Degree of Doctor of Philosophy in Botany of the University of Calicut by **Mr. Santhosh Lal. P.S.**, under my guidance and supervision and that no part of the thesis has been submitted for any other degree.

I, further, certify that such help or source of information, as has been availed of in this connection, has been duly acknowledged.

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(K.PAVITHRAN)

D E C L A R A T I O N

This is to declare that the thesis entitled **"Genetical and transformation studies in rice, Oryza sativa Linn.,"** submitted by me for the degree of Doctor of Philosophy in Botany of the University of Calicut has not formed the basis for the award of any other degree/ diploma.

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C O N T E N T S

INTRODUCTION

Exploitation of the vast potentialities of the crop plants like rice or wheat depends on continuous genetic upgradation of the crop. This is possible by generating new variabilities and exploiting them for crop improvement with full understanding of their genetical behaviour. However, rice genetics is still considered lagging behind that of several other crops. Rice exhibits a highly complex picture of gentic systems for the control of morphological characters. The genetic systems consist of basic genes, complementary genes, duplicate factors, epistatic and hypostatic genes, inhibitory and anti-inhibitory genes. The interactions of these genes with different combinations lead to various genetical ratios ranging from monogenic to pentagenic interactions to control different morphological characters. It is estimated that at least nearly 10% of the total genes known in rice exhibit pleiotropism. The studies on interrelationships of genes or linkage associations of genes remain grossly incomplete in several respects. So is the case with identification and location of mutant genes so far known in rice. The other significant strategy adopted for crop improvement was induced mutation for useful and desirable mutant characters/plants. Survey of studies on mutation have also revealed several lacunae in terms of genetical analysis of mutant characters in rice. The FAO and IAEA, the international co-ordinating agencies for mutation

breeding researches, have reviewed 20 years of their co-ordination. It is stated that before 1961 the Asia-Oceania farmers have received only 4 mutants in total, and till 1985 nearly 300 mutant cultivars have been released to them with a maximum of over 100 during the period 1976-1980. The position of released mutants in rice from India appears to be very meagre, less than 10% of the total rice mutants released so far. Mutation breeding has advanced much in China in the past two decades in various crops. Out of the released mutants, it seems, less than 15% alone have been used in the cross breeding programmes. Estimates indicate that gamma-induced mutants are comparatively high among those released until 1985, amounting to 75%, whereas chemically induced mutant cultivars released during the same period are only 5%. Since 1986 number of cultivars obtained through mutation breeding is rapidly increasing in the case of cereals and legumes. In India until 1985 only six rice mutants have been released out of 47 mutants recorded in different crops (Chopra and Sharma, 1985). An excellent network of collaborative mutation breeding has been effectively organised in China in contrast to the situation prevailing in India and other countries. In the wake of recent advances in genetics, mutation studies are to be approached from different angles so as to elicit adequate knowledge on generating genetic variabilities or on the probable alterations/transformation

likely to occur in the genetic systems in response to various mutagenic treatments. In the present context, transformation refers to genetic transformation of alleles induced by mutagens in pollen grains, which, when used in crosses, can be identified only through altered/transformed genetical ratios in relation to the morphological characters concerned. Such studies are lacking in rice.

In view of the above facts the need for advancing genetical analysis in rice including that of induced mutations was well realised. The present study was undertaken, hence, to fill in this lacunae in our genetical understanding of the rice plant. The following are the specific objectives thus envisaged in the present investigation.

1. To study and compare the patterns of inheritance of morphological characters in a normal cross and in the same cross combination wherein the irradiated pollen grains were used for hybridization.

2. To study genic interrelationships of morphological characters studied in the above crosses for comparative analysis in relation to linkage and pleiotropic situations, if any.

3. To study the mutagenic effectiveness and efficiency of EMS in the rice variety Japan Violet, used as the male parent in the

above corsses.

4. To study the inheritance of morphological mutants induced by EMS treatments.

5. To study the genic interrelationships of certain mutant morphological characters induced by EMS treatments.

6. To study morphometric variations due to micromutations induced by EMS treatments.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

The present study refers to mutagenesis, gene transformation through irradiated pollen grains, inheritance and interrelationships of morphological characters in rice. Relevant literature on the areas of mutagenesis and transformation, inheritance and interrelationships of morphological characters with particular reference to linkage and pleiotropy are reviewed below under different heads.

A. NATURAL MUTAGENESIS

The occurrence of natural or spontaneous mutations has been reported in rice by many workers as reviewed below under appropriate heads.

1. Chlorophyll mutations

The occurrence of spontaneous chlorophyll deficiencies has been reported in rice as albinos (Morinaga, 1927, 1932; Codd, 1934; Ramiah and Ramanujam, 1935; Pal and Ramanujam, 1941), lethal yellow (Imai, 1935; Ramiah and Ramanujam, 1935; Kadam, 1941), zebra white (Kadam, 1939, c.f. Ramiah and Rao, 1953) and lethal zebra yellow (Ramiah and Parthasarathy, 1938).

Spontaneous occurrences of stunted yellow, tip brown yellow, white virescent, light green virescent, golden yellow virescent (Kadam, 1941), spontaneous yellow virescent, green and yellow striped, green and white striped patterns (Ramiah and Ramanujam, 1935) and green and white persistent (Pal and Ramanujam, 1941) have also been reported earlier.

Natural occurrences of green and white variegation (Imai, 1928; Ramiah and Ramanujam, 1935), green and yellow fading (Morinaga and Kondo, 1932, c.f. Ramiah and Rao, 1953; Ramiah and Ramanujam, 1935), variable green and white (Imai, 1928) and spotted leaf mutants (Omura and Iwata, 1972; Iwata and Omura, 1975) have also been reported.

2. Plant type/plant height mutants

Spontaneous plant type mutations reported include "lazy" mutation in rice (Jones and Roy, 1938) and different types of dwarf mutants (Parnell et al., 1922; Sugimoto, 1922; Akemine, 1925; Jones, 1933; Ramaih, 1933; Kadam, 1937; Nandi and Ganguli, 1941).

3. Foliar and panicle mutations

Spontaneous mutants for leaf characters such as rolled leaf (Nagai, 1926), narrow leaf, twisted leaf (Isshiki, 1933), liguleless

and auricleless leaf (Jones, 1933; Chiapelli 1936) and panicle mutants such as long glume (Kadam et al.,1941), open hull and male sterile (Takeda, 1987) have also been reported in rice.

4. Sterile mutants

Various types of spontaneous sterile mutants have been reported by different workers and these include paleaceous sterile (Nagai, 1926), male sterile (Ishikawa, 1929; Kadam, 1932; Miyazava, 1935; Ramanujam, 1935 and Srinivasan, 1937), awned sterile and staminodal sterile (Nagai, 1926), barren sterile (Anandan and Krishnaswamy (1934), pistillate sterile, slender semi-sterile and sterile shrivelled stamens (Miyazava, 1932), semi-sterile-ever-splitting (Terao, 1921), asynaptic sterile (Ramanujam and parthasarathy, 1935), semi-sterile large grained (Kegawa, 1939) and female sterile mutant (Razzaque, 1975).

Shi (1986) reported a spontaneous photosensitive genic male sterile and Rao and Rao (1986) reported a spontaneous multipistillate mutant in rice.

B. INDUCED MUTAGENESIS

Various types of physical and/or chemical mutagens have been used by different workers for induction of mutations in plants since

Muller (1927) and Auerbach et al. (1947). Studies relevant to mutagenesis in rice induced by physical and chemical mutagens are reviewed below.

1. Physical mutagenesis

The physical mutagens in use are ionizing radiations viz; X-rays, gamma-rays, beta-rays, fast and thermal neutrons, deutrons and alpha particles. Laser lights and ultra-violet rays (non-ionizing) have also been frequently used for the induction of mutation. Radio-isotopes of phosphorus, ^{16}P and sulphur, ^{32}S, as physical mutagens are also used.

Effects of ionizing radiations in plants have been studied in detail by Sparrow, et al. (1958), Zimmer (1959) and Sparrow and Evans (1961). Fast and slow neutrons have been reported to be more effective and efficient than X-rays or gamma rays (Adams and Nilan, 1958). Neutrons have been reported to produce more uniform biological effects on seeds (Mackey, 1951,1952; Ehrenberg and Nybom, 1954; Caldecott, 1955 and Beard et al., 1958).

Delay in germination has been reported by several authors due to high doses of radiations (Goud, 1967 and Kumar and Mallick, 1986), while Myttenare et al. (1965) and Goud, (1967) have reported that

germination is little affected by radiations. Reduced germination with gamma-rays has been reported by Kumar and Mallick (1986). Survival of seedlings was generally found to decrease with increasing doses of radiation and chemical mutagens (Rao and Ayangar, 1964; Siddiq, 1967; Siddiq and Swaminathan, 1968; Swaminathan et al., 1970 and Ganashan, 1970; Nair, 1977 and Shobha, 1993).

Singh (1970) reported high frequency of viable mutations in M_2 generation. Kawai (1968) reported that more than 1000 mutant types were induced in a single variety by radiation treatment. About 1400 mutant lines with stable viable characters from gamma-ray treatments of growing plants (Bekendam, 1961; Tanaka, 1969a), 478 types in other instances (Kawai, 1963a) and yet another 238 types with ^{32}P (Masima and Kawai, 1958), 287 types with X-rays (Kawai, 1963b) and 66 types with gamma rays (Swaminathan et al., 1970) have been reported earlier.

Many viable mutants of economic value have been reported in rice (Bora and Rao, 1958; Hu et al., 1960; Bekendam, 1961 and Li et al., 1962 and Awan et al., 1977). However, only few of these viable mutants are of use in breeding programme(Gustaffson and Gadd, 1966; Tanaka, 1969b and Marie, 1970). The first radiation induced productive rice mutant, 'Reimei' was released in Japan in 1966 as a national registered variety (Futsuhara et al., 1967).

Higher frequencies of chromosomal aberrations and genic

mutations have been reported earlier (Ehrenberg and Nybom, 1954 and Nybom, 1956; Caldecott, 1958). Radiation induced chromosomal aberrations have been reported in rice by many (Carpena and Ramirez, 1960; Chao and Chai, 1961; Shastry and Ramiah, 1961; Hsieh, 1962; Siddiq, 1967; Siddiq and Swaminathan, 1968 and Singh, 1970). The earliest reports on irradiation of rice with ionizing radiations are those of [illegible], Nakamura (1918) and Komuro (1919), who also reported the stimulating effects of X-rays on yield. Ramiah and Parthasarathy (1936, 1938) were the first in India to use X-rays for producing mutations and chromosomalaberrations in rice. However, well established mutations reported in rice by physical mutagenes are reviewed below:

a. Chlorophyll mutations

Shastry and Ramiah (1961) reported chlorophyll deficient sectors in M_1 plants of rice following irradiation with X-rays, thermal neutrons and beta particles. Horvat (1961), Siddiq (1967) and Tanaka (1970) reported chlorophyll deficient plants in haploid rice derived from chronic gamma irradiation.

Several induced mutations by physical mutagens have been reported by others (Imai 1935; Kanna, 1935; Bora and Rao, 1958; Soriano, 1959; Kawai, 1963a,b). Among these mutants albino appears

predominant (Bekendam, 1961; Chao and Chai, 1961; Kawai, 1966 and Basu and Basu, 1969) possibly, due to the large number of loci governing this phenotype (Swaminathan et al ., 1970). Ramiah and Parthasarathy (1938) reported X-ray induced albino, lethal yellow, lethal yellow with green sheath, zebra marked, zebra yellow (lethal), zebra white, yellow virescent, green and white striped, green and yellow striped, blotched white and rolled leaf with low fertility.

Chlorophyll mutants including albinos, viridis and striped mutants have been reported by many workers (Swaminathan, 1966; Siddiq, 1967; Siddiq and Swaminathan, 1968; Viado, 1968; Ismail, 1969; Singh, 1970; Vimala and Reddy, 1972; Ram, 1973; Chaudhury et al., 1986 and Maekawa et al., 1990).

b. Plant type/Plant height mutations

A good number of gamma ray induced reduced culm length (Kawai et al., 1961; Kawai, 1967; Miah and Bhatti, 1968; Tanaka, 1968, 1969a; Haq et al., 1971; Ram, 1974; Gangadharan and Misra,1976; Rutger et al., 1976; Mackill and Rutger, 1979) and X-ray induced short culm/ dwarf mutants (Li et al., 1968; Majumdar, 1969; Narahari, 1969; Okuno and Kawai, 1978a,b and Dasgupta, 1979) have been reported in rice earlier. Mutants with reduced plant height are more frequent than those with increased plant height (Kawai,1962 and Tanaka, 1968). Dwarf mutants varied in their appearance from extreme grassy dwarfs

to semi-dwarf (Gustaffson and Gadd, 1966). Ghosh and Bhattacharya (1978) reported a semi-dwarf mutant with midribless leaves using gamma rays. Narahari (1969) reported an X-ray induced dwarf, non-shedding rice with changed husk colour and virido-alba traits. Dominant dwarf mutants have also been reported in rice (Kumar and Sreerangaswamy, 1987; Singh et al., 1989a). Semi-tall-early maturing-high yielding mutant induced with gamma rays and EMS has been reported (Singh and Singh, 1991). Early matuaring, short culm and finer grain mutants (Schwe and Shaikh, 1993), mutants with reduced culm number (Takamure and Kinoshita, 1993a), mutants with increased culm number, short panicle and brown spotted leaves (Takamure and Kinoshita, 1993b) and brittle culm mutant (Tanaka, 1967; Takahashi et al., 1968; Iwata and Omura, 1977, Takamure and Kinoshita, 1993b) have also been reported in rice..

c. High tillering mutants

Nair and Ninan (1973) reported high tillering mutants using gamma rays. Semidwarfs with increased tillering have been reported by several others (Bose, 1968; Miah and Bhatti, 1968; Misra et al., 1971; Reddy and Reddy, 1973 and Reddy et al., 1975). Bhan and Kaul (1974) reported a rice mutant with increased plant height with reduced panicle length, number of grains per panicle and tillering, late maturing and reduced yield with increased 1000 grain weight.

Ismail and Ahmed (1979) reported a mutant with increased tiller number. Takamure and Kinoshita (1985) reported occurrence of reduced culm number mutants with gamma-ray treatment.

d. Leaf mutations

Various types of leaf mutations with size, shape, arrangement, pubescence and colour have been reported in rice earlier. Narrow leaf mutants are most frequent (Hsieh, 1962; Shastry, 1965; Siddiq, 1967; Tanaka, 1968; Singh, 1970 and Swaminathan et al., 1970). Boot leaf mutants have been described differently like incurved lamina or rolled leaf (Narahari and Bora, 1963; Tanaka, 1968; Singh, 1970). Ghosh and Bhattacharya (1978) reported gamma ray induced semi-dwarf mutant with new type of leaf arrangement. Ramiah and Parthasarathy (1938) reported mutants with narrow leaf and malformed spikelet sparsely arranged on the panicle. Venkatanandachari (1963) obtained mutants for leaf width and Roy and Jana (1975) a rice mutant with broad leaves. Majumdar (1980) isolated mutants with forked leaf, extra leaf in the panicle and chlorina leaf.

e. Early/late flowering mutants

Early heading mutants in rice have been reported earlier using gamma rays (Kawai, 1966; Miah and Bhatti, 1968; Tanaka, 1969a; Miah et al., 1970; Ram, 1974; Ismachin and Mikaelsen, 1976a,b; Rutger et

al., 1976; Kaul, 1978; Qin, 1987 and Ismail and Ahmad, 1979), X-rays and neutrons (Ree, 1968; Viado, 1968; Mikaelsen et al., 1971; Hu, 1973 and Dasgupta, 1979) and ^{32}P (Majumdar, 1965,1969; Kawai and Narahari, 1971). Other mutants such as early maturing with short stature (Mallick and Bairagi, 1979) with reduced yield (Majumdar, 1965, 1969), with drought resistance (Singh and Sinha, 1985) and with increased grain per panicle and 1000 grain weight (Rao and Ayengar,1963) have also been reported.

f. Panicle /grain mutations

Mutations affecting panicle characters have also been reported in rice (Rao and Ayangar, 1963; Tanaka, 1968; Singh, 1970; Basu and Basu, 1970; Das et al., 1979). Non-shattering mutants have also been reported (Majumdar, 1969 and Nair, 1972).

Neutron /gamma ray induced spikelet abnormalities (Narahari and Bora, 1963 and Ganashan, 1970), mutants with long sterile glume (Kadam, 1941; Narahari and Bora, 1963; Ganashan, 1970 and Swaminathan et al., 1970), open spikelet mutant (Narahari and Bora, 1963; Siddiq, 1967 and Gill et al., 1969), claw hull and triangular hull types (Chandraratna, 1964), mutants with short grains (Masima and Kawai, 1958; Kawai, 1962 and Ganashan, 1970), awned mutant (Siddiq, 1967 and Omura and Satoh, 1984), small grain mutant

(Takamure and Kinoshika, 1994) and environmental induced male steriles (Shen et al., 1994) have also been reported in rice.

2. Chemical mutagenesis

Induction of mutation by chemicals has been demonstrated with mustard gas (Auerbach et al., 1947), and with epoxides and imines (Rapoport, 1948, c.f. Muzynski and Guzewski, 1986). However, alkylating agents have been found most efficient in a wide array of organisms (Auerbach, 1961). Among this group EMS appears to be more efficient (Swaminathan et al., 1969). Heslot et al. (1959) and Ehrenberg (1960) were the first to demonstrate the mutagenic efficiency of EMS.

a. Germination and viability

Ganashan (1970), Kaul and Bhan (1971), Ramesh (1984), Mohanan (1988) and Shobha (1993) reported that seed germination is greatly affected by EMS. However, seedling survival was also affected with EMS treatment (Swaminathan et al., 1970; Shobha, 1993). Xu (1987) reported that seed viability and germination decreased with DMS treatment.

b. Leaf mutations

EMS induced chlorophyll mutations (Swaminathan, 1966; Siddiq, 1967; Siddiq and Swaminathan, 1968; Viado, 1968; Ismail,

1969 and Singh, 1970), albinos and viridis mutants (Vimala and Reddy, 1972; Iwata and Omura, 1978), NMU induced chlorophyll mutations (Chaudhury et al., 1986, 1987), striped mutant with EMS (Maekawa et al., 1990) and N-nitroso-N-Methylurea induced spotted leaf mutants (Maekawa et al., 1990) have also been reported in rice.

c. Plant type/ plant height mutations

Short statured, early flowering/maturing mutants with EMS treatments (Mallick and Bairagi,1979), dwarf mutant with resistance to lodging and with high yield by using EMS and DES (Kaul, 1978) and mutants for plant height, number of panicles per plant, number of spikelets per panicle and grain size by using ethylimine (Okuno and Kawai, 1978a,b) are known in rice. Further, EMS induced dwarfs have been reported (Mani et al., 1986) and dwarfs with increased yield and late flowering have been reported by many (Marie, 1967, 1970; Li et al., 1968; Siddiq and Swaminathan, 1968; Hu et al., 1970; Swaminathan et al., 1970; Misra et al., 1971) and also dominant brittle culm, mutant (Singh et al., 1994).

d. Tillering mutants

EMS induced high tillering mutants with changes in grain size

and yielding capacity have been reported in rice (Rao and Siddiq, 1977). Mishra and Sahu (1962) reported Maleic hydrazide induced mutants for high tiller number. Ethylimine and DES induced-tillering mutant has been recorded in Oryza rufipogon (Sampath and Jachuck, 1969).

e. Early/late flowering mutants

Early flowering/maturing rice mutants induced with EMS treatments have been reported by many workers (Marie, 1967; 1970; Siddiq and Swaminathan, 1968, Li et al., 1971; Misra et al.., 1971; Miah and Awan, 1971; Sharma et al., 1974; Reddy et al., 1975; Roy and Jana, 1975 and Mallick, 1978). Misra et al. (1971) reported late flowering mutants with EMS.

f. Panicle/Spikelet/Grain mutations

Panicle/spikelet/grain mutations reported in rice are EMS, DES and NMU induced for grain mutants (Reddy and Reddy, 1973, 1974; Kaul, 1978; Chaudhuri et al., 1986), EMS and NMU-induced mutants with high panicle number and panicle weight (Das et al., 1979), EMS-induced high yielding mutant with small grain, high tillering and longer panicles (Hajra et al., 1986), NMU-induced endosperm mutants (Satoh et al., 1984) and phylogenetic mutants for grain types in japonica (Chaudhury et al., 1986), EMS-induced high yielding mutants (Marie,

1967, 1970 and Kaul, 1978), EI, DES and NMU-induced early flowering/late flowering and high yielding mutants (Misra et al., 1971) and EMS-induced short culm high yielding mutants (Sreedharan, 1979).

g. Sterile mutants

Male sterile mutant from EMS treatment has been reported in rice (Pavithran and Mohandas, 1976a; Shobha, 1993). Ethylimine has been found effective to induce male sterile mutants with no pollen grains (Fujimaki et al ., 1977; Hiraiwa and Fujimaki, 1977 Singh and Ikehashi, 1981 and Ko and Yamagata, 1980 and 1987). Studies on male steriles have been reviewed elsewhere.

3. Dosage/Pre-treatment/Mutagenic effectiveness and efficiency

a. Dosage

Effectiveness and efficiency of different dosages of mutagens in inducing mutations has been reported by many workers. Mutagenic effectiveness and efficiency decreased with increasing dose of mutagens (Siddiq, 1967; Siddiq and Swaminathan, 1968; Swaminathan et al., 1970 and Singh, 1970). A linear relationship between decrease in pollen and seed fertility based on dosage has also been reported (Beachell, 1957; Chang and Hsieh, 1957; Yamaguchi, 1964; Siddiq, 1967

and Singh, 1970). Nair and Ninan (1973) reported increased tillering with increased dosage of mutagen and 30-35 kr gamma rays have been reported to be the effective dose range for rice (Haq et al., 1970 and Ram, 1973).

b. Pre-treatment

The soaking of seeds with distilled water before exposing to the mutagen for specific duration has been reported widely in chemical mutagenesis (Misra et al., 1971; Vimala and Reddy, 1972; Nair, 1972; Gangadharan et al., 1975; Kaul, 1978 and Nayar and Ninan, 1979, 1983). Duration of pre-treatment varied from 4 hours (Gangadharan et al., 1975) to 24 hours (Vimala and Reddy, 1972). Nair (1978) reported the enhancement of the mutagenic effects of NMU in rice through alteration of the period of pre-soaking. Possibilities of enhancing mutation frequency and specificity have also been reported (Siddiq, 1969). Increase of radio sensitivity following pre-soaking in water has also been documented (Ota et al., 1957; Matsuo et al., 1958; Yamagata and Syakudo,1960 and Shastry and Ramiah, 1961).

Pre-soaking increased sensitivity to chemical mutagens such as diethyl sulphate, ethyl methane sulfonate and nitrosomethyl urea (Ando, 1968; Ismail, 1969; Ayengar et al., 1969; Swaminathan et al., 1970; Siddiq et al., 1970). Swaminathan et al. (1970) reported that dehulled seeds responded drastically after pre-soaking for 18-22

hours. Kawai (1962) found increase in mutation frequencies with pre-soaking prior to X-ray or gamma ray treatment and Yamaguchi (1958) reported maximum mutation frequency at 58 to 60 hours of soaking and a decrease thereafter. Ismail (1969) found higher mutation frequency with prominent peaks at 21 to 24 hours with seeds pre-soaked beyond 20 hours.

Pre-soaking of seeds for different periods shows a distinct alteration in the spectrum of chlorophyll mutations. Siddiq et al., (1970) reported EMS induced albino and NMU induced viridis mutants with higher frequencies with short periods of pre-soaking.

The positive effect of pre-soaking treatments of seeds with growth regulators and other chemicals have been well documented. Treatment with ascorbic acid stimulated seed germination (Chinoy, 1967) and seedling growth (Chinoy et al., 1957; Chinoy 1967, 1968; Chinoy et al., 1970; Mehta and Chinoy, 1978; Asthana and Srivastava, 1975, 1978). At molecular level, it activates the biosynthesis of RNA in seedlings of Sinapsis alba (Schopfer, 1967) and Cicer arietinum (Chinoy and Saxena, 1971). Ascorbic acid activates the biosynthesis of total nucleic acid, protein and consequent acceleration of cell division and other processes (Chinoy, 1962, 1969; Chinoy and Mansuri, 1966). Studies on the use of Vit. C as a pre-soaking agent for mutagenic treatments are scanty. However, this approach appears desirable.

c. Mutagenic effectiveness and efficiency

Both mutagenic effectiveness and efficiency are the essential pre-requisites for the usefulness of any mutagen in plant breeding. Efficient mutagenesis is the product of desirable changes unassociated with undesirable changes (Konzak et al., 1965). The most effective agent may not always be the most efficient one. Efficient treatments are essential for economical use of mutagens as tools for direct improvement of crops (Ehrenberg and Nybom, 1954; Bora, 1961 and Evans, 1962).

Loveless and Howarth (1959) reported that ethylmethane sulfonate has a higher mutagenic efficiency than radiations. Gaul (1962, 1963) observed that the maximum mutagenic effectiveness of EMS is much greater than that for the mutagens applied to seeds.

Siddiq (1967), Siddiq and Swaminathan (1969) and Swaminathan et al. (1970) reported that among radiations, neutrons are the most effective. Soriano (1968) found fast neutrons are more effective than thermal neutrons. The high mutagenic potency of ethylmethane sulphonate and ethylimine in comparison with sparsely ionizing radiations has been documented (Kawai and Satoh, 1965; Swaminathan, 1966; Matsuo and Yamaguchi, 1967).

Siddiq and Swaminathan (1968) found gamma rays more effective than EMS. High mutagenic effectiveness of nitroso-methylurea has been reported (Swaminathan, 1966; Siddiq and Swaminathan, 1968 and Singh, 1970). Mutagenic efficiency and effectiveness of EMS, DES and gamma rays in rice has been reported by Kaul and Bhan (1977). Swaminathan et al. (1970) concluded that chemicals have no particular advantage over ionizing radiations with reference to either mutation frequency or spectra.

According to Auerbach (1961) alkylating agents are more capable of inducing mutations among known chemicals. Within the alkylating group, monofunctional agents in general and EMS in particular, appear to be more efficient (Swaminathan et al., 1962). Fujimoto and Yamagata (1982) reported that effectiveness and efficiency of chemical mutagens are in the order NEUA > NMUA > EI > NMUT > EMS, and their results showed NEUA as the best mutagen in practical plant breeding. Misra (1990) showed gamma ray and maleic hydrazide as more effective than EMS and sodium azide in rice. Konzak et al., (1965) pointed out that among the chemical mutagens EMS possesses many properties favourable to high mutagenic effectiveness and efficiency.

The efficiency of a mutagen also depends on the physiological state of the tissue, structure of the tissue, genetic composition, capacity for growth, synthetic ability and capacity for tissue repair. Dry seeds are not affected by diethylsulphate (Cervigni and

Belli, 1962 and Heiner, 1963). The mutagenic effects also vary with treatment conditions like temperature and concentration of the mutagen (Ehrenberg et al., 1956; Ehrenberg and Gustaffson, 1957; Ehrenberg, 1960; Konzak et al., 1961, 1964; Heiner, 1963; Nilan et al., 1964).

The presence of hull in rice reduces the effectiveness of the mutagen applied (Mikaelsen and Navarathna, 1968), probably due to slow diffusion of chemical mutagens (Siddiq and Swaminathan, 1968 and Ayengar et al., 1969). Mutation frequencies in M_2 and M_3 population seem to be dependent upon genotype and previous selection history (Bhatia, 1973; Gupta, 1976; Kaushik, 1974).

Relative biological effectiveness (RBE) of various mutagens has also been reported. Fuji (1962) reported the RBE of X-rays and gamma rays to be approximately the same. Kawai (1968) showed varying RBE values for the different criteria such as germination, seedling growth, survival, fertility and mutation frequency. Matsumura (1964) calculated the RBE value for M_1 seed fertility and M_2 chlorophyll mutation frequency. Siddiq (1967) observed that the RBE value to be lowest for seed fertility and highest for seed germination.

C. STUDIES ON TRANSFER/TRANSFORMATION OF GENES THROUGH IRRADIATED POLLEN GRAINS

1. Transfer/transformation of genes with irradiated pollen grains

The use of irradiated pollen to transfer single gene from the pollen parent to the seed parent in cross pollination was first proposed by Pandey (1975). However, transfer of transformed genes via pollen irradiation has not been given much attention.

Artificial cross pollination using pollens treated with ionizing radiation has been carried out for interspecific crosses (Stettler, 1968), for mutation breeding (Gager and Blakslee, 1927; Goodspeed, 1929; Catcheside, 1937; Stubbe, 1937; Barton, 1954; Brewbaker and Emery, 1962; Yamagata and Syakudo, 1963; Donini et al., 1970; Devreux et al., 1972), for haploid plant production (Lacadena, 1974) and for parthenogenesis (Cao et al., 1979). Pandey (1975, 1978, 1980a, b, 1981) in a series of papers reported successful transfer of gene/s through radiation treatment of pollen grains in Nicotaiana. Several workers have transferred a part of the male nuclear complement rather than the whole male nucleus from one plant to another by the use of irradiated pollen grains: (in Nicotiana rustica, Jinks et al., 1981; in Barley, Caligari et al., 1981, 1984; in Zea maize, Pandey 1983; in barley, Powell et al., 1983; in Triticum aestivum, Snape et al., 1983; in Tomato Zamir 1983; in Brascica juncea, Banga et al., 1984; in Pisum sativum, Davies, 1984; and in Capsicum, Daskalov, 1984). Virk et al., (1977) reported that in Nicotiana rustica, single genes can be transferred from irradiated pollen to T_1 progeny.

Pandey (1975) suggested that irradiated pollen would mediate transfer of limited genetic materials, instead of the complete genome from the pollen parent to the female parent, the progeny of which might be considered matromorph arising from diploid parthenogenesis. Thus in a maternal background, a few paternal characteristics from the pollen parent were apparently incorporated and expressed in the M_1 plants and the eggs were said to be transformed. The biological basis to these assumptions seems to lie with the fact that pollen germination and pollen tube growth are highly tolerant to irradiation (Vassileva-Dryanovska, 1966; Gillissen 1978), and that despite extensive nuclear aberrations caused by radiation, fusion of the fragmented paternal genetic materials with the egg nuclei was still possible (Brewbaker and Emary, 1962), as the nuclic acids are not functionally damaged and physically incapacitated, and normal fertilisation occurs in various crosses (Jinks et al., 1981; Caligari et al., 1981; Pandey, 1983; Pandey and Phung, 1982). Thus the irradiated pollen functioned as the source of donor DNA fragments as well as a vector for delivering the genetic fragments to the embryosac.

Various mechanisms were proposed for explaining the phenomenon of limited gene transfer. Pandey (1976) put forward a mechanism of gene transfer which involved incorporation of paternal DNA fragments into the egg nucleus by psuedo fertilisation followed by a

parthenogenetic doubling of the egg genome. Werner et al. (1984), established that the irradiated progenies were the products of a conventional fertilisation mechanism through the cytological studies of an interspecific cross in Nicotiana, and also showed the occurrence of aneuploids and chromosomal rearrangements. Snape et al. (1983) proposed a 'meiotic sieve' acting at or after M_1 meiosis to limit transmission of paternal genes. They suggested that either damaged paternal chromosomes are selectively lost during meiosis or their presence results in pollen or zygote lethality. Sarigorla et al. (1987) stated the excess number of maternal types are due to the result of radiation damage and subsequent selection of irradiated paternal genome.

Inheritance of the transferred characters has also been attempted and showed that in M_2 the number of maternal types are increased than in F_2 (Powell et al., 1983; Caligari et al., 1981; Werner and Cornish, 1985). However, a detailed study of inheritance has not yet been done so far.

2. Dosage for pollen irradiation

For overcoming the incompatibility as well for gene transfer, high doses of radiation (100 kr) were used by Pandey (1975). Sub-lethal doses of radiation varying between 2 kr and 30 kr have been used in Indian mustard (Banga et al., 1984) while 0.5 kr to 20 kr

gamma rays in rice (Chin and Gordon, 1989a,b), 2 to 5 kr gamma rays in wheat (Snape et al.,1983), 10 to 20 kr in Nicotiana(Jinks et al., 1981; Werner and Cornish, 1985) and 20 kr gamma ray in tomato (Zamir, 1983) have been used earlier.

Chin and Gordon (1989a) reported that at 20 kr there was no germination of F_1 seeds in rice and also more shrivelled seeds were produced as the dosage of the radiation increased. Werner et al. (1984) reported that the number of viable hybrid seeds per capsule was decreased with increase of radiation dosage for pollen grains and also that length of chromosome increased or decreased in hybrids obtained from irradiated pollen grains.

D. GENETICAL STUDIES ON MORPHOLOGICAL CHARACTERS AND MUTANTS IN RICE

1. Studies on inheritance of morphological characters

Presence/absence of anthocyanin pigmentation in rice has received considerable attention of different workers. Anthocyanin pigmentation does occasionally show genetic association with characters like spikelet sterility, vigour and even yield (Ramiah, 1935).

Parnell et al. (1917) reported that the presence of anthocyanin

pigmentation in any part of the plant is controlled by two complementary genes. Kadam and Ramiah (1943) designated it as C and A corresponding to the chromogen base and its oxidizing enzyme. Besides the basic genes, there are other genes which localise pigmentation in particular organs, intensifying or diluting the pigment and producing various pigment patterns (Ramiah and Rao, 1953). Takahashi (1964) and Kinoshita (1984a) attributed expression of anthocyanin pigmentation to complementary interaction of three genes C A and P. Both C (chromogen) and A (activator) comprising of multiple allelic series, 10 at C locus and 6 at A locus, and the localisation gene P with 4 alleles control pigmentation in plant parts in rice. Takahashi (1964) and Kinoshita (1984a) established the existence of CAP genic system in japonica and Setty and Misro (1973) and Pavithran (1986) established the same in indica rice. Inheritance patterns of anthocyanin in different plant parts relevant to the present study are reviewed below.

a. Leaf axil pigmentation

Generally leaf axil pigmentation is simple dominant over green and is intimately associated with colour in stigma (Ghose et al, 1960). Simple dominance of purple leaf axil has been reported by many workers (Nair, 1958; Butany et al., 1959; Ghose et al., 1960; Panda, 1962; Misro, 1963; Saran and Srivastava, 1969; Setty and Misro, 1973 and Annie, 1986). A digenic complementary ratio 9:7 (Nair, 1958;

Iyer, 1959; Ghose et al, 1960; Panda, 1962; Misro, 1963 and Annie, 1986) and trigenic complementary ratios 45:19 (Panda, 1962) and 54:10 (Misro and Sastry, 1962; Rao 1965, Setty and Misro, 1973; Dhulappanavar, 1973b) have also been reported.

An inhibitary ratio 27:229 has been reported earlier (Dhulappanavar, 1981) and another ratio 567:457, where three of the four complementary genes interact with the basic gene to produce pigmentation has also been reported (Dhulappanavar 1975a), while a tetragenic complementary ratio 162:94 has been reported by others (Misro, 1963; Ghose et al., 1960; Dhulappanavar 1973d; Dhulappanavar et al., 1973b), weherein two of the three complementary genes with a basic gene are responsible for anthocyanin pigmentation.

b. Leaf sheath pigmentation

Purple leaf sheath has been reported as dominat over green and complementary factors are involved for pigmentation and its expression is suppressed often by inhibitory factors. Various genetical ratios have been reported for inheritance of leaf sheath pigmentation: 3:1 (Parnell et al., 1917; Hector, 1922; Mitra et al., 1928; Kuang et al., 1946; Ramiah and Rao, 1953; Shafi and Khan, 1958; Butany et al., 1959; Ghose et al, 1960; Uzir, 1961; Misro and Kulkarni, 1965; Sastry and Reddy, 1967; Kolhe and Bhat, 1979;

Eurotor, 1986; Annie, 1986), 9:7 (Parnell et al., 1917; Hector, 1922; Chao, 1928; Ramiah and Rao, 1953; Dhulappanavar, 1973a; Dhulappanavar and Hiremath, 1974a; Pavithran, 1977; Tripathi and Rao, 1979; Annie, 1986, Sukeskumar, 1990), 54:10 (Richharia *et al.*, 1960; Dhulappanavar and Mensinkai, 1970; Dhulappanavar, 1973 c, d; Dhulappanavar and Hiremath, 1974a; Shyla and Pavithran, 1989), 27:37 (Hector, 1922, Ghose *et al.*, 1960; Kadam and D´Cruze, 1960; Tripathi and Rao, 1979; Annie, 1986) 81:175 (Ghose *et al.*, 1960), 15:1 (Hector, 1922, Chao, 1928, Ramiah and Rao, 1953) and 255:1 (Thimmappiah, 1975).

Inhibitory ratios 3:13, 9:55 (Sastry and Patnaik, 1962) 3:253 (Dhulappanavar *et al.*, 1975b); 63:193 (Dhulappanavar, 1981), 117:139 (Richharia *et al.*, 1960) have also been reported for leaf sheath pigmentation in rice.

c. Leaf blade pigmentation

Genetical ratios reported for leaf blade pigmentation are 3:1 (Parnell *et. al.*, 1917; Matsura, 1933; Nakayama, 1935; Morinaga, 1938; Dave,1948; Hsieh and Chang, 1962; Butany and Bhattacharya, 1962; Kumar and Rangaswamy, 1989; Nadaf, 1989), 9:7 (Parnell *et al.*, 1917; Jones, 1930; Butany *et al.*, 1959; Hsieh, 1960, 1961; Ghose *et al.*, 1963), 15:1 (Ramiah and Rao, 1953), 3:13 (Kadam, 1936; Yamaguchi, 1937; Dave, 1948; Chakravarthy, 1948; Nagao and Takahashi,

1951; Khan, 1953; Shafi and Khan, 1958; Aziz and Shafi, 1958; Butany and Bhattacharya, 1962; Ghose et al., 1963; Butany and Ghose, 1965; Yadav, 1971; Sastry and Seetharaman, 1980; Kinoshita and Maekawa, 1986; Sahu, 1988; Sahu and Sahu, 1989; Kumar and Rangaswamy, 1989), 27:37 (Ramiah and Rao, 1953; Panda, 1962; Ghose et al., 1963; Saran and Srivastava, 1969; Singh et al., 1989a), 9:55 (Iyer, 1959, Ghose et al., 1963; Butany and Ghose, 1965; Setty and Misro, 1973), 39:25 (Nadaf, 1989); 117:139 (Hsu and Lu, 1943), 27:229 (Iyer, 1959; Ghose et al., 1963; Butany and Ghose, 1965) and 15:241 (Dhulappanavar, 1973b, 1975a).

d. Leaf margin

Various genetic ratios reported for the leaf margin pigmentation are 3:1 (Mitra et al.,1928; Mitra and Ganguli, 1932; Kuang, 1951; Nair,1958; Panda, 1962; Nadaf, 1989; Pinson, 1994), 9:7 (Kuang, 1951; Dubey, 1955; Butany et al., 1959; Panda, 1962; Sastry and Patnaik, 1962; Dhulappanavar, 1973a, Pavithran, 1977; Shyla, 1984; Nadaf, 1989; Shobha, 1993), 27:37 (Panda, 1962; Singh et al., 1989), 45:19 (Setty and Misro, 1973; Hedagal, 1980; Hedagal et al .,1981; Rao and Misro, 1986), 54:10 (Nair, 1958; Shyla, 1984), 9:55 (Iyer, 1959; Sastry and Patnaik, 1962), 81:175 (Dubey, 1955), 117:139 (Dubey, 1955; Misro, 1963; Srivastava and Saran, 1971), 162:94 (Panda, 1962; Saran and Srivastava, 1969; Setty and Misro 1973; Setty et al., 1973; Singh

et al., 1989b), 15:1 (Kuang, 1951; Nadaf, 1989), 63:1 (Kuang, 1951), 27:229 (Iyer, 1959) and 207:49 (Kadam, 1974).

e. Leaf tip pigmentation

Various genetical ratios have been reported for inheritance of leaf tip pigmentation. They are 3:1 (Mitra and Ganguli, 1932; Ghose et al, 1960; Nadaf 1989), 9:7 (Ghose et al., 1960; Manjunath, 1973; Pavithran, 1977), 27:37 (Ghose et al., 1960); Saran and Srivastava, 1969, Singh et al., 1989b), 54:10 (Ghose et al., 1960), 45:19 (Ghose et al., 1960 ; Rahman, 1964; Hedagal, 1980; Rao and Misro, 1986), 162:94 (Ghose et al., 1960; Panda, 1962; Singh et al., 1989b), 189:67 (Setty and Misro, 1973; Setty et al., 1973), 255:1 (Thimmappaiah, 1975) and 702:322 (Setty and Misro, 1973; Setty et al., 1973).

f. Junctura Proper pigmentation

Pigmentation in junctura proper varies from light purple dots to dark purple. Ratios reported earlier for its inheritance include 3:1 (Parnell et al.,1917; Jones, 1930; Ghose et al., 1960; Saran and Srivastava, 1969), 1p:3g (D´Cruz and Dhulappanavar, 1963, Dhulappanavar, 1973b, 1975a), 9:7 (Parnell et al., 1917; Jones 1930; Kuang, 1951; Ghose et al., 1960), 27:37 (Jones, 1930, Butany et al., 1959; Setty and Misro, 1973), 45:19 (Ghose et al., 1960); Sastry and

Patnaik, 1962; Shyla, 1984) and 54:10 (Ghose et al., 1960; Setty and Misro, 1973; Thimmappiah, 1975).

Further, tetragenic complementary ratio 81:175 (Rahman and Srivastava, 1968), 162:94 (Ghose, et al., 1960; Dhulappanavar et al., 1973b., Pavithran and Annie, 1980 and Annie, 1986) and pentagenic complementary ratio 243p:781g (Dhulappanavar, 1979) and an inhibitory ratio 117:139 (Ghose et al., 1960) have also been reported.

g. Ligule pigmentation

Various genetical ratios reported earlier for inheritance of ligule pigmention are 3:1 (Mitra et al., 1928; Mahta and Dave, 1931; Mitra and Ganguli, 1932; Kuang,1951; Ramiah and Rao, 1953; Ghose et al., 1960; Srivastava et al., 1968; Nadaf, 1989), 1:3 (Singh et al., 1989b), 9:7 (Hector, 1922, Mitra et al., 1928, Jones, 1930; Mitra and Ganguli, 1932; Kuang, 1951; CRRI, 1956-57; Ghose et al., 1960; Pavithran, 1977; Rao and Misro, 1986; Ahmed and Das, 1990), 27:37 (Hector, 1922; Chao, 1928; Jones, 1930; Kuang, 1951, Butany and Bhattacharya, 1962; Pavithran and Mohandas, 1976a,b; Prasad et al., 1987; Singh et al., 1989b), 63:1 (Kuang, 1951), 45:19 (Ghose et al., 1960; Pavithran and Mohandas, 1976a, b), 54:10 (Ghose et al., 1960), 81:175 (Kuang, 1951), 117:139 (Dubey, 1955; CRRI, 1956-57; Dhulappanavar, 1979), 162:94 (Ghose et al., 1960; Dhulappanavar,

1973b, 1975a, 1976b; Setty and Misro, 1973), 189:67 (Manjunath, 1973), 27:229 (Dhulappanavar et al., 1975b) and 243:13 (Dhulappanavar, 1981).

h. Auricle pigmentation

Auricle pigmentation has been reported to be simple dominant over green (Parnell et al., 1917; Mitra et al.,1928; Nakayama, 1932; Kuang, 1951, CRRI, 1956-57; Ghose et al., 1960). Further 9:7 (Hector, 1922; Jones, 1930; Mitra and Ganguli, 1932; Dave, 1948; Kuang, 1951; Butany and Bhattacharya, 1962; Dhulappanavar et al., 1973b; Shyla, 1984, Ahmed and Das, 1990; Sukeshkumar, 1990), 15:1 (Kuang, 1951; Ramiah and Rao, 1953; Thimmappiah, 1975), 27:37 (Jones, 1930; Butany et al., 1959; Dhulappanavar, 1973a, 1973c, 1975a; Nadaf, 1989; Singh et al., 1989b), 45:19 (Setty and Misro, 1973; Dhulappanavar, 1981; Shyla, 1984; Rao and Misro 1986), 54:10 (Ghose et al., 1960; Shyla, 1984; Shyla and Pavithran, 1989), and 63:1 (Kuang, 1951) have also been reported.

Tetragenic complementary ratios 81:175 (Kuang et al., 1946; Prasad et al., 1987) and 162:94 (Ghose et al., 1960; Saran and Srivastava, 1969; Pavithran and Annie, 1980; Shyla, 1984; Annie, 1986), have also been recorded.

Inhibitory ratios 3:13 (Shastry and Patnaik, 1962), 9:55 (Prasad

et al., 1987), 117:139 (Dubey, 1955; Dhulappanavar, 1979; Singh et al., 1989b) have also been reported for auricle pigmentation.

i. Node pigmentation

Different genetical ratios reported earlier for nodal pigmentation are 3:1 (Mitra and Ganguli, 1932; Nagao and Takahashi, 1950, 1951; Nagao, 1951; Takahashi, 1957; Butany et al., 1959), 9:7 (Jones, 1930; Silva and Vianna, 1950; Butany and Bhattacharya, 1962; Misro, 1963; Manjunath, 1973; Singh et al., 1989b; Sukeshkumar, 1990), 15:1 (Thimmappaiah, 1975), 27:37 (Lee, 1927), 117:139 (Dhulappanavar et al., 1975b), 162:94 (Singh et al., 1989b) and 111:145 (Nadaf, 1989). Both complementary and inhibitory factors are involved and in certain crosses duplicate recessive control has been assumed.

j. Internode pigmentation

Internode pigmentation has been reported to be simple dominant over green. Different genetical ratios reported so far are 3:1 (Hector, 1922; Mitra et al., 1928; Hsieh, 1960; Misro, 1963; Kondo, 1963; Mori et al., 1981), 9:7 (Hector, 1922; Mitra et al., 1928; Jones, 1930, Ganguli, 1942; Kuang, 1951; Iyer, 1959; Ghose et al., 1960; Majumdar, 1969; Dhulappanavar 1973a, 1975b, Dhulappanavar et

al., 1974; Ahmed and Das, 1990; Singh et al., 1989b), 27:37 (Hector, 1922; Jones, 1930; Ghose et al., 1960; Butany and Bhattacharya, 1962; Sastry and Patnaik, 1962; Dhulappanavar et al., 1973b; Kolhe and Bhat, 1979, 1982), 3:13 (Dhulappanavar, 1981), 45:19 (Rahman and Srivastava, 1968; Hedagal, 1980; Hedagal et al., 1981), 81:175 (Ghose et al., 1960), 117:139 (Ghose et al., 1960; Sadananda, 1981), 147:109 (Nadaf, 1989), and 27:229 (Singh et al., 1989b) involving three complementaries and inhibitory/ anti-inhibitory factors for control of pigmentation in internode.

k. Stigma pigmentation

Anthocyanin pigmentation in stigma is one of the most widely studied characters in rice. Various genetical ratios reported earlier for inheritance include 3:1 (Hector, 1916, 1922; Chao, 1928; Mitra et al., 1928; Mitra and Ganguli, 1932; Khan, 1953; Ramiah and Rao, 1953; Dubey, 1955; Aziz and Shafi, 1958; Shafi and Khan, 1958; Shafi and Aziz, 1959; Ghose et al., 1960; D´Cruz and Dhulappanavar, 1963; Kondo, 1963; Misro, 1963; Srivastava et al., 1968; Yadav, 1984; Yadav and Tomar, 1984; Shyla, 1984, Annie, 1986, Nadaf, 1989, Sukeskumar, 1990; Shobha, 1993), 9:7 (Parnell et al., 1917; Mitra et al., 1928; Jones, 1930; Nakayama, 1932; Ramiah and Rao, 1953; Dubey, 1955; CRRI, 1956-57; Butany et al., 1959; Ghose et al., 1960; Misro, 1963, Rao and Misro, 1968b; Majumdar et al., 1969; Ratho and Rao, 1972; Pavithran and Annie, 1980; Annie, 1986; Prasad et al., 1987;

Ahmed and Das, 1990), 9g:7p (Nagai et al., 1962), 15:1 (Ramiah and Rao, 1953; CRRI, 1956-57), 27:37 (Hector, 1922; Dubey, 1955; Ghose et al., 1960, Kadam and D'cruz, 1960; Ramirez et al., 1960; Misro, 1963; Pavithran, 1977; Ramesh, 1984, Prasad et al., 1987), 45:19 (Dubey, 1955; Ghose et al., 1960; Sastry and Patnaik, 1962; Hedagal, 1980; Sadananda, 1981; Shyla, 1984; Prasad et al., 1981), 54:10 (Manjunath, 1973; Setty and Misro, 1973; Dhulappanavar, 1973d; Dhulappanavar and Hiremath, 1974a,b; Shyla, 1984; Annie, 1986; Shyla and Pavithran, 1989), 81:175 (Hector, 1922; Dubey, 1955; CRRI, 1956-57; Ghose et al.,1960; Kadam and D'cruz, 1960), 162:94 (Dubey, 1955; D'Cruz, 1960; Ghose et al., 1960; Butany and Bhattacharya, 1962; Saran and Srivastava, 1969; Kadam and Desale, 1972; Setty and Misro, 1973; Dhulappanavar et al., 1973c, 1975a; Kolhe and Bhat, 1979, 1982; Shyla, 1984; Annie, 1986; Singh et al., 1989b), and 189:67 (Kadam and Desale, 1972).

Inhibitory ratios like 3:13 (Hsieh, 1980; Panda et al., 1965), and 9:55 (Hsieh, 1960; Srivastava and Saran, 1971) have also been reported for stigma pigmentation.

1. Apiculus pigmentation

Apiculus pigmentation has been studied in greater detail in rice. Various genetical ratios reported for its inheritance are 3:1

(Hector, 1916, 1922; Parnell et al., 1917; Mitra et al.,1928; Jones, 1930; Mitra and Ganguli, 1932; Nakayama, 1932; Jodon, 1940, 1957; Morinaga et al., 1943; Kuang et al., 1946; Jodon and Chilton, 1946; Silva and Vianna, 1950; Kuang, 1951; Chandraratna, 1953; Dubey, 1955; Alim and Sen, 1956; Butany et al., 1959; Aziz and Shafi, 1958; Shafi and Aziz 1959; Hsieh, 1960; Misro, 1963; Hsieh and Chang, 1964; Misro and Kulkarni, 1965; Panda et al., 1965; Sastry and Reddy, 1967; Srivastava et al., 1968; Yen and Hsieh, 1968; Saran and Srivastava, 1969; Setty et al., 1973; Eunus et al., 1974; Nascimento, 1977; Shyla, 1984; Rao and Misro, 1986; Annie, 1986; Nadaf, 1989; Sukeskumar, 1990; Ahmed and Das 1990), 9:7 (Hector, 1916, 1922; Mitra et al., 1928; Chao, 1928; Jones, 1933; Jodon and Chilton, 1946; Ramiah and Rao, 1953; Dubey, 1955; Aziz and Shafi, 1958; Shafi and Aziz, 1959; Hsieh, 1960, 1961; Kadam and D´Cruz, 1960; Ramirez et al., 1960; Nagai et al., 1962; Misro, 1963; Hsieh and Chang, 1964; Rao and Misro, 1968b; Yen and Hsieh, 1968; Akbar et al., 1975; Pavithran, 1977; Paramashivan, 1986; Annie, 1986; Prasad et al., 1987), 9P:3r:4g (Ramiah and Rao, 1953; Dhulappanavar et al., 1975b), 15:1 (Van der Stok, 1908; Hector, 1922; Chao, 1928; Ramiah and Rao, 1953; Nagai et al., 1962; Ratho and Rao, 1972; Setty et al., 1973; Manjunath, 1973; Dhulappanavar, 1973c; Eunus et al., 1974), 13P:3g (Tripathi and Rao, 1979; Sahu and Sahu, 1989), 27:37 (Hector, 1922; Chao, 1928; Jodan and Chilton, 1946; Ramiah and Rao, 1953; Dubey, 1955; Ramesh, 1984; Yadav and Tomar, 1984, Tripathi and Rao, 1985; Prasad et al., 1987), 54:10 (Dubey, 1955, Ghose et al., 1960; Sastry

and Patnaik, 1962; Misro, 1963; Dhulappanavar and Mensinkai, 1970; Setty and Misro, 1973; Shyla, 1984; Annie, 1986; Prasad et al., 1987; Shyla and Pavithran, 1989), 45:19 (Ghose et al., 1960; Butany and Bhattacharya, 1962; Sastry and Patnaik, 1962; Venkataswamy, 1964; Kolhe and Bhat, 1979, 1982; Hedagal, 1980; Pavithran and Annie, 1980; Shyla, 1984; Annie, 1986), 39:25 (Dhulappanavar, 1977), 162:94 (Chao, 1928; Dubey, 1955; Ghose et al., 1960; Misro, 1963; Dhulappanavar, 1975a, b, 1976b; Shyla, 1984; Singh et al., 1989b), 189:67 and 135:121 (Sadananda, 1981), and 171:85 (Jodon, 1955).

m. Awning

Awn is greatly influenced by environmental factors for its expression. Van der stok (1910 c.f Ikeno, 1927) was the first to note awning to be either dominant or recessive. Various genetical ratios later reported are 3:1 (Nagai, 1926; Majid, 1939; Hara, 1942; Ramiah and Rao, 1953; Kuang, 1951; Seetharaman, 1965; Jodon, 1965; Pavithran, 1986; Sahu, 1991), 9:7 (Ramiah, 1935; Grant, 1935; Sastry, 1977; Pavithran, 1986), 9 awned : 6 slgihtly awned, 1 awnless (Ikeno, 1927; Majid, 1939; Ramiah and Rao, 1953), 15:1 (Chao, 1928; Alam, 1932; Mitra and Ganguli, 1932; Kuang, 1951; Ramiah and Rao, 1953; Pavithran, 1986), 3:13 (Breaux, 1940; Housaye and de La, 1942; Misro and Misro, 1954; Jodon, 1957; Pavithran, 1986), 27:37 (Ghose et al.,

1960, Sastry, 1977; Pavithran, 1986), 54:10 (Pavithran, 1986), 63:1 (Ikeno, 1927; Sethi et al., 1937; Ramiah and Rao, 1953; Pavithran, 1986), 81:175 (Ghose et al., 1960; Pavithran, 1986), 117:139 (Thimmappaih, 1975), and 243:781 (Ghose et al., 1960; Pavithran, 1986).

n. Tip sterility /Sponginess

Spikelets at the panicle tips or base remain rudimentary or underdeveloped/undeveloped giving a white feathery appearance. It has been reported to be simple recessive to normal (Ramiah, 1931; Ramiah and Parthasarathy, 1938). This appears to be one of the least studied characters in rice.

2. Inheritance of rice mutants

Mutants are particularly suitable for the study of genetics and gene interactions (Kawai, 1968). Generally most of the induced mutants reported are recessive to the normal types. Spontaneous dwarfs have been simple recessive to normal (Parnell et al., 1922; Sugimoto, 1922; Akemine, 1925; Jones, 1933; Ramiah, 1933; Nandi and Ganguli, 1941).Dwarf Japan I (Akemine, 1925; Kadam, 1937) dwarf Japan II and dwarf kolamba have been reported as spontaneous simple recessive dwarfs.

Monogenic recessive mutations recorded in rice are ageotropic mutant (Ramiah and Parthasarathy, 1936; Jones and Roy, 1938), plastid mutation (Pal and Ramanujam, 1941), spontaneous lethal yellow (Imai, 1935; Ramiah and Ramanujam, 1935 and Kadam, 1941), spontaneous zebra yellow -lethal (Ramiah and Ramanujam, 1935), virescents (Kadam, 1941), albinos (Iwata and Omura, 1978), green and white variegation (Ramiah and Ramanujam, 1935; Maekawa et al., 1990), green and yellow persistent (Ramiah and Ramanujam, 1935), chlorina (Iwata and Omura, 1976; Iwata et al., 1978; Omura et al., 1978), spotted leaf (Morinaga and Fukushima, 1943; Iwata and Omura, 1975; Iwata et al., 1978; Iwata and Omura 1977; Iwata et al., 1978, 1981; Yoshimura et al., 1982), narrow leaf (Ramiah and Parthasarathy, 1938), paleaceous sterile (Nagai, 1926), barren sterile (Anandan and Krishnaswamy, 1934), spontaneous male steriles (Ishikawa, 1929; Miyazava, 1935; Srinivasan, 1937), awned sterile mutant (Nagai, 1926), staminodal sterile (Nagai, 1926), pistillate sterile (Miyazava, 1935), male sterile mutants (Ishikawa, 1929; Miyazava, 1935; Ramanujam, 1935; Srinivasan, 1937; Trees, 1975; Pavithran and Mohandas, 1976a; Ko and Yamagata, 1980; Singh and Ikehashi, 1981; Shyla, 1984; Rutger et al., 1985; Singh and Sinha, 1989; Suh et al., 1989), slender semi-sterile and sterile shrivelled stamens (Kagawa, 1939), sterile open lemma (Miyazava, 1932), prostrate mutant (Sreedharan and Sreevastava, 1979), non-shedding changed husk colour and virido-alba (Narahari, 1969), dwarfness, spreading panicle, lazy

growth habit and narrow rolled leaf (Hsieh, 1962), open hull male sterility (Takeda, 1987), palealess mutant (Jachuck and Sampath, 1969), dwarf mutant (Hsieh, 1962; Kitano and Futsuhara, 1981; Kocoyama et al., 1985; Aruna and Reddy, 1988), dwarf stature with small grain size (Mallick and Bairagi, 1979; Takamure and Kinoshita, 1985), dwarfness, spreading panicle, laziness and narrow leaf (Tranh and Trinh, 1985), semidwarfism (Narahari, 1985), tall recessive (Okuno and Kawai, 1978a,b; Rutger and Carnahan, 1981), compact variant, hull spots, short internode, rolled leaf, neck leaf and coarse culm (Jones, 1952), dense panicle (Futsuhara et al., 1979), resistance to bacterial leaf blight (Nakai et al., 1988), root growth inhibition (Kitano and Futsuhara, 1989), hydroxy-L-proline resistance (Hasegawa et al., 1985), early mutants (Yamagata, 1964), claw hull (Takahashi, 1950), triangular hull (Morinaga and Fukushima, 1943), open spikelet (Gill et al., 1969), long glumed (winged) spikelets (Butany and Bhattacharya, 1962), multiple husk (Seetharaman and Srivastava, 1969), multipistillate mutant (Rao and Rao, 1986; Pavithran et al., 1989; Shobha, 1993), photosensitive male sterile mutant (Li et al., 1988) and brittle culm (Jones, 1933; Morinaga and Fukushima, 1943; Nagao, 1951; Nagao and Takahashi,1963; Takahashi et al., 1968; Iwata and Omura, 1977).

Simple dominant mutants reported in rice are dark violet stem and leaf sheath (Tranh and Trinh, 1985), early maturing (Mckenzie et

al., 1978), blast resistance mutant (Kawai, 1963b; 1974; Kaur et al., 1976, 1977) and brittle culm (Singh et al., 1994).

Some other mutants have been reported to be duplicate recessive such as albino (Morinaga, 1927), rolled leaf mutant with long flag leaf and reduced floret (Ramiah and Parthasarathy, 1938). Triple recessive albinos (Codd, 1934), asynaptic mutants and zebra white (Ramiah and Parthasarathy, 1938; Kadam, 1941), photosensitive mutant (Matsuo and Onozawa, 1961), male sterility (Pavithran and Mohandas, 1976a), complementary dominant genes for dwarfism (Kumar and Sreerangaswamy, 1987; Awan et al., 1985), multiple allelic interaction for male sterility (Ko and Yamagata, 1980); 13 normal : 3 multipistillate (Rao and Rao, 1986), sex linked lethals for semi-sterile eversplitting mutants (Terao, 1921) and heterozygous dominant gene causing ovule sterility in female sterile mutant (Razzaque, 1975) are the other reports on induced mutants in rice.

Grain dimensions such as grain length, breadth and L/B ratio are generally considered under polygenic control (Ramiah and Parthasarathy, 1933; Reddy and Reddy, 1974). Grain size and shape in rice are least influenced by environmental variations and show high heritability (Chandraratna, 1964). More than one gene has been reported for grain size (Kawai, 1968). Eventhough grain dimensions are controlled by polygenes different grain size mutants induced,

suggest that true breeding mutants with altered length, breadth and L/B ratio could be recovered in the M_2 generation (Reddy and Reddy, 1972, 1973), which might possibly be due to induction of mutations in single gene with distinct effects on grain dimensions. Grain size mutations are reported polydireactional in nature.

Pleiotropic action has been reported for plant height and breadth of leaves (Venkatanandhachari, 1963), short grain, panicle length, short culm and panicle density (Kawai, 1968). The simultaneous effects on a number of characters in macromutations were attributed to the pleiotropic action of a single mutated gene (Kawai, 1963 a,b). However, Ree (1970) explained this to be due to the compound effect of two or more neighboring linked genes which changed simultaneously during the mutagen treatment.

3. Studies on interrelationship of genes governing morphological characters

a. Studies on linkage relationship of genes governing morphological characters

Parnell et al. (1917) observed for the first time in rice an association between purple lining of internode and purple glumes and between purple stigma and purple axil. Takahashi (1923) reported linkage between awn color and non-glutinous factor and Chao (1928)

established three linkage groups and also gave an indication about the fourth group based on his observation.

The first review on linkage relationship in rice was published in 1948 based on available information till then (Jodon, 1948). Nagao and Takahashi (1963) constructed 12 linkage groups in Japonica for the first time, while Misro et al. (1966) presented the first linkage map of indica rice. Based on Misro et al. (1966), Takahashi and Kinoshita (1968) revised the linkage map in japonica and compared it with that of indica. Thus the 12 linkage groups corresponding to the haploid number of chromosomes could be established in rice (Jodon, 1956; Nagao and Takahashi, 1963 and Misro et al.,1966). Takahashi and Kinoshita (1977) published revised linkage map of japonica rice. Misro (1981) reviewed linkage studies in indica rice and assigned 76 genes out of 100 genes identified in indica to 12 linkage groups. Takahashi (1982) summarised linkage studies in rice with reference to anthocyanin pigmentation, non-anthocyanin colour and various other morphological characters studied for the last forty years and also discussed certain problematic situations in linkage studies such as search for linkage of major genes with polygenes, compilation of cases of correlated response and proving position effects, linkage between marker genes and agronomic characters and so on.

Khush et al. (1984) established complete correspondence between

linkage groups and cytologically identifiable chromosomes through trisomic analysis and presented a revised linkage map of rice. The authors observed complete correspondence between the linkage groups of japonica and indica and similarity in genic order in both sub-species. Variable distances between the genes were attributed to genetic background or environmental factors. An extensive review on inheritance and linkage studies in japonica rice and a revised linkage map have been presented by Kinoshita (1984b). Pavithran et al. (1991) reviewed and presented an extensive integrated genetic map of rice taking into consideration both the linkage maps of Japanica and indica. However, the present position of linkage association of genes governing morphological characters in rice is reviewed below based on reports relevant to the present study.

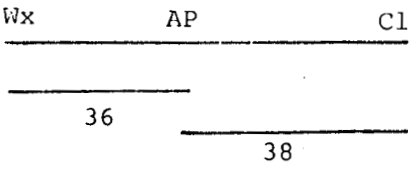
Parnell et al., (1917) proposed the first linkage in rice between black hull and purple internode, which remained unassigned to any linkage group till Shyla and Pavithran (1989) assigned it to the group IV (Fig. 1). Yamaguchi (1926) reported linkage between apiculus colour and waxy endosperm, the first linkage group in rice, the waxy group, and it was subsequently established (Chao, 1928; Ramiah et al., 1931; Jodon, 1940; Nagao and Takahashi, 1942). Saran and Srivastava (1969) studied linkage between clustering and anthocyanin pigmentation in seven plant parts, and assigned them to linkage group I. Kinoshita et al., (1975) through reciprocal

Fig. 1. Linkage maps of indica and japonica rices

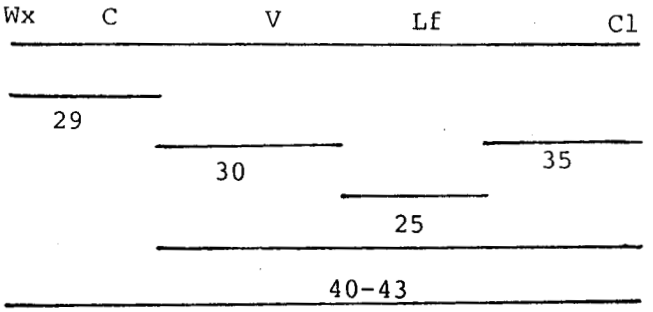
(Revised)

INDICA

I 'Wx' Group

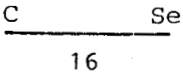


Seetharaman 1964



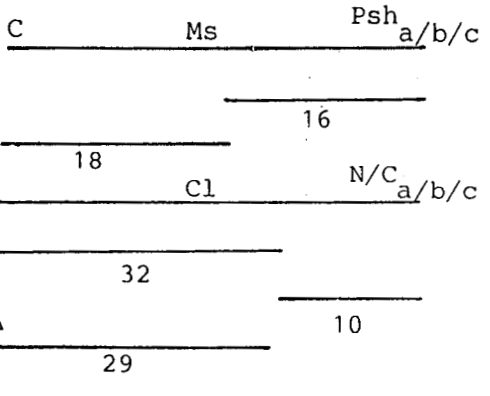
Jodon 1948-56

Nagao and Takahashi 1960



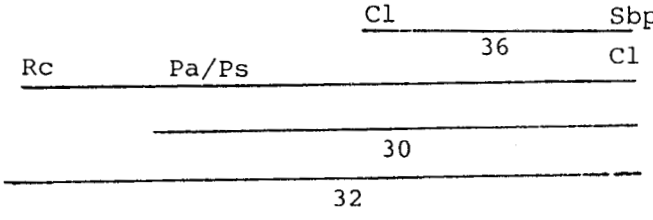
Chandraratna, 1953

Thakur & Roy 1975



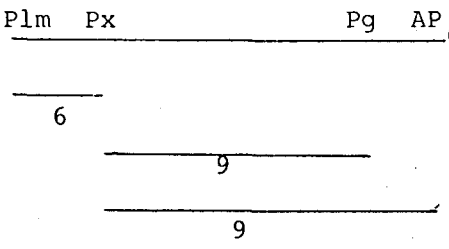
Shyla, 1984

Annie, 1986 (Unpublished)
Sukeskumar, 1990

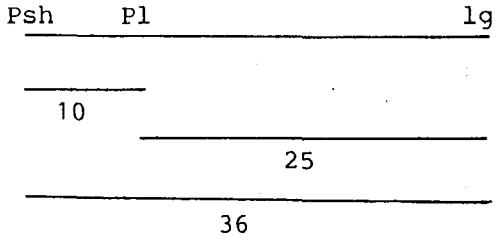


Anitha, 1994 (unpublished)

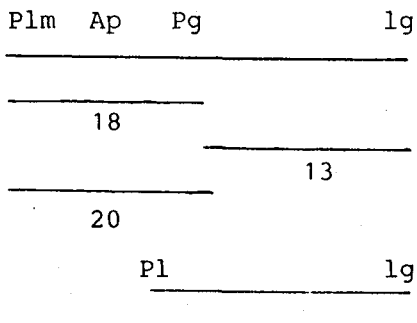
II 'lg' Group



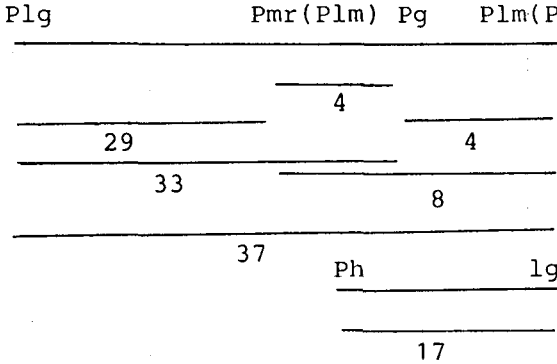
(a) Richharia et al., 1960



Rao, 1965

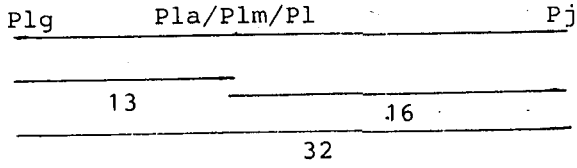


Jodon, 1948-56



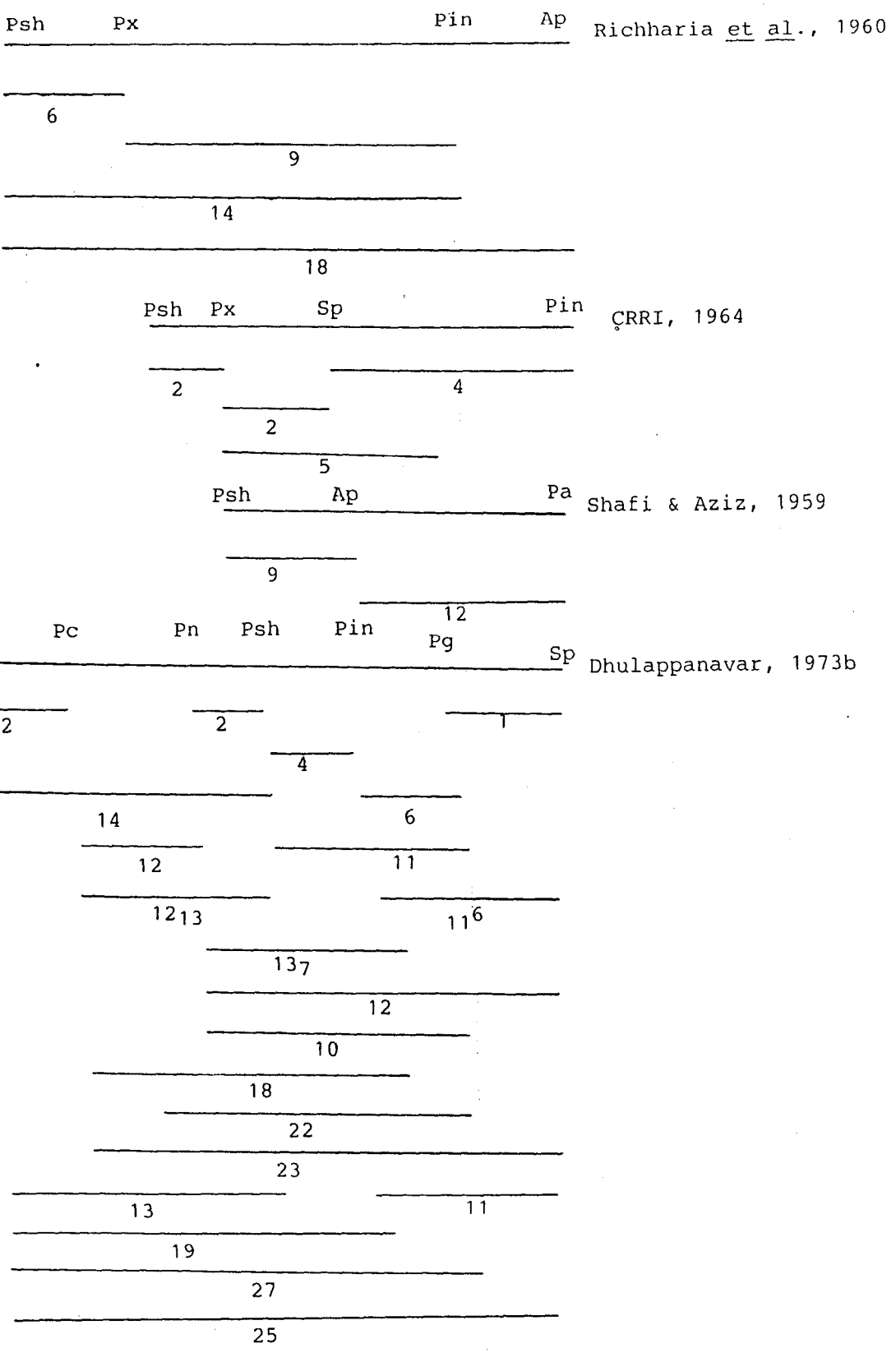
Dhulappanavar, 1973b

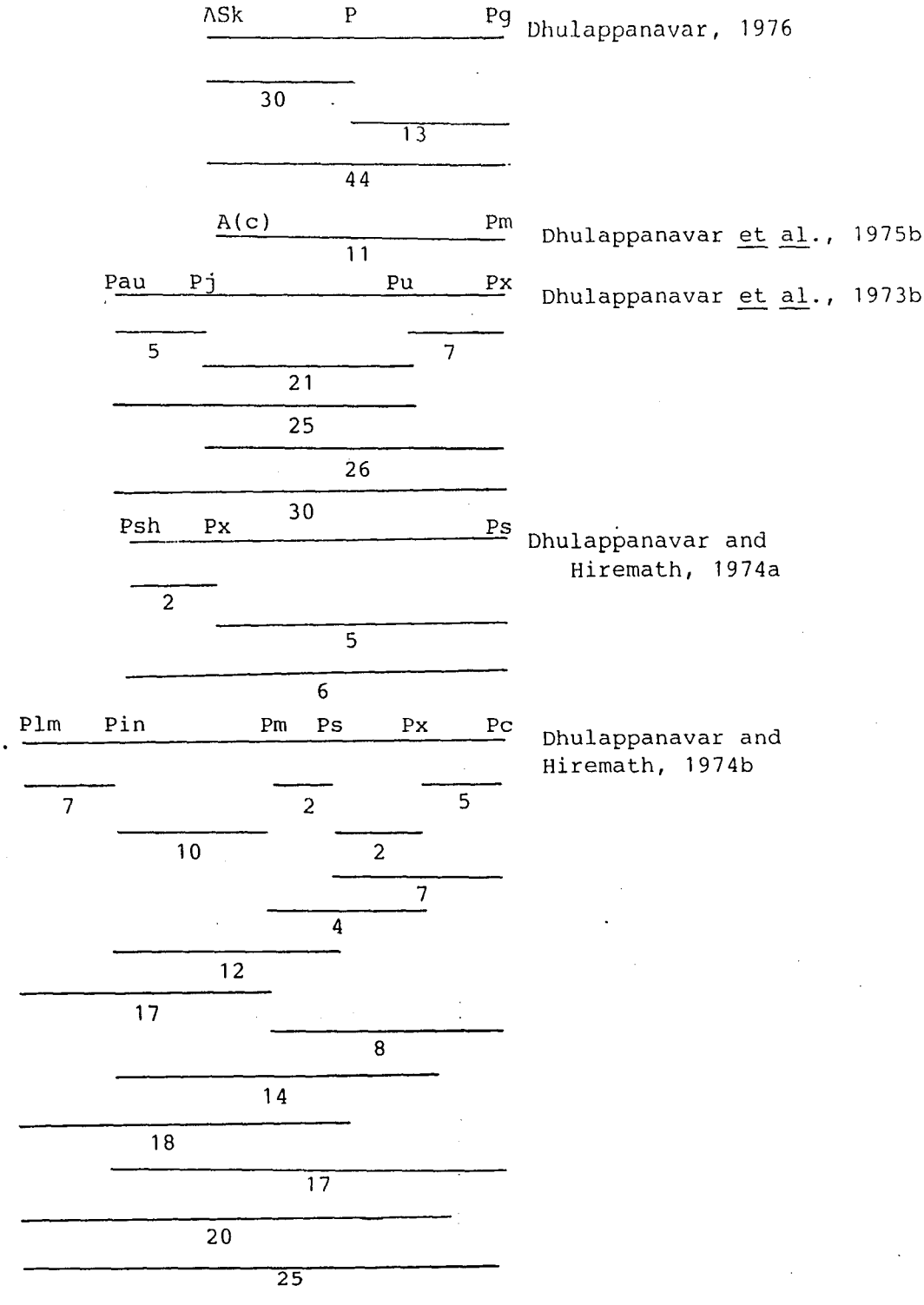
Annie, 1986 (unpublished)



$P_{a/b}$ Anitha, 1994 (unpublished)

III 'Sp' Group


Psh Px Pin Ap
Richharia et al., 1960
6
9
14
18
Psh Px Sp Pin
CRRI, 1964
2
4
2
5
Psh Ap Pa
Shafi & Aziz, 1959
9
12
Pau Pc Pn Psh Pin Pg Sp
Dhulappanavar, 1973b
2
2
1
4
14
6
12
11
12 13
11 6
13 7
12
10
18
22
23
13
11
19
27
25



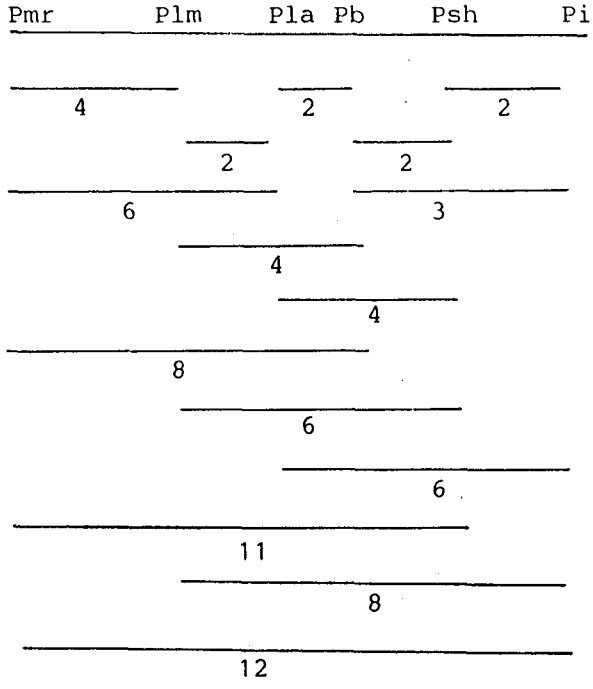
Dhulappanavar, 1976

Dhulappanavar et al., 1975b

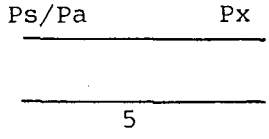
Dhulappanavar et al., 1973b

Dhulappanavar and Hiremath, 1974a

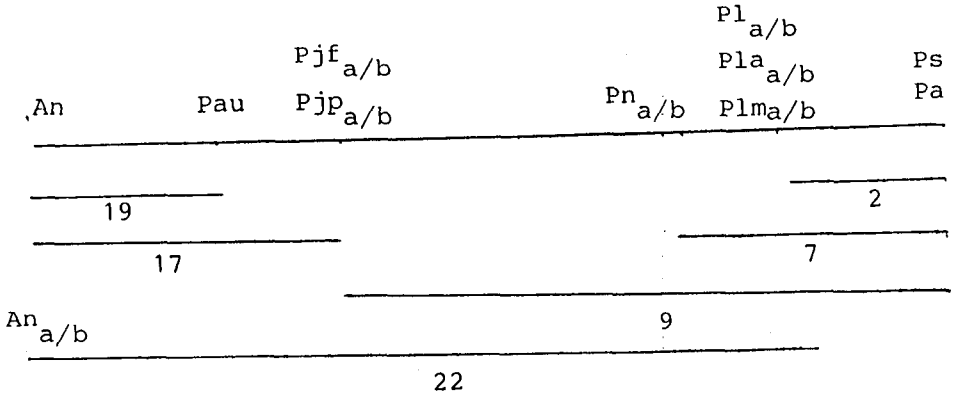
Dhulappanavar and Hiremath, 1974b



Hedegal et al., 1981

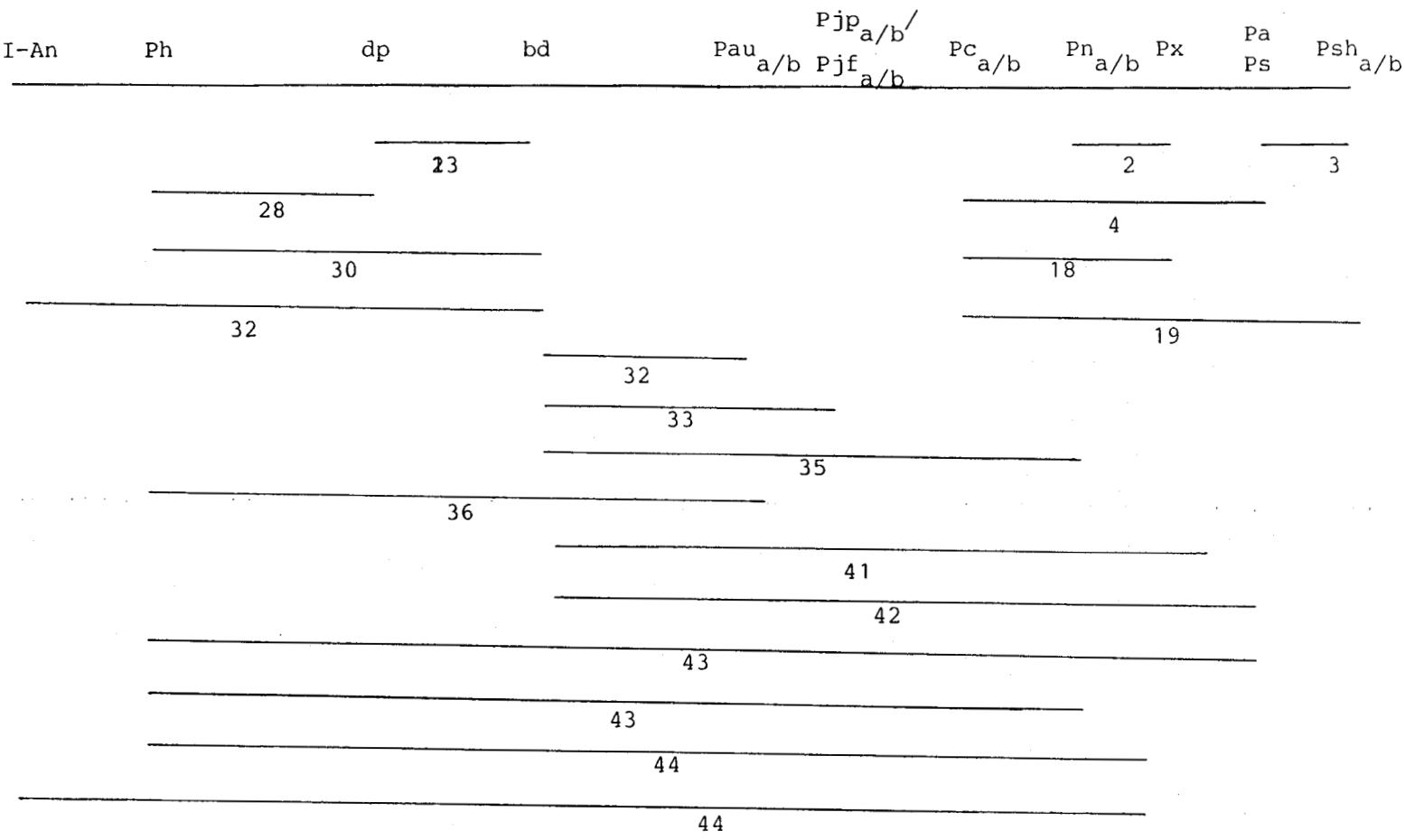


Annie, 1986 (Unpublished)

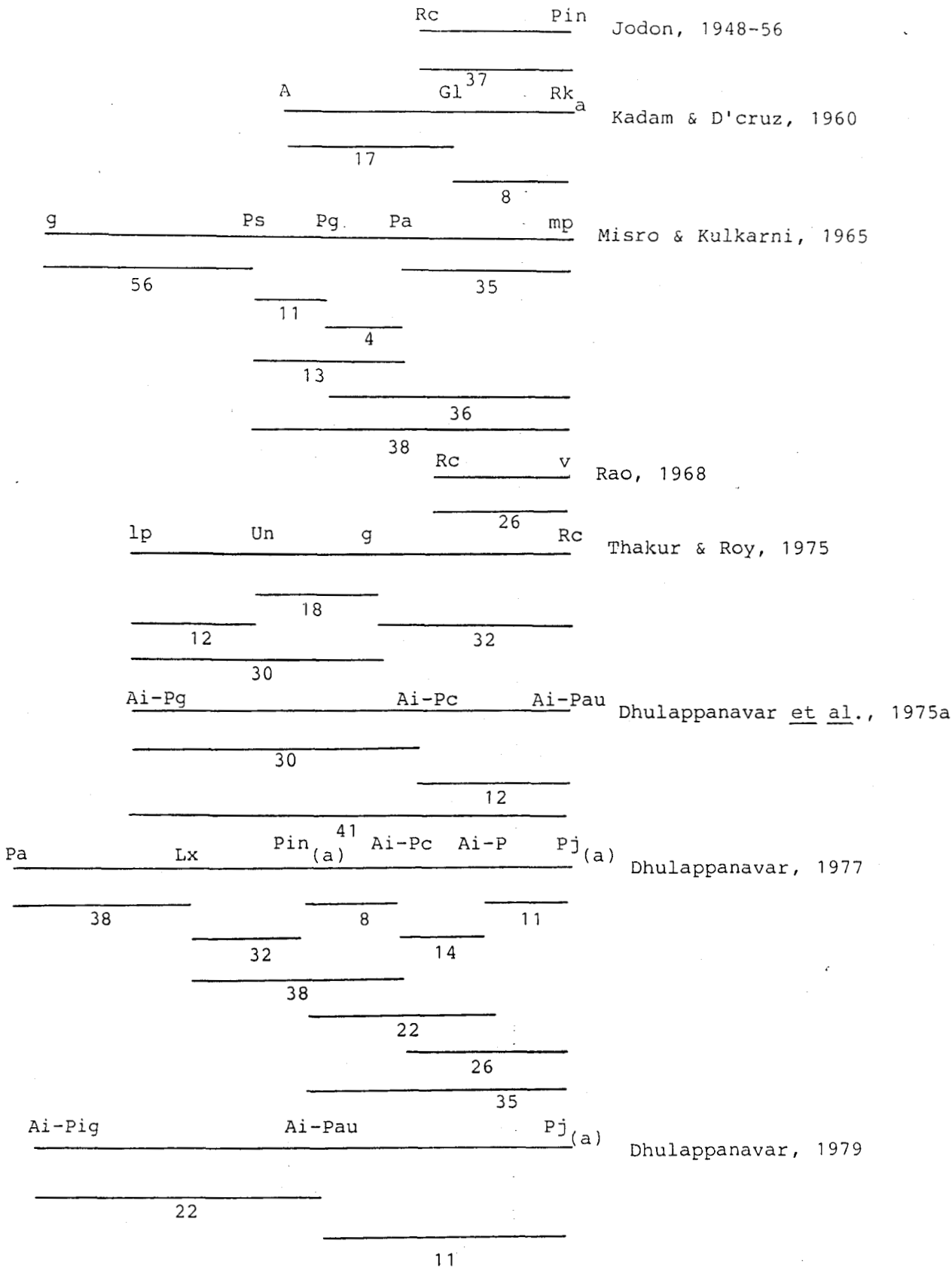


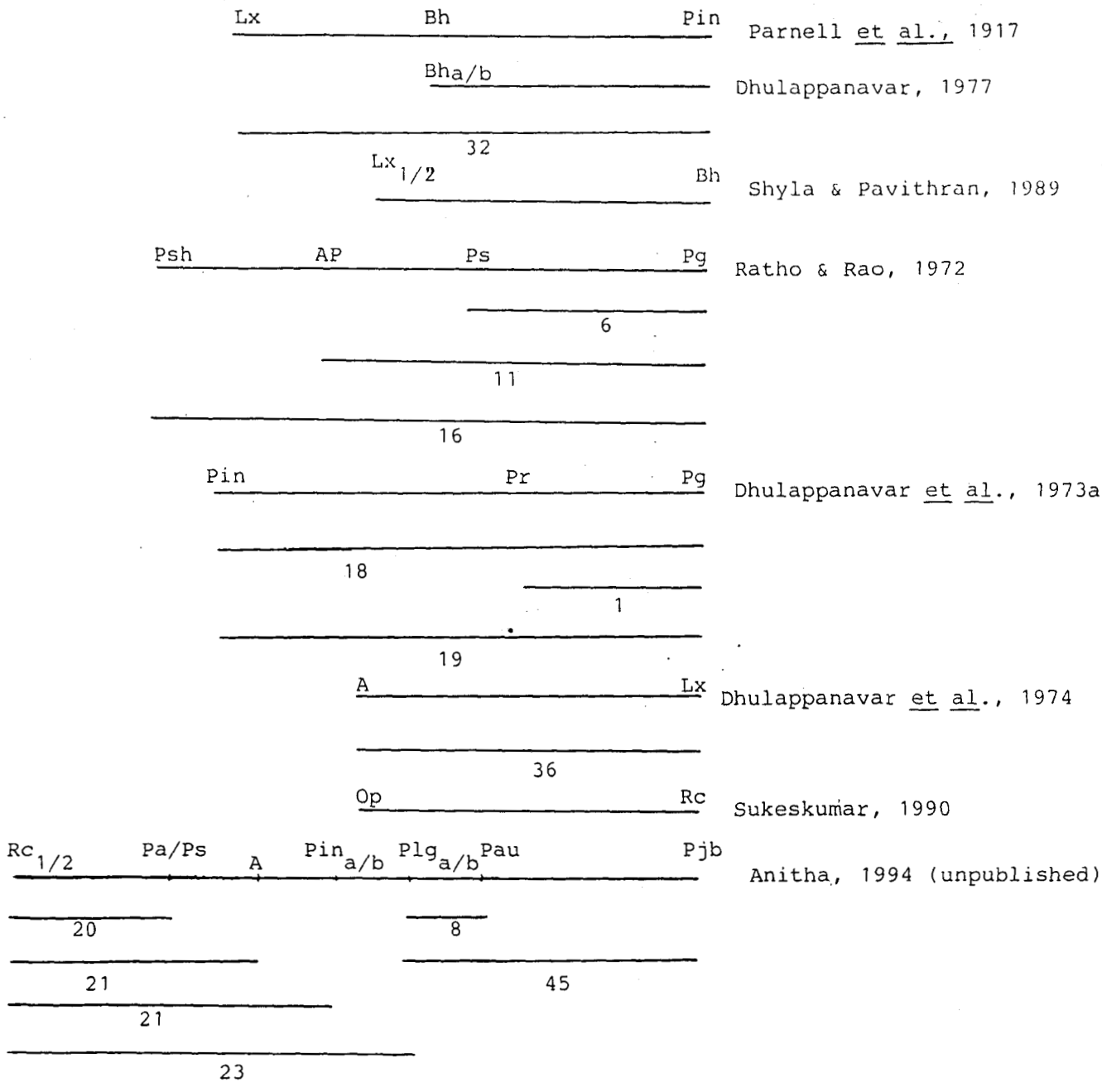
Anitha, 1994 (unpublished)

Sukeskumar, 1990

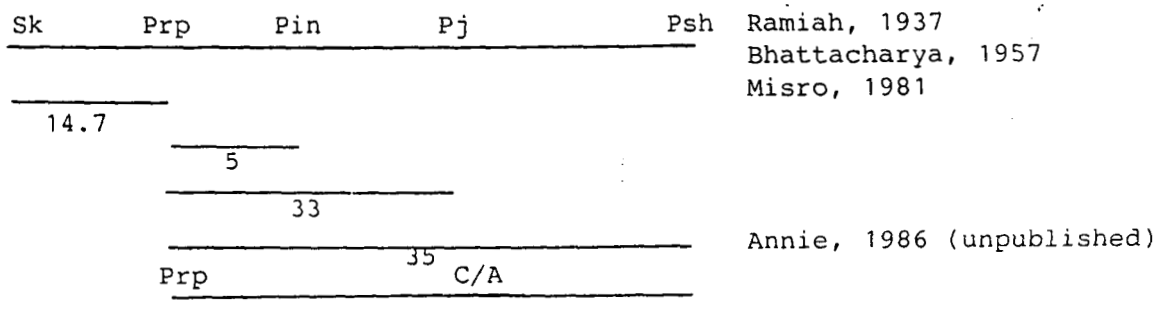


IV 'mp' Group

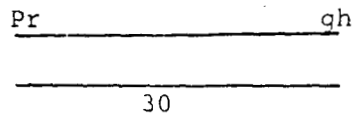

Rc Pin
Jodon, 1948-56
A Gl 37 Rk a
Kadam & D'cruz, 1960
17
8
g Ps Pg Pa mp
Misro & Kulkarni, 1965
56
35
11
4
13
36
38
Rc v
Rao, 1968
26
lp Un g Rc
Thakur & Roy, 1975
18
12
32
30
Ai-Pg Ai-Pc Ai-Pau
Dhulappanavar et al., 1975a
30
12
Pa Lx Pin (a) 41 Ai-Pc Ai-P Pj (a)
Dhulappanavar, 1977
38
8
11
32
14
38
22
26
35
Ai-Pig Ai-Pau Pj (a)
Dhulappanavar, 1979
22
11



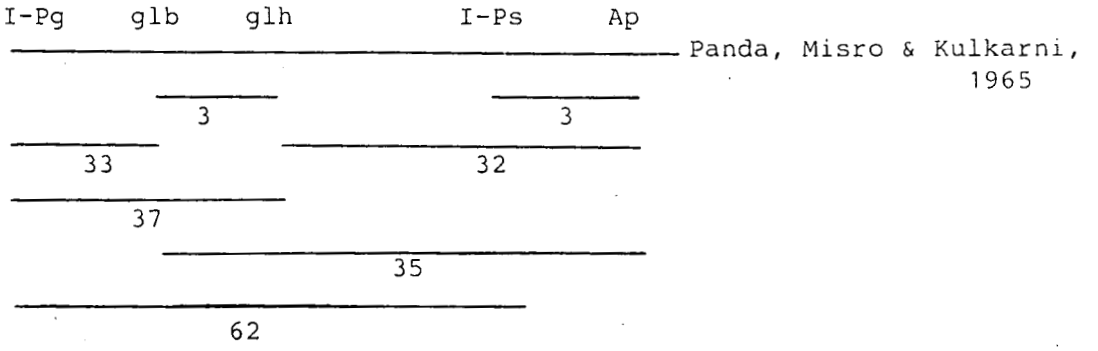
V 'Prp' Group



VI 'gh' Group

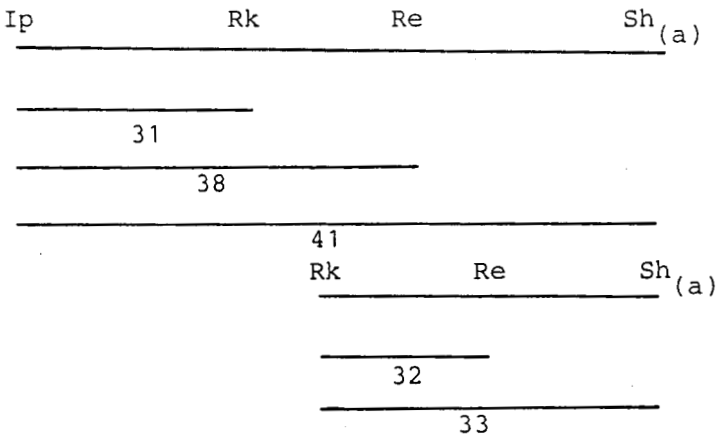


VII 'I-Pg' Group



Panda, Misro & Kulkarni, 1965

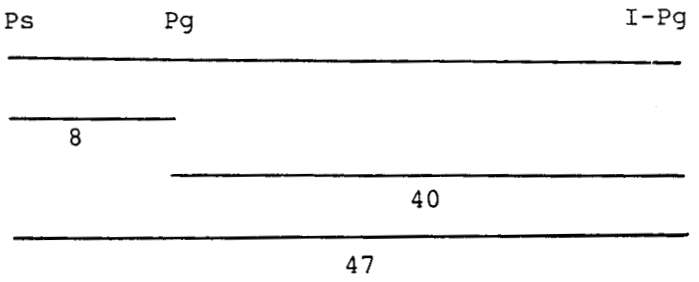
VIII 'Lp' Group



Rao & Misro, 1968a

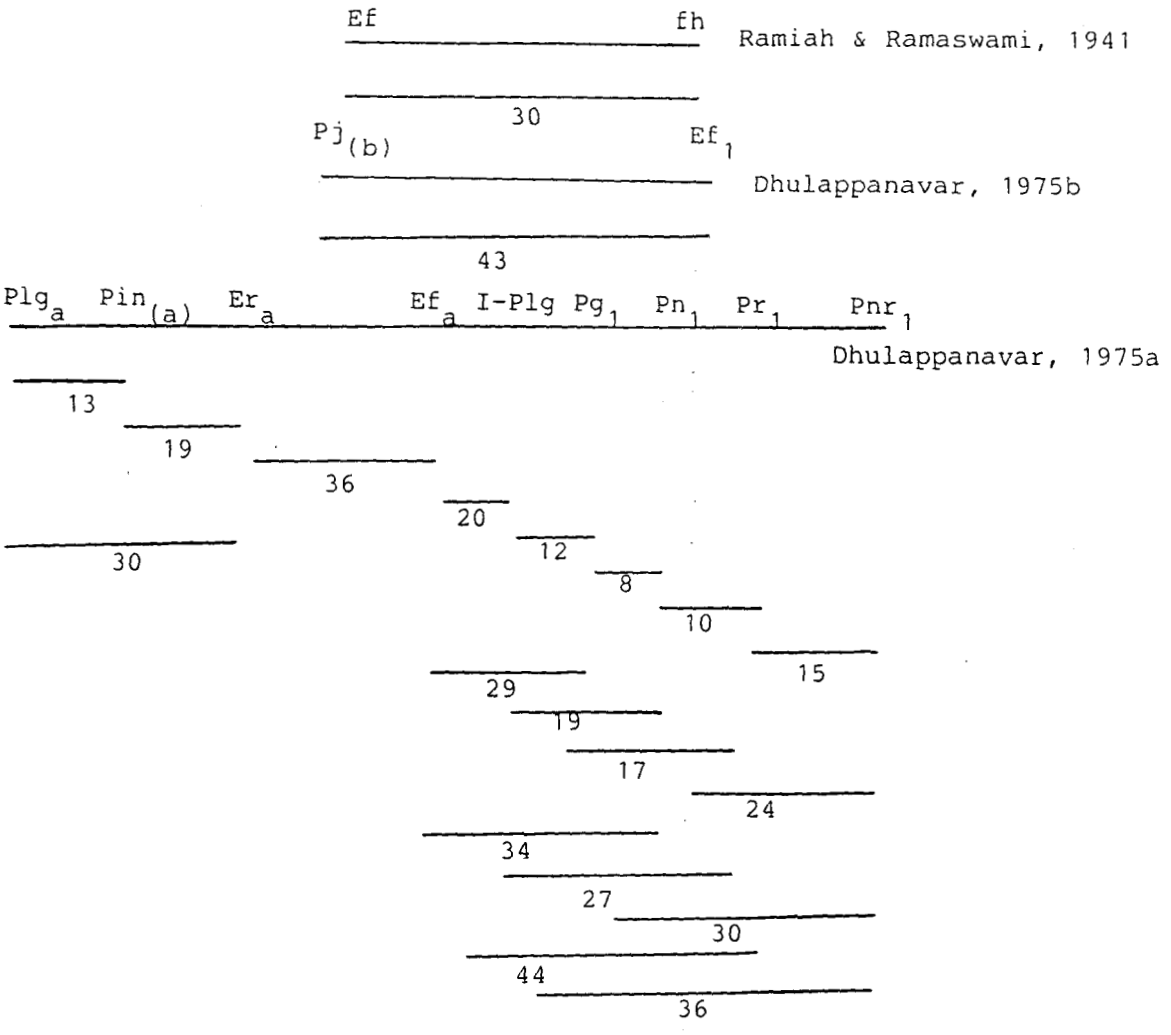
Rao, 1965

IX 'I-Pj' Group



D'Cruz & Dhulappanavar, 1963

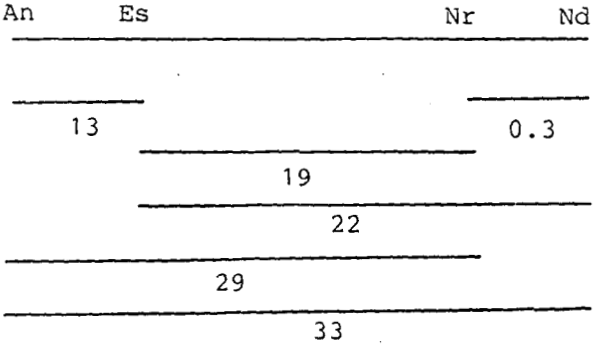
X 'fh' Group



Ramiah & Ramaswami, 1941

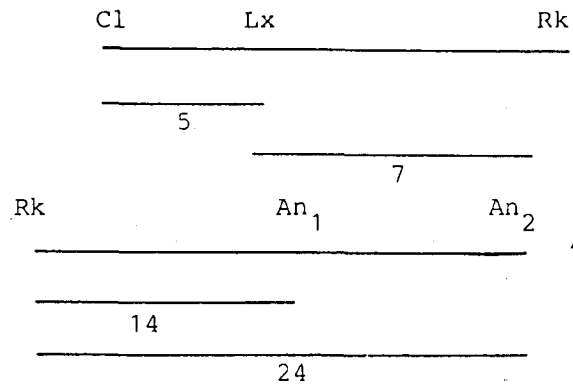
Dhulappanavar, 1975b

Dhulappanavar, 1975a



Tripathi & Rao, 1985

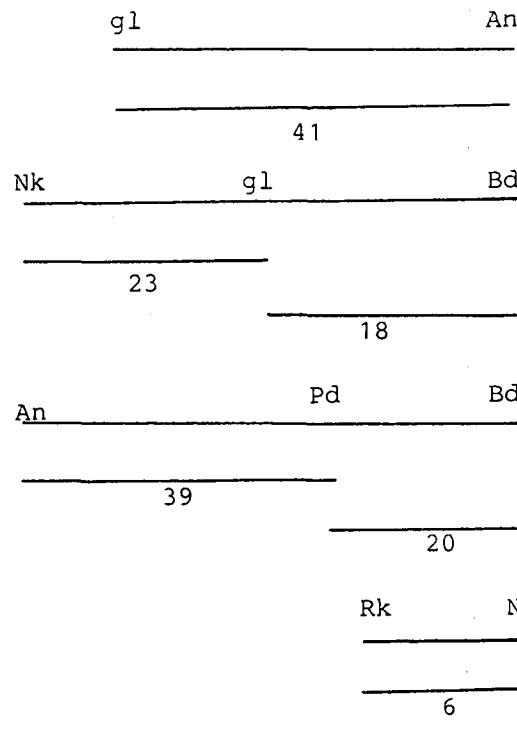
XI 'Lx' Group



Rao & Misro, 1965

Anandan, 1933

XII 'Nk-Bd' Group



Breaux, 1940

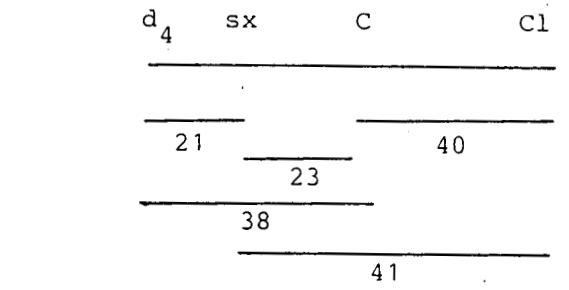
CRRI, 1965

Rao & Misro, 1968b

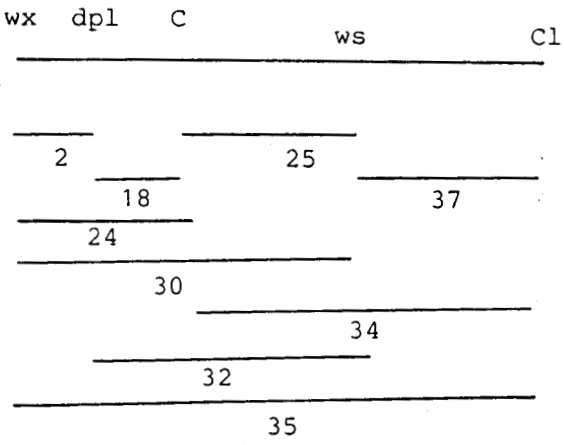
Pavithran, 1977

JAPONICA

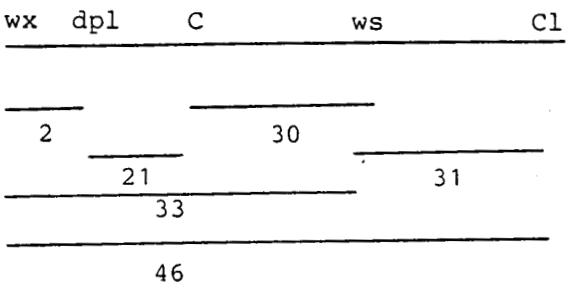
1 'wx' Group



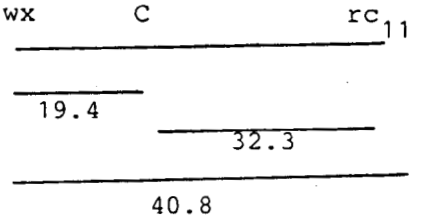
Takahashi 1964



Takahashi & Morimura, 1968

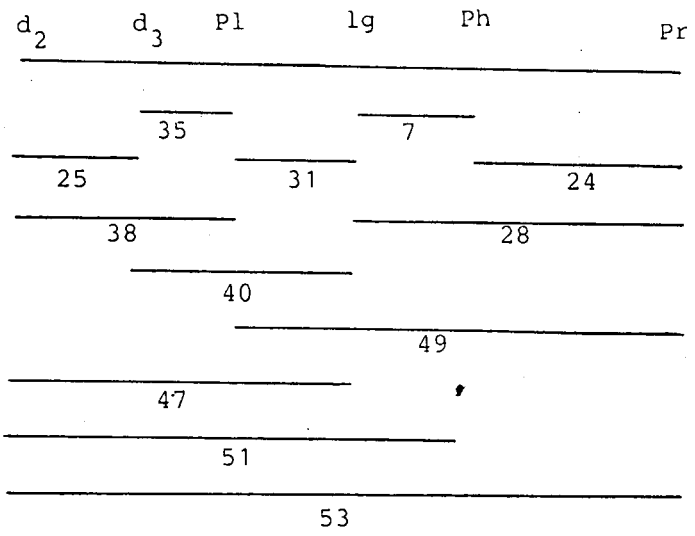


Iwata & Omura, 1971b

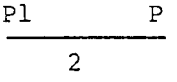


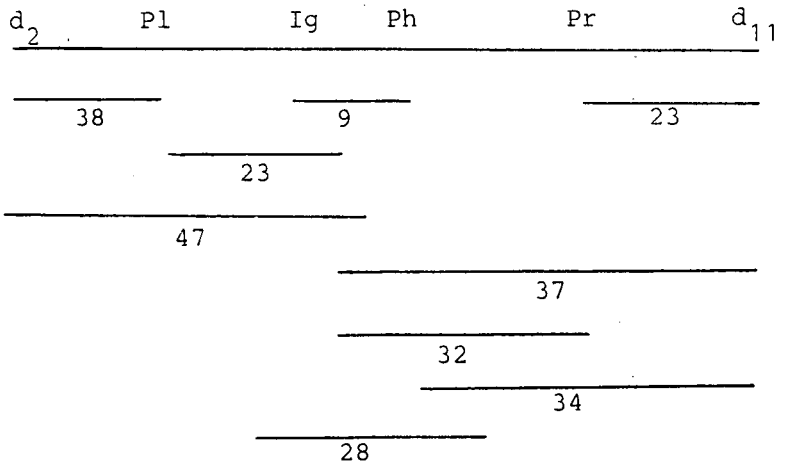
11 Takamure & Kinoshita, 1985

II 'Pl' Group

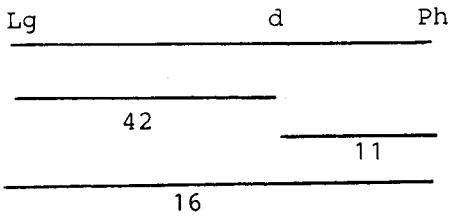


Nagao & Takahashi, 1960
Misro et al., 1966

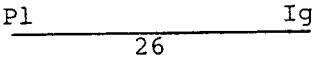




Iwata & Omura, 1971b

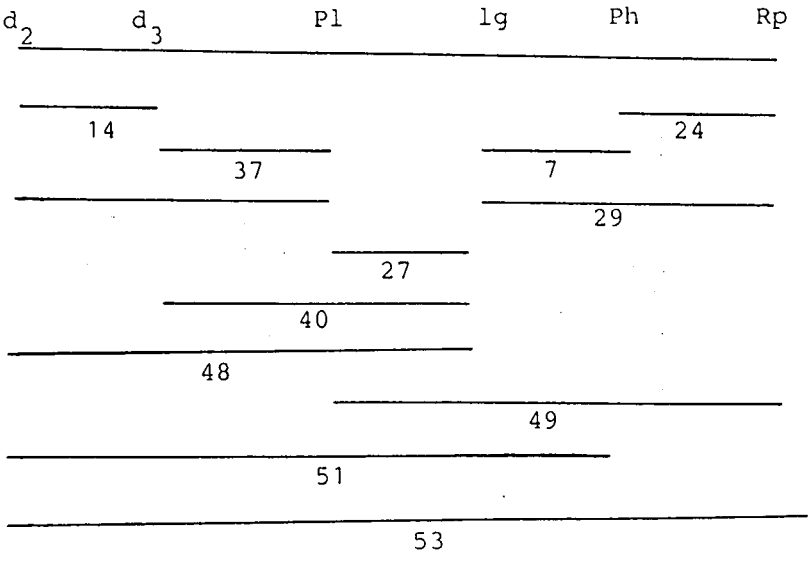


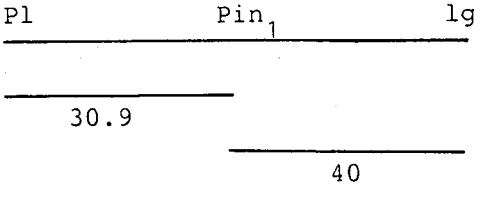
Hsieh & Yen, 1966



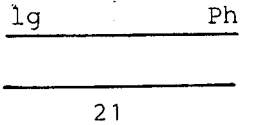
Shastry & Seetharaman, 1980

Nagao & Takahashi, 1952

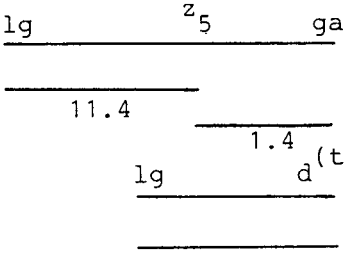




Mori et al., 1981

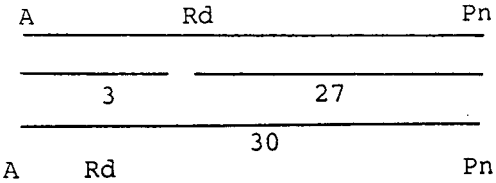


Morinaga et al., 1943

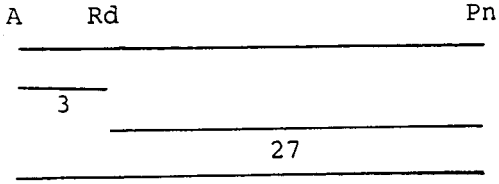


Kinoshita & Takamura, 1984

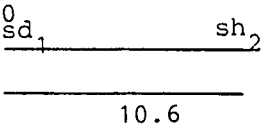
III 'A' Group



Nagao & Takahashi, 1960

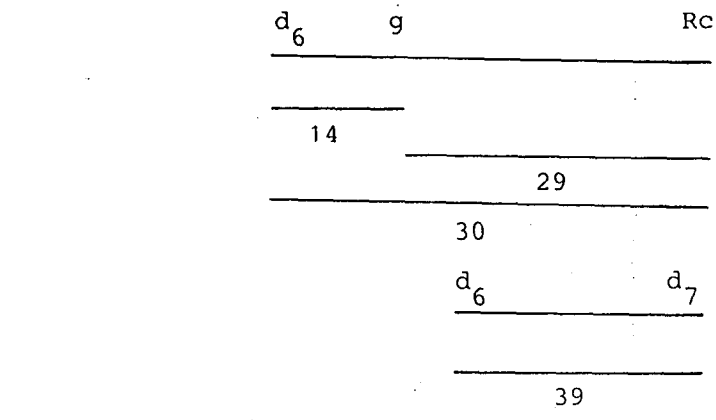


Takahashi, 1964

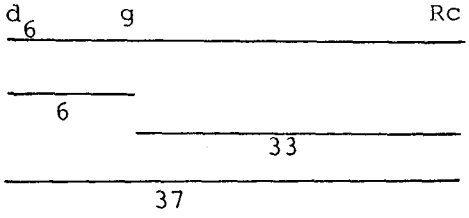


Oba & Kikuchi, 1989

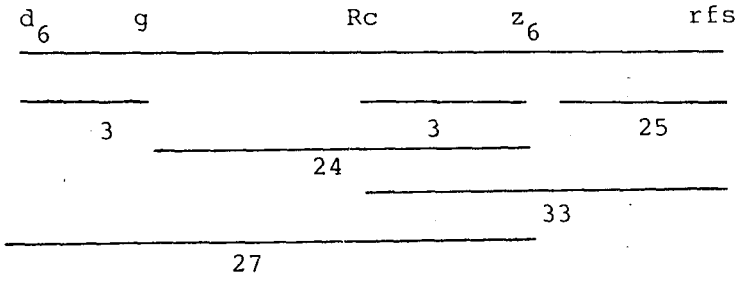
IV 'g' Group



Takahashi, 1964

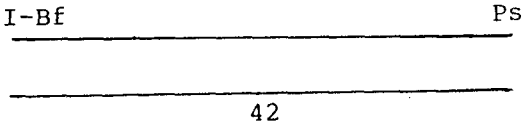


Iwata & Omura, 1971b



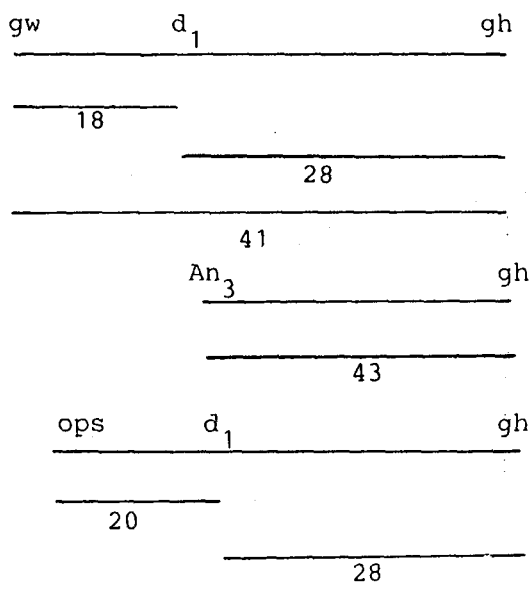
Maekawa, 1987

V '1-Bf' Group



Nagao & Takahashi 1960
Takahashi 1964

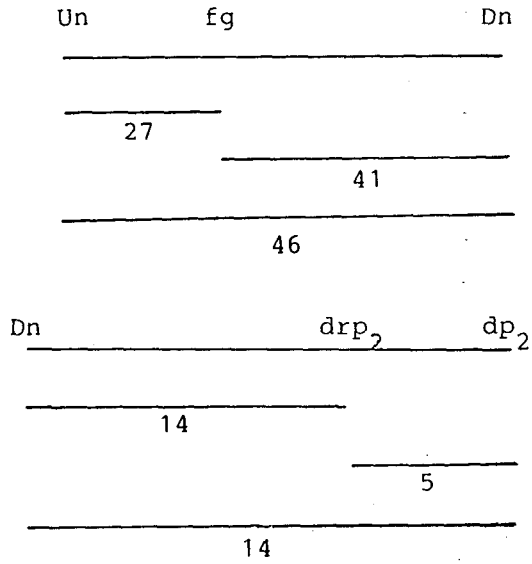
VI 'd' Group



Nagao & Takahashi, 1960
Takahashi, 1964

Iwata & Omura, 1971b

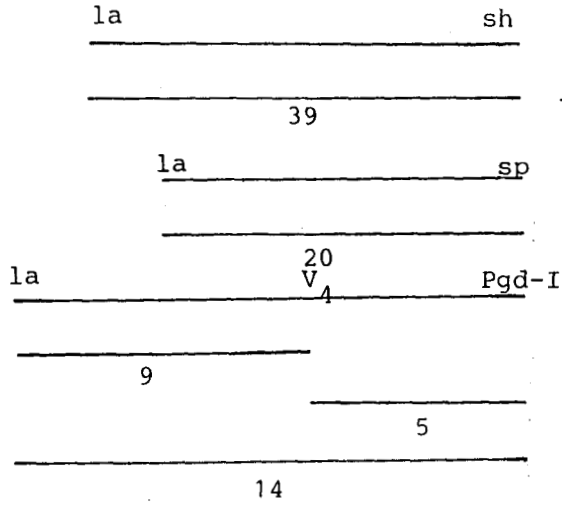
VII 'fs' Group



Nagao & Takahashi, 1960
Takahashi, 1964

Iwata & Omura, 1971

VIII 'la' Group

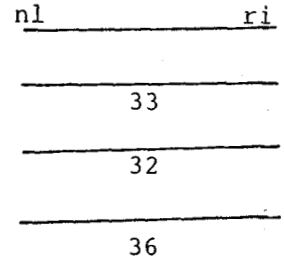


Nagao & Takahashi 1960-63
Takahashi, 1964
Misro et al., 1966

Iwata & Omura, 1971b

Ishikawa et al., 1988

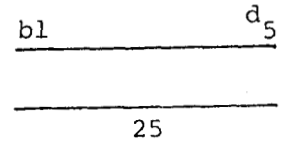
IX 'nl' Group



Nagao & Takahashi, 1960

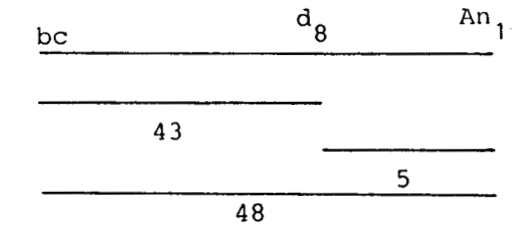
Takahashi, 1964
Misro et al., 1966

X 'bl' Group

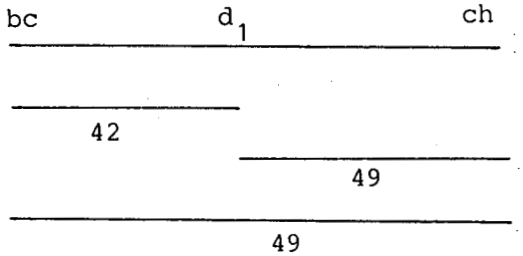


Nagao & Takahashi, 1960
Takahashi, 1964
Misra et al., 1966

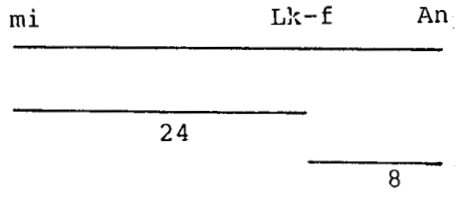
XI 'bc' Group



Takahashi 1964
Misro et al., 1966

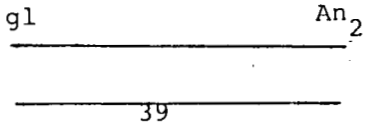


Iwata & Omura, 1971b

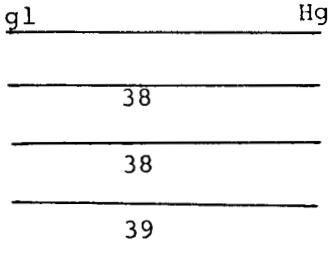


Takamure & Kinoshita, 1983

XII 'gf' Group



Takahashi 1964



Misro et al., 1966
Kinoshita & Takahashi 1968

translocation studies, assigned the gene fs, for fine stripe to I group. Kolhe and Bhat (1982) reported linkage of five anthocyanin genes, Psh (Ext) - Psh (Int) - Ps - AP and Pin, which correspond with sub-group 2 under I linkage of Jodon (1955-56), as reported by Takahashi (1964). Maekawa (1982) reported three complementary duplicate dominant genes for black hull (Bh_1, Bh_2 and Bh_3) and a gametophytic gene, ga, and reported linkage between ga and A, one of the basic genes for apiculus colour. The linkage relationship between clustered spikelets and the basic or one of the complementary genes for pigmentation in plant parts has been reported earlier (Jodon, 1948, 1956; Nagao and Takahashi , 1960; Seetharaman, 1964; Shyla, 1984; Annie, 1986 and Sukeskumar, 1990) (Fig. 1).

Morinaga and Nagamatsu (1942) reported linkage between purple leaf, ligulelessness and phenol staining and established the II linkage group. Dhulappanavar (1973b) located four genes and assigned them to linkage group II of Misro et al., (1966) in the sequence Plg-Plm-Pg-Pla. Misro (1981) modified the map as Plg-Pmr (Plm)-Pg-Plm (Pla) incorporating alternate symbols for genes observed by Dhulappanavar. Misro (1981) established linkage between a basic compelmentary gene for ligule colour Plg and one of the complementary genesfor pigmentation in mid-rib, Plm (Pmr), glume (Pg) and leaf apex-margin, Pla (Plm). Two new genes z^5 (zebra chlorosis) and zn (Zebra necrosis) were identified by Kinoshita and Takamure (1984) and established the linkage between z^5 and ga 10 (t), and

between lg and d (t). These linkages were assigned to the II linkage group. Tripathi and Rao (1985) suggested linkage between red pericarp Rd and grain dormancy, Gdr, with c.o. value = 32.61. They also reported Rd being linked with different characters, including short round grains, long palea, liguleless, phenol staining and anthocyanin pigmentation in various plant parts and were assigned to II, III and VIII linkage groups. Linkage between Spr (spreading panicle) and Pl (purple leaf), reported by Kinoshita and Takmure (1986) has been assigned to the II linkage group. Linkage relationship between lg and Ph with c.o = 16.78 was reported by Annie (1986) and assigned to the II linkage group (Fig. 1).

Linkage relationship between Pc and other six genes was first reported by Dhulappanavar (1973a) and located in the III group. The sequence of these genes is Pau-Pc-Pn-Psh-Pin-Pg-Pm in the 'Sp' group of Misro et al., (1966). Dhulappanawar and Hiremath (1974b) reported linkage between Psh-Px-Ps and between Pc-Px-Ps-Plm-Pin-Psh. It forms part of group III of Misro et al., (1966) but it was not included by Misro (1981). Dhulappanavar et al., (1975b) reported the linkage relationship between a pleiotropic basic gene for anthocyanin colour (A/C) and a complementary gene for purple septum with c.o. = 11.00 and was located in the III linkage group of Misro et al., (1966). Dhulappanavar (1976b) noted the interrelationship of genes Sk, Pg and P and assigned them in the sequence Sk-P-Pg to the

III group. The linkage relationship of genes for pigmentation in ligule, apiculus, stigma, and leaf axil was also reported by Dhulappanavar (1976a) and arranged them in the sequence Plg-Pa-Ps-Px. Misro (1981) adopted Pa for apiculus instead of CAP or AP or PA. Maekawa and Kita (1985) added two gametophytic genes to group III in the sequence ga1-dl-Bh-Pn-A-ga^{7}-lax. Maekawa (1986) studied the linkage relationship between Shp-1 Rd and A and assigned them to III group with recombination values of 2.4 ± 1.4% and 3.1 ± 1.6% respectively. Linkage between Px and Pa/Ps have been obtained with c.o = 5.0 and assigned to the III group (Annie, 1986). Sukeskumar (1990) reported linkage between Ph, I-An, $dp_{1/2}$ and bd and assigned them to the linkage group III along with other genes which were already located in the group III in the sequence I-An-Ph-dp-bd-$Pau_{a/b}$-$Pjp_{a/b}$-/$Pjf_{a/b}$-$Pc_{a/b}$-$Pn_{a/b}$-Px-Pa/Ps-$Psh_{a/b}$ (Fig. 1).

Rao and Misro (1968b) studied linkage between Ap and Ps with c.o. = 13.0 and assigned the genes to group IV. Rao (1968) reported linkage between red kernel and virescence with c.o. = 25.88 and suggested their assignment either to group I or IV of Misro et al. (1966). The linkage relationship of glume length, clustered grain, pericarp colour, palea colour, and uneven kernel shape was observed by Thakur and Roy (1975) and the authors located them to group IV in the sequence Ip_{1}-un_{b}-g-Rc. Dhulappanavar et al., (1975a) reported interrelationship of genes governing pigmentation in six plant parts and established the linkage between Ai-Pg--Ai-Pc--Ai-Pau and assigned

them to group IV. The linkage relationship between panicle density and anthocyanin pigmentation in coleoptile, internode, junctura and apiculus has been reported by Dhulappanavar (1977) and assigned to group IV of Misro *et al.*,(1966) in the sequence Pa--Lx--Pin--(Ai-Pc)--(Ai-P)--Pja. In another study Dhulappanavar (1979) reported linkage between ligule, auricle and junctura and assigned them in the sequence Ai-Plg--Ai-Pau--Pj - and located in the IV group (Fig. 1).

Ramiah (1937) and Bhattacharya (1957) updated interrelationship between *Sk*, *Prp*, *Pin* and *Pj* and located them in the V group. Linkage between *Prp* and *Psh* with c.o = 35.0 has been assigned to the linkage group V in the sequence Sk--Prp--Pin--Pj--Psh (Misro, 1981). Linkage between Prp and C/A with C.O = 23-25 has been established recently and assigned to the group V (Annie, 1986) (Fig. 1).

Linkage relationship between red pericarp (Rd) and gold hull (gh) has been reported (Jodon and Chilton, 1946) and assigned to the IV group. Sato *et al.*, (1982) located the marker genes gh and d1 (daikoku dwarf) and included in the VI group, while $n1^7$ (neck leaf) and $g1^7$ (glabrousness) were inlcuded in group IX and XII respectively (VI+1X of Kinoshita, 1984a). Iwata *et al.* (1984) reported that *bgl* (bright green leaf) was closely linked with *ri* in linkage group IX and with both *d1* and *ops* in linkage group VI. Based on this study the authors proposed combining the group VI and IX (Fig. 1).

Gene for dense panicle has been located in VII group of japonica (Nagao and Takahashi, 1959). Rao (1965) noted linkage between lax panicle (Lx) and grain size (Kl) with c.o = 7.67 and assigned them to VII group of indica, the sh group. The interrelationship of five genes I-Pg--glb--glh--I-Ps--Ap has been assigned to group VII (Panda et al., 1965) (Fig. 1).

Takahashi (1964) observed the gene sh(a) for grain shattering and located it in the VIII group - 'Lp' group. Rao and Misro (1968) reported a new gene Re (Red pericarp), being basic to Rc and Rd and established the linkage between genes R(e), Rk, Lp(a) and Sh(a) in the sequence Lp(a)--Rk--R(e)--Sh(a) in the VIII group (Fig. 1).

D'Cruz and Dhulappanavar (1963) observed linkage between Ps and Pg with c.o = 8 and between Pg and I-Pj with c.o = 40 and assigned them to the IX group, I-Pj group (Fig. 1).

Ramiah and Ramaswamy (1941) reported linkage between Ef and fh. Dhulappanavar (1975a) studied the relationship of genes governing pigmentation with flowering and established linkage between $Pjb_{a/b}$, one of the complementary genes for junctura back pigmentation, and Ef1 (Ef2), one of the duplicate genes for flowering and located these genes in X group. Linkage relationships between awning and nodal rooting (An-Nr), awning and spreading habit (An-Es),

nodal rooting and nodal differentiation (Es-Nr-Nd) and Es-Nd and An-Nd have been reported by Tripati and Rao (1985) and located in X linkage group of Indica (Fig. 1).

Rao and Misro (1965) reported linkage between Cl and Lx with c.o. = 5.0 and Misro (1981) located the genes Lx and Rk in the XI linkage group, Lx - group of indica rice. The linkage between $Rk\text{-}An_1$ and $Rk\text{-}An_2$ has been reported by Anandan (1933). Takamure and Kinoshita (1983) reported linkage between awning (An) and long grain (Lk-f) with c.o. = 7.6, between Lk-f and Mi with c.o.= 24.1 and between Lk-f and bc-1 with c.o = 19.2 and assigned them to XI group of Japonica. These genes occurred in the sequence, Mi--Lk-f-An. Kinoshita (1986) revised the linkage map of rice, adding 119 marker genes.

Breaux (1940) identified the gene gl for hairiness and was located in XII group by Nagao and Takahashi (1959) and to that of indica by Misro (1981), while it was placed in the VII group of Jodon (1955). Panda et al., (1965) reported linkage between I-Pg and I-Ps with c.o = 61.58 and linkage between glh and glb with c.o = 3.36. Further studies are needed for locating these genes to VII and/or XII group. Linkage between An and Pd with c.o = 38.83 and between Pd and Bd with c.o = 19.67 have been assigned to XII linkage group (Rao and Misro, 1986). Pavithran (1977) reported linkage relationship

between Nk (Notched kernel) and Rk (Short grain) and located to the XII linkage group (Fig. 1).

Eventhough several studies have been made on linkage groups of rice, several reported linkage associations of morphological characters remain yet to be assigned to appropriate linkage groups. These are briefly reviewed below.

The unassigned linkage relationships are between Lgp and Hg (Hector, 1922), fst (staminodal sterility) and fp (paleaceous sterility) Nagai (1926), wx and Ap (Ramiah et al., 1931), Ap, Wx and ma (Matsura, 1933), Pl and lg (Morinaga, 1938), Pj and Pg (Jodon,1948), Pg and Ap (Richharia et al., 1960; Misro and Kulkarni, 1965; Rao, 1965), AP-Hb/Hpb-Hf (Ramiah and Rao, 1953), CAP and Ps, Ps and Pin (Dubey, 1955; Richharia et al., 1960), Ntp and Prp and Ap-An/lg/J (Bhattacharya, 1957), Px and Pg/Pjb/Pla/Pin/Plg, Pj and Pla/Pg, I-JP and CAP, Pjb and Pg, Pjb and Pin, Pg and Pin (Nair, 1958 and Richharia et al., 1960), Px and Pj, Plg and Pg, Pla and CAP (Nair, 1958); Px and CAP, Px and Pin (Nair, 1958; Jodon, 1948), Plm and CAP (Richharia et al., 1960), Pg and Ps (D´Cruz 1960; Dhulappanavar, 1973a), Ap and Pg, Pg and Pau, Ap and Pau (Ghose et al., 1960), I-Pj and Pg/Ps (D´Cruz and Dhulappanvar, 1963), Pl and Ps, Ap and Pl (Takahashi, 1964), Px and Ps/An, Pr and Pin, Plm and Pg, CAP and Pin, Ps and An (Rao, 1965); Cl with inhibitor for sparse

awn I-An (Kadam and Pant, 1968), r and Rc (Rao, 1968); Glh_3 and bph_4, Sd and bh_4 (Sidhu and Khush, 1979); Psh and Psh(Int)-Ps-Ap-Pin (Kolhe and Bhat, 1979), I-St (inhibitor for straw colour) with Ce/Px, Straw lemma with flowering gene (Kadam, et al.,1980), Prp and SK (Ramiah, 1937); Prp and Pjp/Psh (Misro, 1981).

b. Pleiotropic genic relationship of morphological characters

Parnell et al. (1917) observed for the first time that purple lining of internode is closely associated with purple lemma-palea and purple leaf axil with purple stigma. Hector (1922) found that purple apiculus is always associated with purple leaf sheath or with leaf sheath and stigma or with leaf sheath, stigma and intermode or with glume, lemma - palea, leaf sheath, intermode, junctura and auricle or with stigma and internode. Ramiah and Rao (1953) considered it as close linkage or pleiotropy. The first evidence of pleiotropism in rice might be the report of Chao (1928), who found one of the complementary genes of apiculus colour is also responsible for purple stigma and purple sheath.

Nagao (1951) and Takahashi (1957) reported that the localization gene P causes anthocyanin pigmentation in apiculus and also acts as a basic gene for other localization genes in assocation with C and A. Setty and Misro (1973) postulated that any two of three dominant complementary genes produce pigmentation in leaf sheath, leaf margin, leaf tip, ligule and junctura back. They also reported that any two

of the three genes determine pigmentation in leaf axil, leaf axil extension and stigma. A localization gene Plm for leaf margin and tip is pleiotropic for ligule and junctura back pigmentation.

Pleiotropy of genes controlling anthocyanin pigmentation in leaf sheath, ligule, leaf blade and apiculus has been reported earlier (Dhulappanavar and Mensinkai, 1970). The basic gene A or A/C is pleiotropic in producing pigment in coleoptile, leaf sheath, ligule, auricle, node, internode, glume, lemma and stigma (Dhulappanavar et al., 1973c).

Further, Dhulappanavar (1975a) reported that A or C is pleiotropic to eleven plant parts such as coleoptile, leaf sheath, ligule, auricle, junctura, node, internode, leaf axil, glume, lemma and apiculus. Dhulappanavar et al. (1975a) reported that an inhibitory gene is pleiotropic for control of pigmentation in coleoptile, leaf sheath, auricle, leaf tip and node. Dhulappanavar (1979) reported two complementary pleiotropic genes for pigmentation in ligule, auricle and junctura, two duplicate pleotropic genes governing pigment in glume and another two duplicate pleiotropic genes for node and nodal ring pigmentation.

Dhulappanavar (1975a) reported that one of the duplicate genes for early flowering Ef_1 or Ef_2 is pleiotropic in producing

pigmentation in 14 plant parts and also that the gene showed differential pleiotropy. In ten plant parts such as coleoptile, leaf sheath, internode, septum, auricle, glume, ligule, leaf axil, stigma and apiculus, it acts as a complementary gene, while in four other plant parts such as junctura proper, junctura back, leaf blade and node it functions as inhibitory gene, thereby showing differential pleiotropy

Differential pleiotropic expression of genes has been reported earlier (Dhulappanavar, 1973b), where two pleiotropic genes act as complementary in producing pigment in junctura back, while acting as duplicate genes in leaf blade and also as inhibitory pleiotropic gene in producing pigment in junctura, junctura back, leaf blade and node. Dhulappanavar et al (1973b) also reported 3 genes common to produce pigmentation in junctura, pulvinus and leaf axil. Dhulappanavar and Hiremath (1974b) found that two duplicate complementary genes are common in coleoptile, leaf sheath, internode, septum, axil and stigma.

Goud and kullaiswamy (1984) made a review on pleiotropy and differential action of genes in qualitative characters, especially on pigmentation characters in rice. Shyla and Pavithran (1989) reported the pleiotropic interrelationship of genes for anthocyanin pigmentation in six plant parts such as Ps, Psh,Plm,Pa,Pau and Pla in rice.

c. Pleiotropic relation of quantitative morphological characters

Information on the pleiotropy of quantitative morphological characters has also been established. Sakai and Susuki (1964) reported that the number of panicles and panicle length are controlled mostly by the same pleiotropic genes with an inverse relationship.

Jennings and Beachell (1964) and Gangadharan and Misra (1976) surmised that the plant type concept might be an instance of successful association of many desirable characterstics under pleiotropic gene control. In another context Takeda (1985) reported the effects of glabrousness gene, gl-1, on agronomic traits and postulated that the gene has some pleiotropic effects on the development of panicles and spikelets.

Sakai and Suzuki (1964) stated that under the term pleiotropy one might include at present all possible varation of association of characters based on pathway differences in development of characters under pleiotropic control. Misro (1968) expressed the opinion that although the absence of cross over type is a proof of pleiotropy of the gene, the crucial test would be subjecting the material to X-ray to break down such association of characters.

Stebbins (1950, 1974) suggested the possible mutational origin of pleiotropism in plants or higher organisms. Postlethwait and Schneederman (1973) and Scandalios (1980) stated that although the exact mechanism by which the cell regulates differential expression of macromolecules during developmental cycle is not fully understood, the level of regulation would be either at the gene or in the pathway between the gene and the final product, the functional enzyme or protein. The catalaze-gene-enzyme system forms an attractive model for the study of post-translational regulation of differential gene expression in a complex eukaryote. Goud and Kullaiswamy (1984) indicated the possible role of a pleiotropic gene for a common enzyme possibly essential in different biochemical pathways operated by several other genes. Similarly, the synthetic pathways of anthocyanin production may be regulated by 2 or more common genes at different stages of synthesis.

E. MICROMUTATIONS AND INHERITANCE OF QUANTITATIVE CHARACTERS

The classic studies on quantitative inheritance refer to those by Johannsen (1903), Yule (1906) and Nilsson Ehle (1908), even though the idea is rooted deep in the original thoughts of Gregor Mendel on the "additive effects" of genes on floral colour variation (Strickberger, 1968).

East (1916) stated that minor variations within each group of

self-pollinating varieties were apparently caused by environmental differences, the large difference between the groups were undoubtedly genetic. The term polygene was first proposed by Mather (1941) in lieu of multiple factors or multiple alleles proposed earlier by Nilsson Ehle(1909) and East (1916).

Mather and Jinks (1982) and many others have stated that micromutation in agronomic characters has great significance in crop breeding. Oka et al. (1958), analysing induced micromutations in rice, reported no significant differences in mean values, although extensive variability was observed with the irradiated materials both for the positive and negative effects in the segregating population. Bateman (1959) observed no symmetrical induction of positive and negative mutations around the means. Heading date varied unidirectionally towards increased lateness, while plant height towards a positive directional increase. Tanaka (1968) observed panicle number and heading date showing shift in both positive and negative directions after gamma irradiation.

Studies on induced variability of quantitative morphological characters have shown the posibility of genetic manipulation of agronomic characters using mutation breeding (Gaul, 1958, 1964; Narahari, 1969; Basu and Basu, 1970; Nair, 1972; Nayar and Ninan, 1974; Roy and Jana, 1975; Gangadharan and Misra, 1976; Sreedharan, 1979; Mather

and Jinks, 1982; Hajra et al., 1986; Mohanan, 1988; Shobha, 1993). Chemical mutagenesis in relation to quantitative characters and the effects of radiation and other factors on mutagenesis have been reviewed by several authors in crop plants, including rice (Nayar, 1964; Fishbein et al., 1970; Jana and Roy, 1975; Omura and Sato, 1984). Sarawgi and Soni (1993) reported that mutagenic treatment did not affect plant height nor panicle length in M_1, but in M_2 higher doses of gamma rays and longer duration of EMS treatment increased panicle length, fertile spikelet per panicle and length/ breadth ratio. Lopez and Virmani (1990) reported the association of some isozyme markers with quantitative characters in rice and suggested that these isozyme loci can be used as markers of quantitative traits.

Mukai and Cockerham (1977) recorded that most of the induced variations must have arisen from mutations not of structural genes, but of controlling elements. This points to the fact that the controlling elements play a greater role than the structural genes in the mediation of continuous variation. It has been reported that the polygenes are no exception to mutations or recombinations and they also follow Mendelian principles in their inheritance like the major genes (Mather, 1941; Mather and Wigan, 1942; Clayton and Robertson, 1955; Paxman, 1957; Mather and Jinks, 1982). Mather and Jinks (1982) also pointed that the intragenic mutation and intergenic recombination will not be directly distinguishable, when genes have

physiologically small, similar and supplementary actions. However, information on the fundamental aspects of polygenes and their patterns of inheritance in relation to many agronomic characters in crop plants including rice is still scanty .

MATERIALS AND METHODS

MATERIALS AND METHODS

Present investigation included (1) mutagenic studies with different concentrations of EMS in rice with presoaking in distilled water or aqueous Vit. C. solution to elicit the combined effects if any, with reference to Vit. C and EMS, (2) genetics of mutants elicited therefrom and (3) studies on the effects of X-ray irradiated pollen grains used for pollination in a hybridization program in rice with particular reference to transformation of genetical ratios and genic interrelationships of morphological characters. The materials used for various experiments, methodology followed and statistical techniques used for analysis of the experimental data are described below under appropriate heads.

A. MUTAGENIC STUDIES OF JAPAN VIOLET WITH EMS AND GENETICS OF THE MUTANTS ISOLATED THEREFROM

1. Materials

Japan Violet, a popular local selection of Wynad supplied by the Rice Research Station (Kerala Agricultural University), Ambalavayal, Kerala, was originally used for making single plant panicle collection of pure seeds for the experiment. Japan Violet showed purple pigmentation in all plant parts except, junctura front,

junctura back and node with semi-dwarf stature. The chemical mutagen Ethylmethane sulfonate, said to be the most efficient (Loveless and Howarth,1959, Gaul, 1965, 1964) was used for treating seeds under different conditions of pre-treatments with distilled water/vit. C solution. Celin - a pharmaceutical formulation of Glaxo, Bombay, India, was used as vitamin source.

2. Procedure/Methodology followed

Pure seeds of uniform size and colour of Japan Violet were selected on single-plant-panicle basis and divided into two lots of 450 each for using 100 seeds per treatment finally. One lot was soaked in distilled water and the other in 0.01% aqueous Vit. C solution (Clinoy 1962, 1969; Chinoy and Saxena, 1971) for 12 h prior to treatments with different dosages of aqueous EMS solution. Details of mutagenic treatments are furnished in Table 1. The control (untreated) and treated seeds were washed thoroughly in running tap water for 2 h (Nair and Ninan, 1979; Shobha, 1993) before placing for germination in petridishes lined with wet filter paper. One hundred seeds were used per treatment and control and were followed up further for the studies as described below. The details of duration of experiment on mutagenesis are furnished in Table 2.

Table 1. Details of different mutagenic treatments given to Japan Violet with EMS and the subsequent population considered for study

Sl. No.	Variety	Pre-soaking solution	Pre-soaking/ treatment period	Mutagen concentration	No. of seed treated	Population studied in		
						M_1	M_2 lines/ total plants	M_3 families/ Total plant
1	Japan Violet	Distilled water	Untreated 12 h	Untreated (control)	100	100	100	100
2	"	"	12/12 h	EMS 0.5%	100	84	74/2885	155/2310
3	"	0.01% Vit. C	"	" 0.5%	100	78	64/2883	160/2440
4	"	DW	"	" 0.75%	100	76	64/1781	167/2338
5	"	0.01% Vit. C	"	" 0.75%	100	78	69/3056	188/2444
6	"	DW	"	" 1.0%	100	62	52/1687	147/3125
7	"	0.01% Vit. C	"	" 1.0%	100	69	62/2693	168/2600

Table 2. Details of the duration of experiments on mutagenesis of Japan violet with EMS

Sl No.	Particulars	Date/period
1.	Date of EMS treatment to Japan violet	10-12-1990
2.	Date of harvest of M_1^* seeds	18-03-1991
3.	Date of sowing of M_1 seeds	15-04-1991
4.	Period of harvest of M_2^* population	Late July 1991
5.	Date of sowing of M_2^* seeds	21-09-1991
6.	Period of harvest of M_3^* population	Late Dec. 1991
7.	Date of sowing of M_3^* seeds	22-1-1992
8.	Period of harvest of M_4^* population	Early May 1992
9.	Period of crossing and raising of F_1, F_2 and F_3 of Japan violet and its mutants	Jan. 1992- Feb. 1994

*M_1 - $M_{3/4}$ populations considered based on early workers (Gaul, 1964; Nair and Nainan, 1977 and others).

a. Study on germination

Rate (%), nature of germination of seeds and root/shoot-growth were observed in M_1, M_2, M_3 and in control. M_1 plants were raised on treatment-to-row method along with control.

b. Study of M_1 plants

M_1 and control plants (Table 1) were grown in 30.5 cm pots filled with soil and farm yard manure in 3:1 proportion under net house condition. Observations on variations induced by EMS treatments were recorded on different agronomically important morphological characters on single plant basis. On maturity the first four panicles each of the M_1 plants were harvested plant-wise. Panicles were sun-dried, observed and stored temporarily in packets till sowing to raise M_2 populations.

c. Study of M_2 generation

Seeds of M_1 plants were sown in well prepared nursery bed for raising the M_2 generation on plant-to-row method (Table 1). These were observed for percentage of germination, root-shoot ratio and survival percentage. One month old seedlings were transplanted to well-puddled field with usual farm yard manuring, following panicle-to-row and plant-to-row method in order to screen mutants in M_2 and

for observations on quantitative morphological characters of agronomic significance. Sterile mutants isolated were maintained in the net house effectively through vegetative propagation (Richharia et al., 1962, 1964, Pavithran and Richharia 1972, 1982) and utilized for further studies. At maturity M_2 plants were harvested plant-wise for post-harvest observations and for raising M_3 population.

d. Study of M_3 generation

M_3 populations (Table 1) were raised from the seeds of random M_2 plants on plant-to-row method for the genetic analysis of isolated mutants and also for studying quantitative morphological variation in M_3. Observations on morphological characters were recorded on individual plant basis as usual.

e. Hybridization between Japan Violet and its mutants

Seeds of Japan Violet and mutants (Table 3) were grown for hybridization separately. The source plant Japan Violet used for mutagenesis with EMS was crossed with different true bred recessive mutants isolated from different treatments for studying the genetic control of the mutants. Each cross was studied upto F_3 generation for confirmation as detailed in Table 3.

Table 3. Details of crosses between Japan violet and its mutants induced by EMS

Sl. No.	Cross		True bred Mutant derived from	Population studied
	Female parent	Male parent (Mutant)		
1	2	3	4	5
1.	Japan violet	x Tall recessive mutant	Vit.C+0.75% EMS M_2	F_1 - F_3
2.	Japan violet	x Beaked lemma depressed palea mutant	1% EMS - M_2	F_1 - F_3
3.	Japan violet	x Deformed palea mutant	Vit.C+0.75% EMS M_2	F_1 - F_3
4.	Japan violet	x Abnomorphic spikelet mutant	Vit.C+1% EMS M_2	F_1 - F_3
5.	Japan violet	x High tillering Dwarf mutant	Vit.C+0.75% EMS M_3	F_1 - F_3
6.	Japan violet	x Complete green mutant	Vit.C+1% EMS M_2	F_1 - F_3
7.	Japan violet	x Brittle culm mutant	0.75% EMS M_3	F_1 - F_3

1	2	3	4	5
8.	Japan violet	x Spotted leaf mutant	Vit.C+1% EMS M_3	$F_1 - F_3$
9.	Japan violet	x Chlorina leaf mutant	Vit.C+1% EMS M_2	$F_1 - F_3$
10.	Japan violet	x Multipistil mutant	Vit.C+0.5% EMS M_3	$F_1 - F_3$
11.	Japan violet	x Long sterile glume mutant	1% EMS M_2	$F_1 - F_3$
12.	Japan violet	x Procumbent mutant	1% EMS M_2	$F_1 - F_3$
13.	Japan violet	x Male sterile mutant	1% EMS M_2	$F_1 - F_3$
14.	Japan violet	x Striped mutant	Vit.C.+1% EMS M_2	$F_1 - F_3$

B. STUDIES ON THE EFFECTS OF X-RAY IRRADIATED POLLEN GRAINS USED IN HYBRIDIZATION OR TRANSFORMATION STUDIES WITH X-RAY IRRADIATED POLLEN GRAINS

1. Materials

The parental materials selected for studying the effects of X-ray irradiated pollen grains used for pollination/hybridization are Cherumodan and Japan Violet. The morphological descriptions of the varieties are furnished in Table 4. A normal cross, Cherumodan X Japan Violet, as control and other three crosses between them, wherein 1500, 2000 and 5000 rad X-ray irradiated pollen grains were used for pollination in the respective crosses, were studied.

The female parent Cherumodan is a local popular green upland rice which was available in the Department of Botany (Genetics Division) of the University of Calicut, Kerala, India and Japan Violet, a purple rice variety, used as a marker for anthocyanin pigmentation was obtained from the Ambalavayal Rice Research Station of Kerala Agricultural University, Trichur, Kerala, as stated earlier.

2. Methodology/Procedure followed

Pure seeds of Cherumodan and Japan Violet were sown in separate petridishes and a few one-week-old seedlings were transplanted to

Table 4. Morphological description of parental varieties used in hybridisation experiment

Sl. No.	Character	Cherumodan	Japan Violet
1.	Panicle height	108 cm	84 cm
2.	Panicle length	17.5 cm	19 cm
3.	No. grains/panicle	60	85
4.	Tillering habit	low	moderate
5.	Duration	80-85 days	90-95 days
Anthocyanin pigmentation			
6.	Leaf axil	P	P
7.	Leaf sheath	G	P
8.	Leaf lamina	G	P
9.	Leaf margin	G	P
10.	Leaf tip	G	P

Sl. No.	Character	Cherumodan	Japan Violet
11.	Junctura proper	G	P
12.	Junctura front	G	G
13.	Junctura back	G	G
14.	Ligule	G	P
15.	Auricle	G	P
16.	Node	G	G
17.	Internode	G	P
18.	Apiculus	G	P
19.	Stigma	G	P
20.	Awning	-	+
21.	Tipsterility	-	+

+ = present
- = absent
P = purple
G = Green

22.50 cm pots and a few in 15 cm polythene bags, filled with soil and farm yard manure in 3:1 proportion. Cherumodan was sown late by a week to synchronize flowering.

At the time of flowering, the control cross was made by pollinating the female plant with normal pollen (untreated) grains of Japan Violet by usual procedure. On the other hand, in the case of other crosses, pollen grains in the nearly-blooming spikelets were irradiated with X-rays of 1500, 2000 and 5000 rad respectively for pollinating the respective female plants. Each cross so made was followed up for F_1, F_2 and F_3 or M_1F_1, M_2F_2 and M_3F_3 generations respectively for genetical analysis. Details of the crosses, dosages of X-rays used for pollen irradiation and the populations studied are presented in Table-5. The procedure followed for the experiment is detailed below.

a. Hybridization procedure

Panicle/s which initiated blooming in the female parent were selected and spikelets that bloomed the previous day, if any, were removed carefully. The spikelets so selected were emasculated by gently opening the lemma-palea and removing the anthers carefully well before anthesis (6-8.00 am) by using a fine forceps. All other younger spikelets were removed from the panicle leaving the emasculated

Table 5. Details of crosses, dosages of X-rays used for pollen irradiation and generation studied

No.	Cross			Dosages of X-rays used for pollen irradiation	Generations* studied
	Female parent		Male parent		
1.	Cheromodan	x	Japan Violet (Normal cross)	0.00	F_1 - F_3
2.	Cherumodan	x	Japan Violet (1500)	1500 rad	M_1F_1-M_3F_3
3.	Cherumodan	x	Japan Violet (2000)	2000 rad	
4.	Cherumodan	x	Japan Violet (5000)	5000 rad	M_1F_1-M_3F_3

*Populations studied in each generation are detailed in Tables 39a & 40.

spikelets free intact.

These spikelets were pollinated subsequently (8-9 am) by dusting the pollen grains of the male parent/the X-ray irradiated pollen grains (see p.80) over the hairy stigmatic lobes of the female parent with the help of a fine brush. A hand lens of 10x was used to ascertain successful emasculation and pollination.

After pollination the panicle was carefully bagged with butter paper cover to protect from external contaminants and labelled properly with details of the cross made. The panicle was held intact with a stick. The butter paper cover was retained for a few more days after ascertaining the effectiveness of the cross the next day by holding the spikelets against sun light. The cross-seeds were harvested at 30 days maturity, dried in the sun and stored for sowing later to raise the F_1 plants.

b. F_1/M_1F_1 to F_3/M_3F_3 generations

Details of the crosses from date of sowing of parents to the date of harvest of the F_3/M_3F_3 population are furnished in Table 6.

The cross-seeds of each cross were sown in separate petridishes along with parental seeds as control. One-week-old seedlings were transplanted in 22.5 cm pots and, during growth, the F_1 hybrids were

Table 6. Details of crosses made and duration of growing F_1/M_1F_1 to F_3/M_3F_3 populations

Particulars of crosses	Cherumodan x Japan violet	Ch x JV 1500 rad	Ch x JV 2000 rad	Ch x JV 5000 rad
1	2	3	4	5
Date of sowing of parents	5-5-90 16-5-90	5-5-90 16-5-90	5-5-90 16-5-90	5-5-90 16-5-90
Flowering date of parents	6-7-90 5-7-90	6-7-90 7-7-90	7-7-90 4-7-90	6-7-90 5-7-90
Period of crossing	Early July 90	Early July 90	Early July 90	Early July 90
Total spikelets crossed	12	20	32	57
Date and No. of cross-seeds Harvested	7-8-90 8	10-8-90 5	10-8-90 7	12-8-90 5
Date of sowing of cross-seeds	28-8-90	28-8-90	28-8-90	28-8-90
No. of cross-seeds sown	5	4	7	5
No. of F_1 plants obtained	4	3	3	3
Date of flowering of F_1	5-11-90	6-11-90	5-11-90	14-11-90

1	2	3	4	5
Date of harvest of F_1	2-12-90	2-12-90	2-12-90	10-12-90
No. of F_1 seeds obtained	310	204	142	76
Date of sowing of F_1 seeds	25-12-90	25-12-90	25-12-90	25-12-90
No. of F_2 plants obtained	287	185	87	45
Period of F_2 harvest	26-3-91	26-3-91	28-3-91	28-3-91
Date of sowing of F_2 seeds	20-5-91	20-5-91	20-5-91	20-5-91
No. of F_3 families studied	30	46	30	25
Period of harvest of F_3	Late Aug. 1991	Late Aug. 1991	Late Aug. 1991	Late Aug. 1991

identified based on diagnostic parental characters. The F_1 or M_1F_1 plants and parent plants were harvested at maturity and the seeds stored temporarily for raising the F_2/M_2F_2 populations of the crosses. The F_1/M_1F_1 plants and the respective parents were observed for morphological characters at appropriate stages of growth (Misro, 1963).

F_2/M_2F_2 populations of the crosses were raised from the F_1/M_1F_1 seeds sown in shallow pans on plant-to-pan (plant-to-row) basis and transplanted to the field on plant-to-row basis with 23 cm x 30 cm spacing,under usual manurial conditions. Top-dressing was done with paddy mixture in recommended dosage at the tillering phase. Plants were observed on individual basis for pigmentation in plant parts and other morphological variations/characters. Plants of F_2/M_2F_2 were harvested at maturity plant-wise for post-harvest observations and also for raising the F_3/M_3F_3 families for confirmation of genetical ratios obtained in F_2/M_2F_2 for morphological characters.

The F_2/M_2F_2 plants of each cross were selected at random and their seeds were sown in the nursery to raise the F_3/M_3F_3 families on plant-to-row basis along with parents. 30 days-old seedlings were transplanted to well-puddled field later. Observations on various morphological characters were made as in previous generations on individual plant basis at appropriate stages of development.

c. Pollen grain irradiation with X-rays

The male parent Japan violet grown in polythene bags for treating the pollen grains with X-rays were transferred along with the female parent, prior to flowering, to the Department of Radiology, Medical College, Calicut, Kerala. Expectant spikelets (intact) for blooming on the day of treatment were exposed to X-rays emitting at a rate of 420 rad/min with the help of the X-ray machine-Maximar-100 of the Medical College, Calicut. The dosages given for the pollen treatment were calibrated to 1500, 2000 and 5000 rad (Table 5).

Hybridization was carried out later (during 8-9 am) with the pollens collected from the irradiated spikelets by dusting them over the female stigmatic lobes with a fine brush after ensuring successful emasculation of the female flowers. The plants were maintained at the Medical College and transferred to the net house of the University of Calicut after 10 days and cross-seeds were later harvested after 30 days of maturity.

3. Crosses and characters studied

The crosses effected and presently studied are Cherumodan x Japan Violet (control), Cherumodan x Japan Violet-1500 rad X-rayed pollens, Cherumodan x Japan Violet - 2000 rad X-rayed pollens and

Cherumodan x Japan Violet - 5000 rad X-rayed pollens (Table 5). The morphological characters observed (Misro, 1963) and studied in the cross/es are awning, tip sterility and anthocyanin pigmentation in leaf axil, leaf sheath, leaf blade, leaf tip, leaf margin, junctura proper, ligule, auricle, node, internode, apiculus and stigma (Table-4).

C. STATISTICAL ANALYSIS

1. Inheritance and interrelationship of genes

Chi-square test was applied to test the goodness of fit of individual segregation ratios for inheritance of morphological characters in F_2/M_2 or M_2F_2 generation and in F_3/M_3 or M_3F_3 for confirmation.

For analysis of interrelationship between character combinations, joint or combined segregation of character pairs was worked out with Chi-square test on the basis of expected independent assortment. Wherever the joint Chi-square value showed statistical significance, linkage was estimated on the F_2 data on minimum discrepancy (Haldane, 1953; Murthy, 1954, as applied by Richharia et al., 1966). In other situations, where joint Chi-square gave very high value and did not yield any linkage, pleiotropy was assumed and

Chi-square test for goodness of fit was applied on expected pleiotropic ratio to determine pleiotropy.

2. Micromutations/Quantitative variations

Micromutations or quantitative variations were studied by using frequency distribution of variants, analysis of variance and co-efficient of dispersion (variance index) of variation relative to control in Japan violet treated with different concentrations of EMS as detailed below.

a. Frequency distribution

The frequency distribution of variations in quantitative morphological characters in M_1, M_2 and M_3 generations of Japan Violet treated with different concentrations of EMS was recorded in respect of plant height, culm length, EBT, percentage of EBT, days to flower, panicle length, total spikelet per panicle, total grains per panicle, panicle density and percentage of spikelet sterility on random bulked population basis.

b. Analysis of Variance

Analysis of variance of morphological characters was done based

on the random selected samples of the treated populations in M_2 and M_3 of Japan Violet for the above mentioned nine morphological characters in comparison with their respective control. In the case of M_1 all survived plants were considered for the purpose.

c. Co-efficient of dispersion of variation relative to control or Variance index

Co-efficient of dispersion of variation (variance index) was calculated using the formula given below.

$$\text{Co-efficient of dispersion} \quad \frac{\bar{x} - i}{i}$$

where $\bar{x}$ = mean value for the treatments and i = control value.Degree of variance = Percentage of +/- variation relative to control

3. Mutagenic effectiveness and efficiency

Effectiveness and efficiency of the mutagens were calculated on the basis of chlorophyll mutation frequency in M_2 following the method of Konzak et al. (1965).

Mutagenic effectiveness was calculated from

Msp/tc or Msp/Krad and

Msd/tc or Msd/Krad

where

Msp = frequency of panicle mutation

Msd = frequency of seedling mutation

c = Concentration of mutagen in millimoles

t = treatment duration in hours

K rad = dosage of radiation used

Efficiency was calculated from the formula

Msp/L, Msp/S; Msd/L, Msd/S

where

L = Lethality (percentage reduction in seedling survival)

S = Sterility (percentage reduction in M_1 seed fertility)

Particulars of measurements

Plant height : Total height/length of the plant (cm) upto

leaf tip/panicle tip whichever is greater.

Culm length : Length of culm up to the basal node of the pancile (cm)

Days to flower : Number of days taken for emergence of inflorescence

Panicle density : Total spikelets/panicle divided by panicle length

Survival : Percentage of seedlings finally survived out of the total seeds germinated.

E.B.T : Ear bearing tillers: number and percentage

EXPERIMENTAL RESULTS

R E S U L T S

Present results comprise of (1) mutagenesis in an indica rice, Japan Violet, treated with different concentrations of Ethylmethane sulfonate, studied upto M_3 generation, (2) hybridization experiments using the cross Cherumodan x Japan Violet as control and the same cross combination using Japan violet pollen grains irradiated with 1500, 2000 and 5000 rad X-rays studied upto F_3/M_3F_3 generations and (3) 14 crosses involving respective mutants isolated from the mutagenic studies and the parental source variety Japan violet studied upto F_3 generation as detailed elsewhere (Table 3, 5 & 6).

The morphological characters studied upto M_3 generation in mutagenesis with Japan violet included 19 morphological mutants isolated and certain morphometric characters like plant height, culm length, total tillers, EBT, EBT %, days to flower, panicle length, total spikelets/ panicle, number of grains/panicle, percentage of sterility and panicle density.

The morphological characters studied in hybridization using irradiated pollen grains included 12 anthocyanin pigmentation characters, awning and tip-sterility.

The mutant morphological characters studied upto F_3 generation in the 14 crosses are recessive tall, beaked lemma-depressed palea, procumbent plant, deformed palea, long sterile glume, high tillering dwarf, anther sterile, spotted leaf, chlorina leaf, brittle culm, complete green, multipistil, broad seed and striped leaf. The results on mutagenesis, inheritance and interrelationships of the characters studied are presented in detail under appropriate heads as follows.

A. MUTAGENIC EFFECTS OF EMS ON JAPAN VIOLET

The mutagenic effects of 0.5, 0.75 and 1% of EMS on Japan violet pre-soaked for 12h in distilled water with or without 0.01% Vitamin C are described below.

1. Rate of germination

Data on seed germination in Japan Violet, pre-treated with distilled water/Vitamin C solution of 0.01% and treated with 0.5%, 0.75% and 1% aqueous solution of EMS are furnished in Table 7. The control was germinated in distilled water and maintained along with the treatments.

The rate of germination in M_1 showed considerable reduction in all treatments. Greater reduction of 17% was observed with DW + 1% EMS treatment, while in other cases 8-12% reduction was observed, the

Table 7. Mean values of rate of germination, seedling growth, surival, % of reduction in germination and % of reduction in seedling survival in M_1, M_2 and M_3 of Japan violet treated with different concentrations of EMS and in combination with 0.01% vitamin C pre-treatment.

			Control			DW + 0.5%EMS			Vit.C + 0.5% EMS			DW + 0.75% EMS			Vit.C + 0.75% EMS			DW + 1% EMS			Vit.C + 1% EMS		
Sl. No.	Character	Variety	M_1	M_2	M_3	M_1	M_2	M_3	M_1	M_2	M_3	M_1	M_2	M_3	M_1	M_2	M_3	M_1	M_2	M_3	M_1	M_2	M_3
1	Germination %	Japan violet	100.00	98.00	100.00	92.00*	67.56*	86.30*	90.00*	70.27*	86.45*	90.00*	59.67*	76.56*	89.00*	55.08*	72.56*	83.00*	49.83*	79.68*	88.00*	50.83*	79.63*
2.	Root length, cm	"	10.32	9.02	8.10	10.97	7.39*	7.98	11.46*	7.71*	5.90*	6.52*	6.81*	8.27	7.25*	7.37*	4.67*	6.00*	4.91*	8.4	4.98*	6.87*	7.24*
3.	Shoot length, cm	"	7.18	5.44	4.89	7.48	4.90	5.96	7.33	4.78	4.67	4.69*	3.40*	5.93*	4.59*	4.96	4.41*	3.91*	3.35	5.16*	2.97*	5.11*	6.56*
4.	Root/shoot ratio	"	1.44	1.66	1.66	1.47	1.51	1.34	1.56	1.61	1.26	1.39	2.00*	1.39	1.58	1.49	1.06*	1.53	1.47	1.63	1.68*	1.34	1.10*
5.	Survival %	"	100.00	98.50	98.00	92.39*	96.25	96.03	86.67*	98.41	96.27	83.33*	98.17	95.50	78.65*	94.47	96.38	74.70*	97.23	96.64	84.09*	98.24	96.00
6.	% of reduction in germination	"	0.00	2.00	0.00	8.00	31.06	13.98	10.00	28.30	13.55	10.00	42.17	23.44	11.00	43.80	27.44	17.00	49.15	20.32	12.00	47.17	20.32
7.	% of reduction in seedling survival	"	-	-	-	7.61	2.28	2.01	13.33	0.009	1.80	16.67	0.003	2.62	21.35	4.09	1.65	25.30	1.29	1.39	15.91	0.003	2.00

* Significant at 5% level

o See page 85

least with DW + 0.5% EMS treatment in comparison with 100% germination in the control (Table 7).

In M_2 the percentage of germination was further reduced in all treatments. Maximum reduction was observed in DW + 1% EMS with 49.15% germination. In other cases percentage of germination was reduced to 31.06%, 28.30%, 42.17%, 43.80% and 47.17% with the respective treatments of DW + 0.5%, Vit. C + 0.5%, DW + 0.75%, Vit. C + 0.75% and Vit. C + 1% EMS, in comparison with 2% reduction in control (Table 7).

However, in M_3 the percentage of germination was increased than in M_2 in all cases. Maximum reduction was observed in the treatment of Vit. C + 0.75% EMS with 27.44% and least reduction was in Vit. C + 0.5% EMS treatment with 13.55%. In other cases it ranged from 13.98 - 23.44 as furnished in Table 7.

Among the three generations greater reduction was observed in M_2 of all treatments. In M_3 the rate of germination was enhanced than in M_2 but lesser than in M_1 generation in all treatments (Table 7).

2. Cleisto-viviparous germination

Out of 74 survived M_1 plants of the treatment with 0.5% EMS, 5

showed abnormal cleisto-viviparous germination during the rainy season, while 28% control plants showed normal vivipary (Fig. 2a).

Out of 347 seeds obtained from the above 5 plants, 43 seeds germinated viviparously and 43.8% showed cleisto-viviparous germination of three types.

In the first type plumule penetrated through the lemmar space horizontally and emerged through the anterior end of the grains (Fig. 2b), thereby exhibiting cleistopary. This cleistopary coupled with viviparous germination being a unique situation could be termed as cleisto-vivipary; 11.63% of the germinated seeds showed this type of germination. In the second type both plumule and radicle traversed through the lemmar space and emerged through the anterior end of the grain or through the side of the grain by break-opening the lemma-palea (Fig. 2d); 32.56% of seeds showed this type of germination. In the third type, plumule grew more strongly and pushed the radicle and endosperm out of lemma-palea causing ejection of young seedlings (Figs. 2 e & f); 4.65% seeds showed this type of germination. In the first two types kernel appeared slightly reduced in size and laterally compressed, probably, due to the pressure exerted by the horizontally emerging shoot. In the third type kernel appeared to be normal.

Fig. 2. Cleisto-viviparous germination in Japan violet

a. Viviparously germinating panicle of Japann violet

b. Cleistoparous germination type I with normal radicle - emergence

c. Different stages of normal (upper row) and cleistoparous germination (lower row) type II showing anterior emergence of both plumule and radicle

d. Single grain with cleistoparous emergence, type II enlarged

e. Cleistoparous germination type III in which extrusion of endosperm and radicle occurs as a result of torsion by the emerging plumule

f. The fallen seedling with naked endosperm resulted from type III.

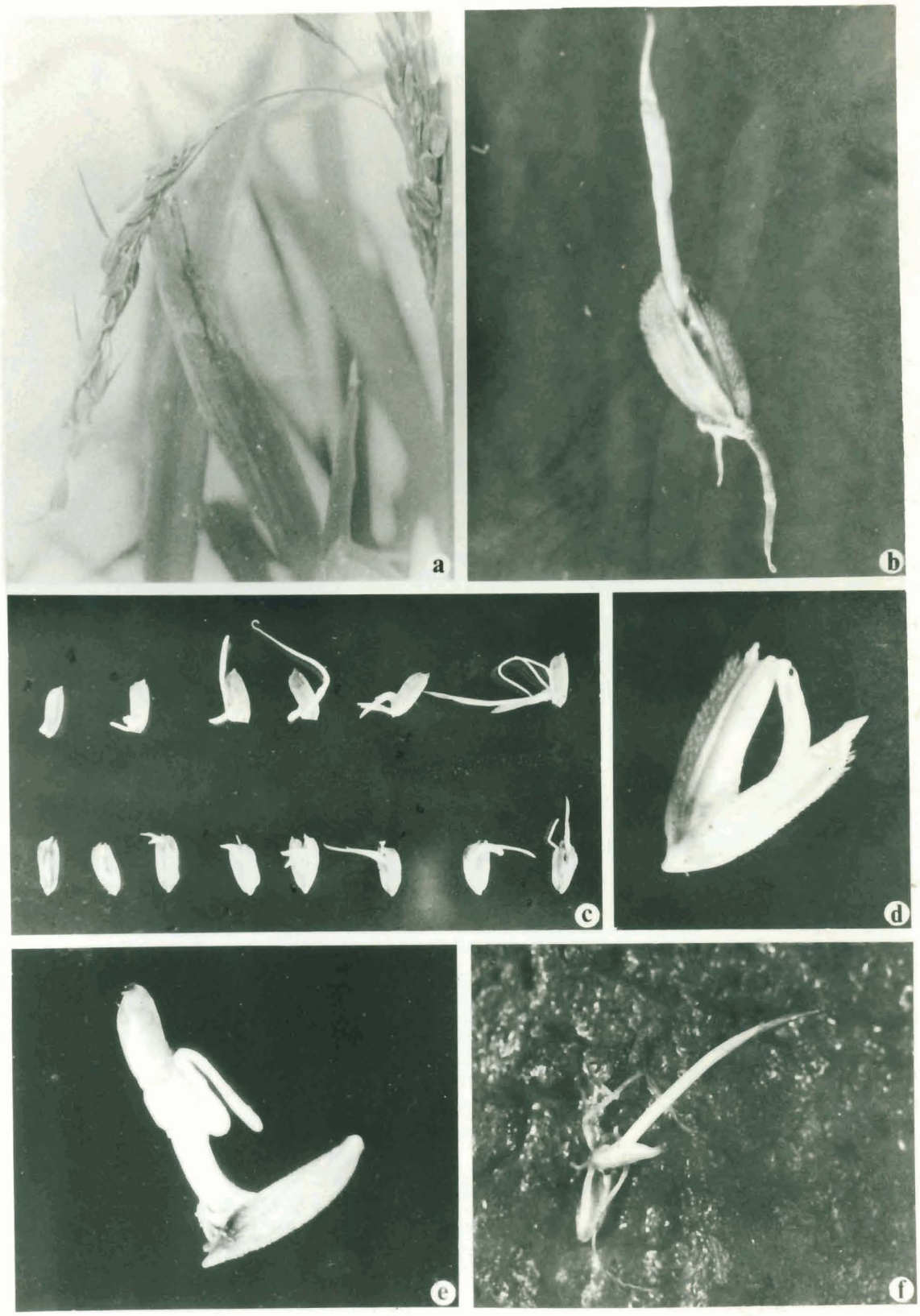


Fig 2

3. Root-shoot growth and root/shoot ratio

Root length showed statistically significant variation in M_1, M_2 and M_3 of most treatments (Table 7). Root length reduced in response to increase in concentration of the mutagen. Maximum reduction was in M_1 of Vit. C + 1% EMS with 51.74% and in M_2 of DW + 1% EMS with 45.56% and in M_3 of Vit. C + 0.75% EMS with 42.35% (Table 8).

Shoot length showed significant variation in all treatments except DW + 0.5% and Vit. C + 0.5% treatments (Table 7). Maximum reduction in M_1 was shown by Vit. C + 1% treatment with 58.64%, in M_2 by DW + 1% EMS with 38.42% and in M_3 by Vit.C + 0.75% EMS with 9.82% (Table 8). Plumule growth was more affected by the treatments than the radicle growth. Maximum inhibition of plumule was observed with DW + 1% EMS treatment and maximum plumule and radicle inhibition was observed with Vit. C + 0.75% EMS treatment in M_1 (Tale 9)

Root/shoot ratio showed significant variation in M_2 of DW + 0.75% EMS, DW + 1% EMS and in M_3 of Vit. C + 0.75% EMS and in M_1, M_2 and M_3 of Vit. C + 1% EMS. In the case of DW + 0.5% treatment and Vit. C + 0.5% treatment variation appeared to be not significant in any of the generations (Table 7).

Table 8. Mean percentage of reduction or increase in root and shoot length due to different concentrations of EMS and in combination with 0.01% Vitamin C pre-treatment

Treatment	M_1				M_2				M_3			
	RL		SL		RL		SL		RL		SL	
	-	+	-	+	-	+	-	+	-	+	-	+
DW+0.5% EMS	0.00	6.30	0.00	4.18	18.07	0.00	9.93	0.00	1.48	0.00	0.00	21.80
Vit. C+0.5% EMS	0.00	11.05	0.00	2.05	14.52	0.00	12.13	0.00	27.16	0.00	4.50	0.00
DW+0.75% EMS	36.82	0.00	34.68	0.00	24.50	0.00	37.50	0.00	0.00	2.10	0.00	21.27
Vit.C+0.75% EMS	29.74	0.00	36.07	0.00	22.39	0.00	8.82	0.00	42.35	0.00	9.82	0.00
DW+1% EMS	41.86	0.00	45.54	0.00	45.56	0.00	38.42	0.00	0.00	3.70	0.00	5.52
Vit.C+1% EMS	51.74	0.00	58.64	0.00	23.84	0.00	6.07	0.00	10.62	0.00	0.00	34.15

RL - Root length
SL - Shoot length

- reduction
+ increase

Table 9. Number of seeds showing radicle and plumule or radicle-cum-plumule inhibition in M_1 due to different treatments with EMS

Sl No.	Variety	R/P/ R&P	Control	DW+0.5% EMS	Vit.C + 0.5% EMS	DW+0.75% EMS	Vit.C + 0.75% EMS	DW+1% EMS	Vit.C + 1% EMS
1.	Japan violet	R	0.00	0.00	0.00	1.00	0.00	0.00	3.00
2.	"	P	0.00	0.00	5.00	6.00	3.00	7.00	4.00
3.	"	R&P	0.00	3.00	7.00	9.00	12.00	8.00	8.00

R = Radicle R & P = Radicle-cum-plumule

P = Plumule

4. Survival of seedlings

Rate of seedling survival after 30 days was recorded in M_1, M_2 and M_3 of Japan Violet. Observations are furnished in Table 7. Results showed reduction in seedling survival in M_1 of all treatments with maximum reduction of 25.3% in DW + 1% EMS treatment. In other cases it ranged from 7.61 - 21.35%. In M_2 and M_3 percentage of seedling survival was more than in M_1 (Table 7).

5. Mutagenic effectiveness and efficiency of EMS

Mutagenic effectiveness and efficiency of Ethylmethane sulfonate (EMS) in relation to the pre-treatment with distilled water/0.01% Vit C in Japan violet are presented in Table 10.

Effectiveness (rate of chlorophyll mutation in relation to dosages) and efficiency (rate of chlorophyll mutation in relation to other biological effects) of the mutagen were calculated on the basis of panicles and seedlings in M_2. The data are presented in Table 10.

Seeds received pre-treatment with 0.01% Vit. C solution before EMS treatment were affected more by EMS treatment than those had no pre-treatment. Vit. C + 1% EMS treatment showed maximum effectiveness

Table 10. Mutagenic effectiveness and efficiency of different concentration of EMS and in combination with 0.01% Vitamin C pre-treatment in Japan Violet

				Percentage of				Mutagenic effectiveness		Mutagenic Efficiency			
Sl. No.	Pre-treatment/ mutagen	Treat-ment duration	Con-centra-tion	Letha-lity (L)	Steri-lity (S)	Mutated principles (Msp)	Total mutated seedlings (Msd)	MSP/tc	Msd/tc	Msp/L	Msp/S	Msd/L	Msd/S
1	DW+EMS	12/12 hr	0.5%	7.61	43.48	0.00	0.38	0.000	0.0008	0.00	0.00	0.05	0.009
2	V.C+EMS	12/12hr	0.5%	13.33	47.20	0.00	0.59	0.000	0.0012	0.00	0.00	0.04	0.013
3	DW+EMS	12/12hr	0.75%	16.67	45.97	0.68	1.01	0.0009	0.0014	0.04	0.02	0.06	0.022
4	V.C+EMS	12/12hr	0.75%	21.35	35.91	2.56	1.03	0.004	0.0014	0.12	0.07	0.05	0.029
5	DW+EMS	12/12hr	1%	25.30	28.70	4.90	0.71	0.0051	0.0010	0.19	0.17	0.03	0.025
6	V.C+EMS	12/12hr	1%	15.91	23.57	6.18	1.49	0.0064	0.0015	0.39	0.26	0.09	0.063
7	Control	--	--	0.00	20.39	0.00	0.00	0.000	0.000	0.00	0.00	0.00	0.000

on the basis of mutated panicles (0.0064) and seedlings (0.0015). In the case of efficiency, Vit. C + 1% EMS treatment showed maximum efficiency for mutated spikelets (0.39 and 0.26) or mutated seedlings (0.09 and 0.06) on the basis of M_1 sterility and lethality (Table 10).

B. MACROMUTATIONS INDUCED BY EMS TREATMENTS

Macromutations of Japan violet were derived from M_2 or M_3 generations following various treatments of EMS (Table 11). The mutants thus obtained were subsequently crossed with the original source variety, Japan Violet for studying inheritance. The occurrence, description and pattern of inheritance of the mutant characters are detailed below under appropriate heads.

1. Chlorophyll mutations/leaf mutations

Four types of chlorophyll mutations such as albinos, lethal yellow, striped and chlorina leaf were isolated from M_2 generations of various treatments and certain others like, purple spotted, partial green and complete green mutant isolated from the treatments at M_3 level as detailed below.

a. Albino

Albinos were induced by almost all treatments of EMS and it

Table 11. Morphological macromutations obtained from M_1, M_2 and M_3 populations of Japan Violet treated with different concentrations of EMS and in combination with 0.01% Vit. C pre-treatment.

Sl. No.	Treatment	Source	Generation		
			M_1	M_2	M_3
1.	Control	Japan Violet	-	-	-
2.	DW + 0.5% EMS	,,	-	albino, lethal yellow, chlorina, anther sterile	-
3.	Vit. C + 0.5% EMS	,,	-	albino, lethal yellow, striped mutant	multipistil mutant,
4.	DW + 0.75% EMS	,,	-	albino, striped mutant, lethal yellow	-
5.	Vit. C + 0.75% EMS	,,	-	albino, striped mutant, tall recessive, deformed palea	high tillering
6.	DW + 1% EMS	,,	-	albino, beaked lemma-depressed palea, lethal yellow, long sterile glume, procumbent, broad seeded, mutant, grassy rhizomatous	partial green mutant
7.	Vit. C + 1% EMS	,,	-	albino, abnomorphic spikelet, striped mutant, chlorina, anther sterile mutant	brittleculm mutant, complete

DW - Distilled water
Vit. C - Vitamin C

mostly occurred in the M_2 generation (Table 11). Observations on the number and percentage of segregating M_2 lines of Japan Violet subjected to different pre-treatments and treated with various concentrations of EMS are presented in Table 12. As inferred from the segregating M_2 lines for albinos, 0.5% EMS with distilled water pre-treatment induced albinism in 2.70% segregating lines. Treatments of 0.75% EMS with distilled water pre-treatment and 1% EMS with Vit. C pre-treatment induced albinism in the highest range of 9.38% and 12.90% respectively. 1% EMS with distilled water pre-treatment showed low induction of albinism in 1.92% segregating line. However, 0.5%, 0.75% and 1% EMS with Vit. C pre-treatment resulted in linear increase of 3.13%, 7.25% and 12.90% segregating lines for albinos respectively (Table 12). On the whole, Vit. C pre-treatment with EMS dosages showed more increase in the rate of induction of albinism except in the case of 0.75% EMS. However, distilled water + 0.75% EMS showed an optimum level of induction of albinism as indicated by the drop at DW + 1% EMS treatment (Table 12), Comparatively, induction of albinism appears feasible with all the treatments of EMS, with or without Vit. C, as no other mutation recorded in the present study had the universality of occurrence with the different treatments studied.

All the albinos showed characteristically similar phenotype with 3-4 non-chlorophyllated or white leaves and were short-lived for 7-10 days. The albinos also showed fading light purple shade due to anthocyanin pigmentation, as the original parent, Japan Violet, had

Table 12. Number and percentage of M_2 lines of Japan Violet segregated for different mutants produced by different treatments with EMS

Sl. No.	Mutant character	Treatment					
		DW + EMS			Vit. C + EMS		
		0.5%	0.75%	1%	0.5%	0.75%	1%
1	2	3	4	5	6	7	8
1	Albino	2 2.70%	6 9.38%	1 1.92%	2 3.13%	5 7.25%	8 12.9%
2	Lethal yellow	1 1.35%	3 4.69%	1 1.92%	1 1.56%	-- 0.0%	-- 0.0%
3	Striped mutant	- 0.0%	- 0.0%	0 0.0%	1 1.56%	1 1.45%	1 1.61%
4	Chlorina (with purple shade)	1 1.35%	- 0.0%	- 0.0%	- 0.0%	- 0.0%	1 1.61%
5	Beaked lemma depressed palea	- 0.0%	- 0.0%	1 1.92%	- 0.0%	- 0.0%	- 0.0%
6	Long sterile glume	- 0.0%	- 0.0%	1 1.92%	- 0.0%	- 0.0%	- 0.0%
7	Deformed palea	- 0.0%	1 1.56%	- 0.0%	- 0.0%	- 0.0%	- 0.0%
8	Procumbent	- 0.0%	1 0.0%	1 1.92%	- 0.0%	- 0.0%	- 0.0%
9	Abnomorphic spikelet	- 0.0%	- 0.0%	- 0.0%	- 0.0%	- 0.0%	1 1.61%
10	Broad seed	- 0.0%	- 0.0%	1 1.92%	- 0.0%	- 0.0%	- 0.0%
11	Tall recessive	- 0.0%	- 0.0%	- 0.0%	- 0.0%	1 1.45%	- 0.0%
12	Grassy rhizomatous	- 0.0%	- 0.0%	1 1.92%	- 0.0%	- 0.0%	- 0.0%
13	Anther Sterile	- 0.0%	1 1.56%	- 0.0%	- 0.0%	- 0.0%	2 3.23%

1	2	3	4	5	6	7	8
14	Total M_2 lines segregated	4	11	7	4	7	13
15	Total M_2 lines studied	74	64	52	64	69	62
16	Total % of segregating M_2 lines	5.40%	17.19%	13.46%	6.25%	10.14%	20.97%

purple pigmentation (Fig. 3).

Data on the pattern of inheritance of albinos of Japan Violet are presented in Table 13. One out of 74 M_2 lines of DW + 0.5% EMS treatment, 3 out of 64 M_2 lines of DW + 0.75% EMS treatment, 1 out of 64 M_2 lines of Vit. C + 0.5% EMS treatment, 5 out of 64 M_2 lines of Vit.C+0.75% EMS treatment, 1 out of 62 M_2 lines of Vit. C + 1% EMS treatment segregated for monogenic ratio 3:1 with recessive genic control of albinism.

However, 3 out of 64 M_2 lines of DW + 0.75% EMS treatment, 1 out of 52 M_2 lines of DW + 1% EMS, 1 out of 64 M_2 lines of Vit. C + 0.5% EMS and 7 out of 62 M_2 lines of Vit. C + 1% EMS segregated for the ratio 15:1 indicating duplicate recessive control of albinism as shown in Table 13. However, the preponderance of albinos or higher rate of mutation has occurred with EMS treatment preceded by Vit. C pre-treatment.

No M_3 confirmation could be obtained for albinos in the M_3 populations studied except in the two cases of DW + 0.75% EMS treatment and Vit. C + 0.75% EMS treatment (Table 14). The absence of albinos in the M_3 families might, probably, be due to genotypic elimination of the albino phenotypes in the small population that could be studied.

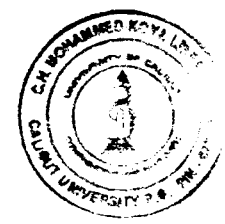


Fig. 3. Purple shaded albino seedlings.

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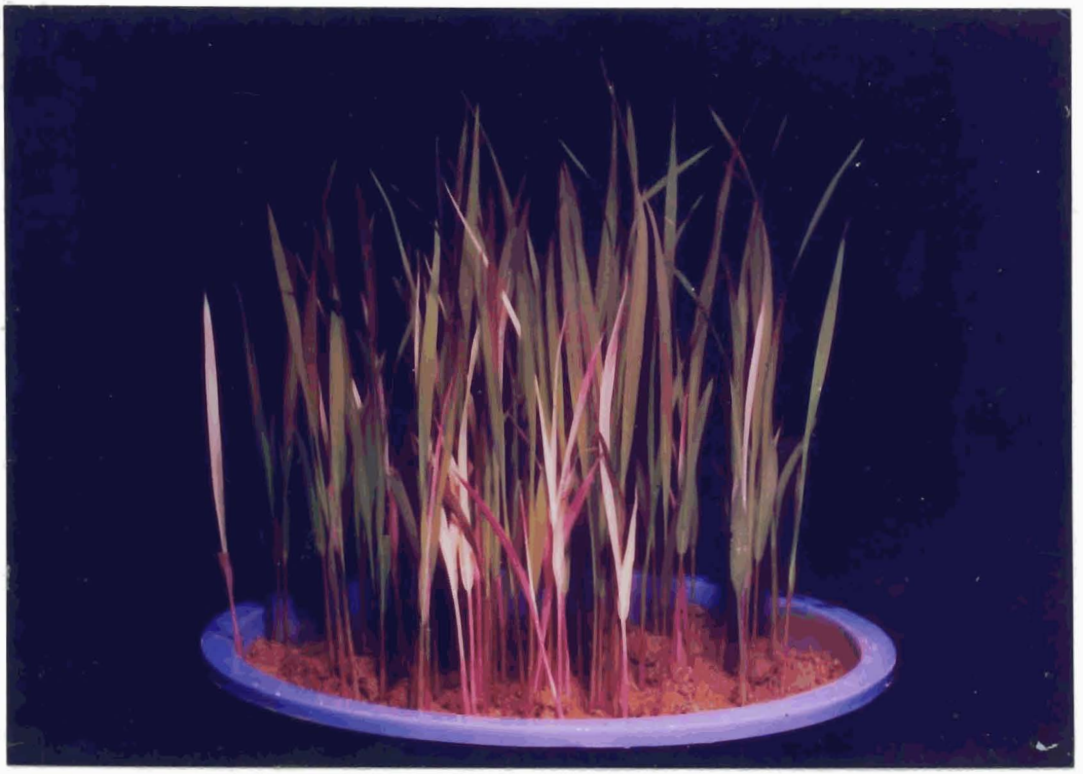

Fig 3

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Table 13. Details of segregation for albinos in M_2 lines of EMS treated Japan violet

Sl. No.	Mutant Character studied	Treatment No.	Line No	M_1	O/E	M_2 Frequency -	M_2 Frequency +	Total	Ratio	X^2	P
1	2	3	4	5	6	7	8	9	10	11	12
1.	Albino	I	8-2	N	O	12	2	14			
					E	10.5	3.5	14	3:1	0.85	0.50-0.30
2.	"	I	20-1	N	O	31	1	32			
					E	30	2	32	15:1	0.53	0.50-0.30
3.	"	II	88-2	N	O	14	3	17			
					E	12.75	4.25	17	3:1	0.49	0.50-0.30
4.	"	II	116-1	N	O	42	2	44			
					E	41.25	2.75	44	15:1	0.21	0.70-0.50
5.	"	III	134-1	N	O	21	2	23			
					E	21.56	1.44	23	15:1	0.24	0.70-0.50
6.	"	III	136-1	N	O	18	1	19			
					E	17.81	1.19	19	15:1	0.03	0.90-0.80
7.	"	III	174-1	N	O	15	3	18			
					E	13.5	4.5	18	3:1	0.16	0.70-0.50
8.	"	III	175-1	N	O	5	1	6			
					E	4.5	1.5	6	3:1	0.81	0.50-0.30
9.	"	III	183-2	N	O	13	3	16			
					E	12	4	16	3:1	0.33	0.70-0.50

1	2	3	4	5	6	7	8	9	10	11	12
10.	"	III	185-2	N	O	23	3	26			
					E	24.38	1.62	26	15:1	1.26	0.30-0.20
11.	"	IV	125-1	N	O	19	4	23			
					E	17.25	5.75	23	3:1	0.71	0.50-0.30
12.	"	IV	125-2	N	O	20	7	27			
					E	20.25	6.75	27	3:1	0.01	0.95-0.90
13.	"	IV	125-3	N	O	18	6	24			
					E	18	6	24	3:1	0.00	
14.	"	IV	235-2	N	O	15	4	19			
					E	14.25	4.75	19	3:1	0.16	0.70-0.50
15.	"	IV	250-3	N	O	27	7	34			
					E	25.5	8.5	34	3:1	0.35	0.70-0.30
16.	"	V	40-2	N	O	26	2	28			
					E	26.25	1.75	28	15:1	0.04	0.90-0.80
17.	"	VI	53-1	N	O	22	1	23			
					E	21.56	1.44	23	15:1	0.14	0.80-0.70
18.	"	VI	53-3	N	O	23	7	30			
					E	22.50	7.50	30	3:1	0.04	0.90-0.80
19.	"	VI	54-2	N	O	34	2	36			
					E	33.75	2.25	36	15:1	0.03	0.80-0.70
20.	"	VI	54-3	N	O	21	2	23			
					E	21.56	1.44	23	15:1	0.23	0.70-0.50
21.		VI	64-1	N	O	20	2	22			
					E	20.62	1.38	22	15:1	0.30	0.70-0.50

1	2	3	4	5	6	7	8	9	10	11	12
22.	"	VI	84-1	N	O	28	3	31			
					E	29.06	1.94	31	15:1	0.62	0.50-0.30
23.	"	VI	85-2	N	O	38	3	41			
					E	38.44	2.56	41	15:1	0.08	0.80-0.70
24.	"	VI	82-1	N	O	17	5	22			
					E	16.5	5.50	22	3:1	0.00	

I - DW + 0.5% EMS

II - Vit.C+0.5% EMS

III - DW + 0.75% EMS

IV - Vit.C+0.75% EMS

V - DW + 1% EMS

VI - Vit.C+1% EMS

Table 14. Breeding behaviour of M_3 families for albino mutants of Japan Violet treated with EMS treatment DW + 0.75% (1) and Vit. C + 0.75% (2)

Sl No.	Character studied	Expected M_3 ratio	O/E	Breeding behaviour of M_3 families TD	3:1	TR	Total	X^2	P
1.	Albino	1:2	O	7	9	-	16		
			E	5.33	10.67	-	16	0.78	0.90-0.80
2.	"	1:2	O	2	10	-	12		
			E	4	8	-	12	1.50	0.70-0.50

TD - True dominant TR = True recessive

b. Lethal yellow

Lethal yellow mutants were observed in M_2 of the treatments such as DW + 0.5% EMS, Vit. C + 0.5% EMS, DW + 0.75% EMS and DW + 1% EMS. Observations on the number and percentage of segregating M_2 lines of Japan violet treated with different concentrations of EMS after seed pre-treatment are presented in Table 12 . Maximum number of M_2 segregating lines of 4.69% was observed for lethal yellow with DW + 0.75% EMS treatment. DW + 0.5% and DW + 1% EMS treatments showed 1.35% and 1.92% M_2 segregating lines respectively for lethal yellow, while in the case of Vit. C pre-treatment, lethal yellow was induced by the treatment Vit. C + 0.5% with 1.56% of M_2 lines as shown in (Table 12).

All the lethal yellow mutants expressed phenotypically similar characters with 3-4 yellow leaves and had a life span of 10-14 days. Data on the pattern of inheritance of lethal yellow of Japan Violet are furnished in Table 15.

One out of 74 M_2 lines of DW + 0.5% EMS and 3 out of 64 M_2 lines of DW + 0.75% EMS treatment segregated for 15:1 indicating duplicate recessive control of lethal yellow (Table 15). However, one out of 64 M_2 lines of Vit. C + 0.5% EMS treatment and 1 out of 52 M_2 lines of DW + 1% treatment segregated for monogenic ratio 3:1 with

TABLE 15. Details of segregation for lethal yellow in M_1 and M_2 of EMS treated Japan violet

Sl. No.	Character studied	Treatment No.M_1 & Line No.	O/E	M_2 Frequency -	M_2 Frequency +	Total	Ratio	X^2	P
1.	Lethal yellow	I 4-1	N O	54	2	56			
			E	52.50	3.5	56	15:1	0.68	0.50-0.30
2.	Lethal Yellow	II 80-1	N O	25	10	35			
			E	26.25	8.75	35	3:1	0.18	0.70-0.50
3.	"	III 136-1	N O	18	1	19			
			E	17.81	1.19	19	15:1	0.03	0.90-0.80
4.	"	III 155-1	N O	29	3	32			
			E	30.00	2.0	32	15:1	0.53	0.50-0.30
5.	"	III 183-1	N O	12	1	13			
			E	12.19	0.81	13	15:1	0.05	0.90-0.80
6.	"	V 80-1	N O	42	10	52			
			E	39	13	52	3:1	0.92	0.50-0.30

I = DW + 0.5% EMS
II = Vit. C + 0.5% EMS
III = DW + 0.75% EMS
V = DW + 1% EMS

recessive genic control of lethal yellow. Induction of lethal yellow appeared reduced with the Vit. C pre-treatments. No M_3 confirmation was observed for lethal yellow in any of the M_3 families studied, as discussed elsewhere.

c. Striped mutant

Striped mutant was obtained from M_2 of the treatment Vit C + 0.5% EMS, Vit. C + 0.75% EMS and Vit. C + 1% EMS. All mutant plants died away before flowering except those obtained from Vit. C + 1% EMS treatment. The mutant showed white/rose stripes on leaf sheath and on both sides of lamina (Fig. 4). In seedlings the first formed leaf appeared normal, while leaf sheath showed pink coloured stripes. Stripes develop from the leaf margin of second or third leaf to leaf tip. In some cases stripes extend to the panicles. In such cases spikelets and most of the rachilla appeared white, while apiculus remaining purple. Other morphological/morphometric characters are almost similar to the control, except sterility. Sterility ranged from 15.79 to 49.18 with a mean of 33.57 (Table 16), against the mean value of 22.60 % of the control.

Striped mutants obtained from the treatments, Vit.C + 0.5% EMS, Vit. C + 0.75% EMS and Vit.C + 1% EMS, were studied for inheritance. In the first two cases M_2 showed segregation of 12 normal : 2 striped and 14 normal : 3 striped, and in the later case M_2 showed

Fig. 4. Striped mutant

a. Mutant at tillering stage

b&c. Mutant at flowering and maturing stage

d. Close-up view of the rose stripes in the leaf.

Fig4

Table 16. Range and mean of certain morphometric characters in striped mutant and its source parent Japan Violet

Population/ Character	Range / Mean / SE	Plant height cm	Culm length cm	Total tiller	EBT	% of EBT	Panicle length cm	Total spikelet/ panicle	No. of st. spikelet/ panicle	% of sterility	Panicle density
Control	Range	71.0-89.0	38.5-51.5	7.0-16.0	6.0-12.0	62.5-100	17.5-22.0	75.5-120.5	9.5-32	8.44-35.60	3.78-6.19
	Mean	82.20	44.82	10.92	9.32	82.67	19.28	105.02	21.16	22.60	5.08
	SE	± 0.28	± 0.19	± 0.15	± 0.11	± 0.61	± 0.08	± 0.65	± 0.28	± 0.28	± 0.03
Striped	Range	67.0-81.0	35.0-41.0	8.0-15.0	6.0-12.0	53.33-100	17.25-20.75	61-95.0	14.0-44.0	15.79-49.18	3.12-4.63
mutant	Mean	75.12	40.02	10.44	7.84	76.18	19.75	84.01	27.74	33.57	4.25
	SE	± 0.17	± 0.15	± 0.08	± 0.06	± 0.39	± 0.03	± 0.36	± 0.18	± 0.24	± 0.02

segregation of 45 normal: 10 striped, all indicating simple recessive genic control (Table 17). The latter M_2 was followed up for M_3 analysis and the breeding behavior of the M_3 families confirmed the M_2 ratio 3:1, with segregation of the families conforming to the expected 1:2:1 ratio (Table 18).

Inheritance of the striped mutant was further studied in the cross Japan Violet x Striped mutant. F_1 showed dominance of purple colour over striped, resembling Japan Violet.

F_2 generation

Out of 368 F_2 plants 283 were normal and 85 striped, giving good fit to the ratio 3:1 with $X^2 = 0.71$ which is not significant for 1 d.f at 5% level (Table 19).

F_3 generation

Breeding behavior of F_3 families of the above cross confirmed the F_2 ratio. Out of 32 F_3 families studied, 8 bred true for normal, 18 segregated for 3:1 and 6 bred true for striped with $X^2 = 0.75$ for 2 d.f. at 5% level in conformity with the expected F_3 ratio 1:2:1 (Table 20).

TABLE 17. Inheritance of mutant morphological characters in M_1 and M_2 of EMS treated Japan violet

Sl. No.	Mutant Studied	Mutant derived from	M_1	O/E	M_2 Frequency -	M_2 Frequency +	Total	Ratio	X^2	P
1	2		3	4	5	6	7	8	9	10
1.	Striped mutant	VI	N	O	12	2	14			
				E	10.5	3.5	14	3:1	0.86	0.50-0.30
	"		N	O	14	3.0	17			
				E	12.75	4.25	17	3:1	0.49	0.50-0.30
	"		N	O	45	10	55			
				E	41.25	13.75	55	3:1	1.36	0.30-0.20
2.	Chlorina	VI	N	O	28	6	34			
				E	25.50	8.50	34	3:1	0.98	0.50-0.30
3.	Abnomorphic spikelet	VI	N	O	38	12	50			
				E	37.5	12.5	50	3:1	0.03	0.90-0.80
4.	Beaked lemma and depressed palea mutant	V	N	O	22	4	26			
				E	19.50	6.50	26	3:1	1.28	0.30-0.20
5.	Long sterile glume mutant	V	N	O	20	2	22			
				E	20.62	1.38	22	15:1	0.50	0.50-0.30
6.	Deformed palea mutant	IV	N	O	19	6	25			
				E	18.75	6.25	25	3:1	0.01	0.90-0.80
7.	Broad seed mutant	V	N	O	17	5	22			
				E	16.50	5.50	22	3:1	0.60	0.50-0.30

1	2	3	4	5	6	7	8	9	10
8.	Anther sterile mutant	I	N	O	21	3	24			
				E	18	6	24	3:1	2.00	0.20-0.10
	"		N	O	21	4	25			
				E	18.75	6.25	25	3:1	0.85	0.50-0.30
9.	Grassy rhizomatous mutant	V	N	O	15	6	21			
				E	15.75	5.25	21	3:1	0.51	0.50-0.30
10.	Tall recessive mutant	IV	N	O	22	4	26			
				E	19.50	6.50	26	3:1	1.28	0.30-0.20
11.	Procumbent mutant	V	N	O	18	4	22			
				E	16.50	5.50	22	3:1	0.55	0.50-0.30

I = DW + 0.5% EMS
II = Vit. C + 0.5% EMS
III = DW + 0.75% EMS
IV = Vit. C + 0.75% EMS
V = DW + 1% EMS
VI = Vit. C + 1%EMS

TABLE 18. Breeding behavior of mutant morphological characters studied in respective M_3 families of EMS treated Japan violet

Sl. No.	Character Studied	Expected M_3 ratio	O/E	Breeding behavior M_3 Families				Total	X^2	P
				TD	3:1	15:1	TR			
1	2	3	4	5	6	7	8	9	10	11
1.	Striped mutant	1:2:1	O	11	11	-	10	32		
			E	8	16	-	8	32	3.19	0.50-0.30
2.	Chlorina	1:2:1	O	9	11	-	10	30		
			E	7.5	15	-	7.5	30	2.19	0.70-0.50
3.	Abnomorphic spikelet mutant	1:2:1	O	10	14	-	10	34		
			E	8.50	17.00	-	8.50	34	1.05	0.80-0.70
4.	Beaked lemma and depressed palea mutant	1:2:1	O	3	12	-	4	19		
			E	4.75	9.5	-	4.75	19	1.42	0.80-0.70
5.	Long sterile glume mutant	7:4:4:1	O	10	4	3	2	19		
			E	8.31	4.75	4.75	1.19	19	1.65	0.70-0.50
6.	Deformed palea mutant	1:2:1	O	3	12	-	5	20		
			E	5	10	-	5	20	1.20	0.80-0.70
7.	Broad seed mutant	1:2:1	O	3	12	-	5	20		
			E	5	10	-	5	20	1.20	0.80-0.90
8.	Anther sterile mutant	1:2	O	6	16	-	-	22		
			E	7.33	14.67	-	-	22	0.36	0.95-0.90

1	2	3	4	5	6	7	8	9	10	11
9.	Grassy rhizomatous mutant	1:2	O	9	11	-	-	20	1.22	0.80-0.70
			E	6.67	13.33	-	-	20		
10.	Tall recessive mutant	1:2:1	O	6	12	-	4	22	0.30	0.98-0.95
			E	5.5	11	-	5.5	22		
11.	Procumbent mutant	1:2:1	O	5	10	-	9	24	2.00	0.70-0.50
			E	6	12	-	6	24		

TD = True dominant TR = True recessive

TABLE 19. Inheritance of mutant characters in F_1 and F_2 of 14 crosses studied

Sl. No.	Mutant character studied	Cross studied	Parents		F_1	O/E	F_2 Frequency -	F_2 Frequency +	Total	Ratio	X^2	P
1	2	3	4		5	6	7	8	9	10	11	12
1.	Striped character	Japan Violet x Striped mutant	+	-	-	O	283	85	368			
						E	276	92	368	3:1	0.71	0.50-0.30
2.	Chlorina with purple shade	Japan Violet x Chlorina	+	-	-	O	168	54	222			
						E	166.5	55.5	222	3:1	0.05	0.90-0.80
3.	Spotted leaf	Japan Violet x Spotted leaf mutant	+	-	-	O	172	54	226			
						E	169.50	56.50	226	3:1	0.15	0.70-0.50
4.	Complete green	Japan Violet x C. green mutant	+	-	-	O	175	53	228			
						E	171	57	228	3:1	0.37	0.70-0.50
5.	Beaked lemma depressed palea	Japan Violet x Beaked lemma depressed palea	+	-	-	O	224	72	296			
						E	222	74	296	3:1	0.07	0.80-0.70
6.	Long sterile glume	Japan Violet x Long sterile glume	+	-	-	O	175	7	182			
						E	170.62	11.38	182	15:1	1.80	0.20-0.10
7.	Deformed palea	Japan Violet x Deformed palea	+	-	-	O	206	57	263			
						E	197.25	65.75	263	3:1	1.55	0.30-0.20
8.	Broad seed	Japan Violet x Broad seed	+	-	-	O	174	52	226			
						E	169.5	56.5	226	3:1	0.48	0.50-0.30

1	2	3	4	5	6	7	8	9	10	11	12
9.	Multipistil	Japan Violet x Multipistil	+	-	O	211	64	275			
					E	206.25	68.75	275	3:1	0.44	0.70-0.50
10.	Anther sterile	Japan Violet x Anther sterile	+	-	O	142	40	182			
					E	136.50	45.50	182	3:1	0.88	0.50-0.30
11.	Brittle culm	Japan Violet x Brittle culm	+	-	O	182	54	236			
					E	177	59	236	3:1	0.56	0.50-0.30
12.	Tall recessive	Japan Violet x Tall recessive	+	-	O	172	64	236			
					E	177	59	236	3:1	0.56	0.50-0.30
13.	High tillering dwarf	Japan Violet x High tillering dwarf	+	-	O	318	111	429			
					E	321.75	107.25	429	3:1	0.17	0.70-0.50
14.	Procumbent character	Japan Violet x Procumbent	+	-	O	236	76	312			
					E	234	78	312	3:1	0.07	0.80-0.70

+ = Presence
- = Absence

TABLE 20. Breeding behaviour of F_3 families for mutant characters in 14 crosses studied

Sl. No.	Character studied	Cross	Expected F_3 ratio	O/E	Breeding behaviour of F_3 Families segregated for TD	3:1	15:1	TR	Total	x^2	P
1	2	3	4	5	6	7	8	9	10	11	12
1.	Striped	Striped mutant x Japan Violet	1:2:1	O	8	18		6	32		
				E	8.0	16		8.0	32	0.75	0.90-0.80
2.	Chlorina with purple shade	Chlorina with x Japan Violet	1:2:1	O	9	11	-	8	28		
				E	7	14		7	28	1.36	0.80-0.70
3.	Spotted leaf	Spotted leaf mutant x Japan Violet	1:2:1	O	6	14	-	7	27		
				E	6.75	13.5		6.75	27	0.19	0.98-0.95
4.	Complete green mutant	Complete green mutant x Japan Violet	1:2:1	O	6	16	-	10	32		
				E	8	16		8	32	1.00	0.90-0.80
5.	Beaked lemma and depressed palea	Beaked lemma depressed palea mutant x Japan Violet	1:2:1	O	3	12	-	5	20		
				E	5	10		5	20	1.20	0.70-0.50
6.	Long sterile glume	Long steriled glume x Japan Violet	7:4:4:1	O	11	3	5	3	22		
				E	9.62	5.5	5.5	1.38	22	2.56	0.50-0.30
7.	Deformed palea	Deformed palea mutant x Japan Violet	1:2:1	O	3	15	-	6	24		
				E	6	12		6	24	2.25	0.70-0.50
8.	Broad seed	Broad seed mutant x Japan Violet	1:2:1	O	9	11	-	4	24		
				E	6	12		6	24	2.25	0.70-0.75

1	2	3	4	5	6	7	8	9	10	11	12
9.	Multipistil	Multipistil mutant x Japan Violet	1:2:1	O	10	12	-	6	28		
				E	7	14		7	28	1.71	0.80-0.70
10.	Anther sterile	Anther sterile mutant x Japan Violet	1:2:0	O	8	15	-	-	23		
				E	7.67	15.33		-	23	0.02	0.99
11.	Brittle culm	Brittle culm mutant x Japan Violet	1:2:1	O	6	11	-	8	25		
				E	6.25	12.5		6.25	25	0.20	0.98-0.95
12.	Tall recessive	Tall recessive mutant x Japan Violet	1:2:1	O	10	12	-	8	30		
				E	7.5	15		7.5	30	1.61	0.70-0.50
13.	High tillering Dwarf	High tillering dwarf mutant x Japan Violet	1:2:1	O	3	17	-	5	25		
				E	6.25	12.5		6.25	25	3.13	0.50-0.30
14.	Procumbent	Procumbent mutant x Japan Violet	1:2:1	O	9	10	-	5	24		
				E	6	12	-	6	24	2.00	0.70-50

TD - True Dominant
TR - True Recessive

d. Chlorina-leaf mutant (with purple shade)

Chlorina-leaf mutant of Japan violet occurred in M_2 of the treatment Vit.C + 1% EMS. It showed yellowish- green colour (resembling chlorina) with slight purple tinge throughout the lamina and purple colour in leaf sheath (Fig. 5b). In all other morphological characters it resembled Japan Violet except in certain quantitative characters. Mean and range of morphometric characters of the mutant and of the parent are furnished in Table 21.

M_2 plants showed segregation pattern of 28 normal : 6 chlorina leaf giving 3:1 for recessive genic control (Table 17). The breeding behavior of 30 M_3 families studied, confirmed the M_2 ratio, as 9 bred true for purple plants : 11 segregated for 3:1 and 10 bred true for chlorina in conformity with the expected ratio 1:2:1 (Table 18).

Inheritance of the mutant character was further studied in the cross Japan violet x Chlorina leaf mutant. F_1 showed purple colour of Japan violet as dominant over chlorina leaf (Table 19).

F_2 generation

F_2 population of 222 plants segregated into 168 normal : 54 mutant types giving good fit to the ratio 3:1 with $X^2 = 0.05$ which is

Fig. 5. a. Japan violet (control)

b. Chlorina mutant

c. Purple spotted mutant

d. Close-up view of the purple spots in the leaves

Fig 5

Table 21. Range and mean of certain morphometric characters of chlorina leaf mutant and its source parent Japan Violet.

Population/ Character	Range / Mean /	Plant height cm	Culm length cm	Total tiller	EBT	% of EBT	Panicle length cm	Total spikelet/ panicle	No. of st. spikelet/ panicle	% of sterility	Panicle density
Control	Range	71.0-89.0	38.5-51.5	7.0-16.0	6.0-12.0	62.5-100	17.5-22.0	75.5-120.5	9.5-32	8.44-35.60	3.78-6.19
	Mean	82.20	44.82	10.92	9.32	82.67	19.28	105.02	21.16	22.60	5.08
	SE	± 0.28	± 0.19	± 0.15	± 0.11	± 0.61	± 0.08	± 0.65	± 0.28	± 0.28	± 0.03
Chlorina leaf mutant	Range	63-78	33.0-43.0	7.0-16.0	6.0-16.0	63.44-100	17.25-21	64.0-104.5	18-40	23.81-43.59	3.71-4.95
	Mean	68.72	40.16	12.44	9.88	79.53	18.78	83.84	28.58	33.29	4.46
	SE	± 0.15	± 0.34	± 0.14	± 0.12	± 0.47	± 0.04	± 0.43	± 0.27	± 0.24	± 0.01

not significant for 1 d.f. at 5% level (Table 19) showing recessive monogenic control of the mutant character.

F_3 generation

Breeding behavior of 28 F_3 families showed that 9 bred true for normal plants, 11 segregated for 3:1 and 8 bred true for the mutant character with X^2 = 1.36 for 2 d.f. at 5% level in conformity with the expected F_3 ratio 1:2:1 (Table 20).

e. Purple spotted leaf mutant

Purple spotted leaf mutant occurred in M_3 of Japan violet treated with Vit C + 1% EMS. It showed number of purple pigment spots of various sizes throughout the leaf (Fig. 5 c&d), which could be clearly seen on both sides of the lamina. Morphometric characters of the mutant resembled the source plant. 1.19% of the M_3 segregating lines showed this type of mutants (Table 22).

Purple spotted leaf mutants were observed in two M_3 lines of Vit C + 1% EMS treatment of which first line segregated into 60 normal : 24 mutant types and the second line segregated into 33 normal: 15 mutant types (Table 23). Both lines showed monogenic ratio for its recessive genic control, which could be confirmed by

Table 22. Number and percentage of M_3 families segregated for mutant characters in Japan Violet induced by different treatments with EMS

Sl. No.	Mutant character	Treatment						
		Control	DW + EMS			Vit. C + EMS		
			0.5%	0.75%	1%	0.5%	0.75%	1%
1.	Spotted leaf	- 0.0%	- 0.0%	- 0.0%	- 0.0%	- 0.0%	- 0.0%	2 1.19%
2.	Partial green mutant	- 0.0%	- 0.0%	- 0.0%	- 0.0%	- 0.0%	1 0.68%	- 0.0%
3.	Complete green mutant	- 0.0%	- 0.0%	- 0.0%	- 0.0%	- 0.0%	- 0.0%	1 0.60%
4.	Multipistil	- 0.0%	- 0.0%	1 0.60%	- 0.0%	- 0.0%	- 0.0%	- 0.0%
5.	Brittle culm mutant	- 0.0%	- 0.0%	- 0.0%	- 0.0%	- 0.0%	- 0.0%	1 0.60%
6.	High tillering dwarf	- 0.0%	- 0.0%	- 0.0%	- 0.0%	- 0.0%	1 0.53%	- 0.0%
7.	Total No. of M_3 families studied		155	167	147	160	188	168
8.	Total No. of M_3 families segregated		0.0	1	0.0		2	4
9.	Total % of M_3 families segregated	0.0	0.0	0.60%	0.0	0.0	1.21%	2.39%

TABLE 23. Inheritance of mutants obtained in M_3 of EMS treated Japan violet

Sl. No.	Mutant studied	M_2	O/E	M_3 Frequency -	M_3 Frequency +	Total	Ratio	X^2	P
1.	Purple spotted leaf	N	O	60	24	84			
			E	63	21	84	3:1	0.57	0.50-0.30
	"	N	O	33	15	48			
			E	36	12	48	3:1	1.0	0.30-0.20
2.	Partial green	N	O	52	15	67			
			E	50.25	16.75	67	3:1	0.24	0.70-0.50
3.	Complete green	N	O	64	18	82			
			E	61.50	20.50	82	3:1	0.40	0.70-0.50
4.	Multipistil	N	O	22	4	26			
			E	19.50	6.50	26	3:1	1.28	0.30-0.20
5.	Brittle culm	N	O	51	13	64			
			E	48.50	16.00	64	3:1	0.75	0.50-0.30
6.	High tillering	N	O	42	10	52			
			E	39	13	52	3:1	0.92	0.50-0.30

N = Normal.
- = Absence
+ = Presence

the breeding behavior of the 34 M_4 families (Table 24), of which 10 bred true for normal, 14 segregated for 3:1 and 10 bred true for spotted leaf and, in the second case, out of 26 families 6 bred true for normal, 15 segregated for 3:1 and 5 bred true for spotted leaf in conformity with the expected ratio 1:2:1 with X^2 = 1.05 and 0.69 respectively for 2 d. f. at 5% level (Table 24).

Inheritance of spotted leaf was further studied in the cross, Japan violet x Spotted leaf mutant. F_1 showed normal leaf colour of Japan violet indicating dominance of purple colour over spotted leaf (Table 19).

F_2 generation

Out of 226 F_2 plants, 172 were normal and 54 spotted leaf giving good fit to the ratio 3:1 with X^2 = 0.15 which is not significant for 1 d.f. at 5% level (Table 19).

F_3 generation

Breeding behavior of 27 F_3 families confirmed the F_2 ratio, as 6 of them bred true for normal plants, 14 segregated for 3:1 and 7 bred true for mutant character with X^2 = 0.19 for 2 d.f. at 5% level in conformity with the expected F_3 ratio 1:2:1 (Table 20).

TABLE 24. Breeding behaviour of mutant morphological characters studied in respective M_4 families of Japan Violet treated with EMS

Sl. No.	Character Studied	Expected ratio	O/E	Breeding behaviour M_4 Families TD	3:1	TR	Total	χ^2	P
1.	Spotted leaf mutant	1:2:1	O	10	14	10	34		
			E	8.50	17	8.50	34	1.05	0.50-0.30
	"	1:2:1	O	6	15	5	26		
			E	6.50	13	6.5	26	0.69	0.50-0.30
2.	Partial green mutant	1:2:1	O	9	13	10	32		
			E	8.00	16	8.00	32	1.19	0.30-0.20
3.	Complete green	1:2:1	O	10	14	10	34		
			E	8.50	17	8.50	34	1.05	0.50-0.30
4.	Multipistil mutant	1:2:1	O	7	8	5	20		
			E	5.00	10	5	20	1.70	0.20-0.10
5.	Brittle culm mutant	1:2:1	O	7	10	6	23		
			E	5.75	11.50	5.75	23	0.48	0.90-0.80
6.	High tillering dwarf mutant	1:2:1	O	8	12	10	30		
			E	7.50	15	7.50	30	1.46	0.30-0.20

TD - True Dominant
TR - True Recessive

f. Partial green mutant

Partial green mutant plant (with a few plant parts turned green) occurred in M_3 of DW + 1% EMS treatment. The mutant showed purple colour in leaf axil, leaf sheath, leaf tip, apiculus, stigma and junctura proper like Japan Violet and green in all other plant parts (Fig. 6). Other morphometric characters of this mutant such as plant height, number of tillers and EBT resembled Japan Violet and showed high sterility percentage of 47.38 (Table 25).

The M_3 line showed segregation for 52 normal : 15 partial green plant, showing monogenic recessive control of the mutant character (Table 23) which was confirmed by the breeding behaviour of the M_4 families, of which 9 bred true for normal plants, 13 segregated for 3:1 and 10 bred true for the mutant character in conformity with the expected ratio 1:2:1 with X^2 = 1.19 for 2 d.f. at 5% level (Table 24).

Further analysis of interrelationships of genes controlling pigmentation in 5 plant parts that could be studied in the cross between Japan Violet x Partial green mutant showed that all the morphological characters, (leaf margin, leaf blade, ligule, auricle and internode) showed a common gene pleiotropic for these characters as detailed in Table 26.

Fig. 6. a. Partial green mutant

b. Showing purple colouration in junctura proper, leaf tip, apiculus, stigma, leaf axil and leaf sheath

Fig 6

Table 25. Range and mean of certain morphometric characters of partial green mutant and its source parent Japan Violet

Population/ Character	Range / Mean /	Plant height cm	Culm length cm	Total tiller	EBT	% of EBT	Panicle length cm	Total spikelet/ panicle	No. of st. spikelet/ panicle	% of sterility	Panicle density
Control	Range	71.0-89.0	38.5-51.5	7.0-16.0	6.0-12.0	62.5-100	17.5-22.0	75.5-120.5	9.5-32.0	8.44-35.60	3.78-6.19
	Mean	82.20	44.82	10.92	9.32	82.67	20.71	105.02	21.16	22.60	5.08
	SE	± 0.16	± 0.17	± 0.37	± 0.40	± 0.46	± 0.08	± 0.65	± 0.28	± 0.28	± 0.03
Partial green mutant	Range	74.0-81.5	42.0-51.5	11.21	8.0-13.0	47.37-86.67	17.25-21.25	71.0-120.0	22.5-56.0	31.69-58.88	4.11-6.00
	Mean	77.85	45.65	14.35	9.3	62.94	20.05	93.60	44.70	47.38	4.67
	SE	± 0.12	± 0.09	± 0.15	± 0.12	± 0.42	± 1.51	± 0.76	± 0.35	± 0.30	± 0.02

Table 26. Interrelationship of certain mutant morphological characters studied in Partial green and Complete green mutants.

sl No.	F_2 ratio/ Character combination	crosses	Population O/E	F_2 Frequency AB	Ab	aB	ab	Total	X^2	P
	(3:1) (3:1)									
1.	Plm vs Pl/Plg/ Pau/Pin	Japan Violet x	O	118.00	0.00	0.00	46.00	164		
		partial green	E on indep	92.25	30.75	30.75	10.25	164	193.38	
		mutant	E on pleio	123.00	0.00	0.00	41.00	164	0.81	0.90-0.80
	Pl vs Plg/Pau/Pin									
	Plg vs Pau/Pin									
	Pau vs Pin									
2.	Px vs Psh/Plm/Pl/Pla/Plg/ Pjp/Pau/Pn/Pin/Pa/Ps	Japan Violet x	O	175	0.00	0.00	53	228		
		complete green	E on indep	128.25	42.75	42.75	14.25	228	207.97	
		mutant	E on pleio	171.00	0.00	0.00	16.75	228	0.37	0.95-0.90
	Psh vs Plm/Pl/Pla/Plg/ Pjp/Pau/Pn/Pin/Pa/Ps									
	Plm vs Pl/Pla/Plg/Pjp/Pau/ Pin/Pa/Ps									
	Pl vs Pla/Plg/ Pjp/Pau/Pn/Pin/Pa/Ps									
	Pla vs Plg/Pjp/Pau/Pn Pin/Pa/Ps									
	Plg vs Pjp/Pau/Pn/Pin/Pa/Ps									
	Pjp vs Pau/Pn/Pin/Pa/Ps									
	Pau vs Pn/Pin/Pa/Ps									
	Pn vs Pin/Pa/Ps									
	Pin vs Pa/Ps									
	Pa vs Ps									

g. Complete green mutant

Complete green mutant occurred in M_3 of Vit C + 1% EMS treatment. All plant parts showed complete green colour in contrast to the source plant, Japan Violet (Fig. 7). Seeds of the green mutant plant are smaller in size and the mutant produced more tillers and spikelets/panicle, and its height appeared slightly reduced than Japan Violet, (Table 27). Mean and range of morphometric characters of the mutant and the source plant are given in Table 27.

Data on inheritance of complete green mutant of Japan Violet are furnished in Table 23. Out of 82 plants of the M_3 line 58-2-26-1 of the treatment Vit C + 1% EMS, 64 were normal pigmented plants and 18 complete green giving 3:1 ratio (Table 23), which was confirmed by the breeding behavior of 34 M_4 families, of which 10 bred true for normal, 14 segregated for 3:1 and 10 bred true for the mutant character in conformity with the expected ratio 1:2:1 with $X^2 = 1.05$ for 2 d.f. at 5% level (Table 24).

Inheritance of complete green mutant was further studied in the cross, Japan Violet x Complete green mutant. F_1 showed dominance of purple colour.

Fig. 7. a. Complete green mutant

b. Apiculus, stigma, junctura proper, ligule, auricle, leaf tip, leaf axil and leaf sheath showing green colouration

c. Segregating line of complete green mutant

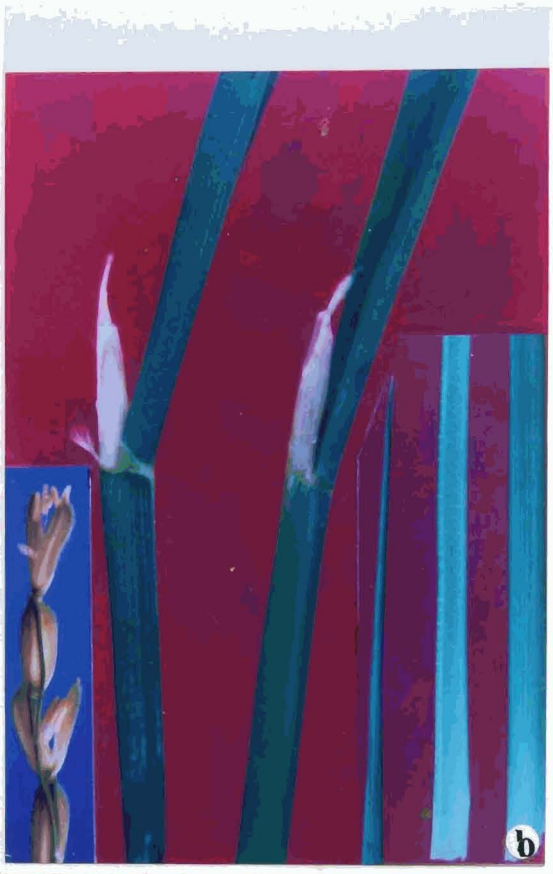

Fig 7

Table 27. Range and mean of cetain morphometric characters of complete green mutant and its source parent Japan Violet

Population/ Character	Range / Mean /	Plant height cm	Culm length cm	Total tiller	EBT	% of EBT	Panicle length cm	Total spikelet/ panicle	No. of st. spikelet/ panicle	% of sterility	Panicle density
Control	Range	71.0-89.0	38.5-51.5	7.0-16.0	6.0-12.0	62.5-100	17.5-22.0	75.5-120.5	9.5-32.0	8.44-35.60	3.78-6.19
	Mean	82.20	44.82	10.92	9.32	82.67	20.71	105.02	21.16	22.60	5.08
	SE	± 0.28	± 0.19	± 0.15	± 0.11	± 0.61	± 0.08	± 0.65	± 0.28	± 0.28	0.03
Complete green mutant	Range	55.0-72.0	36.0-43.0	21.0-47.0	20-42	57.89-97.22	17.0-22.0	76.0-158.0	19.0-35.5	17.0-25.0	4.47-7.18
	Mean	66.85	40.85	32.45	27.20	82.1	20.11	120.55	24.55	20.36	5.72
	SE	± 0.16	± 0.17	± 0.37	± 0.40	± 0.46	± 0.08	± 1.08	± 0.24	± 0.22	± 0.04

F_2 generation

The population of 228 F_2 plants segregated into 175 purple plants and 53 complete green plants, giving good fit to the ratio 3:1 with X^2 = 0.37 which is not significant for 1 d.f. at 5% level (Table 19).

F_3 generation

Breeding behavior of 32 F_3 families of the above cross confirmed the F_2 ratio, as 6 of them bred true for normal purple character, 16 segregated for 3:1 and 10 bred true for complete green mutant character with X^2 = 1.0 for 2 d.f. at 5% level in conformity with the expected F_3 ratio 1:2:1 (Table 20).

Further, analysis of interrelationships of genes controlling pigmentation in 12 plant parts that could be studied in the cross between Japan Violet x Complete green mutant showed that all the morphological characters, (leaf axil, leaf sheath, leaf margin, leaf blade, leaf tip, ligule, junctura proper, node, internode, apiculus and stigma) showed a common gene pleiotropic for these characters as detailed in Table 26.

2. Spikelet mutations

a. Abnomorphic spikelet mutant*

This mutant characterised by its abnormal spikelet parts occurred in M_2 of the treatment Vit C + 1% EMS. This showed multiple abnormalities in the spikelet. These are long sterile glume, depressed palea, twin kernels, open spikelet, long lemma, beaked lemma, extra long glume, long lemma-palea, multipistillate condition (multiple kernels) and deformed palea /palealess condition (Fig. 8). However, these characteristics of the spikelet mutant showed variation in expressivity with lemma-palea abnormalities having the maximum expressivity of 65.64% (Table 28). Mean and range of other morphometric characters of the parent and mutant plant are furnished in Table 29.

Data on inheritance of abnomorphic spikelets are furnished in Table 17. One out of 62 M_2 lines of Japan Violet treated with Vit C + 1% EMS segregated into 38 plants with normal spikelets and 12 abnomorphic spikelet mutant giving 3:1, suggesting recessive gene control of abnomorphic spikelets (Table 17). The M_2 ratio was subsequently confirmed by the breeding behavior of 34 M_3 families, of which 10 bred true for normal spikelets, 14 segregated for 3:1 and 10 bred true for the mutant plant with $X^2 = 1.05$ which is not significant against the table value for 2 d.f. at 5% level (Table

*spikelets with abnormal morphological floral parts having different expressions.

Fig. 8. Abnomorphic spikelet mutant

a. Panicle of control and abnomorphic spikelet mutant

b. Extra long glume

c. Depressed palea

d. Twin kernel

e. Open spikelet

f. Long lemma

g. Beaked lemma

h. Long sterile glume

i. Long lemma palea

j. Multiple kernels

k. Deformed palea/palealess condition

Fig 8

Table 28. Range, mean and expressivity (%) of different spikelet parts of the abnomorphic spikelet mutant induced by Vit. C + 1% EMS treatment in M_2 of Japan Violet

Range / Mean / %	Normal spikelets	Open spikelets	multi pistil	multi husk	extra long glume	multiple kernel	Lemma palea abnormalities				Expressivity (%)
							b. lemma	d. palea	l. lemma palea	df. palea	
Range	2-3	10-24	2-5	1-3	2-5	2-3	12-32	4-15	5-12	5-25	
Mean	2.5	17.1	3.4	2.0	2.75	2.25	16.90	18.9	7.80	13.70	
%	2.86	19.59	3.89	2.29	3.16	2.58	19.36	21.65	8.94	15.69	65.64

Table 29. Range and mean of certain morphometric characters of abnomorphic spikelet mutant and its source parent Japan Violet

Population/ Character	Range / Mean / SE	Plant height cm	Culm length cm	Total tiller	EBT	% of EBT	Panicle length cm	Total spikelet/ panicle	No. of st. spikelet/ panicle	% of sterility	Panicle density
Control	Range	71.0-89.0	38.5-51.5	7.0-16.0	6.0-12.0	62.5-100	17.5-22.0	75.5-120.5	9.5-32.0	8.44-35.60	3.78-6.19
	Mean	82.20	44.82	10.92	9.32	82.67	19.28	105.02	21.16	22.60	5.08
	SE	± 0.28	± 0.19	± 0.15	0.11	0.61	0.08	0.65	0.28	0.28	0.03
Abnomorphic spikelet mutant	Range	73.0-84.0	40.0-57.0	12.0-26.0	10.0-23.0	70-91.67	18.75-20.50	70.0-99.0	27.0-51.0	27.54-52.33	3.73-4.95
	Mean	78.6	46.90	16.35	13.55	82.69	19.50	81.10	35.2	42.70	4.15
	SE	± 0.16	+ 0.24	± 0.16	± 0.15	± 0.33	± 0.05	± 0.42	± 0.42	± 0.39	± 0.02

b. lemma - beaked lemma
d. palea - depressed palea
l. lemma palea - long lemma palea
df. palea - deformed palea

18). As the segregation pattern was so discrete between the normal and the abnomorphic mutant phenotypes, it could be surmised to have a major gene responsible for the overall development of the spikelet, which consequently gave into differential developmental entities in respect of the various organs of the spikelet in tune with the extent of interaction with the localization genes. However, the said oligogene might also be pleiotropic in its effects on several characters.

b. Beaked lemma-depressed palea mutant

Beaked lemma-depressed palea mutant occurred in M_2 of Japan violet treated with DW + 1% EMS. The mutant seeds showed beaked lemma and depressed palea (Fig. 9 a,b). Other morphological characters of the mutant resembled that of the source plant except high sterility of 77.31 (Table 30).

Data on the pattern of inheritance of the mutant character in M_2 and M_3 are furnished in (Tables 17 & 18). Out of 52 M_2 lines, one showed segregation with 22 normal plants and 4 mutant plants, suggesting recessive genic control 3:1 of the mutant character (Table 17). The M_2 ratio was confirmed by the breeding behavior of M_3 families studied. Out of 19 M_3 families, 3 bred true for normal spikelets, 12 segregated for 3:1 and 4 bred true for mutant character

Fig. 9. Beaked lemma depressed palea mutant

a. Mutant panicle

b. Close-up view of a mutant grain

Fig. 10. Long sterile glume mutant

a. mutant plant

b. upper row - long sterile glume in the palea side

middle row - long sterile glumes in both sides upto the middle of the spikelet

lower row - long sterile glume reaching upto the tip of the spikelets in both sides

Fig 9

Fig 10

Table 30. Range and mean of certain morphometric characters in beaked-lemma-depressed-palea mutant and its source parent Japan Violet

Population/ Character	Range / Mean / SE	Plant height cm	Culm length cm	Total tiller	EBT	% of EBT	Panicle length cm	Total spikelet/ panicle	No. of st. spikelet/ panicle	% of sterility	Panicle density
Control	Range	71.0-89.0	38.5-51.5	7.0-16.0	6.0-12.0	62.5-100	17.5-22.0	75.5-120.5	9.5-32.0	8.44-35.60	3.78-6.19
	Mean	82.20	44.82	10.92	9.32	82.67	19.28	105.02	21.16	22.60	5.08
	SE	± 0.28	0.19	± 0.15	0.11	0.61	± 0.08	± 0.65	± 0.28	± 0.28	± 0.03
Beaked lemma depressed palea mutant	Range	61.0-80.0	35.0-46.0	7-16	3.0-12.0	42.86-100	18.5-22.0	60.0-126.5	51.0-112.0	85-88.54	3.24-5.90
	Mean	69.72	4.88	11.88	9.04	78.61	20.19	98.46	76.18	77.31	4.88
	SE	± 0.22	± 0.42	± 0.11	± 0.11	± 0.59	± 0.04	± .51	± 0.60	± 0.36	± 0.02

with X^2 = 1.42 which is not significant against the table value for 2 d.f. at 5% level (Table 18).

Inheritance of beaked lemma-depressed palea mutant was further studied in the cross Japan violet x Beaked lemma-depressed palea mutant. The F_1 showed normal spikelets indicating its dominance over abnomorphic spikelets.

F_2 generation

Out of 296 F_2 plants 224 were normal and 72 showed mutant character, giving good fit to the ratio 3:1 with X^2 = 0.07, which is not significant for 1 d.f. at 5% level (Table 19).

F_3 generation

Breeding behavior of 20 F_3 families studied confirmed F_2 segregation ratio, as 3 bred true for normal plants, 12 segregated for 3:1 and 5 bred true for mutant character with X^2 = 1.2 for 2 d.f. at 5% level in conformity with the expected F_3 ratio 1:2:1 (Table 20).

c. Long sterile glume mutant

Long sterile glume mutant occurred in M_2 of Japan Violet treated

with DW + 1% EMS. The mutant showed grains with long sterile glumes on lemma side or on both sides (Fig. 10). The length of sterile glume varied from middle to the tip of the grain. Percentage of sterility was high with 83.17% and in all other morphological characters it resembled Japan Violet (Table 31).

Data on inheritance of long sterile glume are furnished in Table 17. Out of 52 M_2 lines one segregated into 20 normal : 2 mutant plants giving 15:1, indicating duplicate recessive gene control of the mutant character.

The M_2 ratio 15:1 was confirmed by the breeding behavior of 19 M_3 families, of which 10 bred true for normal, 4 segregated for 3:1, 3 for 15:1 and 2 bred true for the mutant character with $X^2 = 1.65$, which is not significant for 3 d.f. at 5% level (Table 18).

Inheritance of long sterile glume mutant was further studied in the cross Japan violet x Long sterile glume mutant. F_1 showed normal sterile glumes.

F_2 generation

Out of 182 F_2 plants, 175 were normal and 7 long sterile glumes giving good fit to the ratio 15:1 with $X^2 = 1.80$ which is not significant for 1 d.f. at 5% level (Table 19).

Table 31. Range and mean of certain morphometric characters in long sterile glume mutant and its source parent Japan Violet

Population/ Character	Range / Mean / SE	Plant height cm	Culm length cm	Total tiller	EBT	% of EBT	Panicle length cm	Total spikelet/ panicle	No. of st. spikelet/ panicle	% of sterility	Panicle density
Control	Range	71.0-89.0	40.0-51.5	7.0-16.0	6.0-12.0	62.5-100.0	18.0-22.0	75.5-120.5	9.5-32.0	8.44-35.60	3.78-6.19
	Mean	82.20	44.82	10.92	9.32	82.67	20.71	105.02	21.16	22.60	5.08
	SE	± 0.28	± 0.19	± 0.15	± 0.11	± 0.61	± 0.08	0.65	± 0.28	± 0.28	± 0.03
Long St. glume mutant	Range	60-75	31.0-44.0	6.0-15.0	5.0-12.0	63.6-100.0	17.5-21.5	50-73	38.0-62.0	76.0-84.93	2.94-3.70
	Mean	67.55	36.88	10.98	8.85	78.28	17.53	54.65	45.45	83.17	3.12
	SE	± 0.39	± 0.31	± 0.13	± 0.11	± 0.77	± 0.04	0.38	± 0.33	± 0.23	± 0.02

F_3 generation

Breeding behavior of 22 F_3 families of the cross confirmed the F_2 ratio, as 11 bred true for normal plants, 3 segregated for 3:1 and 5 for 15:1 and 3 bred true for the recessive with X^2 = 2.56 for 3 d.f. at 5% level in conformity with the expected F_3 ratio 7:4:4:1 (Table 20).

d. Deformed/palealess mutant

The mutant showed either complete absence of palea or appeared to be a narrow appendage and the lemma got transformed into an incurved sheath for the whole seed (Fig. 11). Deformed palea mutant occurred in one of the M_2 lines of Japan Violet treated with Vit C + 0.75% EMS. All other morphological characters resembled that of Japan Violet, except high sterility of 91.66% (Table 32).

The said M_2 line segregated for 19 normal : 6 mutant giving 3:1 suggesting recessive gene control for deformed palea (Table 17). The M_2 ratio was confirmed by the breeding behavior of 20 M_3 families of which 3 bred true for normal spikelets, 12 segregated for 3:1 and 5 bred true for mutant character in conformity with the expected M_3 ratio 1:2:1 with X^2=1.20 for 2 d.f. at 5% level (Table 18).

Fig. 11. Deformed palea mutant

a. Mutant panicle

b. Side view of the mutant grain

c. View at the palea side

Fig. 12. Broad seed mutant

a. Mutant plant

b. Control and mutant panicles

c. Panicles of the mutant

d. Upper row - grains of the mutant

Lower row - grains of the Japan violet

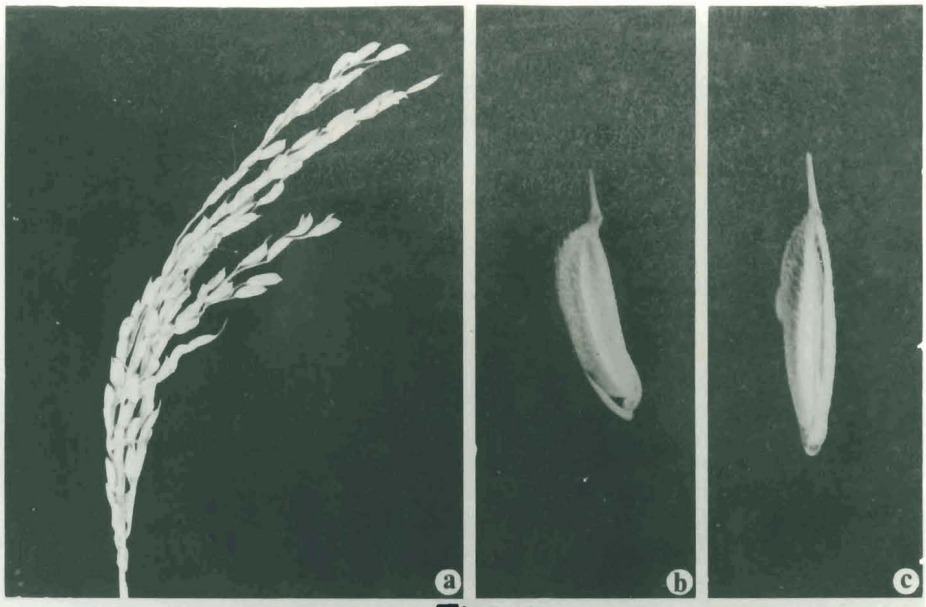

Fig 11

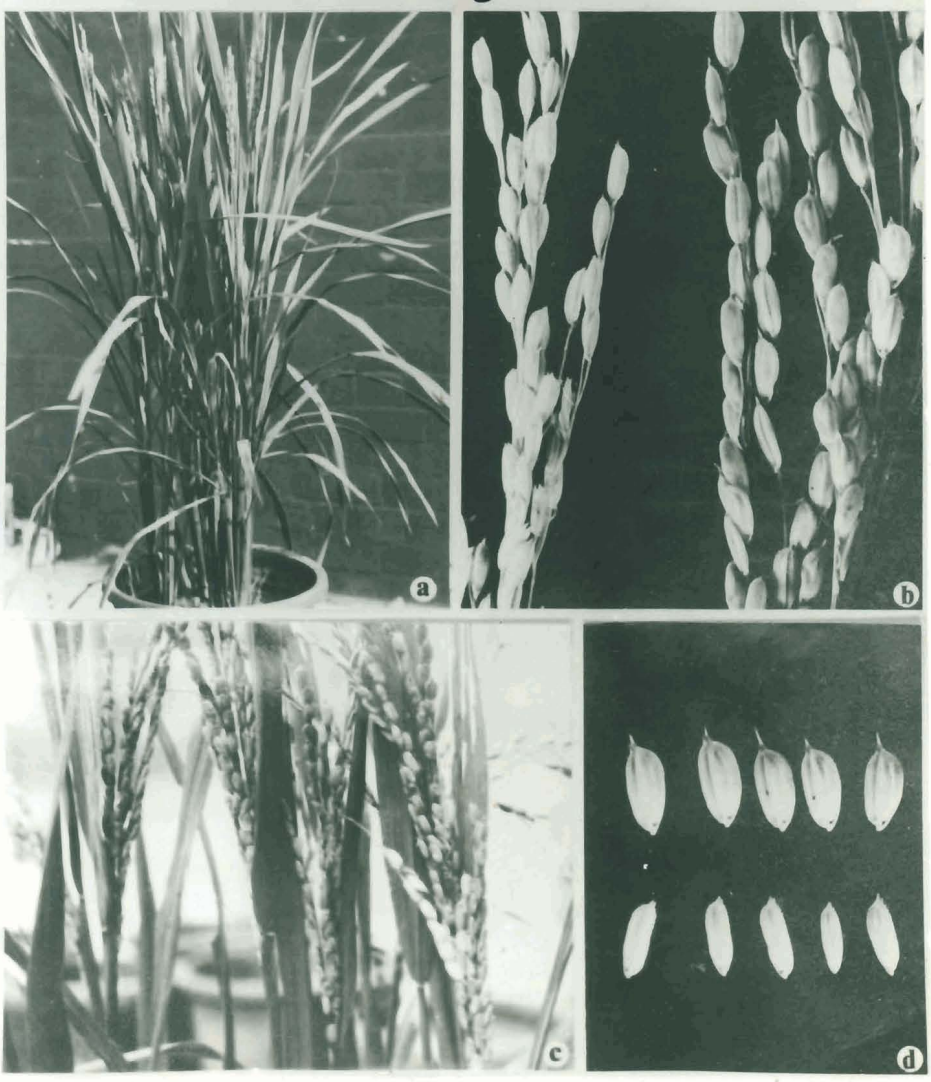

Fig 12

Table 32. Range and mean of certain morphometric characters in deformed palea mutant and its source parent Japan Violet.

Population/ Character	Range / Mean /	Plant height cm	Culm length cm	Total tiller	EBT	% of EBT	Panicle length cm	Total spikelet/ panicle	No. of st. spikelet/ panicle	% of sterility	Panicle density
Control	Range	71.0-89.0	38.5-51.5	7.0-16.0	6.0-12.0	62.5-100	17.5-22.0	75.5-120.5	9.5-32.0	8.44-35.60	3.78-6.19
	Mean	82.20	44.82	10.92	9.32	82.67	19.28	105.02	21.16	22.60	5.08
	SE	± 0.28	± 0.19	± 0.15	± 0.11	± 0.61	± 0.08	± 0.65	± 0.28	± 0.28	± 0.03
Deformed palea	Range	73.0-85.5	38.0-46.5	8.0-16.0	5-11	46.15-87.5	15.5-20.0	58-98	54.0-73.5	85.11-95.23	3.13-4.89
	Mean	75.81	44.22	12.36	8.08	65.91	18.22	72.78	66.48	91.66	3.99
	SE	± 0.10	± 0.09	± 0.09	± 0.06	± 0.57	± 0.05	± 0.40	± 0.58	± 0.11	± 0.01

Inheritance of deformed palea was further studied in the cross, Japan violet x Deformed palea mutant. F_1 showed spikelets with normal lemma palea.

F_2 generation

Out of 263 F_2 plants 206 were normal and 57 showed mutant character, giving good fit to the ratio 3:1 with X^2 = 1.55 which is not significant for 1 d.f. at 5% level (Table 19).

F_3 generation

Breeding behavior of F_3 families of the above cross confirmed the F_2 ratio. Out of 24 F_3 families studied 3 bred true for normal plants, 15 segregated for 3:1 and 6 bred true for mutant character with X^2 = 2.25 for 2 d.f. at 5% level in conformity with the expected F_3 ratio 1:2:1 (Table 20).

e. Broad seed mutant

Broad seed mutant occurred in one of the M_2 lines of DW + 1% EMS treatment. The mutant showed broad seeds of 0.40-0.55 cm breadth against 0.30 cm of the control (Fig. 1%). The mutant also showed increase in leaf width and plant height, while decrease in number of tillers to 4-8 with a mean of 5.4% (Table 33).

Table 33. Range and mean of certain morphometric characters in Broad seed mutant and its source parent Japan Violet

Population / characters	Range/ mean	Plant hieght cm	Culm length cm	Total tiller	EBT	% of EBT	Panicle length cm	Total spikelet / panicle	No. of St. / spikelet / panicle	% of sterility
Control	Range	71.0-89.0	38.5-51.50	7.0-16.0	6.0-12.0	62.50-100.00	17.5-22.0	75.5-120.5	9.5-32.0	8.44-35.60
	Mean	82.20	44.82	10.92	9.32	82.67	19.28	105.02	21.16	22.67
	SE	± 0.28	± 0.19	± 0.15	± 0.11	± 0.61	± 0.08	± 0.65	± 0.28	± 0.28
Broad seed mutant	Range	84.0-102.0	52.0-60.0	6.0-10.0	4.0-8.0	70.0-100.0	17.5-22.5	72.0-115.0	11.0-35.0	14.28-41.16
	Mean	95.20	56.10	7.12	5.44	76.4	19.20	79.55	21.35	26.08
	SE	± 0.23	± 0.13	± 0.07	± 0.06	± 0.85	± 0.06	± 0.56	± 0.34	± 0.51

Population / characters	Range/ mean	Panicle density	grain length cm	grain width cm	grain thickeness cm	grain index cm	leaf width cm
Control	Range	3.78-6.19	0.8-0.9	0.3-0.31	0.2-0.2	0.53-0.59	1.3-1.8
	Mean	5.08	0.87	0.30	0.2	0.58	1.5
	SE	± 0.03	± 0.002	± 0.00	± 0.00	± 0.001	± 0.04
Broad seed mutant	Range	4.11-5.48	0.98-1.3	0.4-0.55	0.2-0.35	0.49-0.77	2.1-2.9
	Mean	4.14	1.0	0.45	0.30	0.65	2.41
	SE	± 0.02	± 0.01	± 0.003	± 0.0001	± 0.005	± 0.01

The M_2 plants segregated into 17 normal: 5 mutants giving 3:1, suggesting recessive gene control of the mutant character (Table 17). Breeding behavior of 20 M_3 families confirmed the M_2 ratio, as 3 bred true for normal spikelets, 12 segregated for 3:1 and 5 bred true for the mutant character in conformity with the expected M_3 ratio 1:2:1 with X^2 = 1.2 for 2 d.f. at 5% level (Table 18).

Inheritance of broad seed was further studied in the cross, Japan violet x Broad seed mutant. F_1 showed normal seeds.

F_2 generation

Out of 226 F_2 plants 174 were normal and 52 broad seeded, giving good fit to the ratio 3:1 with X^2 = 0.48 which is not significant for 1 d.f. at 5%level (Table 19).

F_3 generation

Breeding behavior of 24 F_3 families of the above cross confirmed F_2 ratio, as 9 bred true for normal seeded plants, 11 segregated for 3:1 and 4 bred true for mutant character with X^2 = 2.25 for 2 d.f.at 5% level in conformity with the expected F_3 ratio 1:2:1 (Table 20).

f. Multipistillate mutant

Multipistillate mutant occurred in one of the M_3 families of Japan violet treated with Vit. C + 0.5% EMS. The mutant showed multiple pistil ranging from 1-8 with a mean of 4.24 and stigmatic lobes ranged from 2-4 with a mean of 3.11 (Figs. 13 a-f). Stamens appeared rudimentary or non-fertile, but some late formed tillers had some normal spikelets and seeds. The progenies of these seeds are similar to Japan Violet. In all other morphological characters the mutant plant appeared similar to its source parent Japan violet.

The said M_3 family segregated into 22 normal : 4 mutant giving 3:1 suggesting recessive gene control of the mutant character (Table 23), which was confirmed by the breeding behavior of 20 M_4 families, of which 7 bred true for normal plants, 8 segregated for the 3:1, 5 bred true for recessive mutant on the expected M_4 ratio 1:2:1 with $\chi^2 = 1.70$ for 2 d.f. at 5% level (Table 24).

Inheritance of multipistil mutant was further studied in the cross Japan Violet x Multipistil mutant. F_1 showed normal pistil like the source parent Japan violet.

Fig. 13. Multipistils of the multipistillate mutant

a. Twin pistil (ovary fused)

b. Twin pistil (ovary separated)

c&d. Six pistils (ovary fused)

e. Eight pistils (ovary fused)

f. Five pistils in four groups

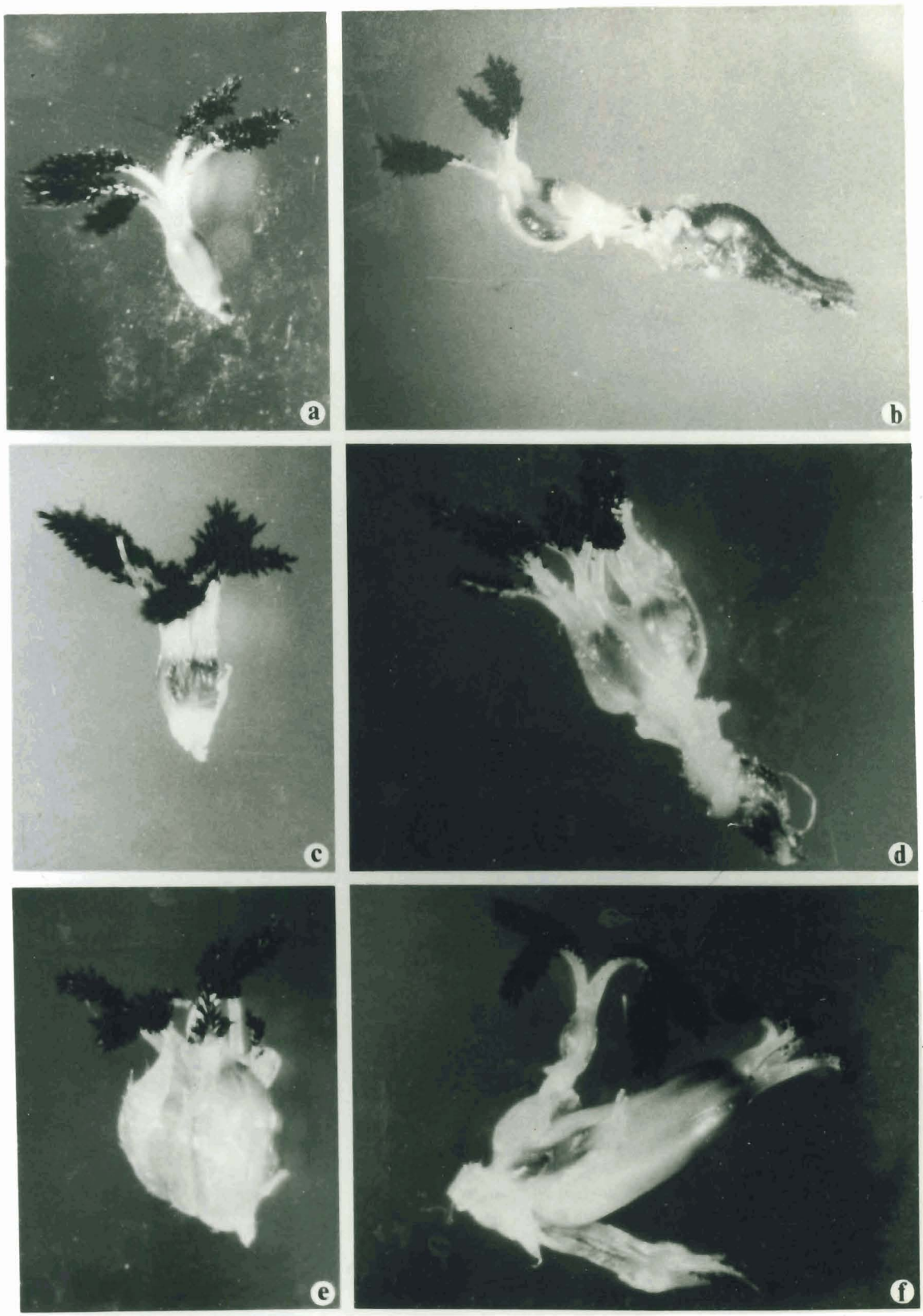

Fig 13

F_2 generation

Out of 275 F_2 plants 211 were normal types and 64 multipistil types, giving good fit to the ratio 3:1 with $X^2 = 0.44$, which is not significant for 1 d.f. at 5% level (Table 19).

F_3 generation

Breeding behavior of 28 F_3 families of the above cross confirmed F_2 ratio, as 10 bred true for single pistil condition and 12 segregated for 3:1 and 6 bred true for the multipistil condition with $X^2 = 1.71$ for 2 d.f. at 5% level in conformity with the expected F_3 ratio 1:2:1 (Table 20).

g. Anther sterile or male sterile mutant

Anther sterile mutant occurred in certain M_2 lines of the treatment . DW + 0.5% EMS and Vit C + 1% EMS . These mutants showed normal spikelets with shrivelled yellowish anthers, without dehiscence, but with 100% sterility. Pollen grains indicated very low mean fertility of 8.13% on acetocarmine staining.

M_2 segregation showed 21 normal: 3 mutants suggesting recessive gene control for the mutant character (Table 17). It was confirmed in the respective M_3 families. Out of 22 M_3 families 6

bred true for normal plants, 16 segregated for 3:1 on the expected M_3 ratio 1:2 with X^2 = 0.36 for 1 d.f.at 5% level (Table 18).

Inheritance of anther sterile mutant was further studied in Japan violet x Anther sterile mutant. F_1 had fertile spikelets like the source parent Japan Violet.

F_2 generation

Out of 182 F_2 plants 142 were normal and 40 mutant types, giving good fit to the ratio 3:1 with X^2 = 0.88 which is not significant for 1 d.f. at 5% level (Table 19).

F_3 generation

Breeding behavior of 23 F_3 families of the above cross confirmed F_2 ratio, as 8 bred true for normal plants, 15 segregated for 3:1 with X^2 = 0.02 for the expected F_3 ratio 1:2 for 1 d.f. at 5% level (Table 20).

3. Plant type mutants

a. Grassy rhizomatous mutant

Grassy rhizomatous mutant occurred in one of the M_2 lines of

Japan Violet treated with DW + 1% EMS. The mutant plant showed grassy rhizomatous stature with pigmentation, shorter leaves, short and rooted internodes, reduced panicles and reduced number of spikelets (Table 34 and Fig. 14). The mutant does not flower during normal duration of 60-65 days, but flowers very rarely on prolonged growth.

Panicles are axillary in position (Fig 14), spikelets are subnormal in size having a length of 0.64 cm and a width of 0.25 cm and completely sterile. Panicle length varied from 4-6.5cm with 5-6 spikelets/panicle. Internode length varied from 1.5 - 4.2 cm, leaf sheath varied from 5.5 cm - 10.7 cm and lamina varied from 1.5 - 10.8 with a mean of 5.29 cm (Table 34). Axillary shoots arise often from the growing main culm. By separating the rooted axillary shoots, it could be propagated vegetatively.

The M_2 population of 21 plants, segregated into 15 normal and 6 mutants giving 3:1 showing recessive monogenic control (Table 17). The M_2 ratio was confirmed by the breeding behavior of 20 M_3 families, of which 9 bred true for normal and 11 segregated for 3:1 on the expected M_3 ratio 1:2 with X^2 = 1.22 for 1 d.f. at 5% level (Table 18). As the mutant was instantly sterile, no hybridization could be effected with Japan Violet.

Fig. 14. Grassy rhizomatous mutant

a. Mutant plant in the field

b. Mutant plant growing in pot

c. Showing the emergence of roots from every node

d. Leaves with long sheath and short lamina

e. Showing the short internodes

f. Close-up view of the roots from the nodes

g&h. Axillary development of the panicle

Fig 14

Table 34. Range and mean of certain morphometric characters in grassy mutant and its source parent Japan Violet

Population/ Character	Range / Mean / SE	Plant height cm	Inter nodel length cm	Panicle length cm	Total No. of spikelet / panicle	No. of st. spikelet	% of sterility	Panicle density	Length of leaf sheath
Control	Range	69.0-89.0	0.5-33.0	17.0-22.50	75.0-120.0	9.5-32.0	8.4-33.56	3.78-6.19	14.50 - 7.60
	Mean	82.20	11.53	20.25	115.80	21.60	20.48	5.08	16.78
	SE	± 0.28	± 0.05	± 0.08	± 0.65	± 0.28	± 0.28	±0.03	± 0.05
Grassy mutant	Range	20.0-26.0	1.5-4.2	4.0-6.5	3.0-8.0	3.0-8.0	100%	0.55-1.33	5.5 - 10.7
	Mean	24.16	2.49	4.88	4.00	4.00	100%	0.82	0.82
	SE	±0.08	± 0.05	± 0.04	± 0.7	± 0.07	--	± 0.01	± 0.01

Population/ Character	Range/ Mean/ SE	Length of leaf lamina cm	Leaf width cm	Length of spikelet cm	Width of spikelet cm
Control	Range	38.5-45.5	1.30-1.80	0.80-0.90	0.30-0.31
	Mean	41.72	1.50	0.87	0.30
	SE	± 0.11	± 0.04	± 0.002	± 0.01
Grassy mutant	Range	1.50-10.80	0.30-0.70	0.60-0.70	0.23-0.25
	Mean	5.29	0.50	0.64	0.25
	SE	± 2.19	± 0.12	± 0.05	± 0.01

b. Brittle culm mutant

Brittle culm mutant occurred in M_3 of Japan Violet treated with Vit. C + 1% EMS. Plant parts like culm, leaf, leaf sheath, rachis showed brittleness. The brittle nature appeared more prominent after flowering and seed setting. The plant parts break quickly on slight force. All other morphometric characters are similar to Japan Violet.

Data on inheritance of brittle culm in the M_3 line studied are furnished in Table 23. Out of 64 plants of the M_3 line 91-1-46-2 of Vit. C + 1% EMS treated-Japan Violet, 51 showed normal culm and 13 brittle culm giving 3:1 ratio (Table 23), which was confirmed by the breeding behavior of 23 M_4 families, of which 7 bred true for normal plants, 10 segregated for 3:1 and 6 bred true for mutant character in conformity with the expected ratio 1:2:1 with $X^2 = 0.48$ for 2 d.f.at 5% level (Table 24).

Inheritance of brittle culm was further studied in the cross Japan violet x Brittle culm mutant. F_1 showed normal culm as dominant over brittle culm.

F_2 generation

Out of 236 F_2 plants 182 were normal and 54 showed brittle culm

character, giving good fit to the ratio 3:1 with X^2 = 0.56 which is not significant for 1 d.f. at 5% level (Table 19).

F_3 generation

Breeding behavior of F_3 families of the cross confirmed the F_2 ratio, as out of 25 F_3 families studied, 6 bred true for normal plants, 11 segregated for 3:1 and 8 bred true for mutant character with X^2 = 0.20 for 2 d.f.at 5% level in conformity with the expected F_3 ratio 1:2:1 (Table 20).

c. Tall recessive mutant

Tall recessive mutant occurred in one of the M_2 lines of Japan Violet treated with Vit.C + 0.75% EMS. Height of the mutant remained almost same as that of the parent upto flowering. By the emergence of panicles, internodes elongated further to attain a mean height of 108.12 cms. (Fig. 15). Mean and range of the morphometric characters studied in the mutant in comparison with Japan Violet are presented in Table 35. In the mutant 1st, 2nd, 3rd and 4th internodes showed elongation of 77.40%, 142.00%, 60.70%, and 66.70% respectively over control. Panicle length and spikelet length also showed significant increase over the control, while significant reduction was observed in spikelet sterility (Table 35). Other

Fig. 15. Tall recessive mutant

a. Tall recessive mutant

b. Control - Japan violet

c. Pancile of the control

d. Panicle of the mutant

Fig. 16. High tillering dwarf mutant

a. Control - Japan violet

b. High tillering dwarf mutant

c. Close-up view of the mutant

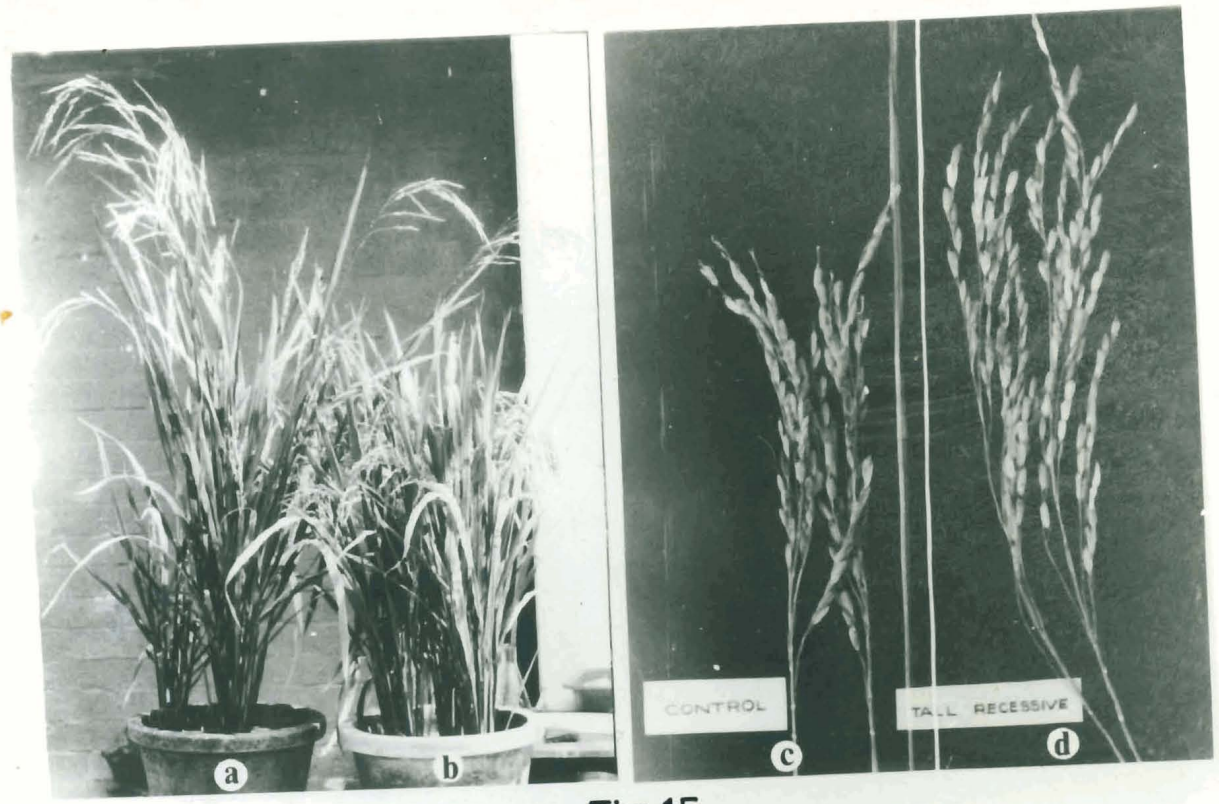
a
b
CONTROL
c
TALL RECESSIVE
d

Fig 15


CONTROL
a
M3VI-73-1-26
b
M3VI-73-1-26
c

Fig 16

Table 35. Range and mean of certain morphometri characters in tall recessive mutant and its source parent Japan Violet

Population / characters	Range/ mean	Plant hieght cm	Culm length cm	Total tiller	EBT	% of EBT	Panicle length cm	Total spikelet / panicle	% of sterility	Panicle density
Control	Range	71.0-89.0	38.5-51.50	7.0-16.0	6.00-12.00	62.50-100.00	17.50-22.00	75.50-120.50	8.44-35.60	3.78-6.19
	Mean	82.20	44.82	10.92	9.22	82.67	19.28	105.02	22.60	5.08
	SE	± 0.28	± 0.19	± 0.15	± 0.11	± 0.61	± 0.08	± 0.65	± 0.28	± 0.03
Tall recessi ve mutant	Range	100.00-116.00	78.00-94.00	7.00-15.00	7.00-12.00	72.00-100.00	21.30-24.00	86.00.121.00	6.94-29.71	3.30-5.09
	Mean	108.12	86.08	9.44	8.68	92.20	22.72	97.22	18.16	5.04
	SE	± 0.15	± 0.23	± 0.09	± 0.07	± 0.53	± 0.03	± 0.48	± 0.22	± 0.02

Population / characters	Range/ mean	grain length cm	grain width cm	grain thickness cm	grain index	inter nodal length, cm (top to bottom)			
						1	2	3	4
Control	Range	0.80-0.90	0.30-0.31	0.20-0.20	0.53-0.59	28.00-33.00	10.00-13.50	1.50-4..00	0.50-2.00
	Mean	0.87	0.30	0.20	0.58	29.60	11.23	2.78	1.21
	SE	± 0.002	± 0.00	± 0.00	± 0.001	± 0.04	± 0.01	± 0.01	± 0.01
Tall recessi ve mutant	Range	0.90-1.10	0.30-0.31	0.20-0.24		46.00-57.00	21.00-30.00	2.00-6.00	1.00-3.00
	Mean	0.92	0.30	0.23	0.74	52.46	27.12	4.50	2.02
	SE	± 0.05	± 0.003	± 0.02		± 0.13	± 0.13	± 0.05	± 0.03

morphological characters such as total tillers and ear bearing tillers (EBT) showed slight reduction than the control. Further, the mutant showed panicle emergence of 8-12 cm above the flag leaf unlike the sheathed panicle character of Japan violet. The mutant also showed nonlodging habit like the control

One of the M_2 lines segregated into 22 normal: 4 mutants giving 3:1, showing recessive gene control of the mutant character (Table 17). M_2 ratio was confirmed by the breeding behavior of 22 M_3 families of which 6 bred true for normal, 12 segregated for 3:1 and 4 bred true for mutant character in conformity with the expected M_3 ratio 1:2:1 with $X^2 = 0.30$ for 2 d.f.at 5% level (Table 18).

Inheritance of tall recessive mutant was further studied in the cross, Japan violet x Tall recessive mutant F_1 resembled Japan violet.

F_2 generation

Out of 236 F_2 plants of the cross, 172 were normal and 64 mutant type, giving good fit to the ratio 3:1 with $X^2 = 0.56$ for 1 d.f. at 5% level, showing recessive gene control of the mutant character (Table 19).

F_3 generation

Breeding behavior of 30 F3 families confirmed the F_2 ratio, as

10 bred true for normal plants, 12 segregated for 3:1 and 8 bred true for mutant character with X^2 = 1.61 for 2 d. f. at 5% level in conformity with the expected F_3 ratio 1:2:1 (Table 20).

d. High tillering dwarf mutant

High tillering dwarf mutant occurred in M_3 of Vit C + 0.75% EMS treatment. These plants were small and plant height ranged from 23-48 cm with a mean of 40.04 cm (Fig. 16), culm length ranged from 11-21 cm with a mean of 15.75 cm and total tiller number ranged from 43-165 with a mean of 94.24 (Table 36). Panicle length, total number of spikelets/panicle and number of fertile spikelets/panicle, length of leaf sheath, lamina and internode also showed reduction when compared to the control plant (Table 36).

The M_3 family concerned segregated for 42 normal: 10 mutant, giving 3:1 (Table 23), which was confirmed by the breeding behavior of 30 M_4 families of which 8 bred for true normal plants, 12 segregated for 3:1 and 10 bred true for the mutant character in conformity with the expected ratio 1:2:1 with X^2 = 1.46 for 2 d.f. at 5% level (Table 24).

Inheritance of high tillering dwarf mutant was further studied in the cross, Japan Violet x High tillering dwarf . F_1 was normal like the source parent.

Tabel 36. Range and mean of certain morphometric characters in high tlleing dwarf mutant and its source parent Japan Violet.

Population / characters	Range/ mean	Plant hieght cm	Culm length cm	Total tiller	EBT	% of EBT	Panicle length cm	Total spikelet / panicle	% of sterility	Panicle density
Control	Range	71.00-89.00	38.5-51.50	7.00-16.00	6.00-12.00	62.50-100.00	17.50-22.00	75.50-120.50	8.44-35.60	3.78-6.19
	Mean	82.20	44.82	10.92	9.32	82.67	19.28	105.02	22.60	5.08
	SE	± 0.28	± 0.19	± 0.15	± 0.11	± 0.61	± 0.08	± 0.65	± 0.28	± 0.03
High tillering dwarf	Range	23.00-48.00	11.00-21.00	43.00-165.00	39.00-144.00	77.92-100.00	8.75-15.25	28.00-62.00	30.00-76.78	3.20-4.13
	Mean	40.04	15.75	94.24	86.68	91.98	12.02	46.15	42.13	3.84
	SE	± 0.22	± 0.12	± 1.47	± 1.40	± 0.65	± 0.06	± 0.29	± 0.41	± 0.02

Population / characters	Range/ mean	grain length cm	grain width cm	grain thickness cm	grain index	length of leaf sheath cm	length of leaf lamina cm	inter nodal length, cm (top to bottom)			
								1	2	3	4
Control	Range	0.80-0.90	0.30-031	0.20-0.20	0.53-0.59	14.50-17.60	38.50-45.50	28.00-33.00	10.00-13.50	1.50-4..00	0.50-0.20
	Mean	0.87	0.30	0.20	0.58	16.78	41.72	29.60	11.23	2.78	1.21
	SE	± 0.002	± 0.01	± 0.00	± 0.001	± 0.05	± 0.11	± 0.08	± 0.05	± 0.04	± 0.02
High tillering dwarf	Range	0.50-0.62	0.25-0.30	0.15-0.20	0.30-0.41	6.00-12.00	4.00-9.00	10.00-13.50	1.80-2.30	0.10-1.70	0.10-0.60
	Mean	0.53	0.27	0.17	0.34	8.57	7.50	12.58	2.01	1.32	0.52
	SE	± 0.001	± 0.001	± 0.001	± 0.001	± 0.08	± 0.07	± 0.04	± 0.01	± 0.01	± 0.01

F_2 generation

Out of 429 F_2 plants, 318 were normal and 111 high tillering dwarf mutant types giving good fit to the ratio 3:1 with X^2 = 0.17 which is not significant for 1 d.f. at 5% level (Table 19).

F_3 generation

Breeding behavior of 25 F_3 families of the above cross studied confirmed the F_2 ratio, as 3 bred true for normal plants, 17 segregated for 3:1 and 5 bred true for the mutant character with X^2 = 3.13, which is not significant for 2 d. f. at 5% level (Table 20).

e. Procumbent plant type

Japan Violet presents a short erect stature. The procumbent mutant occurred in one of the M_2 lines of Japan Violet treated with DW + 1% EMS. The tillers of the mutant appeared slanding to spread around with panicles without tip-sterility (Fig. 17). Grains showed devoid of awn tipped condition. Other morphometric characters are presented in Table 37.

M_2 plants segregated into 18 normal : 4 mutants giving 3:1 suggesting recessive gene control for the mutant character (Table

Fig. 17. Procumbent mutant

a. Mutant plant

b. Panicles of control and mutant, showing tip sterility in control and its absence in the mutant's panicle

Fig 17

Table 37. Range and mean of certain morphometric characters in procumbent mutant and its source parent Japan Violet

Population/ Character	Range / Mean / SE	Plant height cm	Culm length cm	Total tiller	EBT	% of EBT	Panicle length cm	Total spikelet/ panicle	No. of st. spikelet/ panicle	% of sterility	Panicle density
Control	Range	71.0-89.0	38.5-51.5	7.0-16.0	6.0-12.0	62.5-100.0	17.5-22.0	75.5-120.5	9.5-32.0	8.44-35.60	3.78-6.19
	Mean	82.20	44.82	10.92	9.32	82.67	19.28	105.02	21.16	22.60	5.08
	SE	± 0.28	± 0.19	± 0.15	± 0.11	± 0.61	± 0.08	± .65	± 0.28	± 0.28	± 0.03
Procumbent mutant	Range	57.0-77.0	31.0-44.0	12.0-27.0	11.0-20.0	62.97-100.0	15.0-17.75	56.5-105.5	32.5-105.5	36.01-62.52	3.35-5.59
	Mean	66.05	38.12	18.48	15.75	85.72	16.44	81.22	40.80	51.00	4.94
	SE	± 0.40	± 0.23	± 0.18	± 0.14	± 0.45	± 0.05	± 0.58	± 0.62	± 0.66	± 0.03

17). Breeding behavior of 24 M_3 families confirmed the ratio, as 5 bred true for normal plants, 10 segregated for 3:1 and 9 bred true for the mutant character in conformity with the expected M_3 ratio 1:2:1 with $x^2 = 2.0$ for 2 d.f at 5% level (Table 18).

Inheritance of procumbent mutant was further studied in the cross Japan Violet x Procumbent mutant. F_1 showed normal plant type.

F_2 generation

Out of 312 F_2 plants, 236 were normal and 76 mutant types, giving good fit to the ratio 3:1 with $X^2 = 0.07$, which is not significant for 1 d.f at 5% level (Table 19).

F_3 generation

Breeding behavior of 24 F_3 families of the above cross confirmed F_2 ratio, as 9 bred true for normal plants, 10 segregated for 3:1 and 5 bred true for mutant character with $X^2 = 2.0$ for 2 d.f. at 5% level in conformity with the expected F_3 ratio 1:2:1 (Table 20).

4. Frequency of occurrence of EMS induced mutants of Japan violet

Frequency of occurrence of mutants of Japan Violet treated with

different concentrations of EMS calculated on percentage basis with reference to either M_2/M_3 population concerned is presented in Table 38. Scrutiny of the data showed that pretreatment with Vit. C enhanced mutation by 22% with 0.5% EMS, by 90 % with 0.75% EMS and by 96% with 1% EMS. However, maximum number of mutations were found to have been induced by 1% EMS in both cases of pretreatments.

C. INHERITANCE OF ANTHOCYANIN PIGMENTATION IN PLANT PARTS AND OTHER CHARACTERS STUDIED IN CROSSES 1-4

Inheritance of anthocyanin pigmentation and other morphological characters were studied in four crosses: (1) Cherumodan x Japan Violet (normal cross as control), (2) Cherumodan x Japan Violet - pollen grains treated with 1500 rads of X-rays, (3) Cherumodan x Japan Violet - pollen grains treated with 2000 rads of X-rays and (4) Cherumodan x Japan Violet - pollen grains treated with 5000 rads of X-rays. All crosses were studied up to F_3/M_3F_3 generation for genetical analysis except in the latter cross where M_4F_4 generation was also incidentally studied. The patterns of inheritance observed for 14 morphological characters (12 anthocyanin and 2 non anthocyanin) are described below :

1. Leaf axil

Inheritance of leaf axil pigmentation was studied in Cherumodan x Japan violet (normal), Cherumodan x Japan Violet -1500 rad,

Table 38. Frequency of occurrence (%) of morphological mutants obtained with different treatments of EMS and in combination with Vit. C pre-treatment

Sl. No.	Population	Mutant	Control	Treatment					
				DW + EMS			Vit.C + EMS		
				0.5%	0.75%	1%	0.5%	0.75%	1%
1	2	3	4	5	6	7	8	9	10
1	M_1	--	--	--	--	--	--	--	--
2	M_2	Albino	--	0.10	0.73	0.12	0.17	0.92	0.93
3	M_2	Lethal yellow	--	0.07	0.28	0.59	0.35	--	--
4	M_2	Chlorina	--	0.21	--	--	--	--	0.22
5	M_2	Striped	--	--	--	--	0.07	0.10	0.37
6	M_2	Deformed palea	--	--	--	--	--	0.20	--
7	M_2	Beaked lemma depressed palea	--	--	--	0.24	--	--	--
8	M_2	Long sterile glume	--	--	--	0.12	--	--	--
9	M_2	Procumbent	--	--	--	0.24	--	--	--
10	M_2	Broad seed	--	--	--	0.30	--	--	--
11	M_2	Grassy rhizomatous	--	--	--	0.36	--	--	--
12	M_2	Anther sterile	--	0.10	--	--	--	--	0.15
13	M_2	Abnomorphic spikelet	--	--	--	--	--	--	0.45
14	M_2	Tall recessive	--	--	--	--	--	0.13	--
15	M_3	Multipistil	--	--	--	--	--	0.16	--

1	2	3	4	5	6	7	8	9	10
16	M_3	High tillering dwarf	--	--	--	--	--	0.41	--
17	M_3	Partial green	--	--	--	0.48	--	--	--
18	M_3	Brittle culm	--	--	--	--	--	--	0.50
19	M_3	Complete green	--	--	--	--	--	--	0.69
20	M_3	Spotted leaf	--	--	--	--	--	--	1.50
21	Total frequency			0.48	1.01	2.45	0.59	1.92	4.81
22	% of increase by Vit. C treatment			--	--	--	22.92	90.09	96.33

Cherumodan x Japan Violet - 2000 rad and Cherumodan x Japan Violet - 5000 rad. Leaf axil of both Cherumodan and Japan Violet showed purple colour. F_1 of all crosses showed dominance of purple leaf axil (Table 39a-1).

F_2/ M_2F_2 generation

F_2 population of Cherumodan x Japan Violet (normal), Cherumodan x Japan Violet - 1500 rad, Cherumodan x Japan Violet - 2000 and 5000 rads segregated into the ratio 15 purple : 1 green with X^2= 1.07, 0.16, 0.34, 0.08 respectively which are not significant for 1 d.f. at 5% level (Table 39a&b-1). The ratio indicates duplicate factors for control of leaf axil pigmentation, the duplicate recessives giving green colour.

F_3/M_3F_3 generation

Breeding behaviour of F_3/M_3F_3 families in the above crosses confirmed the F_2/M_2F_2 ratio. Out of 30 F_3 families of Cherumodan x Japan Violet (normal), 12 bred true for purple leaf axil, 7 segregated for 3:1, 6 for 15:1 and 5 bred true for green leaf axil. Out of 32 M_3F_3 families of Cherumodan x Japan Violet -1500, 12 bred true for purple leaf axil, 8 segregated for 3:1, 7 segregated for 15:1 and 5 bred true for green leaf axil. Out of 32 M_3F_3 families of

Table 39a. Inheritance of different morphological characters in F_1/M_1F_1 and F_2/M_2F_2 of crosses 1-4 Cherumodan x Japan Violet (normal, 1500/2000 and 5000 rad X-ray treatments)

Sl. No.	Characters studied	Crosses	Parents Female	Parents Male	F_1	O/E	F_2 Frequency +	F_2 Frequency -	Total	Ratio	X^2	P
1	2	3	4	5	6	7	8	9	10	11	12	13
1.	Leaf Axil	Cherumodan x Japan Violet (normal)	+	+	+	O	132.00	12.00	144			
						E	135.00	9.00	144	15:1	1.07	0.50-0.30
		Cherumodan x Japan Violet (1500 rad)	+	+	+	O	171.00	10.00	181			
						E	169.69	11.31	181	15:1	0.16	0.70-0.50
		Cherumodan x Japan Violet (2000 rad)	+	+	+	O	70.00	6.00	76			
						E	71.25	4.75	76	15:1	0.34	0.70-0.50
		Cherumodan x Japan violet (5000 rad)	+	+	+	O	36.00	2.00	38			
						E	35.63	2.37	38	15:1	0.08	0.80-0.70
2.	Leaf Sheath	Cherumodan x Japan Violet (normal)	-	+	+	O	111.00	33.00	144			
						E	108.00	36.00	144	3:1	0.33	0.70-0.50
		Cherumodan x Japan Violet (1500 rad)	-	+	+	O	126.00	55.00	181			
						E	135.75	45.25	181	3:1	2.80	0.10-0.05
		Cherumodan x Japan Violet (2000 rad)	-	+	+	O	55.00	21.00	76			
						E	57.00	19.00	76	3:1	0.28	0.70-0.50
		Cherumodan x Japan Violet (5000 rad)	-	+	+	O	23.00	15.00	38			
						E	21.38	16.62	38	9:7	0.28	0.70-0.50

1	2	3	4	5	6	7	8	9	10	11	12	13
3.	Leaf Blade	Cherumodan x Japan Violet (normal)	-	+	+	O	82.00	62.00	144			
						E	81.00	63.00	144	9:7	0.03	0.90-0.80
		Cherumodan x Japan Violet (1500 rad)	-	+	+	O	93.00	88.00	181			
						E	101.81	79.19	181	9:7	1.74	0.20-0.10
		Cherumodan x Japan Violet (2000 rad)	-	+	+	O	38.00	38.00	76			
						E	32.06	43.94	76	27:37	1.90	0.20-0.10
		Cherumodan x Japan Violet (5000 rad)	-	+	+	O	14.00	24.00	38			
						E	16.03	21.97	38	27:37	0.19	0.70-0.50
4.	Leaf Margin	Cherumodan x Japan Violet (normal)	-	+	+	O	100.00	44.00	144			
						E	108.00	36.00	144	3:1	2.37	0.20-0.10
		Cherumodan x Japan Violet (1500 rad)	-	+	+	O	114.00	67.00	181			
						E	101.81	79.19	181	9:7	3.34	0.10-0.05
		Cherumodan x Japan Violet (2000 rad)	-	+	+	O	49.00	27.00	76			
						E	42.75	33.25	76	9:7	2.09	0.20-0.10
		Cherumodan x Japan Violet (5000 rad)	-	+	+	O	19.00	19.00	38			
						E	21.38	16.62	38	9:7	0.60	0.50-0.30

1	2	3	4	5	6	7	8	9	10	11	12	13
5.	Leaf tip	Cherumodan x Japan Violet (normal)	-	+	+	O	103.00	41.00	144			
						E	108.00	36.00	144	3:1	0.93	0.50-0.30
		Cherumodan x Japan Violet (1500 rad)	-	+	+	O	114.00	67.00	181			
						E	101.81	79.19	181	9:7	3.33	0.10-0.05
		Cherumodan x Japan Violet (2000 rad)	-	+	+	O	53.00	23.00	76			
						E	57.00	19.00	76	3:1	1.12	0.30-0.20
		Cherumodan x Japan Violet (5000 rad)	-	+	+	O	20.00	18.00	38			
						E	21.38	16.62	38	9:7	0.19	0.70-0.50
6.	Juncura proper	Cherumodan x Japan Violet (normal)	-	+	+	O	75.00	69.00	144			
						E	81.00	63.00	144	9:7	1.01	0.50-0.30
		Cherumodan x Japan Violet (1500 rad)	-	+	+	O	80.00	101.00	181			
						E	76.36	104.64	181	27:37	0.72	0.50-0.30
		Cherumodan x Japan Violet (2000 rad)	-	+	+	O	33.00	43.00	76			
						E	32.06	43.94	76	27:37	0.05	0.90-0.80
		Cherumodan x Japan Violet (5000 rad)	-	+	+	O	15.00	23.00	38			
						E	16.03	21.97	38	27:37	1.10	0.30-0.20

1	2	3	4	5	6	7	8	9	10	11	12	13
7.	Ligule	Cherumodan x Japan Violet (normal)	-	+	+	O E	89.00 81.00	55.00 63.00	144 144	9:7	1.81	0.20-0.10
		Cherumodan x Japan Violet (1500 rad)	-	+	+	O E	102.00 101.81	79.00 79.19	181 181	9:7	0.001	>0.95
		Cherumodan x Japan Violet (2000 rad)	-	+	+	O E	39.00 42.75	37.00 33.25	76 76	9:7	1.21	0.30-0.20
		Cherumodan x Japan Violet (5000 rad)	-	+	+	O E	15.00 16.03	23.00 21.97	38 38	27:37	1.10	0.30-0.20
8.	Auricle	Cherumodan x Japan Violet (normal)	-	+	+	O E	77.00 81.00	67.00 63.00	144 144	9:7	0.45	0.70-0.50
		Cherumodan x Japan Violet (1500 rad)	-	+	+	O E	85.00 76.36	92.00 104.64	181 181	27:37	3.62	0.10-0.05
		Cherumodan x Japan Violet (2000 rad)	-	+	+	O E	33.00 32.06	43.00 43.94	76 76	27:37	0.05	0.90-0.80
		Cherumodan x Japan Violet (5000 rad)	-	+	+	O E	12.00 16.03	26.00 21.97	38 38	27:37	1.75	0.20-0.10

1	2	3	4	5	6	7	8	9	10	11	12	13
9.	Node	Cherumodan x Japan Violet (normal)	-	-	-	O	30.00	114.00	144			
						E	27.00	117.00	144	3:13	0.41	0.70-0.50
		Cherumodan x Japan Violet (1500 rad)	-	-	-	O	25.00	156.06	181			
						E	33.94	147.06	181	3:13	2.29	0.20-0.10
		Cherumodan x Japan Violet (2000 rad)	-	-	-	O	16.00	60.00	76			
						E	14.25	61.75	76	3:13	0.26	0.70-0.50
		Cherumodan x Japan Violet (5000 rad)	-	-	-	O	7.00	31.00	38			
						E	7.12	30.88	38	3:13	0.002	0.98-0.95
10.	Internode	Cherumodan x Japan Violet (normal)	-	+	+	O	106.00	38.00	144			
						E	108.00	36.00	144	3:1	0.15	0.70-0.50
		Cherumodan x Japan Violet (1500 rad)	-	+	+	O	126.00	55.00	181			
						E	135.75	45.25	181	3:1	2.80	0.10-0.05
		Cherumodan x Japan Violet (2000 rad)	-	+	+	O	54.00	22.00	76			
						E	57.00	19.00	76	3:1	0.63	0.50-0.30
		Cherumodan x Japan Violet (5000 rad)	-	+	+	O	22.00	16.00	38			
						E	21.38	16.43	38	9:7	0.04	0.90-0.80

1	2	3	4	5	6	7	8	9	10	11	12	13
	11. Stigma	Cherumodan x Japan Violet (normal)	-	+	+	O E	108.00 108.00	36.00 36.00	144 144	3:1	0.00	
		Cherumodan x Japan Violet (1500 rad)	-	+	+	O E	138.00 135.75	43.00 45.25	181 181	3:1	0.15	0.70-0.50
		Cherumodan x Japan Violet (2000 rad)	-	+	+	O E	54.00 57.00	22.00 19.00	76 76	3:1	0.63	0.50-0.30
		Cherumodan x Japan Violet (5000 rad)	-	+	+	O E	22.00 21.38	16.00 16.62	38 38	9:7	0.04	0.90-0.80
	12. Apiculus	Cherumodan x Japan Violet (normal)	-	+	+	O E	106.00 108.00	38.00 36.00	144 144	3:1	0.15	0.70-0.50
		Cherumodan x Japan Violet (1500 rad)	-	+	+	O E	127.00 135.75	54.00 45.25	181 181	3:1	2.26	0.20-0.10
		Cherumodan x Japan Violet (2000 rad)	-	+	+	O E	55.00 57.00	21.00 19.00	76 76	3:1	0.28	0.70-0.50
		Cherumodan x Japan Violet (5000 rad)	-	+	+	O E	22.00 21.38	16.00 16.62	38 38	9:7	0.04	0.90-0.80

1	2	3	4	5	6	7	8	9	10	11	12	13
13.	Awning	Cherumodan x Japan Violet (normal)	-	+	-	O E	107.00 108.00	37.00 36.00	144 144	3:1	0.04	0.90-0.80
		Cherumodan x Japan Violet (1500 rad)	-	+	-	O E	128.00 135.75	53.00 45.25	181 181	3:1	1.86	0.20-0.10
		Cherumodan x Japan Violet (2000 rad)	-	+	-	O E	61.00 57.00	15.00 19.00	76 76	3:1	1.12	0.30-0.20
		Cherumodan x Japan Violet (5000 rad)	-	+	-	O E	27.00 28.50	11.00 9.50	38 38	3:1	0.32	0.70-0.50
14.	Tip Sterility	Cherumodan x Japan Violet (normal)	-	+	-	O E	107.00 108.00	37.00 36.00	144 144	3:1	0.04	0.90-0.80
		Cherumodan x Japan Violet (1500 rad)	-	+	-	O E	144.00 135.75	37.00 45.25	181 181	3:1	2.01	0.20-0.10
		Cherumodan x Japan Violet (2000 rad)	-	+	-	O E	57.00 57.00	19.00 19.00	76 76	3:1	0.00	
		Cherumodan x Japan Violet (5000 rad)	-	+	-	O E	33.00 28.50	5.00 9.50	38 38	3:1	2.84	0.10-0.05

TABLE 39b. Transformation of genetical ratios for morphological characters occurred in F_2 of X-ray treated populations of the crosses 2-4 relative to the control cross of Cherumodan x Japan Violet

	Character	Control cross	Treated crosses		
		1	2	3	4
1.	Px	15:1	15:1	15:1	15:1
2.	Psh	3:1	3:1	3:1	9:7
3.	Pl	9:7	9:7	27:37	27:37
4.	Plm	3:1	9:7	9:7	9:7
5.	Pla	3:1	9:7	3:1	9:7
6.	Pjp	9:7	27:37	27:37	27:37
7.	Plg	9:7	9:7	9:7	27:37
8.	Pau	9:7	27:37	27:37	27:37
9.	Pn	3:13	3:13	3:13	3:13
10.	Pin	3:1	3:1	3:1	9:7
11.	Ps	3:1	3:1	3:1	9:7
12.	Pa	3:1	3:1	3:1	9:7
13.	an	3:1	3:1	3:1	3:1
14.	tst	3:1	3:1	3:1	3:1

1 - Cherumodan x Japan violet normal (control)
2 - " " pollen treated with 1500 rad X-ray
3 - " " pollen treated with 2000 rad X-ray
4 - " " pollen treated with 5000 rad X-ray

Cherumodan x Japan Violet - 2000 rad, 14 bred true for purple leaf axil, 12 segregated for 3:1, 5 segregated for 15:1 and 1 bred true for green leaf axil. Out of 25 M_3F_3 families of Cherumodan x Japan Violet-5000 rad, 11 bred true for purple leaf axil, 5 segregated for 3:1 and 7 segregated for 15:1 and 2 bred true for green leaf axil. The respective X^2 value are 5.97, 4.91, 3.63 and 0.46 which are not significant against the table value for 3 d.f.at 5% level and hence the patterns of segregation are in conformity with the expected F_3/M_3F_3 ratio of 7:4:4:1 (Table 40-1).

2. Leaf sheath

Leaf sheath pigmentation was studied in all the above mentioned crosses. Japan Violet had purple leaf sheath and Cherumodan green. F_1 of all crosses showed dominance of purple colour in leaf sheath (Table 39a 2).

F_2/M_2F_2 generation

F_2/M_2F_2 population of Cherumodan x Japan Violet (normal), Cherumodan x Japan violet - 1500 rad, Cherumodan x Japan Violet - 2000 rad segregated into 3 purple : 1 green with X^2 = 0.33, 2.80, 0.28 and Cherumodan x Japan Violet - 5000 rad segregated into 9 purple : 7 green with X^2 = 0.28 which are not significant for 1 d. f. at 5% level (Table 39a-2 & 39b-2)

TABLE 40. **Breeding behaviour of F_3 / M_3 F_3 families for different morphological characters studied in crosses (1-4) Cherumodan x Japan Violet normal/1500/2000/5000 rad X-ray treated pollen grains used for hybridization**

Sl. No.	Characters studied	Crosses	F_2 Ratio	Expetced F_3 Ratio*	O/E	Breeding Behaviour of F_3 families								TR	Total	X^2	P
						TD	3:1	1:3	9:7	15:1	1:15	3:13	27:37				
1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
1.	Leaf Axil	Cherumodan x Japan Violet (normal)	15:1	7:4:4:1	O	12.00	7.00			6.00				5.00	30		
					E	13.33	7.50			7.50				1.87	30	5.97	0.20-0.10
		Cherumodan x Japan Violet (1500 rad)	15:1	7:4:4:1	O	12.00	8.00			7.00				5.00	32		
					E	14.00	8.00			8.00				2.00	32	4.91	0.20-0.10
		Cherumodan x Japan Violet (2000 rad)	15:1	7:4:4:1	O	14.00	12.00			5.00				1.00	32		
					E	14.00	8.00			8.00				2.00	32	3.63	0.50-0.30
		Cherumodan x Japan Violet (5000 rad)	15:1	7:4:4:1	O	11.00	5.00			7.00				2.00	25		
					E	10.94	6.25			6.25				1.56	25	0.46	0.95-0.90
2.	Leaf Sheath	Cherumodan x Japan Violet (normal)	3:1	1:2:1	O	9.00	14.00							7.00	30		
					E	7.50	15.00							7.50	30	0.40	0.90-0.80
		Cherumodan x Japan Violet (1500 rad)	3:1	1:2:1	O	11.00	14.00							7.00	32		
					E	8.00	16.00							8.00	32	1.50	0.50-0.30
		Cherumodan x Japan Violet (2000 rad)	3:1	1:2:1	O	9.00	19.00							4.00	32		
					E	8.00	16.00							8.00	32	2.69	0.30-0.20
		Cherumodan x Japan Violet (5000 rad)	9:7		--	--	--							--	--		

* Expected breeding behaviour of F_3 families

TD - True dominant
TR - True recessive

Table value at 0.5% level for 1 d f. 3.84, 2 d f. 5.99, 3 d f. 7.81, 4 d f. 9.49, 5 d f. 11.07

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
3.	Leaf Blade (Lamina)	Cherumodan x Japan Violet (normal)	9:7	1:4:4:7	O	2.00	6.00		9.00					13.00	30		
					E	1.88	7.50		7.50					13.12	30	0.16	< 0.95
		Cherumodan x Japan Violet (1500 rad)	9:7	1:4:4:7	O	2.00	5.00		7.00					18.00	32		
					E	2.00	8.00		8.00					14.00	32	2.39	0.50-0.30
		Cherumodan x Japan Violet (2000 rad)	27:37	1:6:12:8:37	O	1.00	4.00		5.00				5.00	17.00	32		
					E	0.50	3.00		6.00				4.00	18.50	32	1.25	0.80-0.70
		Cherumodan x Japan Violet (5000 rad)	27:37	1:6:12:8:37	O	1.00	4.00		6.00				3.00	11.00	25		
					E	0.39	2.34		4.69				3.13	14.45	25	3.33	0.50-0.30
4.	Leaf Margin	Cherumodan x Japan Violet (normal)	3:1	1:2:1	O	6.00	13.00							11.00	30		
					E	7.50	15.00							7.50	30	2.20	0.50-0.30
		Cherumodan x Japan Violet (1500 rad)	9:7	1:4:4:7	O	5.00	10.00		6.00					11.00	32		
					E	2.00	8.00		8.00					14.00	32	6.14	0.20-0.10
		Cherumodan x Japan Violet (2000 rad)	9:7	1:4:4:7	O	3.00	12.00		7.00					10.00	32		
					E	2.00	8.00		8.00					14.00	32	3.77	0.30-0.20
		Cherumodan x Japan Violet (5000 rad)	9:7	1:4:4:7	O	2.00	8.00		7.00					7.00	25		
					E	1.56	6.25		6.25					10.94	25	2.84	0.50-0.30

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
5.	Leaf tip	Cherumodan x Japan Violet (normal)	3:1	1:2:1	O	9.00	14.00							7.00	30		
					E	7.50	15.00							7.50	30	0.40	0.90-0.80
		Cherumodan x Japan Violet (1500 rad)	9:7	1:4:4:7	O	4.00	11.00		9.00					8.00	32		
					E	2.00	8.00		8.00					14.00	32	5.82	0.20-0.10
		Cherumodan x Japan Violet (2000 rad)	3:1	1:2:1	O	9.00	18.00							5.00	32		
					E	8.00	16.00							8.00	32	1.50	0.50-0.30
		Cherumodan x Japan Violet (5000 rad)	9:7	1:4:4:7	O	4.00	8.00		7.00					6.00	25		
					E	1.56	6.25		6.25					10.94	25	6.63	0.10-0.05
6.	Junctura proper	Cherumodan x Japan Violet (normal)	9:7	1:4:4:7	O	1.00	5.00		11.00					13.00	30		
					E	1.87	7.50		7.50					13.13	30	2.87	0.50-0.30
		Cherumodan x Japan Violet (1500 rad)	27:37	1:6:12:8:37	O	1.00	6.00		5.00				3.00	17.00	32		
					E	0.50	3.00		6.00				4.00	18.50	32	4.04	0.50-0.30
		Cherumodan x Japan Violet (2000 rad)	27:37	1:6:12:8:37	O	1.00	4.00		2.00				7.00	18.00	32		
					E	0.52	3.09		6.18				4.13	19.08	32	5.59	0.30-0.20
		Cherumodan x Japan Violet (5000 rad)	27:37	1:6:12:8:37	O	1.00	5.00		4.00				3.00	12.00	25		
					E	0.39	2.34		4.69				3.13	14.45	25	4.50	0.50-0.30

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
7.	Ligule	Cherumodan x Japan Violet (normal)	9:7	1:4:4:7	O	1.00	8.00		9.00					12.00	30		
					E	1.87	7.50		7.50					13.13	30	0.84	0.90-0.80
		Cherumodan x Japan Violet (1500 rad)	9:7	1:4:4:7	O	4.00	7.00		6.00					15.00	32		
					E	2.00	8.00		8.00					14.00	32	2.70	0.50-0.30
		Cherumodan x Japan Violet (2000 rad)	9:7	1:4:4:7	O	5.00	10.00		8.00					9.00	32		
					E	2.06	8.25		8.25					14.44	32	6.62	0.10-0.05
		Cherumodan x Japan Violet (5000 rad)	27:37	1:6:12:8:37	O	1.00	5.00		6.00				4.00	9.00	25		
					E	0.39	2.34		4.69				3.13	14.45	25	6.64	0.20-0.10
8.	Auricle	Cherumodan x Japan Violet (normal)	9:7	1:4:4:7	O	1.00	6.00		8.00					15.00	30		
					E	1.87	7.50		7.50					13.13	30	1.00	0.90-0.80
		Cherumodan x Japan Violet (1500 rad)	27:37	1:6:12:8:37	O	2.00	5.00		7.00				2.00	16.00	32		
					E	0.50	3.00		6.00				4.00	18.50	32	7.34	0.20-0.10
		Cherumodan x Japan Violet (2000 rad)	27:37	1:6:12:8:37	O	1.00	2.00		6.00				6.00	17.00	32		
					E	0.52	3.09		6.18				4.13	19.08	32	1.91	0.80-0.70
		Cherumodan x Japan Violet (5000 rad)	27:37	1:6:12:8:37	O	1.00	4.00		4.00				5.00	11.00	25		
					E	0.39	2.34		4.69				3.13	14.45	25	4.17	0.50-0.30

1.	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
9.	Node	Cherumodan x Japan Violet (normal)	3:13	1:2:2:4:7	O	0.00	1.00	4.00				7.00		18.00	30		
					E	1.87	3.75	3.75				7.50		13.13	30	8.74	0.30-0.05
		Cherumodan x Japan Violet (1500 rad)	3:13	1:2:2:4:7	O	0.00	1.00	3.00				8.00		20.00	32		
					E	2.00	4.00	4.00				8.00		14.00	32	7.07	0.20-0.10
		Cherumodan x Japan Violet (2000 rad)	3:13	1:2:2:4:7	O	0.00	3.00	7.00				7.00		15.00	32		
					E	2.00	4.00	4.00				8.00		14.00	32	2.70	0.70-0.50
		Cherumodan x Japan Violet (5000 rad)	3:13	1:2:2:4:7	O	0.00	1.00	4.00				3.00		17.00	25		
					E	1.56	3.13	3.13				6.25		10.93	25	8.31	0.10-0.05
10.	Internode	Cherumodan x Japan Violet (normal)	3:1	1:2:1	O	5.00	16.00							9.00	30		
					E	7.50	15.00							7.50	30	1.20	0.70-0.50
		Cherumodan x Japan Violet (1500 rad)	3:1	1:2:1	O	8.00	17.00							7.00	32		
					E	8.00	16.00							8.00	32	0.19	0.95-0.90
		Cherumodan x Japan Violet (2000 rad)	3:1	1:2:1	O	6.00	16.00							10.00	32		
					E	8.00	16.00							8.00	32	1.00	0.70-0.50
		Cherumodan x Japan Violet (5000 rad)	9:7	1:4:4:7	O	2.00	7.00		9.00					7.00	25		
					E	1.56	6.25		6.25					10.94	25	2.84	0.50-0.30

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
11.	Stigma	Cherumodan x Japan Violet (normal)	3:1	1:2:1	O E	5.00 7.50	16.00 15.00							9.00 7.50	30 30	1.20	0.70-0.50
		Cherumodan x Japan Violet (1500 rad)	3:1	1:2:1	O E	6.00 8.00	17.00 16.00							9.00 8.00	32 32	0.69	0.80-0.70
		Cherumodan x Japan Violet (2000 rad)	3:1	1:2:1	O E	6.00 8.00	16.00 16.00							10.00 8.00	32 32	1.00	0.70-0.50
		Cherumodan x Japan Violet (5000 rad)	9:7	1:4:4:7	O E	2.00 1.56	7.00 6.25		9.00 6.25					7.00 10.94	25 25	2.84	0.50-0.30
12.	Apiculus	Cherumodan x Japan Violet (normal)	3:1	1:2:1	O E	5.00 7.50	16.00 15.00							9.00 7.50	30 30	1.20	0.70-0.50
		Cherumodan x Japan Violet (1500 rad)	3:1	1:2:1	O E	6.00 8.00	17.00 16.00							9.00 8.00	32 32	0.69	0.80-0.70
		Cherumodan x Japan Violet (2000 rad)	3:1	1:2:1	O E	6.00 8.25	16.00 16.50							10.00 8.25	32 32	1.00	0.70-0.50
		Cherumodan x Japan Violet (5000 rad)	9:7	1:4:4:7	O E	2.00 1.56	7.00 6.25		9.00 6.25					7.00 10.94	25 25	2.84	0.30-0.20

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
13.	Awn	Cherumodan x Japan Violet (normal)	3:1	1:2:1	O	11.00	12.00							7.00	30		
					E	7.50	15.00							7.50	30	2.27	0.50-0.30
		Cherumodan x Japan Violet (1500 rad)	3:1	1:2:1	O	8.00	17.00							7.00	32		
					E	8.00	16.00							8.00	32	0.19	0.95-0.90
		Cherumodan x Japan Violet (2000 rad)	3:1	1:2:1	O	7.00	20.00							5.00	32		
					E	8.00	16.00							8.00	32	2.25	0.50-0.30
		Cherumodan x Japan Violet (5000 rad)	3:1	1:2:1	O	8.00	12.00							5.00	25		
					E	6.25	12.50							6.25	25	0.76	0.70-0.50
14.	Tipsterility	Cherumodan x Japan Violet (normal)	3:1	1:2:1	O	8.00	13.00							9.00	30		
					E	7.50	15.00							7.50	30	0.60	0.80-0.79
		Cherumodan x Japan Violet (1500 rad)	3:1	1:2:1	O	7.00	16.00							9.00	32		
					E	8.00	16.00							8.00	32	0.25	0.90-0.80
		Cherumodan x Japan Violet (2000 rad)	3:1	1:2:1	O	7.00	18.00							7.00	32		
					E	8.00	16.00							8.00	32	0.50	0.80-0.70
		Cherumodan x Japan Violet (5000 rad)	3:1	1:2:1	O	7.00	14.00							4.00	25		
					E	6.25	12.50							6.25	25	1.08	0.70-0.50

F_3/M_3F_3 generation

Breeding behaviour of F_3/M_3F_3 families of the above crosses confirmed the monogenic F_2/M_2F_2 ratio. Out of 30 F_3 families of Cherumodan x Japan Violet (normal), 9 bred true for purple leaf sheath, 14 segregated for 3:1 and 7 bred true for green leaf sheath. Out of 32 M_3F_3 families of Cherumodan x Japan Violet - 1500 rad, 11 bred true for purple leaf sheath, 14 segregated for 3:1, and 7 bred true for green leaf sheath. Out of 32 M_3F_3 families of Cherumodan x Japan Violet - 2000 rad, 9 bred true for purple leaf sheath, 19 segregated for 3:1 and 4 bred true for green leaf sheath. The respective X^2 values are 0.40, 1.50, and 2.69 which are not significant for 2 d. f. at 5% level against the table value (Table 40-2), while the M_3F_3 of Cherumodan x Japan Violet - 5000 rad didn't give confirmation for the M_2F_2 ratio 9:7 (Table 40).

3. Leaf blade (Lamina)

Inheritance of anthocyanin pigmentation in leaf blade was studied in the same four crosses. Leaf blade in Cherumodan is green and in Japan Violet purple. F_1 of the crosses showed dominance of purple lamina (Table 39a-3 & 39b-3).

F_2/M_2 F_2 generation

F_2 population of Cherumodan x Japan Violet (normal) and, Cherumodan x Japan violet - 1500 rad segregated into 9 purple : 7 green with X^2 = 0.03, 1.74 respectively which are not significant for 1. d.f. at 5% level (Table 39-4). M_2F_2 of Cherumodan x Japan Violet - 2000 rad and Cherumodan x Japan Violet - 5000 rad segregated into 27 purple : 37 green with X^2 = 1.90 and 0.19 respectively which are not significant for 1.d.f. at 5% level (Table 39a-3). The former crosses showed two complementary genes and the latter three complementary genes for pigmentation in leaf blade.

F_3/M_3F_3 generation

Breeding behaviour of F_3/M_3F_3 families confirmed the F_2/M_2F_2 ratios. Out of 30 F_3 families of Cherumodan x Japan Violet (normal), 2 bred true for purple leaf blade, 6 segregated for 3:1, 9 for 9:7 and 13 bred true for green leaf blade and out of 32 M_3F_3 families of Cherumodan x Japan Violet - 1500 rad, 2 bred true for purple leaf blade, 5 segregated for 3:1, 7 for 9:7 and 18 bred true for green leaf blade, giving good fit to the expected ratio 1:4:4:7 with X^2 = 0.61 and 2.39 respectively, which are not significant for 3 d.f. at 5% level (Table 40-4). Out of 32 M_3F_3 families of Cherumodan x Japan Violet - 2000 rad, 1 bred true for purple leaf blade, 4 segregated for 3:1, 5 for 9:7, 5 for 27:37 and 17 bred true for green leaf blade

and out of 25 M_3 F_3 families of Cherumodan x Japan Violet - 5000 rad, 1 bred true for purple leaf blade, 4 segregated for 3:1, 6 for 9:7, 3 for 27:37 and 11 bred true for green leaf blade with X^2=1.25 and 3.33 respectively which are not significant for 4 d. f. at 5% level and are in conformity with the expected F_3/M_3F_3 ratio 1:6:12:8:37 (Table 40-3).

4. Leaf margin

Inheritance of pigmentation in leaf margin was studied in the same four crosses. Japan Violet showed purple leaf margin and Cherumodan green. F_1 of all crosses showed dominance of purple leaf margin (Table 39a-4).

F_2/M_2F_2 generation

F_2 population of Cherumodan x Japan Violet (normal) segregated into 3 purple : 1 green with X^2 = 2.37 which is not significant for 1 d.f.at 5% level against the table value. Cherumodan x Japan Violet crosses of - 1500 rad, 2000 rad, and 5000 rad treatments segregated into 9 purple : 7 green with X^2 = 3.34, 2.09 and 0.60 respectively which are not significant for 1 d. f. at 5% level (Table 39a-4 & 39b-4).

F_3/M_3F_3 generation

Breeding behaviour of F_3/M_3F_3 families confirmed F_2/M_2F_2 ratios of 3:1 and 9:7 (Table 40-4). Out of 30 F_3 families of Cherumodan x Japan Violet (normal) 6 bred true for purple leaf margin, 13 segregated for 3:1 and 11 bred true for green leaf margin in conformity with the expected ratio 1:2:1 (Table 40-4). Out of 32 M_3F_3 families of Cherumodan x Japan Violet -1500 rad, 5 bred true for purple leaf margin, 10 segregated for 3:1, 6 segregated for 9:7 and 11 bred true for green leaf margin. Out of 32 M_3F_3 families of Cherumodan x Japan Violet - 2000 rad, 3 bred true for purple leaf margin, 12 segregated for 3:1, 7 segregated for 9:7 and 10 bred true for green leaf margin and out of 25 M_3F_3 families of Cherumodan x Japan Violet - 5000 rad 2 bred true for purple leaf margin, 9 segregated for 3:1, 7 segregated for 9:7 and 7 bred true for green leaf margin . The respective X^2 values are 2.20, 6.14, 3.77 and 2.84 which are not significant for 2/3 d. f. at 5% level against the table value (Table 40-4).

5. Leaf tip

Inheritance of leaf tip pigmentation was also studied in the said crosses. Leaf tip is purple in Japan Violet and green in Cherumodan. F_1s showed dominance of purple leaf tip (Table 39a-5).

F_2/M_2 F_2 generation

F_2 of Cherumodan x Japan Violet (normal) and Cherumodan x Japan Violet- 2000 rad segregated for 3 purple : 1 green with X^2 = 0.93 and 1.12 respectively which are not significant for 1 d. f. at 5% level. M_2F_2 of Cherumodan x Japan Violet 1500 rad and 5000 rad, segregated into 9 purple : 7 green with X^2 = 3.33 and 0.19 respectively which are not significant for 1 d. f. at 5% level (Table 39a-5 & 39b-5).

F_3/M_3F_3 generation

Breeding behaviour of F_3/M_3F_3 families confirmed F_2/M_2F_2 ratios of 3:1 and 9:7 (Table 39-5). Out of 30 F_3 families of Cherumodan x Japan Violet (normal), 9 bred true for purple leaf tip, 14 segregated for 3:1 and 7 bred true for green leaf tip with X^2 = 0.40 and out of the 32 F_3 families of Cherumodan x Japan Violet 2000 rad, 9 bred true for purple leaf tip, 18 segregated into 3:1 and 5 bred true for green leaf tip with X^2 = 1.50 which are not significant for 2 d.f. at 5% level (Table 40-5). Out of 32 M_3 F_3 families of Cherumodan x Japan Violet - 1500 rad, 4 bred true for purple leaf tip, 11 segregated for 3:1, 9 segregated for 9:7 and 8 bred true for green leaf tip with X^2 = 5.82 and out of 25 M_3F_3 families of Cherumodan x Japan Violet - 5000 rad, 4 bred true for purple leaf tip, 8 segregated for 3:1, 7 for 9:7 and 6 bred true for green leaf tip with the X^2 = 6.63, which are not significant for 3 d.f. at 5% level (Table 40-5).

6. Junctura Proper

Inheritance of pigmentation in junctura proper was also studied in the said crosses. Junctura proper is purple in Japan Violet and green in Cherumodan. F_1 of all crosses showed dominance of purple junctura proper (Table 39a-6).

F_2/M_2 F_2 generation

F_2/M_2F_2 population of Cherumodan x Japan Violet (normal), segregated into 9 purple : 7 green with X^2 1.01 which is not significant for 1 d. f. at 5% level. M_2F_2 of Cherumodan x Japan Violet - 1500 rad, Cherumodan x Japan Violet 2000 rad, and Cherumodan x Japan Violet - 5000 rad segregated into 27 purple : 37 green with X^2 = 0.72, 0.05, and 1.10 respectively which are not significant for 1 d. f. at 5% level (Table 39a-6 & 39b-6).

F_3/M_3F_3 generation

Breeding behaviour of F_3/M_3F_3 families confirmed the F_2/M_2F_2 ratios of 9:7 and 27:37 in the respective crosses. Out of 30 F_3 families of Cherumodan x Japan Violet (normal) 1 bred true for purple junctura proper, 5 segregated for 3:1, 11 for 9:7 and 13 bred true for green junctura proper in conformity with the expected F_3 ratio

of 1:4:4:7 with X^2=2.87 which is not significant for 3 d. f. at 5% level (Table 40-6). Out of 32 M_3F_3 families of Cherumodan x Japan Violet - 1500 rad, 1 bred true for purple junctura proper, 6 segregated for 3:1, 5 for 9:7, 3 for 27:37 and 17 bred true for green junctura proper; out of 32 M_3 F_3 families of Cherumodan x Japan Violet - 2000 rad, 1 bred true for purple junctura proper, 4 segregated for 3:1, 2 for 9:7, 7 for 27:37 and 18 bred true for green junctura proper and out of 25 M_3 F_3 families of Cherumodan x Japan Violet - 5000 rad, 1 bred true for purple junctura proper, 5 segregated for 3:1, 4 for 9:7, 3 for 27:37 and 12 bred true for green junctura proper in conformity with the expected ratio 1:6:12:8:37 with X^2 =4.04, 5.59 and 4.50 respectively which are not significant for 4 d. f. at 5% level (Table 40-6).

7. Ligule

Inheritance of pigmentation in ligule was studied in the same crosses. Ligule is purple in Japan Violet and green in Cherumodan. F_1 of all crosses showed dominance of purple ligule. (Table 39a-7).

F_2/M_2F_2 generation

F_2/M_2F_2 populations of Cherumodan x Japan violet (normal), Cherumodan x Japan Violet - 1500 rad and Cherumodan x Japan violet - 2000 rad segregated into 9 purple : 7 green with X^2 = 1.81, 0.001 and

1.21, respectively which are not significant for 1 d. f. at 5% level (Table 39a-7). Cherumodan x Japan Violet - 5000 rad segregated into 27 purple : 37 green with X^2 = 1.10, which is not significant for 1 d. f. at 5% level (Table 39a-7 & 39b-7). The former crosses showed two complementary genes and the later three complementary genes for ligule pigmentation.

F_3/M_3F_3 generation

Breeding behaviour of F_3/M_3F_3 families confirmed F_2/M_2F_2 ratios, 9:7 and 27:37. Out of 30 F_3 families of Cherumodan x Japan Violet (normal), 1 bred true for purple ligule, 8 segregated for 3:1, 9 for 9:7 and 12 bred true for the green ligule; out of 32, M_3F_3 families of Cherumodan x Japan Violet - 1500 rad, 4 bred true for purple ligule, 7 segregated for 3:1, 6 for 9:7 and 15 bred true for green ligule and out of 32 M_3F_3 families of Cherumodan x Japan Violet - 2000 rad, 5 bred true for the purple ligule, 10 segregated for 3:1, 8 for 9:7 and 9 bred true for green ligule with X^2 = 0.84, 2.70 and 6.62 respectively which are not significant for 3 d. f. at 5% level (Table 40-7). Out of 25 M_3 F_3 families of Cherumodan x Japan Violet - 5000 rad, 1 bred true for purple ligule, 5 segregated for 3:1, 6 for 9:7, 4 for 27:37 and 9 bred true for green ligule with X^2 = 6.64 which is not significant for 4 d. f. at 5% level and is in conformity with the expected ratio 1:6:12:8:37 (Table 40-7).

8. Auricle

Inheritance of pigmentation in auricle was studied in the same crosses. Auricle is purple in Japan Violet and green in Cherumodan. F_1 of all crosses showed dominance of purple auricle (Table 39a-8).

F_2/M_2F_2 generation

F_2/M_2F_2 population of Cherumodan x Japan Violet (normal) segregated for 9 purple : 7 green with X^2 = 0.45 which is not significant for 1 d. f. at 5% level (Table 39-8). M_2F_2 of Cherumodan x Japan Violet - 1500 rad, Cherumodan x Japan Violet - 2000 rad, Cherumodan x Japan Violet - 5000 rad treatments segregated for 27 purple : 37 green with X^2 = 3.62, 0.05, 1.75 respectively which are not significant for 1 d. f. at 5% level (Table 39a-8 & 39b-8).

F_3/M_3F_3 generation

Breeding behavior of F_3/M_3F_3 families confirmed F_2/M_2F_2 ratios 9:7 and 27:37 (Table 40-8). Out of 30 F_3 families of Cherumodan x Japan Violet (normal), 1 bred true for purple auricle, 6 segregated for 3:1, 8 for 9:7 and 15 bred true for green auricle in conformity with the expected F_3 ratio 1:4:4:7 with X^2 = 1.00 which is not significant for 3 d. f. at 5% level. Out of 32 M_3F_3 families of

Cherumodan x Japan Violet - 1500 rad, 2 bred true for purple auricle, 5 segregated for 3:1, 7 segregated for 9:7, 2 segregated for 27:37 and 16 bred true for green auricle; out of 32 M_3 F_3 families of Cherumodan x Japan Violet - 2000 rad, 1 bred true for purple auricle, 2 segregated for 3:1, 6 for 9:7, 6 for 27:37 and 17 bred true for green auricle and out of 25 M_3F_3 families of Cherumodan x Japan Violet - 5000 rad, 1 bred true for purple auricle, 4 segregated for 3:1, 4 for 9:7, 5 for 27:37 and 11 bred true for green auricle, all in conformity with the expected F_3 ratio 1:6:12:8:37 with X^2= 7.34, 1.91, and 4.17 respectively which are not significant for 4 d. f. at 5% level (Table 40-8).

9. Node

Inheritance of nodal pigmentation was studied in the same crosses. Both Japan Violet and Cherumodan have green node. F_1 of all the crosses showed green node (Table 39a-9).

F_2/M_2F_2 generation

F_2/M_2F_2 populations of the crosses Cherumodan x Japan Violet (normal), Cherumodan x Japan Violet - 1500 rad, Cherumodan x Japan Violet - 2000 rad, Cherumodan x Japan Violet - 5000 rad segregated for 3 purple : 13 green with X^2 = 0.41, 2.29, 0.26, and 0.002

respectively which are not significant for 1 d. f. at 5% level (Table 39a-9 & 39b-9).

F_3/M_3F_3 generation

Breeding behaviour of F_3/M_3F_3 families confirmed F_2/M_2F_2 ratio 3:13. Out of 30 F_3 families of Cherumodan x Japan Violet (normal), 1 segregated for 3:1, 4 for 1:3, 7 for 3:13 and 18 bred true for green node. Out of 32 M_3F_3 families of Cherumodan x Japan Violet-1500 rad, 1 segregated for 3:1, 3 for 1:3 and 8 for 3:13 and 20 bred true for green node. Out of 32 M_3 F_3 families of Cherumodan x Japan Violet - 2000 rad, 3 segregated for 3:1, 7 for 1:3, 7 for 3:13 and 15 bred true for green node. Out of 25 families of Cherumodan x Japan Violet- 5000 rad, 1 segregated for 3:1, 4 for 1:3, 3 for 3:13 and 17 bred true for green node. These gave X^2 = 5.74, 7.07, 2.70 and 8.31 respectively, which are not significant for 4 d. f. at 5% level (Table 40-9) and the segregation was in conformity with the expected F_3/M_3F_3 ratio 1:2:2:4:7.

10. Internode

Inheritance of pigmentation in internode was studied in the same crosses. Internode is purple in Japan Violet and green in Cherumodan. F_1 of all crosses showed dominance of purple internode (Table 39a-10).

F_2/M_2F_2 generation

F_2/M_2F_2 population of Cherumodan x Japan Violet (normal), and of 1500 and 2000 rad segregated for 3 purple : 1 green with X^2 = 0.15, 2.80 and 0.63 respectively , which are not significant for 1 d.f. at 5% level. M_2 F_2 of Cherumodan x Japan Violet - 5000 rad segregated for 9 purple: 7 green with X^2 = 0.04 which is not significant for 1. d.f. at 5% level (Table 39a-10 & 39b-10).

F_3/M_3F_3 generation

Breeding behaviour of F_3/M_3F_3 families of the crosses confirmed the F_2/M_2F_2 ratios 3:1 and 9:7 (Table 40-10). Out of 30 F_3 families of Cherumodan x Japan Violet (normal), 5 bred true for purple internode; 14 segregated for 3:1, 10 bred true for green internode, out of 32 M_3F_3 families of Cherumodan x Japan Violet - 1500 rad, 8 bred true for purple internode, 17 segregated for 3:1 and 7 bred true for green internode, and out of 32 M_3F_3 families of Cherumodan x Japan Violet - 2000 rad, 6 bred true for purple internode, 16 segregated for 3:1 and 10 bred true for green internode in conformity with the expected F_3/M_3F_3 ratio 1:2:1 with X^2 = 1.20, 0.19 and 1.00 respectively, which are not significant for 2 d.f. at 5% level. Out of 25 M_3F_3 families of Cherumodan x Japan Violet - 5000 rad, 2 bred true for the purple internode, 7 segregated for 3:1, 9 for 9:7 and 7

bred true for green internode in conformity with the expected M_3F_3 ratio 1:4:4:7 with X^2 = 2.84 which is not significant for 3 d. f. at 5% level (Table 40-10).

11. Stigma

Inheritance of anthocyanin pigmentation in stigma was studied in the said crosses. Stigma colour in Japan Violet is purple and green in Cherumodan. F_1 of the crosses showed dominance of purple stigma (Table 39a-11).

F_2/M_2F_2 generation

F_2/M_2F_2 population of Cherumodan x Japan Violet (normal), Cherumodan x Japan Violet - 1500 rad, and Cherumodan x Japan Violet - 2000 rad segregated for 3 purple : 1 green with X^2 = 0.00, 0.15, and 0.63 respectively, which are not significant for 1 d. f. at 5% level (Table 39-11). M_2F_2 of Cherumodan x Japan Violet - 5000 rad segregated for 9 purple : 7 green with X^2 = 0.04 which is not significant for 1 d.f. at 5% level (Table 39a-11 & 39b-11).

F_3/M_3F_3 generation

Breeding behaviour of F_3/M_3F_3 families of the crosses confirmed F_2/M_2F_2 ratios 3:1 and 9:7 (Table 40-11). Out of 30 F_3 families of

Cherumodan x Japan Violet (normal) 5 bred true for purple stigma, 16 segregated for 3:1 and 9 bred true for green stigma; out of 32 M_3F_3 families of Cherumodan x Japan Violet - 1500, 6 bred true for purple stigma, 17 segregated for 3:1 and 9 bred true for green stigma and out of 32 M_3F_3 families of Cherumodan x Japan Violet - 2000 rad, 6 bred true for purple stigma, 16 segregated for 3:1 and 10 bred true for green stigma in conformity with the expected F_3/M_3F_3 ratio 1:2:1 with X^2 = 1.20, 0.69, 1.00 respectively which are not significant for 2 d. f. at 5% level (Table 40-11). Out of 25 M_3F_3 families of Cherumodan x Japan Violet - 5000 rad, 2 bred true for purple stigma, 7 segregated for 3:1, 9 for 9:7 and 7 bred true for green stigma in conformity with the expected M_3F_3 ratio 1:4:4:7 with X^2 = 2.84 which is not significant for 3 d. f. at 5% level (Table 40-11).

12. Apiculus

Inheritance of apiculus pigmentation was also studied in the same crosses. Apiculus is purple in Japan Violet and green in Cherumodan. F_1 of the crosses showed purple apiculus as dominant (Table 39a-12).

F_2/M_2F_2 generation

F_2 population of Cherumodan x Japan Violet (normal), Cherumodan

x Japan Violet-1500 rad and Cherumodan x Japan Violet-2000 rad segregated for 3 purple :1 green with X^2 = 0.15, 2.26 and 0.28 respectively which are not significant for 1 d. f. at 5% level. M_2F_2 of Cherumodan x Japan Violet - 5000 rad segregated for 9 purple: 7 green with X^2 = 0.04 which is not significant for 1 d. f. at 5% level (Table 39a-12 & 39b-12).

F_3/M_3F_3 generation

Breeding behavior of F_3/M_3F_3 families of the crosses confirmed the F_2/M_2F_2 ratios 3:1 and 9:7. Out of 30 F_3 families of Cherumodan x Japan Violet (normal), 5 bred true for purple apiculus, 16 segregated for 3:1 and 9 bred true for green apiculus; out of 32 M_3F_3 families of Cherumodan x Japan Violet - 1500 rad, 6 bred true for purple apiculus, 17 segregated for 3:1 and 9 bred true for green apiculus; out 32 M_3F_3 families of Cherumodan x Japan Violet - 2000 rad, 6 bred true for purple apiculus, 16 segregated for 3:1 and 10 bred true for green apiculus in conformity with the expected F_3/M_3F_3 ratio 1:2:1 with X^2 = 1.20, 0.69, and 1.00 respectively which are not significant for 2 d. f. at 5% level (Table 40-12). Out of 25 M_3F_3 families of Cherumodan x Japan Violet - 5000 rad, 2 bred true for purple apiculus, 7 segregated for 3:1, 9 for 9:7 and 7 bred true for green apiculus in conformity with the expected M_3F_3 ratio 1:4:4:7 with X^2 = 2.84 which is not significant for 3 d. f. at 5% level (Table 40-12).

13. Awning

Inheritance of awning was studied in the same four crosses. Cherumodan is awnless and Japan Violet awn-tipped. F_1s of all crosses showed awnless condition of spikelets as dominant.

F_2/M_2F_2 generation

F_2/M_2F_2 population of Cherumodan x Japan Violet (normal), Cherumodan x Japan violet - 1500 rad, Cherumodan x Japan Violet- 2000 rad and Cherumodan x Japan Violet- 5000 rad segregated for 3 awnless: 1 awned with X^2 = 0.04, 1.86, 1.12 and 0.32 which are not significant for 1 d. f. at 5% level (Table 39a-13 & 39b-13).

F_3/M_3F_3 generation

Breeding behavior of F_3/M_3F_3 families of the crosses confirmed the F_2/M_2F_2 ratio 3:1 (Table 40-13). Out of 30 F_3 families of Cherumodan x Japan Violet (normal), 11 bred true for awnless condition, 12 segregated for 3:1 and 7 bred true for recessive awned condition; out of 32 M_3F_3 families of Cherumodan x Japan Violet - 1500 rad, 8 bred true for awnless condition, 17 segregated for 3:1 and 7 bred true for awned condition; out 32 M_3F_3 families in Cherumodan x Japan Violet- 2000 rad, 7 bred true for awnless

condition, 20 segregated for 3:1 and 5 bred true for awned condition and out of 25 M_3F_3 families of Cherumodan x Japan Violet-5000 rad, 8 bred true for awnless condition, 12 segregated for 3:1 and 5 bred true for awned condition with X^2 = 2.27, 0.19, 2.25 and 0.76, respectively which are not significant for 2 d.f. at 5% level (Table 40-13), thereby confirmed the F_2/M_2F_2 ratio.

14. Tip-sterility

Inheritance of tip-sterility was studied in the same four crosses. Japan Violet has tip-sterility and Cherumodan normal panicles (panicles without tip-sterility) condition. F_1/M_1F_1 of the crosses showed absence of tip-sterility or normal panicles as dominant (Table 39a-14).

F_2/M_2F_2 generation

F_2/M_2F_2 population of Cherumodan x Japan Violet (normal), Cherumodan x Japan violet - 1500 rad, Cherumodan x Japan Violet - 2000 rad, Cherumodan x Japan Violet -5000 rad segregated for 3:1 for tip-sterility with X^2 = 0.04. 2.01, 0.00 and 2.84 respectively, which are not significant for 1 d. f. at 5% level (Table 39a-14 & 39b-14), thereby showing tip-sterility as recessive to normal.

F_3/M_3F_3 generation

Breeding behavior of F_3/M_3F_3 families confirmed F_2/M_2F_2 ratio. Out of 30 F_3 families studied in cherumodan x Japan Violet (normal), 8 bred true for normal panicles, 13 segregated for 3:1 and 9 bred true for tip-sterility. Out of 32 M_3F_3 families studied in Cherumodan x Japan Violet - 1500 rad, 7 bred true for normal panicle, 16 segregated for 3:1 and 9 bred true for tip-sterility. Out of 32 M_3F_3 families in Cherumodan x Japan Violet - 2000 rad, 7 plants bred true for normal panicle, 18 segregated for 3:1 and 7 bred true for tip-sterility and out of 25 families studied in Cherumodan x Japan Violet - 5000 rad, 7 bred true for normal panicle, 14 segregated for 3:1 and 4 bred true for tip-sterility in conformity with the expected F_3/M_3F_3 ratio 1:2:1 with X^2 = 0.60, 0.25, 0.5 and 1.08 respectively, which are not significant for 1 d.f. at 5% level (Table 40-14).

D. MUTANTS ELICITED FROM CERTAIN X-RAYED POLLEN TREATED CROSSES OF CHERUMODAN x JAPAN VIOLET

Besides the genomutations described elsewhere as elicited from the X-rayed pollen treated crosses of Cherumodan x Japan Violet (detailed under Chapter III), a few morphomutants were isolated from F_3M_3/M_4M_4 levels of 2000 rad and 5000 rad treatments. Observations on the morphological characters of these morphomutants are furnished below.

1. Recessive clustered spikelet

M_1F_1 of the cross Cherumodan x Japan Violet - 2000 rad irradiated pollen, showed normal panicles, but showed 70.22% spikelet sterility. M_2F_2 population of 78 plants, obtained from 126 F_1 seeds sown, also appeared to be normal. However, in the next generation, out of 32 M_3F_3 families studied, one out of 63 plants of one of the families showed terminal twin clustering of spikelets (Fig. 18a). No further segregant of the type could be obtained in M_4F_4 families studied later. However, the progeny of the said plant which showed terminal twin spikelets segregated for 1 normal : 2 twin clustered : 1 clustered (Fig 18b-d). The later clustered type proved to be true breeding recessive and the twin clustered again segregated into the same ratio. This could be thus identified as the recessive clustering in rice , its morophometric characters are presented in the Appendix I.

2. Recessive long awned green mutant

M_1F_1 of the cross Cherumodan x Japan Violet-5000 rad X-rayed pollen appeared like a normal F_1 but showed 85.85% spikelet sterility. Out of 38 M_2F_2 plants studied, 2 plants appeared completely green and awnless (Fig. 19a), of which one was highly sterile (95.89%) and the other showed low sterility 18.87%. The

Fig. 18. Terminal twin and clustered mutant

a. Panicles showing terminal twin condition

b. Clustered mutant plant

c. Clustered panicles

d. Closeup view of the clustered panicles

Fig 18

Fig. 19. Complete green-awned and awnless mutants

a. Complete green-awnless mutant

b. Complete green-awned mutant

Fig 19

former one could not be followed up further, while the later was studied with its M_3F_3 with 49 plants, which showed a segregation of 39 green awnless : 10 green long awned (Fig. 19b) giving a monogenic ratio 3:1 with X^2 = 0.55. The awnless plants further segregated into 1:2:1 ratio as expected.

E. INTERRELATIONSHIP OF GENES GOVERNING DIFFERENT MORPHOLOGICAL CHARACTERS

Interrelationship of genes governing 14 morphological characters was studied in 4 crosses : Cherumodan x Japan Violet - normal (1), Cherumodan x Japan Violet wherein pollen grains irradiated with 1500 (2), 2000 (3), and 5000 (4)rad x-rays were used for pollination. Ninety one possible character combinations of 14 morphological characters in F_2 / M_2F_2 of these crosses (Table 39-a)were studied for the interrelationships of genes in relation to (1) independent assortment of genes (2) linkage relationships of genes and (3) pleiotropic association of morphological characters (Tables 41-45).

Joint segregation studied for 46 combinations of 14 morphological characters in these crosses showed X^2 values lower than the table value 7.82 for 3 d.f at 5% level, indicating independent assortment of genes involved in the control of the morphological characters concerned (Table 41). Joint segregation for 27

Table - 41 Interrelationship of morphological characters studied: Independent assortment of genes in crosses 1-4

Sl. No.	Charector Combinations/F2 ratio/s			Cross/es	Population O/E	F_2 Frequency AB	Ab	aB	ab	Total	X^2	P
1	2			3	4	5	6	7	8	9	10	11
1.	Px (15:1)	Vs.	Psh (9:7)	Cherumodan x Japan Violet (5000 rad)	O	23.00	13.00	0.00	2.00	38		
					E on independent	20.04	15.58	1.34	1.04	38	3.09	0.50-0.30
2.	Px (15:1)	Vs.	Pl (27:37)	Cherumodan x Japan Violet (2000 rad)	O	38.00	32.00	1.00	5.00	76		
					E on independent	30.06	41.19	2.00	2.75	76	6.49	0.10-0.05
	(15:1)		(27:37)	Cherumodan x Japan Violet (5000 rad)	O	14.00	22.00	0.00	2.00	38		
					E on independent	15.03	20.60	1.00	1.37	38	1.45	0.80-0.70
3.	Px (15:1)	Vs.	Plm (9:7)	Cherumodan x Japan Violet (5000 rad)	O	19.00	17.00	0.00	2.00	38		
					E on independent	20.04	15.58	1.34	1.04	38	2.41	0.50-0.30
4.	Px (15:1)	Vs.	Pla (9:7)	Cherumodan x Japan Violet (5000 rad)	O	20.00	16.00	0.00	2.00	38		
					E on independent	20.04	15.58	1.34	1.04	38	2.24	0.70-0.50
5.	Px (15:1)	Vs.	Pjp (27:37)	Cherumodan x Japan Violet (1500 rad)	O	80	91.00	1.00	9.00	181		
					E on independent	71.59	98.10	4.77	6.54	181	5.41	0.20-0.10
	(15:1)		(27:37)	Cherumodan x Japan Violet (2000 rad)	O	33.00	37.00	1.00	5.00	76		
					E on independent	30.06	41.19	2.00	2.75	76	3.05	0.50-0.30

1		2		3	4	5	6	7	8	9	10	11
	(15:1)		(27:37)	Cherumodan x Japan Violet (5000 rad)	O E on independent	15.00 15.03	21.00 20.60	0.00 1.00	2.00 1.37	38 38	1.29	0.80-0.70
6.	Px (15:1)	Vs.	Plg (9:7)	Cherumodan x Japan Violet (2000 rad)	O E on independent	39.00 40.08	31.00 31.17	1.00 2.67	5.00 2.08	76 76	5.17	0.20-0.10
	(15:1)		(27:37)	Cherumodan x Japan Violet (5000 rad)	O E on independent	15.00 15.03	21.00 20.60	0.00 1.00	2.00 1.37	38 38	1.29	0.80-0.70
7.	Px (15:1)	Vs.	Pau (27:37)	Cherumodan x Japan Violet (1500 rad)	O E on independent	84.00 71.59	87.00 98.10	5.00 4.77	5.00 6.54	181 181	5.39	0.20-0.10
	(15:1)		(27:37)	Cherumodan x Japan Violet (2000 rad)	O E on independent	32.00 30.06	38.00 41.19	1.00 2.00	5.00 2.75	76 76	2.71	0.50-0.30
	(15:1)		(27:37)	Cherumodan x Japan Violet (5000 rad)	O E on independent	12.00 15.03	24.00 20.60	0.00 1.00	2.00 1.37	38 38	2.46	0.50-0.30
8.	Px (15:1)	Vs.	Pn (3:13)	Cherumodan x Japan Violet (Normal)	O E on independent	30.00 25.31	100.00 109.69	2.00 1.69	12.00 7.31	144 144	4.79	0.20-0.10
	(15:1)		(3:13)	Cherumodan x Japan Violet (1500 rad)	O E on independent	24.00 31.82	147.00 137.87	2.00 2.12	8.00 9.19	181 181	2.68	0.70-0.50
	(15:1)		(3:13)	Cherumodan x Japan Violet (2000 rad)	O E on independent	16.00 13.36	58.00 57.89	1.00 0.89	5.00 3.86	76 76	0.70	0.90-0.80
	(15:1)		(3:13)	Cherumodan x Japan Violet (5000 rad)	O E on independent	7.00 6.68	29.00 28.95	0.00 0.45	2.00 1.92	38 38	0.47	0.95-0.90

1		2		3	4	5	6	7	8	9	10	11
9.	Px (15:1)	Vs.	Pin (9:7)	Cherumodan x Japan Violet (5000 rad)	O E on independent	22.00 20.04	14.00 15.58	0.00 1.34	2.00 1.04	38 38	2.58	0.50-0.30
10.	Px (15:1)	Vs.	Ps (9:7)	Cherumodan x Japan Violet (5000 rad)	O E on independent	22.00 20.04	14.00 15.58	0.00 1.34	2.00 1.04	38 38	2.53	0.50-0.30
11.	Px (15:1)	Vs.	Pa (9:7)	Cherumodan x Japan Violet (5000 rad)	O E on independent	22.00 20.04	14.00 15.58	0.00 1.34	2.00 1.04	38 38	2.57	0.95-0.90
12.	Px (15:1)	Vs.	an (3:1)	Cherumodan x Japan Violet (Nomal)	O E on independent	98.00 101.25	34.00 33.75	9.00 6.75	3.00 2.25	144 144	1.10	0.80-0.70
	(15:1)		(3:1)	Cherumodan x Japan Violet (5000 rad)	O E on independent	122.00 127.27	49.00 42.42	6.00 8.48	4.00 2.82	181 181	2.45	0.70-0.50
	(15:1)		(3:1)	Cherumodan x Japan Violet (2000 rad)	O E on independent	57.00 53.44	13.00 17.81	4.00 3.56	2.00 1.19	76 76	2.14	0.70-0.50
	(15:1)		(3:1)	Cherumodan x Japan Violet (5000 rad)	O E on independent	26.00 26.72	10.00 8.91	1.00 0.59	1.00 1.78	38 38	0.78	0.90-0.80
13.	Px (15:1)	Vs.	tst (3:1)	Cherumodan x Japan Violet (Normal)	O E on independent	98.00 101.25	34.00 33.75	9.00 6.75	3.00 2.25	144 144	1.11	0.80-0.70
	(15:1)		(3.1)	Cherumodan x Japan Violet (1500 rad)	O E on independent	136.00 127.27	35.00 42.42	8.00 8.48	3.00 2.83	181 181	1.93	0.70-0.50
	(15:1)		(3:1)	Cherumodan x Japan Violet (2000 rad)	O E on independent	53.00 53.44	17.00 17.81	4.00 3.56	2.00 1.19	76 76	0.65	0.90-0.80

1		2	3	4	5	6	7	8	9	10	11
			Cherumodan x	O	31.00	5.00	2.00	0.00	38		
	(15:1)	(3:1)	Japan Violet	E on	26.72	8.91	1.78	0.59	38	3.02	0.50-0.30
			(Normal)	independent							
14.	Px Vs.	Pn	Cherumodan x	O	14.00	41.00	2.00	19.00	76		
	(3:1)	(3:13)	Japan Violet	E on	10.69	46.31	3.56	15.44	76	3.14	0.50-0.30
			(2000 rad)	independent							
			Cherumodan x	O	7.00	16.00	0.00	15.00	38		
	(9:7)	(3:13)	Japan Violet	E on	4.01	17.37	3.12	13.51	38	5.62	0.20-0.10
			(5000 rad)	independent							
15.	Psh Vs.	an	Cherumodan x	O	83.00	28.00	24.00	9.00	144		
	(3:1)	(3:1)	Japan Violet	E on	81.00	27.00	27.00	9.00	144	0.41	0.95-0.90
			(Normal)	independent							
			Cherumodan x	O	91.00	35.00	37.00	18.00	181		
	(3:1)	(3:1)	Japan Violet	E on	101.81	33.94	33.94	11.31	181	5.41	0.20-0.10
			(1500 rad)	independent							
			Cherumodan x	O	44.00	11.00	17.00	4.00	76		
	(3:1)	(3:1)	Japan Violet	E on	42.75	14.25	14.25	4.75	76	1.43	0.70-0.50
			2000 rad)	independent							
			Cherumodan x	O	15.00	8.00	12.00	3.00	38		
	(9:7)	(3:1)	Japan Violet	E on	16.03	5.34	12.47	4.16	38	1.73	0.70-0.05
			(5000 rad)	independent							
16.	Psh Vs.	tst	Cherumodan x	O	83.00	28.00	24.00	9.00	144		
	(3:1)	(3:1)	Japan Violet	E on	81.00	27.00	27.00	9.00	144	0.41	0.95-0.90
			(Normal)	independent							
			Cherumodan x	O	102.00	24.00	42.00	13.00	181		
	(3:1)	(3:1)	Japan Violet	E on	101.81	33.94	33.94	11.31	181	5.08	0.20-0.10
			(5000 rad)	independent							
			Cherumodan x	O	41.00	14.00	16.00	5.00	76		
	(3:1)	(3:1)	Japan Violet	E on	42.75	14.25	14.25	4.75	76	0.30	0.98-0.95
			(2000 rad)	independent							

1	2	3	4	5	6	7	8	9	10	11
(9:1)	(3:1)	Cherumodan x Japan Violet (5000 rad)	O E on independent	20.00 16.03	3.00 5.34	13.00 12.47	2.00 4.16	38 38	3.15	0.50-0.30
17. Pl (27:37) Vs.	Pn (3:13)	Cherumodan x Japan Violet (5000 rad)	O E on independent	5.00 3.01	9.00 13.03	2.00 4.12	22.00 17.85	38 38	4.62	0.30-0.20
18. Pl (9:7) Vs.	an (3:1)	Cherumodan x Japan Violet (Normal)	O E on independent	61.00 60.75	21.00 20.25	46.00 47.25	16.00 15.75	144 144	0.07	0.99
(9:7)	(3:1)	Cherumodan x Japan Violet (1500 rad)	O E on independent	63.00 76.36	31.00 25.45	65.00 59.39	22.00 19.80	181 181	4.20	0.30-0.20
(27:37)	(3:1)	Cherumodan x Japan Violet (2000 rad)	O E on independent	32.00 24.05	6.00 8.02	29.00 32.95	9.00 10.98	76 76	3.97	0.35-0.20
(27:37)	(3:1)	Cherumodan x Japan Violet (5000 rad)	O E on independent	8.00 12.02	6.00 4.01	19.00 16.48	5.00 5.49	38 38	2.76	0.50-0.30
19. Pl (9:7) Vs.	tst (3:1)	Cherumodan x Japan Violet (Normal)	O E on independent	62.00 60.75	20.00 20.25	45.00 47.25	17.00 15.75	144 144	0.24	0.99-0.95
(9:7)	(3:1)	Cherumodan x Japan Violet (1500 rad)	O E on independent	76.00 76.36	17.00 25.45	68.00 59.39	20.00 19.80	181 181	4.06	0.50-0.30
(27:37)	(3:1)	Cherumodan x Japan Violet (2000 rad)	O E on independent	29.00 24.05	9.00 8.02	28.00 32.95	10.00 10.98	76 76	1.97	0.70-0.50
(3:1)	(3:1)	Cherumodan x Japan Violet (5000 rad)	O E on independent	11.00 12.02	3.00 4.01	22.00 16.48	2.00 5.49	38 38	4.41	0.30-0.20

1		2	3	4	5	6	7	8	9	10	11
20.	Plm (9:7) Vs.	Pn (3:13)	Cherumodan x Japan Violet (5000 rad)	O E on independent	6.00 4.00	13.00 17.37	1.00 3.12	18.00 13.51	38 38	4.03	0.20-0.10
21.	Plm (3:1) Vs.	an (3:1)	Cherumodan x Japan Violet (Normal)	O E on independent	74.00 81.00	26.00 27.00	33.00 27.00	11.00 9.00	144 144	2.42	0.50-0.30
	(9:7)	(3:1)	Cherumodan x Japan Violet (1500 rad)	O E on independent	80.00 76.36	34.00 25.45	48.00 59.39	19.00 19.80	181 181	5.26	0.20-0.10
	(9:7)	(3:1)	Cherumodan x Japan Violet (2000 rad)	O E on independent	40.00 32.06	9.00 10.69	21.00 24.94	6.00 8.31	76 76	3.50	0.50-0.30
	(9:7)	(3:1)	Cherumodan x Japan Violet (5000 rad)	O E on independent	13.00 16.03	6.00 5.34	14.00 12.47	5.00 4.16	38 38	1.01	0.80-0.70
22.	Plm (3:1) Vs.	tst (3:1)	Cherumodan x Japan Violet (Normal)	O E on independent	80.00 81.00	20.00 27.00	27.00 27.00	17.00 9.00	144 144	7.12	0.10-0.05
	(9:7)	(3:1)	Cherumodan x Japan Violet (1500 rad)	O E on independent	93.00 76.36	21.00 25.45	51.00 59.39	16.00 19.80	181 181	6.32	0.10-0.05
	(9:7)	(3:1)	Cherumodan x Japan Violet (2000 rad)	O E on independent	35.00 32.06	14.00 10.69	22.00 24.94	5.00 8.31	76 76	2.56	0.50-0.30
	(9:7)	(3:1)	Cherumodan x Japan Violet (5000 rad)	O E on independent	16.00 16.03	3.00 5.34	17.00 12.47	2.00 4.16	38 38	3.79	0.30-0.20
23.	Plm (9:7) Vs.	Pn (3:13)	Cherumodan x Japan Violet (5000 rad)	O E on independent	6.00 4.00	14.00 17.37	1.00 3.12	17.00 13.51	38 38	3.99	0.30-0.20

1		2	3	4	5	6	7	8	9	10	11
24.	Pla	Vs.	an	Cherumodan x	O	76.00	27.00	31.00	10.00	144		
	(3:1)		(3:1)	Japan Violet	E on	81.00	27.00	27.00	9.00	144	1.01	0.90-0.80
				(Normal)	independent							
				Cherumodan x	O	80.00	34.00	48.00	19.00	181		
	(9:7)		(3:1)	Japan Violet	E on	76.36	25.45	59.39	19.80	181	4.90	0.20-0.10
				(1500 rad)	independent							
				Cherumodan x	O	44.00	9.00	17.00	6.00	76		
	(3:1)		(3:1)	Japan Violet	E on	42.75	14.25	14.25	4.75	76	2.83	0.50-0.30
				(2000 rad)	independent							
				Cherumodan x	O	14.00	6.00	13.00	5.00	38		
	(9:7)		(3:1)	Japan Violet	E on	16.03	5.34	12.47	4.16	38	0.53	0.95-0.90
				(5000 rad)	independent							
25.	Pla	Vs.	tst	Cherumodan x	O	82.00	22.00	25.00	15.00	144		
	(3:1)		(3:1)	Japan Violet	E on	81.00	27.00	27.00	9.00	144	6.93	0.20-0.10
				(Normal)	independent							
				Cherumodan x	O	93.00	21.00	51.00	16.00	181		
	(9:7)		(3:1)	Japan Violet	E on	76.36	25.45	59.39	19.80	181	6.32	0.10-0.05
				(1500 rad)	independent							
				Cherumodan x	O	39.00	14.00	18.00	5.00	76		
	(3:1)		(3:1)	Japan Violet	E on	42.75	14.25	14.25	4.75	76	1.33	0.80-0.70
				(2000 rad)	independent							
				Cherumodan x	O	17.00	3.00	16.00	2.00	38		
	(9:7)		(3:1)	Japan Violet	E on	16.03	5.34	12.47	4.16	38	3.21	0.50-0.30
				(5000 rad)	independent							
26.	Pjp	Vs.	Pn	Cherumodan x	O	5.00	10.00	2.00	21.00	38		
	(29:37)		(3:13)	Japan Violet	E on	3.01	13.03	4.12	17.84	38	3.67	0.30-0.20
				(5000 rad)	independent							
27.	Pjp	Vs.	an	Cherumodan x	O	59.00	16.00	48.00	21.00	144		
	(9:7)		(3:13)	Japan Violet	E on	60.75	20.25	47.25	15.75	144	2.70	0.50-0.30
				(Normal)	independent							

1	2	3	4	5	6	7	8	9	10	11
(27:37)	(3:1)	Cherumodan x Japan Violet (1500 rad)	O E on independent	54.00 57.27	26.00 19.09	74.00 78.48	27.00 26.16	181 181	2.77	0.20-0.10
(27:37)	(3:1)	Cherumodan x Japan Violet (2000 rad)	O E on independent	26.00 24.05	7.00 8.05	35.00 32.95	8.00 10.98	76 76	1.22	0.80-0.70
(27:37)	(3:1)	Cherumodan x Japan Violet (5000 rad)	O E on independent	11.00 12.02	4.00 4.01	16.00 16.48	7.00 5.49	38 38	0.52	0.95-0.90
28. Pjp (9:7) Vs. tst	(3:1)	Cherumodan x Japan Violet (Normal)	O E on independent	58.00 60.75	17.00 20.25	49.00 47.25	20.00 15.75	144 144	1.86	0.70-0.50
(27:37)	(3:1)	Cherumodan x Japan Violet (1500 rad)	O E on independent	64.00 57.27	16.00 19.09	80.00 78.48	21.00 26.16	181 181	2.34	0.70-0.50
(27:37)	(3:1)	Cherumodan x Japan Violet (2000 rad)	O E on independent	28.00 24.05	5.00 8.02	29.00 32.95	14.00 10.98	76 76	3.09	0.50-0.30
(27:37)	(3:1)	Cherumodan x Japan Violet (5000 rad)	O E on independent	3.00 12.02	12.00 4.01	2.00 16.48	21.00 5.49	38 38	3.71	0.30-0.20
29. Plg (9:7) Vs. pn	(3:13)	Cherumodan x Japan Violet (2000 rad)	O E on independent	12.00 8.02	27.00 34.73	4.00 6.23	33.00 27.02	76 76	5.82	0.20-0.10
(27:37)	(3:13)	Cherumodan x Japan Violet (5000 rad)	O E on independent	6.00 3.01	9.00 13.03	1.00 4.12	22.00 17.84	38 38	7.54	0.10-0.05
30. Plg (9:7) Vs. an	(3:1)	Cherumodan x Japan Violet (Normal)	O E on independent	66.00 60.75	23.00 20.25	41.00 47.25	14.00 15.75	144 144	1.84	0.70-0.50

1	2	3	4	5	6	7	8	9	10	11
		Cherumodan x	O	69.00	33.00	59.00	20.00	181		
(9:7)	(3:1)	Japan Violet	E on	76.36	25.45	59.39	19.80	181	2.95	0.50-0.30
		(1500 rad)	independent							
		Cherumodan x	O	33.00	6.00	28.00	9.00	76		
(9:7)	(3:1)	Japan Violet	E on	32.06	10.69	34.94	8.31	76	2.52	0.50-0.30
		(2000 rad)	independent							
		Cherumodan x	O	9.00	6.00	18.00	5.00	38		
(27:37)	(3:1)	Japan Violet	E on	12.02	4.01	16.48	5.49	38	1.93	0.70-0.50
		(5000 rad)	independent							
31. Plg Vs. tst		Cherumodan x	O	70.00	19.00	37.00	18.00	144		
(9:7)	(3:1)	Japan Violet	E on	60.75	20.25	47.25	15.75	144	4.03	0.30-0.20
		(Normal)	independent							
		Cherumodan x	O	83.00	19.00	61.00	18.00	181		
(9:7)	(3:1)	Japan Violet	E on	76.36	25.45	59.39	19.80	181	2.42	0.50-0.30
		(1500 rad)	independent							
		Cherumodan x	O	29.00	10.00	28.00	9.00	76		
(9:7)	(3:1)	Japan Violet	E on	32.06	10.69	24.94	8.31	76	0.77	0.90-0.80
		(2000 rad)	independent							
		Cherumodan x	O	12.00	3.00	21.00	2.00	38		
(27:37)	(3:1)	Japan Violet	E on	12.02	4.01	16.48	5.49	38	3.72	0.30-0.20
		(5000 rad)	independent							
32. Pau Vs. pn		Cherumodan x	O	5.00	7.00	2.00	24.00	38		
(27:37)	(3:13)	Japan Violet	E on	3.01	13.03	4.12	17.85	38	7.32	0.10-0.05
		(5000 rad)	independent							
33. Pau Vs. an		Cherumodan x	O	63.00	14.00	44.00	23.00	144		
(9:7)	(3:1)	Japan Violet	E on	60.75	20.25	47.25	15.75	144	5.56	0.20-0.10
		(Normal)	independent							
		Cherumodan x	O	59.00	30.00	70.00	22.00	181		
(27:37)	(3:1)	Japan Violet	E on	57.27	19.09	78.48	26.16	181	7.26	0.10-0.05
		(1500 rad)	independent							

1	2	3	4	5	6	7	8	9	10	11
(27:37)	(3:1)	Cherumodan x Japan Violet (2000 rad)	O E on independent	28.00 24.05	5.00 8.02	33.00 32.95	10.00 10.98	76 76	1.87	0.70-0.50
(27:37)	(3:1)	Cherumodan x Japan Violet (5000 rad)	O E on independent	8.00 12.02	4.00 4.01	19.00 16.48	7.00 5.49	38 38	2.15	0.70-0.50
34. Pau (9:7) Vs. tst	(3:1)	Cherumodan x Japan Violet (Normal)	O E on independent	59.00 60.75	18.00 20.25	59.00 47.25	8.00 15.75	144 144	7.04	0.10-0.50
(27:37)	(3:1)	Cherumodan x Japan Violet (1500 rad)	O E on independent	71.00 57.27	18.00 19.09	73.00 78.48	19.00 26.16	181 181	5.70	0.20-0.10
(27:37)	(3:1)	Cherumodan x Japan Violet (2000 rad)	O E on independent	27.00 24.05	6.00 8.02	30.00 32.95	13.00 10.98	76 76	1.50	0.70-0.50
(27:37)	(3:1)	Cherumodan x Japan Violet (5000 rad)	O E on independent	9.00 12.02	3.00 4.01	24.00 16.48	2.00 5.49	38 38	6.67	0.10-0.05
35. Pn (3:13) Vs. pin	(3:1)	Cherumodan x Japan Violet (2000 rad)	O E on independent	15.00 10.69	1.00 3.56	39.00 46.31	21.00 15.44	76 76	6.74	0.10-0.05
(3:13)	(3:1)	Cherumodan x Japan Violet (5000 rad)	O E on independent	7.00 4.00	0.00 3.12	15.00 17.37	16.00 13.51	38 38	6.15	0.20-0.10
36. Pn (3:13) Vs. ps	(3:1)	Cherumodan x Japan Violet (1500 rad)	O E on independent	23.00 25.45	2.00 8.48	115.00 110.30	41.00 36.77	181 181	5.87	0.20-0.10
(3:13)	(9:7)	Cherumodan x Japan Violet (5000 rad)	O E on independent	7.00 4.00	0.00 3.12	15.00 17.37	16.00 13.51	38 38	6.15	0.20-0.10

1			2	3	4	5	6	7	8	9	10	11
37.	Pn (3:13)	Vs.	pa (9:7)	Cherumodan x Japan Violet (5000 rad)	O E on independent	7.00 4.00	0.00 3.12	15.00 17.37	16.00 13.51	38 38	6.15	0.20-0.10
38.	Pn (3:13)	Vs.	an (3:1)	Cherumodan x Japan Violet (Normal)	O E on independent	21.00 20.25	10.00 6.75	86.00 87.75	27.00 29.25	144 144	0.82	0.70-0.50
	(3:13)		(3:1)	Cherumodan x Japan Violet (1500 rad)	O E on independent	20.00 25.45	5.00 8.48	108.00 110.30	48.00 36.77	181 181	6.07	0.20-0.10
	(3:13)		(3:1)	Cherumodan x Japan Violet (2000 rad)	O E on independent	15.00 10.69	1.00 1.78	46.00 46.31	14.00 15.44	76 76	3.71	0.30-0.20
	(3:13)		(3:1)	Cherumodan x Japan Violet (5000 rad)	O E on independent	5.00 5.34	2.00 1.78	22.00 23.16	9.00 7.72	38 38	0.32	0.98-0.95
39.	Pn (3:13)	Vs.	tst (3:1)	Cherumodan x Japan Violet (Normal)	O E on independent	24.00 20.25	6.00 6.75	83.00 87.75	31.00 29.25	144 144	1.14	0.70-0.50
	(3:13)		(3:1)	Cherumodan x Japan Violet (1500 rad)	O E on independent	20.00 25.45	5.00 8.48	123.00 110.30	33.00 36.77	181 181	4.44	0.30-0.20
	(3:13)		(3:1)	Cherumodan x Japan Violet (2000 rad)	O E on independent	16.00 10.69	0.00 3.56	41.00 46.31	19.00 15.44	76 76	7.63	0.10-0.05
	(3:13)		(3:1)	Cherumodan x Japan Violet (5000 rad)	O E on independent	5.00 5.34	2.00 1.785	28.00 23.16	3.00 7.72	38 38	3.95	0.30-0.20
40.	Pin (3:1)	Vs.	an (3:1)	Cherumodan x Japan Violet (Normal)	O E on independent	78.00 81.00	28.00 27.00	29.00 27.00	9.00 9.00	144 144	0.29	0.98-0.95

1	2	3	4	5	6	7	8	9	10	11
(3:1)	(3:1)	Cherumodan x Japan Violet (2000 rad)	O E on independent	45.00 42.75	9.00 14.25	16.00 14.25	6.00 4.75	76 76	2.60	0.50-0.30
(9:7)	(3:1)	Cherumodan x Japan Violet (5000 rad)	O E on independent	16.00 16.03	6.00 5.34	11.00 12.47	5.00 4.16	38 38	0.42	0.95-0.90
41. Pin Vs. tst (3:1)	(3:1)	Cherumodan x Japan Violet (Normal)	O E on independent	84.00 81.00	22.00 27.00	23.00 27.00	15.00 9.00	144 144	5.63	0.20-0.10
(3:1)	(3:1)	Cherumodan x Japan Violet (2000 rad)	O E on independent	41.00 42.75	13.00 14.25	16.00 14.25	6.00 4.75	76 76	0.73	0.90-0.80
(9:7)	(3:1)	Cherumodan x Japan Violet (5000 rad)	O E on independent	19.00 16.03	3.00 5.34	14.00 12.47	2.00 4.16	38 38	2.88	0.50-0.30
42. Ps Vs. an (3:1)	(3:1)	Cherumodan x Japan Violet (Normal)	O E on independent	77.00 81.00	31.00 27.00	30.00 27.00	6.00 9.00	144 144	2.12	0.70-0.50
(3:1)	(3:1)	Cherumodan x Japan Violet (1500 rad)	O E on independent	99.00 101.81	39.00 33.94	29.00 33.94	14.00 11.31	181 181	2.19	0.70-0.50
(3:1)	(3:1)	Cherumodan x Japan Violet (2000 rad)	O E on independent	44.00 42.75	10.00 14.25	17.00 14.25	5.00 4.75	76 76	1.84	0.70-0.50
(3:1)	(3:1)	Cherumodan x Japan Violet (5000 rad)	O E on independent	16.00 16.03	6.00 5.34	11.00 12.47	5.00 4.16	38 38	0.42	0.95-0.90
43. Ps Vs. tst (3:1)	(3:1)	Cherumodan x Japan Violet (Normal)	O E on independent	86.00 81.00	22.00 27.00	21.00 27.00	15.00 9.00	144 144	6.57	0.10-0.05

1	2	3	4	5	6	7	8	9	10	11
(3:1)	(3:1)	Cherumodan x Japan Violet (1500 rad)	O E on independent	110.00 101.81	28.00 33.94	24.00 33.94	9.00 11.31	181 181	2.17	0.70-0.50
(3:1)	(3:1)	Cherumodan x Japan Violet (2000 rad)	O E on independent	42.00 42.75	12.00 14.25	15.00 14.25	7.00 4.75	76 76	1.47	0.70-0.50
(9:7)	(3:1)	Cherumodan x Japan Violet (5000 rad)	O E on independent	17.00 16.03	6.00 5.34	10.00 12.47	5.00 4.16	38 38	0.80	0.95-0.90
44. Pa (3:1)	Vs. an (3:1)	Cherumodan x Japan Violet (Normal)	O E on independent	77.00 81.00	29.00 27.00	30.00 27.00	8.00 9.00	144 144	0.78	0.90-0.80
(3:1)	(3:1)	Cherumodan x Japan Violet (1500 rad)	O E on independent	90.00 101.81	37.00 14.25	38.00 33.94	16.00 11.31	181 181	4.08	0.30-0.20
(3:1)	(3:1)	Cherumodan x Japan Violet (2000 rad)	O E on independent	45.00 42.75	10.00 14.25	16.00 14.25	5.00 4.75	76 76	1.61	0.70-0.50
(9:7)	(3:1)	Cherumodan x Japan Violet (5000 rad)	O E on independent	17.00 16.03	5.00 5.34	10.00 12.47	6.00 4.16	38 38	1.38	0.90-0.80
45. Pa (3:1)	Vs. tst (3:1)	Cherumodan x Japan Violet (Normal)	O E on independent	83.00 81.00	23.00 27.00	24.00 27.00	14.00 9.00	144 144	3.75	0.30-0.20
(3:1)	(3:1)	Cherumodan x Japan Violet (1500 rad)	O E on independent	101.00 101.81	26.00 33.94	43.00 33.94	11.00 11.31	181 181	4.29	0.30-0.20
(3:1)	(3:1)	Cherumodan x Japan Violet (2000 rad)	O E on independent	41.00 43.31	13.00 14.44	16.00 14.44	6.00 4.81	76 76	0.73	0.95-0.90

1	2	3	4	5	6	7	8	9	10	11
(9:7)	(3:1)	Cherumodan x Japan Violet (5000 rad)	O E on independent	20.00 16.03	3.00 5.34	13.00 12.47	2.00 4.16	38 38	3.15	0.50-0.30
46. an (3:1) Vs. tst (3:1)		Cherumodan x Japan Violet (Normal)	O E on independent	80.00 81.00	27.00 27.00	27.00 27.00	10.00 9.00	144 144	0.12	0.99-0.00
(3:1)	(3:1)	Cherumodan x Japan Violet (1500 rad)	O E on independent	101.00 101.25	27.00 33.75	43.00 33.75	10.00 11.25	181 181	4.02	0.30-0.20
(3:1)	(3:1)	Cherumodan x Japan Violet (2000 rad)	O E on independent	44.00 42.75	17.00 14.25	13.00 14.25	2.00 4.75	76 76	2.27	0.70-0.50
(3:1)	(3:1)	Cherumodan x Japan Violet (5000 rad)	O E on independent	26.00 21.38	1.00 7.12	7.00 7.12	4.00 2.38	38 38	7.37	0.10-0.05

TABLE 42. Interrelationship of morphological characters studied : Linkage relationship of genes in crosses 1-4

Sl. No	Character combination / F_2 ratio			Crosses	Population O/E	Frequency AB	Ab	aB	ab	Total	X^2	X^2L	P	Co%	Phase
1	2			3	4	5	6	7	8	9	10	11	12	13	14
1.	Psh	Vs	Pl	Cherumodan X	O	82.00	29.00	0.00	33.00	144					
	3:1		9:7	Japan Violet	E on indep	60.75	47.25	20.25	15.75	144	53.26	53.26	0.96	2.02	C
				(Normal)	E on link	79.94	28.06	1.06	34.94	144	1.25	-	0.80-0.70		
				Cherumodan X	O	93.00	33.00	0.00	55.00	181					
	3:1		9:7	Japan Violet	E on indep	76.36	59.39	25.45	19.80	181	103.41	98.86	0.98	1.01	C
				(1500 rad)	E on link	100.97	34.78	0.84	44.41	181	4.09	-	0.30-0.20		
				Cherumodan X	O	38.00	17.00	0.00	21.00	76					
	3:1		27:37	Japan Violet	E on indep	24.05	32.95	8.02	10.98	76	32.97	30.78	0.98	1.01	C
				(2000 rad)	E on link	31.80	25.20	0.27	18.73	76	4.42	-	0.30-0.20		
2.	Psh	Vs	Pla	Cherumodan X	O	101.00	10.00	2.00	31.00	144					
	3:1		3:1	Japan Violet	E on indep	81.00	27.00	27.00	9.00	144	92.57	91.31	0.87	6.74	C
				(Normal)	E on link	103.31	4.69	4.69	31.31	144	7.60	-	0.10-0.05		
3.	Psh	Vs	Pjp	Cherumodan X	O	75.00	36.00	0.00	33.00	144					
	3:1		9:7	Japan Violet	E on indep	60.75	47.25	20.25	15.75	144	45.16	43.81	0.95	2.53	C
				(Normal)	E on link	79.70	28.30	1.30	34.70	144	3.76	-	0.30-0.20		
				Cherumodan X	O	80.00	46.00	0.00	55.00	181					
	3:1		27:37	Japan Violet	E on indep	57.27	78.48	19.09	26.16	181	73.35	70.25	0.98	1.01	C
				(1500 rad)	E on link	75.90	59.85	0.46	44.79	181	6.22	-	0.20-0.10		

1	2		3	4	5	6	7	8	9	10	11	12	13	14
			Cherumodan X	O	33.00	22.00	0.00	21.00	76					
	3:1	27:37	Japan Violet	E on indep	24.05	32.95	8.02	10.98	76	24.12	23.79	0.94	3.05	C
			(2000 rad)	E on link	31.42	25.58	0.65	18.35	76	1.61	-	0.70-0.50		
4.	Psh Vs	Plg	Cherumodan X	O	88.00	23.00	1.00	32.00	144					
	3:1	9:7	Japan Violet	E on indep	60.75	47.25	20.25	15.75	144	59.73	57.59	0.94	3.05	C
			(Normal)	E on link	79.50	28.50	1.50	34.50	144	2.32	-	0.70-0.50		
			Cherumodan X	O	98.00	28.00	4.00	51.00	181					
	3:1	9:7	Japan Violet	E on indep	76.36	59.39	25.45	19.80	181	89.97	87.19	0.92	4.08	C
			(1500 rad)	E on link	99.05	36.70	2.76	42.49	181	4.33	-	0.30-0.20		
			Cherumodan X	O	39.00	16.00	0.00	21.00	76					
	3:1	9:7	Japan Violet	E on indep	32.06	24.94	10.69	8.31	76	34.76	33.72	0.93	3.56	C
			(2000 rad)	E on link	41.78	15.22	0.97	18.03	76	1.68	-	0.70-0.50		
5.	Psh Vs	Pau	Cherumodan X	O	77.00	34.00	0.00	33.00	144					
	3:1	9:7	Japan Violet	E on indep	60.75	47.25	20.25	15.75	144	47.21	46.42	0.95	2.53	C
			(Normal)	E on link	79.76	28.24	1.24	34.76	144	2.60	-	0.50-0.30		
			Cherumodan X	O	33.00	22.00	0.00	21.00	76					
	3:1	27:37	Japan Violet	E on indep	24.05	32.95	8.02	10.98	76	24.12	23.79	0.94	3.05	C
			(2000 rad)	E on link	31.42	25.58	0.65	18.35	76	1.61	-	0.70-0.50		
6.	Psh Vs	Pn	Cherumodan X	O	29.00	82.00	1.00	32.00	144					
	3:1	3:13	Japan Violet	E on indep	20.25	87.75	6.75	29.25	144	9.31	8.57	0.80	10.56	C
			(Normal)	E on link	25.24	82.76	1.76	34.24	144	1.04	-	0.80-0.70		
			Cherumodan X	O	23.00	103.00	2.00	53.00	181					
	3:1	3:13	Japan Violet	E on indep	25.45	110.30	8.48	36.77	181	12.84	7.15	0.72	15.15	C
			(1500 rad)	E on link	30.78	104.97	3.16	42.09	181	5.26	-	0.20-0.10		

1	2			3	4	5	6	7	8	9	10	11	12	13	14
7.	Psh	Vs	Pin	Cherumodan X	O	100.00	11.00	6.00	27.00	144					
	3:1		3:1	Japan Violet	E on indep	81.00	27.00	27.00	9.00	144	66.27	65.79	0.76	12.82	C
				(Normal)	E on link	99.34	8.66	8.66	27.34	144	1.46	-	0.70-0.50		
				Cherumodan X	O	125.00	1.00	1.00	54.00	181					
	3:1		3:1	Japan Violet	E on indep	101.81	33.94	33.94	11.31	181	230.30	224.69	0.96	2.02	C
				(1500 rad)	E on link	133.90	1.85	1.85	43.40	181	3.96	-	0.30-0.20		
				Cherumodan X	O	49.00	6.00	5.00	16.00	76					
	3:1		3:1	Japan Violet	E on indep	42.75	14.25	14.25	4.75	76	38.34	37.43	0.69	16.93	C
				(2000 rad)	E on link	51.19	5.81	5.81	13.19	76	0.81	-	0.90-0.80		
8.	Psh	Vs	Ps	Cherumodan X	O	103.00	8.00	5.00	28.00	144					
	3:1		3:1	Japan Violet	E on indep	81.00	27.00	27.00	9.00	144	77.38	77.05	0.80	10.55	C
				(Normal)	E on link	101.92	7.08	7.08	29.94	144	0.87	-	0.90-0.80		
				Cherumodan X	O	49.00	6.00	5.00	16.00	76					
	3:1		3:1	Japan Violet	E on indep	42.75	14.25	14.25	4.75	76	38.34	37.43	0.69	16.93	C
				(2000 rad)	E on link	51.19	5.81	5.81	13.19	76	0.81	-	0.90-0.80		
9.	Psh	Vs	Pa	Cherumodan X	O	101.00	10.00	5.00	38.00	144					
	3:1		3:1	Japan Violet	E on indep	81.00	27.00	27.00	9.00	144	73.68	73.20	0.79	11.12	C
				(Normal)	E on link	100.38	7.62	7.62	28.38	144	1.65	-	0.70-0.50		
				Cherumodan X	O	120.00	6.00	7.00	48.00	181					
	3:1		3:1	Japan Violet	E on indep	101.81	33.94	33.94	11.31	181	161.61	161.55	0.84	8.35	C
				(1500 rad)	E on link	128.85	6.90	6.90	38.35	181	3.15	-	0.50-0.30		
				Cherumodan X	O	50.00	5.00	5.00	16.00	76					
	3:1		3:13	Japan Violet	E on indep	42.75	14.25	14.25	4.75	76	39.88	39.32	0.71	15.74	C
				(2000 rad)	E on link	51.57	5.43	5.43	13.75	76	0.55	-	0.95-0.90		

1	2			3	4	5	6	7	8	9	10	11	12	13	14
10.	Plm	Vs	Pla	Cherumodan X	O	98.00	2.00	5.00	39.00	144					
	3:1		3:1	Japan Violet	E on indep	81.00	27.00	27.00	9.00	144	144.64	141.35	0.90	5.13	C
				(Normal)	E on link	104.28	3.72	3.75	32.28	144	3.01	-	0.50-0.30		
11.	Plm	Vs	Pjp	Cherumodan X	O	75.00	25.00	0.00	44.00	144					
	3:1		9:7	Japan Violet	E on indep	60.75	47.25	20.25	15.75	144	84.74	81.35	0.97	1.51	C
				(Normal)	E on link	80.22	27.77	0.77	35.23	144	3.57	-	0.50-0.30		
12.	Plm	Vs	Plg	Cherumodan X	O	85.00	15.00	0.00	44.00	144					
	3:1		9:7	Japan Violet	E on indep	60.75	47.25	20.25	15.75	144	102.61	99.79	0.71	15.74	C
				(Normal)	E on link	80.95	27.05	4.95	35.95	144	7.42	-	0.10-0.05		
13.	Plm	Vs	Pau	Cherumodan X	O	77.00	23.00	0.00	44.00	144					
	3:1		9:7	Japan Violet	E on indep	60.75	47.25	20.25	15.75	144	87.71	84.89	0.98	1.01	C
				(Normal)	E on link	80.34	27.66	0.66	35.34	144	3.71	-	0.30-0.20		
14.	Plm	Vs	Pn	Cherumodan X	O	30.00	70.00	0.00	44.00	144					
	3:1		3:13	Japan Violet	E on indep	20.25	87.75	6.75	29.25	144	22.47	19.69	0.94	3.04	C
				(Normal)	E on link	26.50	81.50	0.50	35.50	144	4.61	-	0.30-0.20		
15.	Plm	Vs	Pin	Cherumodan X	O	98.00	2.00	8.00	36.00	144					
	3:1		3:1	Japan Violet	E on indep	81.00	27.00	27.00	9.00	144	121.09	118.57	0.88	6.19	C
				(Normal)	E on link	103.71	4.29	4.29	31.71	144	5.33	-	0.20-0.10		
16.	Plm	Vs	Ps	Cherumodan X	O	97.00	3.00	11.00	33.00	144					
	3:1		3:1	Japan Violet	E on indep	81.00	27.00	27.00	9.00	144	97.98	95.60	0.84	8.35	C
				(Normal)	E on link	102.21	5.79	5.79	30.21	144	6.55	-	0.10-0.05		
17.	Plm	Vs	Pa	Cherumodan X	O	97.00	3.00	9.00	35.00	144					
	3:1		3:1	Japan Violet	E on indep	81.00	27.00	27.00	9.00	144	111.60	109.09	0.85	7.80	C
				(Normal)	E on link	102.58	5.42	5.42	30.58	144	4.39	-	0.30-0.20		

1	2			3	4	5	6	7	8	9	10	11	12	13	14
18.	Pla	Vs	Pjp	Cherumodan X	O	75.00	28.00	0.00	41.00	144					
	3:1		9:7	Japan Violet	E on indep	60.75	47.25	20.25	15.75	144	71.91	69.97	0.97	1.51	C
				(Normal)	E on link	80.07	27.93	0.93	35.07	144	2.25	-	0.70-0.50		
				Cherumodan X	O	33.00	20.00	0.00	23.00	76					
	3:1		27:37	Japan Violet	E on indep	24.05	32.95	8.02	10.98	76	29.58	28.41	0.95	2.53	C
				(2000 rad)	E on link	31.57	25.43	0.49	18.51	76	2.80	-	0.50-0.30		
19.	Pla	Vs	Plg	Cherumodan X	O	84.00	19.00	5.00	36.00	144					
	3:1		9:7	Japan Violet	E on indep	60.75	47.25	20.25	15.75	144	63.31	60.58	0.92	4.08	C
				(Normal)	E on link	78.75	29.25	2.25	33.75	144	7.44	-	0.10-0.05		
				Cherumodan X	O	39.00	14.00	0.00	23.00	76					
	3:1		9:7	Japan Violet	E on indep	32.06	24.94	10.69	8.31	76	42.94	39.98	0.94	3.05	C
				(2000 rad)	E on link	41.95	15.05	0.80	18.20	76	2.35	-	0.70-0.50		
20.	Pla	Vs	Pau	Cherumodan X	O	73.00	30.00	4.00	37.00	144					
	3:1		9:7	Japan Violet	E on indep	60.75	47.25	20.25	15.75	144	50.48	49.10	0.84	8.35	C
				(Normal)	E on link	76.70	31.30	4.30	31.70	144	1.14	-	0.80-0.70		
				Cherumodan X	O	33.00	20.00	0.00	23.00	76					
	3:1		27:37	Japan Violet	E on indep	24.05	32.95	8.02	10.98	76	29.58	28.41	0.95	2.53	C
				(2000 rad)	E on link	31.57	25.43	0.49	18.51	76	2.80	-	0.50-0.30		

1	2			3	4	5	6	7	8	9	10	11	12	13	14
21.	Pla	Vs	Pn	Cherumodan X	O	28.00	75.00	2.00	39.00	144					
	(3:1)		(3:13)	Japan Violet	E on indep	20.25	87.75	6.75	29.25	144	11.41	10.08	0.79	11.12	C
				(Normal)	E on link	25.12	82.88	1.88	34.12	144	1.79	-	0.70-0.50		
				Cherumodan X	O	16.00	37.00	0.00	23.00	76					
	(3:1)		(3:13)	Japan Violet	E on indep	10.69	46.31	3.56	15.44	76	11.78	10.39	0.91	4.61	C
				(2000 rad)	E on link	13.80	43.20	0.45	18.55	76	2.75	-	0.50-0.30		
22.	Pla	Vs	Pin	Cherumodan X	O	96.00	7.00	10.00	31.00	144					
	(3:1)		(3:1)	Japan Violet	E on indep	81.00	27.00	27.00	9.00	144	82.07	81.00	0.76	12.82	C
				(Normal)	E on link	99.30	8.69	8.69	27.31	144	1.14	-	0.80-0.70		
				Cherumodan X	O	51.00	2.00	3.00	20.00	76					
	3:1		3:1	Japan Violet	E on indep	42.75	14.25	14.25	4.75	76	69.96	68.21	0.83	8.90	C
				(2000 rad)	E on link	53.92	3.08	3.08	15.92	76	1.58	-	0.70-0.50		
23.	Pla	Vs	Ps	Cherumodan X	O	99.00	4.00	9.00	32.00	144					
	3:1		3:1	Japan Violet	E on indep	81.00	27.00	17.00	9.00	144	94.37	93.44	0.82	9.45	C
				(Normal)	E on link	101.62	6.38	6.38	29.62	144	2.22	-	0.70-0.50		
				Cherumodan X	O	50.00	3.00	4.00	19.00	76					
	3:1		3:1	Japan Violet	E on indep	42.75	14.25	14.25	4.75	76	60.23	58.48	0.79	11.12	C
				(2000 rad)	E on link	53.02	3.98	3.98	15.02	76	1.47	-	0.70-0.50		

1	2			3	4	5	6	7	8	9	10	11	12	13	14
24.	Pla	Vs	Pa	Cherumodan X	O	96.00	7.00	10.00	31.00	144					
	3:1		3:1	Japan Violet	E on indep	81.00	27.00	27.00	9.00	144	82.07	81.00	0.76	12.82	C
				(Normal)	E on link	99.31	8.69	8.69	27.31	144	1.14	-	0.80-0.70		
				Cherumodan X	O	51.00	2.00	4.00	19.00	76					
	3:1		3:1	Japan Violet	E on indep	42.75	14.25	14.25	4.75	76	62.25	60.84	0.82	9.45	C
				(2000 rad)	E on link	53.60	3.40	3.40	15.60	76	1.25	-	0.80-0.70		
25	Pin	Vs	Ps	Cherumodan X	O	104.00	2.00	4.00	34.00	144					
	3:1		3:1	Japan Violet	E on indep	81.00	27.00	27.00	9.00	144	118.72	118.57	0.90	5.13	C
				(Normal)	E on link	104.40	3.60	3.60	32.40	144	0.84	-	0.90-0.80		
				Cherumodan X	O	52.00	2.00	2.00	20.00	76					
	3:1		3:1	Japan Violet	E on indep	42.75	14.25	14.25	4.75	76	72.02	70.76	0.86	7.27	C
				(2000 rad)	E on link	54.30	2.70	2.70	16.30	76	1.30	-	0.80-0.70		
26	Pin	Vs	Pa	Cherumodan X	O	101.00	5.00	5.00	33.00	144					
	3:1		3:1	Japan Violet	E on indep	81.00	27.00	27.00	9.00	144	104.79	104.49	0.84	8.35	C
				(Normal)	E on link	102.32	5.68	5.68	30.32	144	0.42	-	0.95-0.90		
				Cherumodan X	O	122.00	4.00	5.00	50.00	181					
	3:1		3:1	Japan Violet	E on indep	101.81	33.94	33.94	11.31	181	187.39	182.34	0.89	5.66	C
				(1500 rad)	E on link	130.70	5.08	5.08	40.20	181	3.19	-	0.50-0.30		

1	2			3	4	5	6	7	8	9	10	11	12	13	14
				Cherumodan X	O	52.00	2.00	3.00	19.00	76					
	3:1		3:1	Japan Violet	E on indep	42.75	14.25	14.25	4.75	76	64.16	63.25	0.84	8.35	C
				(2000 rad)	E on link	53.89	3.11	3.11	15.89	76	1.07	-	0.80-0.70		
27.	Ps	Vs	Pa	Cherumodan X	O	105.00	3.00	1.00	35.00	144					
	3:1		3:1	Japan Violet	E on indep	81.00	27.00	27.00	9.00	144	128.59	128.44	0.93	3.56	C
				(Normal)	E on link	105.44	2.56	2.56	33.44	144	1.10	-	0.80-0.70		
				Cherumodan X	O	54.00	0.00	1.00	21.00	76					
	3:1		3:1	Japan Violet	E on indep	42.75	14.25	14.25	4.75	76	85.12	84.21	0.94	3.05	C
				(2000 rad)	E on link	55.78	1.22	1.22	17.78	76	1.89	-	0.70-0.50		

TABLE 43 Inter relationship of morphological characters studied : Pleiotropic relationship of genes in crosses 1-4

Sl. No.	Character Combination/ F_2 ratios/s			Cross/es	Population O/E	F_2 Frequency AB	Ab	aB	ab	Total	X^2	P
1	2			3	4	5	6	7	8	9	10	11
1.	Px (15:1)	Vs	Psh (3:1)	Cherumodan x Japan Violet (Normal)	O	111.00	21.00	0.00	12.00	144		
					E on indep	101.25	33.75	6.75	2.25	144	54.76	
					E on pleio	108.00	27.00	0.00	9.00	144	2.42	0.50-0.30
	(15:1)	Vs	(3:1)	Cherumodan x Japan Violet (1500 rad)	O	126.00	45.00	0.00	10.00	181		
					E on indep	127.27	42.42	8.48	2.83	181	26.82	
					E on pleio	135.75	33.94	0.00	11.31	181	4.46	0.30-0.20
	(15:1)	Vs	(3:1)	Cherumodan x Japan Violet (2000 rad)	O	55.00	15.00	0.00	6.00	76		
					E on indep	53.44	17.81	3.56	1.19	76	23.49	
					E on pleio	57.00	14.25	0.00	4.75	76	0.44	0.95-0.90
2.	Px (15:1)	Vs	Plm (3:1)	Cherumodan x Japan Violet (Normal)	O	100.00	32.00	0.00	12.00	144		
					E on indep	101.25	33.75	6.75	2.25	144	49.12	
					E on pleio	108.00	27.00	0.00	9.00	144	2.52	0.50-0.20
	(15:1)	Vs	(9:7)	Cherumodan x Japan Violet (1500 rad)	O	114.00	57.00	0.00	10.00	181		
					E on indep	95.45	74.24	6.36	4.95	181	19.12	
					E on pleio	101.81	67.88	0.00	11.31	181	3.36	0.50-0.20
	(15:1)	Vs	(9:7)	Cherumodan x Japan Violet (2000 rad)	O	49.00	21.00	0.00	6.00	76		
					E on indep	40.08	31.17	2.67	2.08	76	15.36	
					E on pleio	42.75	28.50	0.00	4.75	76	3.21	0.70-0.50
3.	Px (15:1)	Vs	Pl (9:7)	Cherumodan x Japan violet (Normal)	O	82.00	50.00	0.00	12.00	144		
					E on indep	75.94	59.06	5.06	3.94	144	23.42	
					E on pleio	81.00	54.00	0.00	9.00	144	1.31	0.90-0.80
	(15:1)	Vs	(9:7)	Cherumodan x Japan violet (1500 rad)	O	93.00	78.00	0.00	10.00	181		
					E on indep	95.44	74.24	6.36	4.95	181	11.76	
					E on pleio	101.81	67.88	0.00	11.31	181	2.42	0.50-0.30

1	2		3	4	5	6	7	8	9	10	11
4.	Px (15:1)	Vs Pla (3:1)	Cherumodan x Japan Violet (Normal)	0	104.00	28.00	0.00	12.00	144		
				E on indep	101.25	33.75	6.75	2.25	144	49.70	
				E on pleio	108.00	27.00	0.00	9.00	144	1.19	0.70-0.50
	(15:1)	Vs (9:7)	Cherumodan x Japan Violet (1500 rad)	0	114.00	57.00	0.00	10.00	181		
				E on indep	94.45	74.24	6.36	4.95	181	19.12	
				E on pleio	101.81	67.88	0.00	11.31	181	3.05	0.20-0.10
	(15:1)	Vs (3:1)	Cherumodan x Japan Violet (2000 rad)	0	53.00	17.00	0.00	6.00	76		
				E on indep	53.44	17.81	3.56	1.19	76	23.04	
				E on pleio	57.00	14.25	0.00	4.75	76	1.14	0.80-0.70
5.	Px (15:1)	Vs Pjp (9:7)	Cherumodan x Japan Violet (Normal)	0	75.00	57.00	0.00	12.00	144		
				E on indep	75.94	59.06	5.06	3.94	144	21.62	
				E on pleio	81.00	54.00	0.00	9.00	144	1.61	0.70-0.50
6.	Px (15:1)	Vs Plg (9:7)	Cherumodan x Japan Violet (Normal)	0	89.00	43.00	0.00	12.00	144		
				E on indep	75.94	59.06	5.06	3.94	144	28.16	
				E on pleio	81.00	54.00	0.00	9.00	144	4.03	0.30.-0.20
	(15:1)	Vs (9:7)	Cherumodan x Japan Violet (1500 rad)	0	102.00	69.00	0.00	10.00	181		
				E on indep	94.45	74.24	6.36	4.95	181	12.48	
				E on pleio	101.81	67.88	0.00	11.31	181	0.17	0.99-0.95
7.	Px (15:1)	Vs Pau (9:7)	Cherumodan x Japan Violet (Normal)	0	77.00	55.00	0.00	12.00	144		
				E on indep	75.94	59.06	5.06	3.94	144	21.85	
				E on pleio	81.00	54.00	0.00	9.00	144	1.22	0.90-0.80
8.	Px (15:1)	Vs Pin (3:1)	Cherumodan x Japan Violet (Normal)	0	106.00	26.00	0.00	12.00	144		
				E on indep	101.25	33.75	6.75	2.25	144	51.00	
				E on pleio	108.00	27.00	0.00	9.00	144	1.08	0.90-0.80
	(15:1)	Vs (3:1)	Cherumodan x Japan Violet (1500 rad)	0	126.00	45.00	0.00	10.00	181		
				E on indep	127.27	42.42	8.48	2.83	181	26.82	
				E on pleio	135.75	33.94	0.00	11.31	181	4.46	0.30-0.20
	(15:1)	Vs (3:1)	Cherumodan x Japan Violet (2000 rad)	0	54.00	16.00	0.00	6.00	76		
				E on indep	53.44	17.81	3.56	1.19	76	23.19	
				E on pleio	57.00	14.25	0.00	4.75	76	0.70	0.90-0.80

1	2		3	4	5	6	7	8	9	10	11
9.	Px (15:1)	Vs	Ps (3:1)	Cherumodan x Japan Violet (Normal)	0	108.00	24.00	0.00	12.00	144	
					E on indep	101.25	33.75	6.75	2.25	144	52.27
					E on pleio	108.00	27.00	0.00	9.00	144	1.33	0.80.-070
	(15:1)	Vs	(3:1)	Cherumodan x Japan Violet (1500 rad)	0	138.00	33.00	0.00	10.00	181	
					E on indep	127.27	42.42	8.48	2.83	181	29.64
					E on pleio	135.75	33.94	0.00	11.31	181	0.22	0.98-0.95
	(15:1)	Vs	(3:1)	Cherumodan x Japan Violet (2000 rad)	0	54.00	16.00	0.00	6.00	76	
					E on indep	40.08	31.17	2.67	2.08	76	22.28
					E on pleio	57.00	14.25	0.00	4.75	76	0.70	0.90-0.80
10.	Px (15:1)	Vs	Pa (3:1)	Cherumodan x Japan Violet (Normal)	0	106.00	26.00	0.00	12.00	144	
					E on indep	101.25	33.75	6.75	2.25	144	51.00
					E on pleio	108.00	27.00	0.00	9.00	144	1.08	0.80-0.70
	(15:1)	Vs	(3:1)	Cherumodan x Japan Violet (1500 rad)	0	127.00	44.00	0.00	10.00	181	
					E on indep	127.27	42.42	8.48	2.83	181	26.71
					E on pleio	135.75	33.94	0.00	11.31	181	3.70	0.50-0.30
	(15:1)	Vs	(3:1)	Cherumodan x Japan Violet (2000 rad)	0	55.00	15.00	0.00	6.00	76	
					E on indep	53.44	17.81	3.56	1.19	76	23.49
					E on pleio	57.00	14.25	0.00	4.75	76	0.44	0.95-0.90
11.	Psh (9:7)	Vs	Pl (27:37)	Cherumodan x Japan Violet (5000 rad)	0	14.00	9.00	0.00	15.00	38	
					E on indep	9.02	12.36	7.01	9.61	38	13.72
					E on pleio	16.03	5.34	0.00	16.63	38	2.92	0.50-0.30
12.	Psh (3:1)	Vs	Plm (9:7)	Cherumodan x Japan Violet (2000 rad)	0	49.00	6.00	0.00	21.00	76	
					E on indep	32.06	24.94	10.69	8.31	76	53.40
					E on pleio	42.75	14.25	0.00	19.00	76	5.90	0.20-0.10
	(9:7)	Vs	(9:7)	Cherumodan x Japan Violet (5000 rad)	0	19.00	4.00	0.00	15.00	38	
					E on indep	12.02	9.35	9.35	7.27	38	24.68
					E on pleio	16.03	5.35	5.34	11.28	38	7.46	0.10-0.05
13.	Psh (9:7)	Vs	Pla (9:7)	Cherumodan x Japan Violet (5000 rad)	0	19.00	4.00	1.00	14.00	38	
					E on indep	12.02	9.35	9.35	7.27	38	20.80
					E on pleio	16.03	5.34	5.34	11.28	38	5.07	0.20-0.10

1	2		3	4	5	6	7	8	9	10	11
14.	Psh	Vs	Pjp	Cherumodan x	0	15.00	8.00	0.00	15.00	38	
	(9:7)		(27;37)	Japan Violet	E on indep	9.02	12.36	7.01	9.61	38	15.54
				(5000 rad)	E on pleio	16.03	5.34	0.00	16.63	38	1.55 0.70-0.50
15.	Psh	Vs	Plg	Cherumodan x	0	15.00	8.00	0.00	15.00	38	
	(9:7)		(27:37)	Japan Violet	E on indep	9.02	12.36	7.01	9.61	38	15.54
				(5000 rad)	E om pleio	16.03	5.34	0.00	16.63	38	1.55 0.70-0.50
16.	Psh	Vs	Pau	Cherumodan x	0	12.00	11.00	0.00	15.00	38	
	(9:7)		(27:37)	Japan Violet	E on indep	9.02	12.36	7.01	9.61	38	11.17
				(5000 rad)	E on pleio	16.03	5.34	0.00	16.63	38	7.17 0.10-0.05
17.	Psh	Vs	Pin	Cherumodan x	0	21.00	2.00	1.00	14.00	38	
	(9:7)		(9:7)	Japan Violet	E on indep	12.02	9.35	9.35	7.27	38	26.17
				(5000 rad)	E on pleio	16.04	5.34	5.34	11.28	38	7.81 0.10-0.05
18.	Psh	Vs	Ps	Cherumodan x	0	21.00	2.00	1.00	14.00	38	
	(9:7)		(9:7)	Japan Violet	E on indep	12.02	9.35	0.35	7.27	38	26.17
				(5000 rad)	E on pleio	16.04	5.34	5.34	11.28	38	7.81 0.10-0.05
19.	Psh	Vs	Pa	Cherumodan x	0	21.00	2.00	1.00	14.00	38	
	(9:7)		(9:7)	Japan Violet	E on indep	12.02	9.35	9.35	7.27	38	26.17
				(5000 rad)	E on pleio	16.04	5.34	5.34	11.28	38	7.81 0.10-0.05
20.	Pl	Vs	Plm	Cherumodan x	0	82.00	0.00	18.00	44.00	144	
	(9:7)		(3:1)	Japan Violet	E on indep	60.75	20.25	47.25	15.75	144	96.44
				(Normal)	E on pleio	81.00	0.00	27.00	36.00	144	4.79 0.20-0.10
				Cherumodan x	0	38.00	0.00	11.00	27.00	76	
	(9:7)	Vs	(9:7)	Japan Violet	E on indep	18.04	14.03	24.71	19.22	76	46.87
				(2000 rad)	E on pleio	32.06	0.00	10.69	33.25	76	2.28 0.70-0.50
				Cherumodan x	0	14.00	0.00	5.00	19.00	38	
	(27:37)	Vs	(9:7)	Japan Violet	E on indep	9.02	7.01	12.36	9.61	38	23.32
				(5000 rad)	E on pleio	16.03	0.00	5.34	16.63	38	0.62 0.90-0.80

1	2		3	4	5	6	7	8	9	10	11
21.	Pl	Vs	Pla	Cherumodan x	0	82.00	0.00	21.00	41.00	144	
	(9:7)		(3:1)	Japan Violet)	E on indep	60.75	20.25	47.25	15.75	144	82.73
				(Normal)	E on pleio	81.00	0.00	27.00	36.00	144	2.04 0.70-0.50
				Cherumodan x	0	38.00	0.00	15.00	23.00	76	
	(27:37)	Vs	(9:7)	Japan Violet	E on indep	18.04	14.03	24.71	19.22	76	40.67
				(2000 rad)	E on pleio	32.06	0.00	24.93	19.00	76	5.90 0.20-0.10
	(27:37)	Vs	(9:7)	Cherumodan x	0	14.00	0.00	6.00	18.00	38	
				Japan Violet	E on indep	9.02	7.01	12.36	9.61	38	20.36
				(5000 rad)	E on pleio	16.03	0.00	5.34	16.63	38	0.71 0.90-0.80
22.	Pl	Vs	Pjp	Cherumodan x	0	80.00	13.00	0.00	88.00	181	
	(9:7)		(27:37)	Japan Violet	E on indep	42.95	58.86	33.41	45.78	181	140.04
				(1500 rad)	E on pleio	76.36	25.45	0.00	79.19	181	7.04 0.10-0.05
	(27:37)	Vs	(27:37)	Cherumodan x	0	30.00	8.00	3.00	35.00	76	
				Japan Violet	E on indep	13.52	18.54	18.54	25.40	76	42.73
				(2000 rad)	E on pleio	24.05	8.02	8.02	35.91	76	4.64 0.30-0.20
	(27:37)		(27:37)	Cherumodan x	0	11.00	3.00	4.00	20.00	38	
				Japan Violet	E on indep	6.76	9.27	9.27	12.70	38	14.09
				(5000 rad)	E on pleio	12.02	4.01	4.01	17.96	38	0.57 0.95-0.90
23.	Pl	Vs	Plg	Chermodan x	0	38.00	0.00	3.00	36.00	76	
	(27:37)		(9:7)	Japan Violet	E on indep	18.04	14.03	24.71	19.22	76	69.83
				(2000 rad)	E on pleio	32.06	0.00	10.69	33.25	76	6.85 0.10-0.05
	(27:37)		(27:37)	Cherumodan x	0	12.00	2.00	3.00	21.00	38	
				Japan Violet	E on indep	6.76	9.27	9.27	12.70	38	19.43
				(5000 rad)	E on pleio	12.02	4.01	4.01	17.96	38	1.77 0.70-0.50
24.	Pl	Vs	Pau	Cherumodan x	0	31.00	7.00	2.00	36.00	76	
	(27:37)		(27:37)	Japan violet	E on indep	13.52	18.54	18.54	25.40	76	48.96
				(2000 rad)	E on pleio	24.05	8.02	8.02	35.91	76	6.66 0.10-0.05
	(27:37)	Vs	(27:37)	Cherumodan x	0	10.00	4.00	2.00	22.00	38	
				Japan Violet	E on indep	6.76	9.27	9.27	12.70	38	17.06
				(5000 rad)	E on pleio	12.02	4.01	4.01	17.96	38	2.26 0.70-0.50

1	2		3	4	5	6	7	8	9	10	11
25.	Pl	Vs	Pn	Cherumodan x	0	26.00	56.00	4.00	58.00	144	
	(9:7)		(3:13)	Japan Violet	E on indep	15.19	65.81	11.81	51.19	144	15.21	
				(Normal)	E on pleio	20.25	60.75	6.75	56.25	144	3.18	0.50-0.30
	(9:7)	Vs	(3:13)	Cherumodan x	0	22.00	71.00	3.00	85.00	181		
				Japan Violet	E on indep	19.09	82.72	14.85	64.34	181	18.19	
				(1500 rad)	E on pleio	25.45	76.36	8.48	70.71	181	7.27	0.10-0.05
	(27:37)	Vs	(3:13)	Cherumodan x	0	12.00	26.00	4.00	34.00	76		
				Japan Violet	E on indep	6.01	26.05	8.24	35.70	76	8.23	
				(2000 rad)	E on pleio	8.02	24.05	6.23	37.70	76	3.30	0.50-0.30
26.	Pl	Vs	Pin	Cherumodan x	0	82.00	0.00	24.00	38.00	144		
	(9:7)		(3:1)	Japan Violet	E on indep	60.75	20.25	47.25	15.75	144	70.56	
				(Normal)	E on pleio	81.00	0.00	27.00	36.00	144	0.46	0.95-0.90
				Cherumoden x	0	93.00	33.00	32.00	55.00	181		
	(9:7)	Vs	(3:1)	Japan Violet	E on indep	76.36	25.45	59.39	19.80	181	103.38	
				(1500 rad)	E on pleio	101.81	0.00	33.94	45.25	181	2.89	0.50-0.30
				Cherumoden x	0	38.00	0.00	16.00	22.00	76		
	(27:37)	Vs	(3:1)	Japan Violet	E on indep	24.05	8.02	32.95	10.98	76	35.89	
				(2000 rad)	E on pleio	32.06	0.00	24.94	19.00	76	4.78	0.20-0.10
				Cherumoden x	0	14.00	0.00	8.00	16.00	38		
	(27:37)	Vs	(9:7)	Japan Violet	E on indep	9.02	7.01	12.35	9.62	38	15.53	
				(5000 rad)	E on pleio	16.03	0.00	5.34	16.63	38	1.61	0.70-0.50
27.	Pl	Vs	Ps	Cherumoden x	0	82.00	0.00	26.00	36.00	144		
	(9:7)		(3:1)	Japan Violet	E on indep	60.75	20.25	47.25	15.75	144	60.77	
				(Normal)	E on pleio	81.00	0.00	27.00	36.00	144	0.08	0.98
				Cherumoden x	0	93.00	0.00	45.00	43.00	181		
	(9:7)	Vs	(3:1)	Japan Violet	E on indep	76.36	25.45	59.39	19.80	181	59.75	
				(1500 rad)	E on pleio	101.81	0.00	33.94	45.25	181	4.48	0.30-0.20
				Cherumoden x	0	38.00	0.00	16.00	22.00	76		
	(27:37)	Vs	(3:1)	Japan Violet	E on indep	24.05	8.02	32.95	10.98	76	35.89	
				(2000 rad)	E on pleio	32.06	0.00	24.94	19.00	76	4.78	0.20-0.10

1	2		3	4	5	6	7	8	9	10	11
	(27:37)	Vs	(9:7)	Cherumoden	0	14.00	0.00	8.00	16.00	38	
				Japan Violet	E on indep	9.02	7.01	12.36	9.62	38	15.58	
				(5000 rad)	E on pleio	16.03	0.00	5.34	16.63	38	1.61	0.90-0.80
28.	Pl	Vs	Pa	Cherumoden x	0	82.00	0.00	24.00	38.00	144		
	(9:7)		(3:1)	Japan Violet	E on indep	60.75	20.25	47.25	15.75	144	63.09	
				(Normal)	E on pleio	81.00	0.00	27.00	36.00	144	0.75	0.90-0.80
				Cherumodan x	0	93.00	0.00	34.00	54.00	181		
	(9:7)	Vs	(3:1)	Japan Violet	E on indep	76.36	25.45	59.39	19.80	181	101.70	
				(1500 rad)	E on pleio	101.81	0.00	33.94	45.25	181	2.26	0.70-0.50
				Cherumodan x	0	38.00	0.00	17.00	21.00	76		
	(27:37)	Vs	(3:1)	Japan Violet	E on indep	24.05	8.02	32.95	10.98	76	32.98	
				(2000 rad)	E on pleio	32.06	0.00	24.94	19.00	76	3.84	0.30-0.20
	(27:37)	Vs	(9:7)	Cherumodan x	0	14.00	0.00	8.00	16.00	38		
				Japan Violet	E on indep	9.02	7.01	12.36	9.61	38	15.55	
				(5000 rad)	E on pleio	16.03	0.00	5.34	16.63	38	1.61	0.90-0.80
29.	Plm	Vs	Pla	Cherumodan x	0	114.00	0.00	0.00	67.00	181		
	(9:7)		(9:7)	Japan Violet	E on indep	57.27	44.54	44.54	34.64	181	175.51	
				(1500 rad)	E on pleio	101.81	0.00	0.00	79.19	181	3.34	0.50-0.30
	(9:7)	Vs	(9:7)	Cherumodan x	0	17.00	2.00	3.00	16.00	38		
				Japan Violet	E on indep	12.03	9.35	9.35	7.27	38	22.62	
				(5000 rad)	E on pleio	21.38	0.00	0.00	16.62	38	5.15	0.20-0.10
30.	Plm	Vs	Pjp	Cherumodan x	0	80.00	34.00	0.00	67.00	181		
	(9:7)		(27:37)	Japan Violet	E on indep	42.95	58.86	33.41	45.78	181	87.46	
				(1500 rad)	E on pleio	76.36	25.45	0.00	79.19	181	4.92	0.20-0.10
	(9:7)	Vs	(27:37)	Cherumodan x	0	33.00	16.00	0.00	27.00	76		
				Japan Violet	E on indep	18.04	24.71	14.03	19.22	76	32.65	
				(2000 rad)	E on pleio	32.06	10.69	0.00	33.25	76	3.84	0.30-0.20
	(9:7)	Vs	(27:37)	Cherumodan x	0	15.00	4.00	0.00	19.00	38		
				Japan Violet	E on indep	9.02	12.36	7.01	9.61	38	25.80	
				(5000 rad)	E on pleio	16.03	5.34	0.00	16.63	38	0.74	0.90-0.80

1	2			3	4	5	6	7	8	9	10	11
31.	Plm (9:7)	Vs	Plg (27:37)	Cherumodan x Japan Violet (5000 rad)	0	15.00	4.00	0.00	19.00	38		
					E on indep	9.02	12.36	7.01	9.61	38	25.80	
					E on pleio	16.03	5.34	0.00	16.63	38	0.74	0.90-0.80
32.	Plm (9:7)	Vs	Pau (27:37)	Cherumodan x Japan Violet (1500 rad)	0	89.00	25.00	0.00	67.00	181		
					E on indep	42.95	58.86	33.41	45.78	181	112.10	
					E on pleio	76.36	24.45	0.00	79.19	181	3.98	0.30-0.20
	(9:7)	Vs	(27:37)	Cherumodan x Japan Violet (2000 rad)	0	33.00	16.00	0.00	27.00	76		
					E on indep	18.04	24.71	14.73	19.22	76	32.66	
					E on pleio	32.06	10.69	0.00	33.25	76	3.84	0.30-0.20
	(9:7)	Vs	(27:37)	Cherumodan x Japan Violet (5000 rad)	0	12.00	7.00	0.00	19.00	38		
					E on indep	9.02	12.36	7.01	6.61	38	33.54	
					E on pleio	16.03	5.34	0.00	16.63	38	1.87	0.70-0.50
33.	Plm (9:7)	Vs	Pn (3:13)	Cherumodan x Japan iolet (1500 rad)	0	21.00	93.00	4.00	63.00	181		
					E on indep	19.09	82.72	14.85	64.34	181	8.64	
					E on pleio	25.45	76.36	8.48	70.71	181	7.61	0.10-0.05
	(9:7)	Vs	(3:13)	Cherumodan x Japan Violet (2000 rad)	0	16.00	33.00	0.00	27.00	76		
					E on indep	8.02	34.73	6.23	27.02	76	14.26	
					E on pleio	10.69	32.06	3.56	29.69	76	2.47	0.50-0.30
34	Plm (9;7)	Vs	Pin (3:1)	Cherumodan x Japan Violet (2000 rad)	0	49.00	0.00	6.00	21.00	76		
					E on indep	32.06	10.69	24.94	8.31	76	53.40	
					E on pleio	42.75	0.00	14.25	19.00	76	5.90	0.20-0.10
	(9:7)	Vs	(9:7)	Cherumodan x Japan Violet (5000 rad)	0	17.00	2.00	3.00	16.00	38		
					E on indep	12.03	9.35	9.35	7.27	38	19.34	
					E on pleio	16.04	5.343	5.34	11.28	38	5.15	0.20-0.10
35.	Plm (9:7)	Vs	Ps (3:1)	Cherumodan x Japan Violet (1500 rad)	0	114.00	0.00	24.00	43.00	181		
					E on indep	76.36	25.45	59.39	19.80	181	92.28	
					E on pleio	101.81	0.00	33.94	45.25	181	4.48	0.30-0.20
	(9:7)	Vs	(3:1)	Cherumodan x Japan Violet (2000 rad)	0	49.00	0.00	6.00	21.00	76		
					E on indep	32.06	10.69	24.94	8.31	76	53.40	
					E on pleio	42.75	0.00	14.25	19.00	76	5.90	0.20-0.10

1	2		3	4	5	6	7	8	9	10	11
	(9:7)	Vs	(9:7)	Cherumodan x	0	17.00	2.00	5.00	16.00	38	
				Japan Violet	E on indep	12.03	9.35	9.35	7.27	38	19.34	
				(5000 rad)	E on pleio	16.04	5.34	5.34	11.28	38	5.15	0.20-0.10
36.	Plm	Vs	Pa	Cherumodan x	0	49.00	0.00	6.00	21.00	76		
	(9:7)		(3:1)	Japan Violet	E on indep	32.06	10.69	24.94	8.31	76	53.40	
				(2000 rad)	E on pleio	42.75	0.00	14.25	19.00	76	5.90	0.20-0.10
	(9:7)	Vs	(9:7)	Cherumodan x	0	17.00	2.00	5.00	14.00	38		
				Japan Violet	E on indep	12.03	9.35	9.35	7.27	38	16.12	
				(5000 rad)	E on pleio	21.38	0.00	0.00	16.62	38	2.82	0.50-0.30
37.	Pla	Vs	Pjp	Cherumodan x	0	80.00	34.00	0.00	67.00	181		
	(9:7)		(27:37)	Japan Violet	E on indep	42.95	58.86	33.41	45.78	181	85.71	
				(1500 rad)	E on pleio	76.36	25.45	0.00	79.19	181	4.92	0.30-0.20
	(9:7)	Vs	(27:37)	Cherumodan x	0	15.00	5.00	0.00	18.00	38		
				Japan Violet	E on indep	9.02	12.36	7.01	9.61	38	22.68	
				(5000 rad)	E on pleio	16.03	5.34	0.00	16.63	38	0.20	0.-0.95
38.	Pla	Vs	Plg	Cherumodan x	0	15.00	5.00	0.00	18.00	38		
	(9:7)		(27:37)	Japan Violet	E on indep	9.02	12.35	7.01	9.62	38	22.68	
				(5000 rad)	E on pleio	16.03	5.34	0.00	16.63	38	0.20	0-0.95
39.	Pla	Vs	Pau	Cherumodan x	0	12.00	8.00	0.00	18.00	38		
	(9:7)		(27:37)	Japan Violet	E on indep	9.02	12.35	7.01	9.62	38	16.83	
				(5000 rad)	E on pleio	16.03	5.34	0.00	16.63	38	2.45	0.50-0.30
40.	Pla	Vs	Pn	Cherumodan x	0	26.00	88.00	3.00	64.00	181		
	(9:7)		(3:13)	Japan Violet	E on indep	19.09	82.72	14.85	64.34	181	12.30	
				(1500 rad)	E on pleio	25.45	76.36	8.48	70.71	181	7.60	0.10-0.05
41.	Pla	Vs	Pin	Cherumodan x	0	18.00	2.00	4.00	14.00	38		
	(9:7)		(9;7)	Japan Violet	E on indep	12.03	9.35	9.35	7.27	38	18.03	
				(5000 rad)	E on pleio	21.38	0.00	0.00	16.62	38	3.32	0.50-0.30
42.	Pla	Vs	Ps	Cherumodan x	0	114.00	0.00	24.00	43.00	181		
	(9:7)		(3:1)	Japan Violet	E on indep	76.36	25.45	59.39	19.80	181	92.28	
				(1500 rad)	E on pleio	101.81	0.00	33.94	45.25	181	4.48	0.30-0.20

1	2			3	4	5	6	7	8	9	10	11
	(9:7)	Vs	(9:7)	Cherumodan x	0	18.00	2.00	4.00	14.00	38		
				Japan Violet	E on indep	12.03	9.35	9.35	7.27	38	18.03	
				(5000 rad)	E on pleio	16.03	5.34	5.34	11.29	38	3.32	0.50-0.30
43.	Pla	Vs	Pa	Cherumodan x	0	19.00	1.00	3.00	15.00	38		
	(9:7)		(9:7)	Japan Violet	E on indep	12.03	9.35	9.35	7.27	38	24.03	
				(5000 rad)	E on pleio	16.03	5.34	5.34	11.29	38	6.32	0.10-0.05
44.	Pjp	Vs	Plg	Cherumodan x	0	80.00	0.00	22.00	79.00	181		
	(27:37)		(9:7)	Japan Violet	E on indep	42.95	33.41	58.36	45.78	181	112.15	
				(1500 rad)	E on pleio	76.36	0.00	25.45	79.19	181	0.64	0.90-0.80
	(27:37)	Vs	(9:7)	Cherumodan x	0	33.00	0.00	6.00	37.00	76		
				Japan Violet	E on indep	18.04	14.03	24.71	19.22	76	57.05	
				(2000 rad)	E on pleio	32.06	0.00	10.69	32.25	76	2.78	0.50-0.30
	(27:37)	Vs	(27:37)	Cherumodan x	0	11.00	4.00	4.00	19.00	38		
				Japan Violet	E on indep	6.76	9.27	9.27	12.70	38	11.78	
				(5000 rad)	E on pleio	12.02	4.01	4.01	17.96	38	0.15	0.00-0.95
45.	Pjp	Vs	Pau	Cherumodan x	0	11.00	4.00	1.00	22.00	38		
	(27:37)		(27:37)	Japan Violet	E on indep	6.76	9.27	9.27	12.70	38	19.84	
				(5000 rad)	E on pleio	12.02	4.01	4.01	17.96	38	3.25	0.50-0.30
46.	Pjp	Vs	Pn	Cherumodan x	0	20.00	60.00	5.00	96.00	181		
	(27:37)		(3:13)	Japan Violet	E on indep	14.32	62.04	19.62	85.02	181	14.63	
				(1500 rad)	E on pleio	19.09	57.27	14.85	89.79	181	7.14	0.10-0.05
	(27:37)	Vs	(3:13)	Cherumodan x	0	12.00	21.00	4.00	39.00	76		
				Japan Violet	E on indep	6.01	26.05	8.24	37.70	76	9.44	
				(2000 rad)	E on pleio	8.02	24.05	6.23	37.70	76	3.21	0.50-0.30
47.	Pjp	Vs	Pin	Cherumodan x	0	75.00	0.00	31.00	38.00	144		
	(9:7)		(3:1)	Japan Violet	E on indep	60.75	20.25	47.25	15.75	144	60.61	
				(Normal)	E on pleio	81.00	0.00	27.00	36.00	144	1.15	0.80-0.70
	(27:37)	Vs	(3:1)	Cherumodan x	0	80.00	0.00	46.00	55.00	181		
				Japan Violet	E on indep	57.27	19.09	78.48	26.16	181	73.35	
				(1500 rad)	E on pleio	76.36	0.00	59.39	45.25	181	5.29	0.20-0.10

1	2		3	4	5	6	7	8	9	10	11
	(27:37)	Vs (3:1)	Cherumodan x	0	33.00	0.00	21.00	22.00	76		
			Japan Violet	E on indep	24.05	8.02	32.95	10.98	76	26.74	
			(2000 rad)	E on pleio	32.06	0.00	24.94	19.00	76	1.12	0.80-0.70
	(27:37)	Vs (9:7)	Cherumodan x	0	15.00	0.00	7.00	16.00	38		
			Japan Violet	E on indep	9.02	7.01	12.36	9.61	38	17.55	
			(5000 rad)	E on pleio	16.03	0.00	5.34	16.63	38	0.61	0.90-0.80
48.	Pjp Vs Ps		Cherumodan x	0	75.00	0.00	33.00	36.00	144		
	(9:7)	(3:1)	Japan Violet	E on indep	60.75	20.25	47.25	15.75	144	53.93	
			(Normal)	E on pleio	81.00	0.00	27.00	36.00	144	1.78	0.70-0.50
	(27:37)	Vs (3:1)	Cherumodan x	0	80.00	0.00	58.00	43.00	181		
			Japan Violet	E on indep	57.27	19.09	78.48	26.16	181	44.30	
			(1500 rad)	E on pleio	76.36	0.00	59.39	45.25	181	0.32	0.95-0.90
	(27:37)	Vs (3:1)	Cherumodan x	0	33.00	0.00	21.00	22.00	76		
			Japan Violet	E on indep	24.05	8.02	32.95	10.98	76	26.74	
			(2000 rad)	E on pleio	32.06	0.00	24.94	19.00	76	1.12	0.80-0.70
	(27:37)	Vs (9:7)	Cherumodan x	0	15.00	0.00	7.00	16.00	38		
			Japan Violet	E on indep	9.02	7.01	12.36	9.61	38	17.55	
			(5000 rad)	E on pleio	16.03	0.00	5.34	16.63	38	0.61	0.90-0.80
49.	Pjp Vs Pa		Cherumodan x	0	75.00	0.00	31.00	38.00	144		
	(9:7)	(3:1)	Japan Violet	E on indep	60.75	20.25	47.25	15.75	144	60.61	
			(Normal)	E on pleio	81.00	0.00	27.00	36.00	144	1.15	0.80-0.70
	(27:37)	Vs (3:1)	Cherumodan x	0	80.00	0.00	47.00	54.00	181		
			Japan Violet	E on indep	57.27	19.09	78.48	26.16	181	70.37	
			(1500 rad)	E on pleio	76.36	0.00	59.39	45.25	181	4.44	0.30-0.20
	(27:37)	Vs (3:1)	Cherumodan	0	33.00	0.00	22.00	21.00	76		
			Japan Violet	E on indep	24.05	8.02	32.95	10.98	76	24.13	
			(2000 rad)	E on pleio	32.06	0.00	24.94	19.00	76	0.58	0.95-0.90
	(27:37)	Vs (9:7)	Cherumodan x	0	15.00	0.00	7.00	16.00			
			Japan Violet	E on indep	9.02	7.01	12.36	9.61	38	17.55	
			(5000 rad)	E on pleio	16.03	0.00	5.34	16.63	38	0.61	0.90-0.80

1	2		3	4	5	6	7	8	9	10	11
50.	Plg (9:7)	Vs	Pau (27:37)	Cherumodan x Japan Violet (2000 rad)	0 E on indep E on pleio	33.00 18.04 32.06	6.00 24.71 10.69	0.00 14.03 0.00	37.00 19.22 33.23	76 76 76	 57.05 2.51	 0.50-0.30
	(27:37)	Vs	(27:37)	Cherumodan x Japan Violet (5000 rad)	0 E on indep E on pleio	10.00 6.76 12.02	5.00 9.27 4.01	2.00 9.27 4.01	21.00 12.70 17.96	38 38 38	 14.65 2.11	 0.70-0.50
51.	Plg (9:7)	Vs	Pn (3:13)	Cherumodan x Japan Violet (Normal)	0 E on indep E on pleio	26.00 15.19 20.25	63.00 65.81 60.75	4.00 11.81 6.75	51.00 51.19 56.25	144 144 144	 12.98 3.33	 0.50-0.30
	(9:7)	Vs	(3:13)	Cherumodan x Japan Violet (1500 rad)	0 E on indep E on pleio	22.00 19.09 25.45	80.00 82.72 76.36	4.00 14.85 8.48	75.00 64.34 70.71	181 181 181	 10.23 3.27	 0.50-0.30
52.	Plg (9:7)	Vs	Pin (3:1)	Cherumodan x Japan Violet (Normal)	0 E on indep E on pleio	89.00 60.75 81.00	0.00 20.25 0.00	17.00 47.25 27.00	38.00 15.75 36.00	144 144 144	 84.19 4.61	 0.20-0.10
	(9:7)	Vs	(3:1)	Cherumodan x Japan Violet (2000 rad)	0 E on indep E on pleio	39.00 32.06 42.75	0.00 10.69 0.00	15.00 24.94 14.25	22.00 8.31 19.00	76 76 76	 38.71 0.84	 0.90-0.80
	(27:37)	Vs	(9:7)	Cherumodan x Japan Violet (5000 rad)	0 E on indep E on pleio	15.00 9.02 16.03	0.00 7.01 0.00	7.00 12.35 5.34	16.00 9.62 16.63	38 38 38	 17.52 0.61	 0.90-0.80
53.	Plg (9:7)	Vs	Ps (3:1)	Cherumodan x Japan Violet (Normal)	0 E on indep E on pleio	89.00 60.75 81.00	0.00 20.25 0.00	19.00 47.25 27.00	36.00 15.75 36.00	144 144 144	 76.32 3.16	 0.50-0.30
	(9:7)	Vs	(3:1)	Cherumodan x Japan Violet (1500 rad)	0 E on indep E on pleio	102.00 76.36 101.81	0.00 25.45 0.00	36.00 59.39 33.94	43.00 19.80 45.25	181 181 181	 70.46 0.24	 0.-0.95

1	2			3	4	5	6	7	8	9	10	11
	(9:7)	Vs	(3:1)	Cherumodan x	0	39.00	0.00	15.00	22.00	76		
				Japan Violet	E on indep	32.06	10.69	24.94	8.31	76	38.71	
				(2000 rad)	E on pleio	42.75	0.00	14.25	19.00	76	0.84	0.90-0.80
	(27:37)	Vs	(9:7)	Cherumodan x	0	15.00	0.00	7.00	16.00	38		
				Japan Violet	E on indep	9.02	7.01	12.35	9.62	38	17.52	
				(5000 rad)	E on pleio	16.03	0.00	5.34	16.63	38	0.61	0.90-0.80
54.	Plg	Vs	Pa	Cherumodan x	0	89.00	0.00	17.00	38.00	144		
	(9:7)		(3:1)	Japan Violet	E on indep	60.75	20.25	47.25	15.75	144	84.19	
				(Normal)	E on pleio	81.00	0.00	27.00	36.00	144	4.61	0.30-0.20
	(9:7)	Vs	(3:1)	Cherumodan x	0	102.00	0.00	25.00	54.00	181		
				Japan Violet	E on indep	76.36	25.45	59.39	19.80	181	113.05	
				(1500 rad)	E on pleio	101.81	0.00	33.94	45.25	181	4.05	0.30-0.20
	(9:7)	Vs	(3:1)	Cherumodan x	0	39.00	0.00	16.00	21.00	76		
				Japan Violet	E on indep	32.00	10.69	24.94	8.31	76	34.78	
				(2000 rad)	E on pleio	42.75	0.00	14.25	19.00	76	0.75	0.90-0.80
	(27:37)	Vs	(9:7)	Cherumodan x	0	15.00	0.00	7.00	16.00	38		
				Japan Violet	E on indep	9.02	7.01	12.35	9.62	38	17.52	
				(5000 rad)	E on pleio	16.03	0.00	5.34	16.63	38	0.61	0.90-0.80
55.	Pau	Vs	Pn	Cherumodan x	0	26.00	51.00	4.00	63.00	144		
	(9:7)		(3:13)	Japan Violet	E on indep	15.19	65.81	11.81	51.19	144	18.92	
				(Normal)	E on pleio	20.25	60.75	6.75	56.25	144	5.13	0.20-0.10
	(27:37)	Vs	(3:13)	Cherumodan x	0	12.00	21.00	4.00	39.00	76		
				Japan Violet	E on indep	6.01	26.05	8.24	35.70	76	9.55	
				(2000 rad)	E on pleio	8.02	24.05	6.23	37.70	76	3.20	0.50-0.30
56.	Pau	Vs	Pin	Cherumodan x	0	77.00	0.00	29.00	38.00	144		
	(9:7)		(3:1)	Japan Violet	E on indep	60.75	20.25	47.25	15.75	144	63.08	
				(Normal)	E on pleio	81.00	0.00	27.00	36.00	144	0.46	0.95-0.90
	(27:37)	Vs	(3:1)	Cherumodan x	0	33.00	0.00	21.00	22.00	76		
				Japan Violet	E on indep	24.05	8.02	32.95	10.98	76	26.74	
				(2000 rad)	E on pleio	32.06	0.00	24.94	19.00	76	1.12	0.80-0.70

1	2		3	4	5	6	7	8	9	10	11
	(27:37)	Vs (9:7)	Cherumodan x	0	12.00	0.00	10.00	16.00	38		
			Japan Violet	E on indep	9.02	7.01	12.35	9.62	38	12.67	
			(5000 rad)	E on pleio	16.03	0.00	5.34	16.63	38	5.10	0.20-0.10
57.	Pau	Vs Ps	Cherumodan x	0	77.00	0.00	31.00	36.00	144		
	(9:7)	(3:1)	Japan Violet	E on indep	60.75	20.25	47.25	15.75	144	56.22	
			(Normal)	E on pleio	81.00	0.00	27.00	36.00	144	0.79	0.90-0.80
	(27:37)	Vs (3:1)	Cherumodan x	0	89.00	0.00	49.00	43.00	181		
			Japan Violet	E on indep	57.27	19.09	78.48	26.16	181	58.58	
			(1500 rad)	E on pleio	76.36	0.00	59.39	45.25	181	4.02	0.30-0.20
	(27:37)	Vs (3:1)	Cherumodan x	0	33.00	0.00	21.00	22.00	76		
			Japan Violet	E on indep	24.05	8.02	32.95	10.98	76	26.74	
			(2000 rad)	E on pleio	32.06	0.00	24.94	19.00	76	1.12	0.80-0.70
	(27:37)	Vs (9:7)	Cherumodan x	0	12.00	0.00	10.00	16.00	38		
			Japan Violet	E on indep	9.02	12.35	7.01	9.62	38	12.67	
			(5000 rad)	E on pleio	16.03	0.00	5.34	16.63	38	5.10	0.20-0.10
58.	Pau	Vs Pa	Cherumodan x	0	77.00	0.00	29.00	38.00	144		
	(9:7)	(3:1)	Japan Violet	E on indep	60.75	20.25	47.25	15.75	144	63.08	
			(Normal)	E on pleio	81.00	0.00	27.00	36.00	144	0.46	0.95-0.90
	(27:37)	Vs (3:1)	Cherumodan x	0	33.00	0.00	22.00	21.00	76		
			Japan Violet	E on indep	24.05	8.02	32.95	10.98	76	24.13	
			(2000 rad)	E on pleio	32.06	0.00	24.94	19.00	76	0.58	0.95-0.90
	(27:37)	Vs (9:7)	Cherumodan x	0	12.00	0.00	10.00	16.00	38		
			Japan Violet	E on indep	9.02	7.01	12.35	9.62	38	11.15	
			(5000 rad)	E on pleio	16.03	0.00	5.34	16.63	38	7.17	0.10-0.05
59.	Pn	Vs Pin	Cherumodan x	0	30.00	0.00	76.00	38.00	144		
	(3:13)	(3:1)	Japan Violet	E on indep	20.25	6.75	87.75	29.25	144	18.34	
			(Normal)	E on pleio	27.00	0.00	81.00	36.00	144	1.64	0.70-0.50
	(3:13)	Vs (3:1)	Cherumodan x	0	25.00	0.00	102.00	54.00	181		
			Japan Violet	E on indep	25.45	8.48	110.30	36.77	181	17.19	
			(1500 rad)	E on pleio	33.94	0.00	101.81	45.25	181	4.05	0.30-0.20

1	2			3	4	5	6	7	8	9	10	11
60.	Pn	Vs	Ps	Cherumodan x	0	31.00	0.00	78.00	35.00	144		
	(3:13)		(3:1)	Japan Violet	E on indep	20.25	6.75	87.75	29.25	144	14.67	
				(Normal)	E on pleio	27.00	0.00	81.00	36.00	144	0.73	0.90-0.80
	(3:13)	Vs	(3:1)	Cherumodan x	0	16.00	0.00	38.00	22.00	76		
				Japan Violet	E on indep	10.69	3.65	46.31	15.44	76	10.58	
				(2000 rad)	E on pleio	14.25	0.00	42.75	19.00	76	1.22	0.90-0.80
61.	Pn	Vs	Pa	Cherumodan x	0	31.00	0.00	75.00	38.00	144		
	(3:13)		(3:1)	Japan Violet	E on indep	20.25	6.75	87.75	29.25	144	16.93	
				(Normal)	E on pleio	27.00	0.00	81.00	36.00	144	1.15	0.90-0.80
	(3:13)	Vs	(3:1)	Cherumodan x	0	26.00	0.00	101.00	54.00	181		
				Japan Violet	E on indep	25.45	8.48	110.30	36.77	181	17.35	
				(1500 rad)	E on pleio	33.94	0.00	101.81	45.25	181	3.56	0.30-0.20
	(3:13)	Vs	(3:1)	Cherumodan x	0	16.00	0.00	39.00	21.00	76		
				Japan Violet	E on indep	10.69	3.66	46.31	15.44	76	9.45	
				(2000 rad)	E on pleio	14.25	0.00	42.75	19.00	76	0.75	0.90-0.80
62.	Pin	Vs	Ps	Cherumodan x	0	22.00	0.00	0.00	16.00	38		
	(9:7)		(9:7)	Japan Violet	E on indep	12.03	9.35	9.35	7.27	38	37.45	
				(5000 rad)	E on pleio	21.37	0.00	0.00	16.63	38	0.04	0.99
63.	Pin	Vs	Pa	Cherumodan x	0	22.00	0.00	0.00	16.00	38		
	(9:7)		(9:7)	Japan Violet	E on indep	12.02	9.35	9.35	7.28	38	37.45	
				(5000 rad)	E on pleio	21.37	0.00	0.00	16.63	38	0.04	0.99
64.	Ps	Vs	Pa	Cherumodan x	0	22.00	0.00	0.00	16.00	38		
	(9:7)		(9:7)	Japan Violet	E on indep	12.02	9.35	9.35	7.28	38	37.45	
				(5000 rad)	E on pleio	21.37	0.00	0.00	16.63	38	0.04	0.99

TABLE 44. **Comparative account on transformation in interrelationships of various characters occurred due to X-ray treatment of pollen grains used in crosses 2-4 relative to control**

Character combinations	Crosses				Remarks
	Ch x Jv Control (1)	Ch xJv 1500 rad (2)	Ch x Jv 2000 rad (3)	Ch x Jv 5000 rad (4)	
Px vs Pn/an/tst	I	I	I	I	Independent assortment
Psh vs an/tst	"	"	"	"	unchanged
Pl vs an/tst	"	"	"	"	
Pa vs an/tst	"	"	"	"	
Plm vs an/tst	"	"	"	"	
Pla vs an/tst	"	"	"	"	
Pjp vs an/tst	"	"	"	"	
Plg vs an/tst	"	"	"	"	

Pau vs an/tst	"	"	"	"	
Pn vs an/tst	"	"	"	"	
Pin vs an/tst	"	"	"	"	
Ps vs an/tst	"	"	"	"	
an vs tst	"	"	"	"	
Pl vs Pin/Ps/Pa	P	P	P	P	Pleiotropy unchanged
Pjp vs Pin/Ps/Pa	"	"	"	"	
Plg vs Ps/Pa	"	"	"	"	
Pau vs Ps	"	"	"	"	
Px vs Psh/Plm/Pla/ Pin/Ps/Pa/ Pn/Pa	P	P	P	I	P -- I Changed only with 5000 rad

Px vs Pjp/Pau	P	I	I	I	P – I Changed with all treatments
Px vs Pl/ Plg/ Pin/Pn	P	P	I	I	P – I Changed with 2000 and 5000 rad treatments
Pl vs Plm/Pla/ Pin/ Pau/ Pa	P	-	P	P	Unchanged with 2000 and 5000 rad & did not yield anything with 1500 rad treatment
Pau vs Pn	P	-	P	I	P – I
Pn vs Ps	P	I	P	I	"
Psh vs Pl/Pjp/ Plg/Pin/ Pa/Ps	L	L	L	P	L – P Linkage unchanged upto 2000 rad
Psh vs Ps	L	-	L	P	"
Pin vs Pa/Ps	L	L	L	P	"
Pin vs Ps	L	-	L	P	"

Psh vs Pn	L	L	I	I	L -- I
Plm vs Pn	L	P	P	I	L -- P -- I
Plm vs Pjp/Pau/ Ps	L	P	P	P	L -- P
Psh vs Pla	L	-	P	P	"
Plm vs Pin/Pa	L	-	P	P	"
Plm vs Pla	L	P	-	P	"
Plm vs Plg	L	-	-	P	"
Psh vs Pau	L	-	L	P	L -- P Linkages changed in 5000 rad only
Pla vs Plg/Pau/ Pin/Pa	"	"	"	"	"
Ps vs Pa	"	"	"	"	"

Pla vs Pn	L	P	L	I	L --- P/I
Pla vs Ps/Pjp	L	P	L	P	L --- P
Psh vs Plm	-	-	P	P	Non specific in control
Pl vs Pjp	-	P	P	P	"
Pl vs Plg	-	-	P	P	"
Pl vs Pau	-	-	P	P	"
Pjp vs Plg	-	P	P	P	"
Pjp vs Pau	-	-	-	P	"
Pjp vs Pn	-	P	P	I	"
Plg vs Pau	-	P	P	P	"

I - Independent assortment

L - Linkage

P - Pleiotropy

TABLE 45. Relative frequency of incidence of different interrelationshis of genes controlling 14 morphological characters studied in untreated and treated crosses of Cherumodan and Japan Violet

Interrelationship	Crosses			
	Ch x Jv Normal	Ch x Jv 1500 rad	Ch x Jv 2000 rad	Ch x Jv 5000 rad
1. Independent assortment	26	29	33	46
2. Linkage relations	27	7	18	Nil
3. Pleiotropy	30	32	36	45
4. Neither I/L/P	8	23	4	Nil
Total character combinations	91	91	91	91
Total characters	14	14	14	14

Ch - Cherumodan
Jv - Japan violet

combinations of 11 morphological characters showed highly significant Chi square values against the table value 7.82 for 3 d.f at 5% level (Table 42), which on estimation of linkage on minimum discrepancy showed linkage association of certain genes controlling the characters studied. Another 64 combinations of 12 morphological characters showed highly significant Chi square values against the table value 7.82 on independent assortment, which on estimation for linkage did not show linkage between the genes involved (Table 43). However, joint segregation of these characters on the assumption of pleiotropy in different crosses showed Chi square values lower than the table value 7.82 for 3 d.f at 5% level, thereby establishing pleiotropic relationship of genes governing the morphological characters concerned (Table 43). Observations on genic interrelationships of various morphological characters are detailed below under appropriate heads.

1. Leaf axil pigmentation vs leaf sheath pigmentation

Leaf axil gave 15:1 in 1-4 crosses and leaf sheath showed 3:1 in crosses 1 - 3 and 9:7 in cross 4 (Table 39a-1,2).

Joint segregation of these characters in M_2F_2 of cross 4 gave X^2 = 3.09 which is not significant against the table value 7.82 for 3

d.f at 5% level (Table 41-1), thereby showing independent assortment of the genes controlling these characters.

However, joint segregation of these characters in F_2 and M_2F_2 of crosses 1 - 3 gave X^2 = 54.76, 26.82 and 23.49 respectively which are significant against the table value (Table 43-1), indicating that the genes controlling these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy gave X^2 for linkage = 53.35, 6.01, 22.92 and P = 3.49, 1.94, 3.16 respectively, indicating that leaf axil is not linked with leaf sheath .

However, joint segregation on the assumption of pleiotropic association of these characters in F_2/M_2F_2 gave X^2 = 2.42,4.46 and 0.44 respectively in 1-3 crosses (Table 43-1), suggesting pleiotropic association of these characters. The gene identified as $\underline{Px_1}/\underline{Px_2}$ in leaf axil is pleiotropic for leaf shath, as Psh, showing differential pleiotropy.

2. Leaf axil pigmentation vs leaf blade pigmentation

Leaf axil showed 15:1 in 1-4 crosses and leaf blade gave 9:7 in crosses 1 & 2 and 27:37 in crosses 3 & 4 (Table 39a-1,3).

Joint segregation of these characters in M_2F_2 of crosses 3 & 4

gave X^2 = 6.47 and 1.45 respectively, which are not significant against the table value 7.82 for 3 d.f at 5% level (Table 41-3), thereby showing independent assortment of the genes controlling these characters.

Joint segregation of these characters in F_2/M_2F_2 of the crosses 1 & 2 gave X^2 = 23.42 and 11.76 respectively (Table 43-2) which are significant against the table value, indicating that the genes controlling these characters do not follow independent assortment. Estimation of linkage association gave X^2 for linkage = 22.76 and 10.11 and P = 7.99 and 7.96 respectively, indicating no linkage between them.

However, joint segregation on the assumption of pleiotropic association of these characters in F_2/M_2F_2 of crosses 1&2 gave X^2 = 1.31 and 2.42 respectively (Table 43-2), suggesting pleiotropic genic control of these characters. The pleiotropic gene could be identified as one of the duplicate factors- $\underline{Px_1}/\underline{Px_2}$, in leaf axil functioning as one of the complementaries in leaf blade ($\underline{Pl_{a/b}}$) or vice versa, showing differential pleiotropy.

3. Leaf axil pigmentation vs leaf margin pigmentation

Leaf axil pigmentation gave 15:1 in 1-4 crosses and leaf margin gave 3:1 in cross- 1 and gave 9:7 in crosses 2 - 4 (Table 39a-1,4).

Joint segregation of these characters in M_2F_2 of cross 4 gave X^2 = 2.41 which is not significant against the table value 7.82 for 3 d.f. at 5% level (Table 41-2), thereby showing independent assortment of these characters.

However, joint segregation of these characters in F_2/M_2F_2 of crosses 1,2 & 3 gave X^2 = 49.12, 19.12 and 15.36 respectively (Table 43-3), which are significant against the table value, indicating that the genes controlling these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy gave X^2 for linkage = 45.67, 15.62 and 10.52 with P = 3.51, 7.96 and 5.32 respectively, thereby indicating that leaf axil and leaf margin are not linked.

However, joint segregation on the assumption of pleiotropic association of these characters in F_2/M_2F_2 of the above crosses gave X^2 =2.52, 3.36 and 3.21 respectively in 1-3 crosses (Table 43-3), suggesting pleiotropic association of these characters. The pleiotropic gene could be identified as one of the duplicate factors ($\underline{Px_1}/\underline{Px_2}$) of leaf axil functioning as basic/complementary, $\underline{Plm}$ or $\underline{Plm_{a/b}}$, in leaf margin, showing differential pleiotropy.

4. Leaf axil pigmentation vs leaf tip pigmentation

Leaf axil pigmentation gave 15:1 in 1-4 crosses and leaf tip 3:1 in crosses 1 & 3 and 9:7 in crosses 2 & 4 (Table 39a-1,5).

Joint segregation of these characters in M_2F_2 of cross 4 gave X^2 = 2.24 which is not significant against the table value 7.82 for 3 d.f at 5% level (Table 41-4), thereby showing independent assortment of these characters.

However, joint segregation of these characters in M_2F_2 of crosses 1, 2 and 3 gave X^2 = 49.70, 22.41 and 20.04 respectively (Table 43-4) which are significant against the table value, indicating that the genes controlling these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy gave X^2 for linkage = 46.87, 16.56, 14.71 and P = 3.51, 7.96 and 7.23 respectively, thereby indicating that the gene for leaf axil is not linked with that of leaf tip.

However, joint segregation on the assumption of pleiotropic association of these characters in F_2/M_2F_2 of the above crosses gave X^2 = 1.19, 3.05 and 1.14 respectively in 1-3 crosses (Table 43-4), suggesting the possible pleiotropic association of these characters. The duplicate gene for leaf axil $\underline{Px_1}/\underline{Px_2}$, could be identified as pleiotropic for leaf tip being complementary, $\underline{Pla}$ or $\underline{Pla_{a/b}}$.

5. Leaf axil pigmentation vs junctura proper pigmentation

Leaf axil pigmentation gave 15:1 in crosses 1-4 and junctura

proper gave 9:7 in cross 1 and 27:37 in crosses 2 - 4 (Table 39a-1,6).

Joint segregation of these characters in M_2F_2 of crosses 2-4 gave X^2 = 5.41, 3.05 and 1.29, which are not significant against the table value 7.82 for 3 d.f at 5% level (Table 41-5), thereby showing independent assortment of these characters.

However, joint segregation of these characters in F_2 of cross 1 gave X^2 = 21.62 which is significant against the table value, indicating that the genes controlling these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy gave X^2 for linkage = 19.57 and P = 7.99, indicating no linkage between these characters in the control cross.

However, joint segregation on the assumption of pleiotropy of these characters in F_2 of the above cross 1 gave X^2 = 1.61 (Table 43-5), thereby suggesting pleiotropic association of these characters. One of the genes showed differential pleitropy by being duplicate factor in leaf axil $\underline{Px_1}/\underline{Px_2}$ and complementary gene, $\underline{Pjp_{a/b}}$, in junctura proper.

6. Leaf axil pigmentation vs ligule pigmentation

Leaf axil pigmentation gave 15:1 in crosses 1-4 and ligule gave 9:7 in crosses 1-3 and 27:37 in cross 4 (Table 39a-1,7).

Joint segregation of these characters in M_2F_2 of crosses 3 & 4 gave X^2 = 5.17 and 1.29, which are not significant against the table value 7.82 for 3 d.f at 5% level (Table 41-6), thereby showing independent assortment of these characters.

However, joint segregation of these characters in F_2/ M_2F_2 of crosses 1 and 2 gave X^2 = 28.16 and 12.48 which are significant against the table value, indicating that the genes controlling these characters do not follow independent assortment (Table 43-6). Estimation of linkage on minimum discrepancy gave X^2 for linkage = 25.31, 12.17 and P = 7.99, 7.96 respectively, showing no linkage between leaf axil and ligule pigmentation.

However, joint segregation on the assumption of pleiotropy of these characters in F_2/M_2F_2 of the crosses 1&2 gave X^2 = 4.03 and 0.17 (Table 43-6) thereby suggesting pleiotropic association of these characters. The pleiotropic gene functioned as duplicate in leaf axil $\underline{Px_1}/\underline{Px_2}$ and as one of the complementaries in ligule $\underline{Plg_{a/b}}$.

7. Leaf axil pigmentation vs auricle pigmentation

Leaf axil pigmentation gave 15:1 in crosses 1-4 and auricle gave 9:7 in cross 1 and 27:37 in the crosses 2 - 4 (Table 39-1,8).

Joint segregation of these characters in M_2F_2 of crosses 2 - 4 gave X^2 = 5.39, 2.71 and 2.46 respectively, which are not significant against the table value 7.82 for 3 d.f at 5% level (Table 41-7), thereby showing independent assortment of these characters.

However, joint segregation of these characters in F_2 of cross 1 gave X^2 = 21.85 (Table 43-7) which is significant against the table value, indicating that the genes controlling these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy gave X^2 for linkage = 20.35 with P = 7.99, indicating that leaf axil and auricle are not linked.

However, joint segregation on the assumption of pleiotropic association of these characters in F_2 of the above cross gave X^2 = 1.22 (Table 43-7), thereby suggesting pleiotropic association of these characters. The pleiotropic gene identified functioned as duplicate factor, $\underline{Px}_1/\underline{Px}_2$, in leaf axil and as complementary, $\underline{Pau}_{a/b}$, in auricle.

8. Leaf axil pigmentation vs nodal pigmentation

Leaf axil pigmentation gave 15:1 in crosses 1-4 and nodal pigmentation 3:13 in crosses 1-4 (Table 39a-1,9).

Joint segregation of these characters in F_2/ M_2F_2 of these crosses gave X^2 = 4.79, 2.68, 0.70 and 0.47 respectively, which are not significant against the table value 7.82 for 3 d.f at 5% level (Table 41-8), thereby showing independent assortment of genes controlling pigmentation in leaf axil and node.

9. Leaf axil pigmentation vs internode pigmentation

Leaf axil pigmentation gave 15:1 in crosses 1-4 and internode 3:1 in crosses 1-3 and 9:7 in cross 4 (Table 39a-1,10).

Joint segregation of these characters in M_2F_2 of cross 4 gave X^2 = 2.58 which is not significant against the table value 7.82 for 3 d.f. at 5% level (Table 41-9), thereby showing independent assortment of these characters.

However, joint segregation of these characters in F_2/M_2F_2 of crosses 1 - 3 gave X^2 = 51.00, 26.82 and 23.19 respectively (Table 43-8), which are significant against the table value, indicating that the genes controlling these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy gave X^2 for linkage = 49.79, 23.45, 22.27 and P = 3.50, 3.51, 3.16 respectively in crosses 1-3, showing that the genes for leaf axil and internode are not linked.

However, joint segregation on the assumption of pleiotropic association of these characters in F_2/M_2F_2 of the crosses 1-3 gave X^2 = 1.08, 4.46 and 0.70 respectively (Table 43-8 & 44), suggesting pleiotropic association of these characters. The pleiotropic gene functioned as duplicate ($\underline{Px}_1/\underline{Px}_2$) in leaf axil and as basic gene ($\underline{Pin}$) in internode, showing differential action.

10. Leaf axil pigmentation vs stigma pigmentation

Leaf axil pigmentation gave 15:1 in crosses 1-4 and stigma 3:1 in crosses 1-3 and 9:7 in cross 4 (Table 39a-1,11).

Joint segregation of these characters in M_2F_2 of cross 4 gave X^2 = 2.58, which is not significant against the table value 7.82 for 3 d.f at 5% level (Table 41-9), thereby showing independent assortment of these characters.

However, joint segregation of these characters in F_2/M_2F_2 of crosses 1-3 gave X^2 = 52.27, 29.64 and 22.28 respectively (Table 43-9), which are significant against the table value, indicating that the genes controlling these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy gave X^2 for linkage = 51.20, 29.36, 22.27 and P = 3.50, 3.51, 3.16 respectively in crosses 1-3, suggesting that the gene for leaf axil and stigma are not linked.

However, joint segregation on the assumption of pleiotropic association of these characters in F_2/M_2F_2 of crosses 1-3 gave X^2 = 1.33, 0.22, and 0.70 respectively (Table 43-9), suggesting pleiotropic association of these characters. The pleiotropic gene identified showed differential pleotropy by being duplicate, $\underline{Px_1}/\underline{Px_2}$, in leaf axil and basic, Ps, in the stigma.

11. Leaf axil pigmentation vs apiculus pigmentation

Leaf axil pigmentation gave 15:1 in crosses 1-4 and apiculus 3:1 in crosses 1-3 and 9:7 in cross 4 (Table 39a-1,12).

Joint segregation of these characters in M_2F_2 of cross 4 gave X^2= 2.57 which is not significant against the table value 7.82 for 3 d.f at 5% level (Table 41-11), thereby showing independent assortment of these characters.

However, joint segregation of these characters in F_2/M_2F_2 of crosses 1 - 3 gave X^2 = 51.00, 26.71 and 23.49 respectively (Table 43-10), which are significant against the table value, indicating that the genes controlling these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy gave X^2 for linkage = 49.79, 23.45, 29.92 and P = 3.50, 3.51, 3.16 respectively, suggesting that the genes for leaf axil and apiculus are not linked.

However, joint segregation on the assumption of pleiotropy in F_2/M_2F_2 of crosses 1-3 gave X^2 = 1.08, 3.70 and 0.44, respectively (Table 43-10), suggesting pleiotropic association of these characters. The pleiotropic gene is identified as duplicate $\underline{Px}_1/\underline{Px}_2$, in leaf axil and as basic gene, Pa, in apiculus.

12. Leaf axil pigmentation vs awning

Leaf axil pigmentation gave 15:1 in crosses 1-4 and awning 3:1 in crosses 1-4 (Table 39a-1,13).

Joint segregation of these characters in F_2/ M_2F_2 of these crosses gave X^2 =1.10, 2.45, 2.14 and 0.78 respectively, which are not significant against the table value 7.82 for 3 d.f at 5% level (Table 41-12), thereby showing independent assortment of genes controlling pigmentation in leaf axil and awning.

13. Leaf axil pigmentation vs tip-sterility

Leaf axil pigmentation gave 15:1 in crosses 1-4 and tip sterility 3:1 in crosses 1-4 (Table 39a-1, 14).

Joint segregation of these characters in F_2/ M_2F_2 of 1-4 crosses gave X^2 =1.11, 1.93, 0.65, 3.02 respectively, which are

not significant against the table value 7.82 for 3 d.f. at 5% level (Table 41-13), thereby showing independent assortment of genes controlling these characters.

14. Leaf sheath pigmentation vs leaf blade pigmentation

Leaf sheath pigmentation gave 3:1 in crosses 1-3 and 9:7 in cross 4 and leaf blade 9:7 in crosses 1 & 2 and 27:37 in crosses 3 & 4 (Table 39a-2,3).

However, joint segregation of these characters in F_2/M_2F_2 of crosses 1 - 4 gave X^2 = 53.62, 103.41, 32.97 and 13.72 respectively, which are significant against the table value, (Tables 42-1 & 43-11) indicating that the genes controlling these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy gave X^2 for linkage =53.26, 98.86, and 30.78 with c.o. = 2.52, 1.01 and 1.01 respectively in crosses 1-3 (Table 42-1), showing that the gene Psh for leaf sheath is linked with $Pl_{a/b}$ or $Pl_{a/b/c}$ for leaf blade (Table 42-1). Cross 4 gave X^2 for linkage = 10.97 with P = 1.65, showing no linkage between these characters.

However, joint segregation on the assumption of pleiotropy in M_2F_2 of cross 4 gave X^2 = 2.92 (Table 43-11), suggesting pleiotropic association of these characters. The pleiotropic gene identified as

$\underline{Psh_{a/b}}$ in leaf sheath functioned as one of the complementaries, $\underline{Pl_{a/b/c}}$ in leaf blade, showing differential action.

15. Leaf sheath pigmentation vs leaf margin pigmentation

Leaf sheath pigmentation gave 3:1 in crosses 1-3 and 9:7 in cross 4 and leaf margin gave 3:1 in cross 1 and 9:7 in crosses 2-4 (Table 39-2,4).

Joint segregation of these characters in F_2/M_2F_2 of these 4 crosses gave X^2 = 42.81, 128.60, 53.40, and 24.68 which are significant against the table value 7.82 for 3 d.f. at 5% level (Table 43-12 & Appendix II), thereby indicating that genes controlling these characters do not follow independent assortment. However, estimation of linkage on minimum discrepancy on F_2/M_2F_2 of crosses 1,2, 3 and 4 gave X^2 for linkage = 55.50, 28.92, 52.94 and 23.78 with P = 2.53, 1.01, 1.04 and 1.70 respectively, indicating that the genes controlling these characters are not linked.

However, joint segregation on the assumption of pleiotropy in M_2F_2 of the crosses 3 and 4 gave X^2 = 5.90 and 7.46 respectively (Table 43-12), suggesting pleiotropic association of these characters. The pleiotropic gene could be identified as $\underline{Psh}/\underline{Psh_{a/b}}$ for leaf sheath functioning as one of the complementary

genes $\underline{Plm}_{a/b}$ for leaf margin. Cross 1&2 showed neither independent assortment/linkage nor pleiotropy (Appendix II).

16. Leaf sheath pigmentation vs leaf tip pigmentation

Leaf sheath pigmentation gave 3:1 in crosses 1-3 and 9:7 in cross 4 and leaf tip gave 3:1 in crosses 1 & 3 and 9:7 in crosses 2 & 4 (Table 39a-2,5).

Joint segregation of these characters in F_2/M_2F_2 of these 1-4 crosses gave X^2 = 92.57, 149.62,82.83 and 20.80 respectively which are significant against the table value 7.82 for 3 d.f at 5% level (Tables 42-2,43-13 & Appendix II), thereby indicating that genes controlling these characters do not follow independent assortment. However, estimation of linkage on F_2 of the cross 1 on minimum discrepancy gave X^2 for linkage = 91.31 with c.o. = 6.74 (Table 42-2), indicating that the genes controlling these characters are linked (gene $\underline{Psh}$ of leaf sheath linked with $\underline{Pla}_{a/b}$ of leaf tip). In crosses 2, 3 and 4, estimation of linkage gave X^2 for linkage = 143.02, 51.01, and 24.33 with P = 1.03, 1.02, and 1.01 respectively, suggesting that the genes controlling these characters are not linked.

However, joint segregation on the assumption of pleiotropy in M_2F_2 of crosse 4 gave X^2 = 5.07 (Table 43-13)

suggesting pleiotropic association of the genes controlling these characters. The pleiotropic gene is identified as $\underline{Psh}_{a/b}$ in leaf sheath and as $\underline{Pla}_{a/b}$ in leaf tip. The cross 2&3 showed neither independent assortment/linkage nor pleiotropy between leaf sheath and leaf tip (Appendix II)

17. Leaf sheath pigmentation vs junctura proper pigmentation

Leaf sheath showed 3:1 in crosses 1-3 and 9:7 in cross 4 and junctura proper 9:7 in cross 1 and 27:37 in crosses 2-4 (Table 39a-2,6).

Joint segregation of these characters in F_2/M_2F_2 of the above crosses gave X^2 = 45.16, 73.35, 24.12 and 15.54 respectively, which are significant against the table value 7.82 for 3 d.f at 5% level (Tables 42-3 & 43-14), thereby indicating that genes controlling these characters do not follow independent assortment. However, estimation of linkage by minimum discrepancy in F_2 and M_2F_2 of the crosses 1-3 gave X^2 for linkage = 43.82, 70.76 and 22.47 with c.o. = 2.53 1.01 and 3.06 repectively (Table 42-3) showing linkages. Gene for leaf sheath pigmentation $\underline{Psh}$ is linked with $\underline{Pjp}_{a/b}$ or $\underline{Pjp}_{a/b/c}$ for junctura proper. Cross 4 on estimation of linkage gave X^2 for linkage = 15.14 with P = 1.71, indicating no linkage association.

However, joint segregation on the assumption of pleiotropic association of these characters in M_2F_2 of the cross 4 gave X^2 = 1.55, thereby suggesting pleiotropic association of these characters (Table 43-14). Pleiotropic gene is identified as $\underline{Psh_{a/b}}$ in leaf sheath and $\underline{Pjp_{a/b/c}}$ in junctura proper, showing non differential pleiotropy.

18. Leaf sheath pigmentation vs ligule pigmentation

Leaf sheath showed 3:1 in crosses 1-3 and 9:7 in cross 4 and ligule showed 9:7 in crosses 1-3 and 27:37 in cross 4 (Table 39, 2,7).

Joint segregation of these characters in F_2/M_2F_2 of crosses 1-4 gave X^2 = 59.73, 89.97, 34.76 and 15.54 respectively, which are significant against the table value 7.82 for 3 d.f at 5% level (Tables 42-4 and 43-15), thereby indicating that genes controlling these characters do not follow independent assortment. However, estimation of linkage on minimum discrepancy in F_2/M_2F_2 of the crosses 1-3, gave X^2 for linkage = 57.59, 87.19 and 33.72 with c.o. = 3.05, 4.08 and 3.56 respectively (Table 42-4) showing linkage between these characters. Gene for leaf sheath pigmentation $\underline{Psh}$ is linked with $\underline{Plg_{a/b}}$ for ligule pigmentation. Cross 4 gave X^2 for linkage = 15.14 and P = 1.71, indicating no linkage relation between these characters.

However, joint segregation on the assumption of pleiotropy in M_2F_2 of cross 4 gave X^2 = 1.55, suggesting pleiotropic association of these characters (Table 43-15). Pleiotropic gene is identified as gene $\underline{Psh}_{a/b}$ in leaf sheath and $\underline{Plg}_{a/b/c}$ in ligule showing non differential pleiotropy.

19. Leaf sheath pigmentation vs auricle pigmentation

Leaf sheath showed 3:1 in crosses 1-3 and 9:7 in cross 4, while auricle showed 9:7 in cross 1 and 27:37 in crosses 2-4 (Table 39-2,8).

Joint segregation of these characters in F_2/M_2F_2 of above crosses gave X^2 = 47.21, 72.37, 24.12 and 11.17 respectively, which are significant against the table value 7.82 for 3 d.f at 5% level (Tables 42-5, 43-16 and Appendix 1), thereby indicating that genes controlling these characters do not follow independent assortment.

However, estimation of linkage on minimum discrepancy in F_2/M_2F_2 of crosses 1 & 3 gave X^2 for linkage = 46.42 and 23.79 with c.o = 2.53 and 3.05 respectively (Table 42-5), thereby indicating that gene Psh is inked with $\underline{Pau}_{a/b}$ or $\underline{Pau}_{a/b/c}$. Crosses 2 & 4 gave X^2 for linkage = 79.26 and 9.13 with P = 0.99 and 1.63 respectively, indicating that these genes are not linked.

However, joint segregation on the assumption of pleiotropy in M_2F_2 of cross 4 gave X^2 = 7.16, thereby suggesting pleiotropic association of these characters (Table 43-16). Pleiotropic gene is identified as $\underline{Psh}_{a/b}$ in leaf sheath and $\underline{Pau}_{a/b/c}$ in auricle pigmentation showing non-differential pleiotropy. Cross 2 showed neither independent assortment/ linkage nor pleiotropy (Appendix II).

20. Leaf sheath pigmentation vs nodal pigmentation

Leaf sheath showed 3:1 in crosses 1-3 and 9:7 in the cross 4 and nodal pigmentation 3:13 in 1-4 crosses (Table 39a-2,9).

Joint segregation of these characters in M_2F_2 of crosses 3 and 4 gave X^2 = 3.14 and 5.62, which are not significant against the table value 7.82 for 3 d.f at 5% level (Table 41-14), thereby showing independent assortment of these characters. The crosses 1&2 gave X^2 = 9.31 and 12.84 (Table 42-6) thereby indicating that genes controlling these characters do not follow independent assortment. However, estimation of linkage on minimum discrepancy on F_2/M_2F_2 of crosses 1&2 gave X^2 for linkage = 8.57 & 7.15 with c.o = 10.56 and 15.15 respectively (Table 42-6), thereby indicating that the gene Psh for leaf sheath is linked with Pn for nodal pigmentation.

21. Leaf sheath pigmentation vs internode pigmentation

Leaf sheath and internode pigmentation showed 3:1 in 1-3 crosses and 9:7 in cross 4 (Table 39a-2,10).

Joint segregation of these characters in F_2/M_2F_2 of crosses 1-4 gave X^2 = 66.27, 230.20, 38.34 and 26.17 which are significant against the table value 7.82 for 3 d.f. at 5% level (Table 42-7 and 43-17), thereby indicating that these characters do not follow independent assortment. However, estimation of linkage on minimum discrepancy on F_2/M_2F_2 of the crosses 1,2 & 3 gave X^2 for linkage = 65.79, 224.69 and 37.43 with c.o = 12.82, 2.02, and 16.93 respectively (Table 42-7) showing that gene Psh for leaf sheath is linked with gene Pin for internode. Cross 4, however, did not show linkage on estimation with X^2 for linkage = 39.70 and P = 1.08.

However, joint segregation on the assumption of pleiotropy in M_2F_2 of cross 4 gave X^2 = 7.81, for 3 d.f at 5% level, thereby indicating pleiotropic association of these characters (Table 43-17). However, the X^2 value seems to be close to the table value though not significant. However, the pleiotropic gene is identified as $Psh_{a/b}$ for leaf sheath and $Pin_{a/b}$ for internode pigmentation being non-differential.

22. Leaf sheath pigmentation vs stigma pigmentation

Leaf sheath and stigma pigmentation showed 3:1 in crosses 1-3 and 9:7 in cross 4 (Table 39a-2,11).

Joint segregation of these characters in F_2/M_2F_2 of crosses 1-4 gave X^2 = 77.38, 115.98 38.34 and 26.17 respectively which are significant against the table value 7.82 for 3 d.f at 5% level (Table 42-8 and 43-18), thereby indicating that these characters do not follow independent assortment.

However, estimation of linkage on minimum discrepancy on F_2/M_2F_2 of crosses 1&3 gave X^2 for linkage = 77.05 and 37.43 with c.o = 10.00, 8.35 and 16.93 respectively (Table 42-8), indicating that gene Psh for leaf sheath is linked with gene Ps for stigma. Cross 2&4 did not show linkage on estimation with X^2 =115.31 39.70 and P = 1.84 and 1.08

However, joint segregation on the assumption of pleiotropy in M_2F_2 of cross 4 gave X^2 = 7.81, indicating, perhaps association of these characters (Table 43-18). Pleiotropic gene is identified as gene $Psh_{a/b}$ for leaf sheath and $Ps_{a/b}$ for stigma pigmentation showing non-differential pleiotropy. while cross 2 showed neither independent assortment, linkage/ pleiotropy Appendix II

23. Leaf sheath pigmentation vs apiculus pigmentation

Leaf sheath and apiculus pigmentation showed 3:1 in crosses 1-3 and 9:7 in cross 4 (Table 39-2,12).

Joint segregation of these characters in F_2/M_2F_2 of the crosses 1-4 gave X^2 = 73.68, 166.61, 39.88 and 26.17, which are significant against the table value 7.82 for 3 d.f at 5% level (Table 42-9 and 43-19), thereby indicating that these characters do not follow independent assortment.

However, estimation of linkage on minimum discrepancy on F_2/M_2F_2 of the crosses 1,2 & 3 gave X^2 for linkage = 73.20, 161.55, and 39.32 with c.o = 11.12, 3.35, and 15.74 respectively (Table 42-9), indicating that gene Psh for leaf sheath is linked with gene Pa for apiculus. Cross 4 did not give linkage on estimation with X^2 for linkage = 39.70 P = 1.08.

However, joint segregation on the assumption of pleiotropy of these characters in M_2F_2 of cross 4 gave X^2 = 7.81, indicating, perhaps, pleiotropic association of these characters (Table 43-19). The pleiotropic gene is identified as $Psh_{a/b}$ for leaf sheath and $Pa_{a/b}$ for the apiculus pigmentation.

24. Leaf sheath pigmentation vs awning

Leaf sheath showed 3:1 in crosses 1-3 and 9:7 in cross 4, and awning 3:1 in crosses 1-4 (Table 39-2,13).

Joint segregation of these characters in F_2/M_2F_2 of crosses 1-4 gave X^2 = 0.41, 5.41, 1.43 and 1.73 respectively, which are not significant against the table value 7.82 for 3 d.f at 5% level (Table 41-15), thereby showing independent assortment of these characters.

25. Leaf sheath pigmentation vs tip-sterility

Leaf sheath showed 3:1 in crosses 1-3 and 9:7 in cross 4, and tip-sterility showed 3:1 in crosses 1-4 (Table 39-2,14).

Joint segregation of these characters in F_2/M_2F_2 of the above crosses gave X^2 = 0.41 , 5.08, 0.30, and 3.15 which are not significant against the table value 7.82 for 3 d.f at 5% level (Table 41-16), thereby showing independent assortment of these characters.

26. Leaf blade pigmentation vs leaf margin pigmentation

Leaf blade showed 9:7 in crosses 1 & 2 and 27:37 in crosses 3 & 4. Leaf margin showed 3:1 in cross 1 and 9:7 in crosses 2-4 (Table 39-3,4).

Joint segregation of these characters in F_2/M_2F_2 of the crosses 1-4 gave X^2 = 98.44, 105.66, 46.87 and 23.30 respectively, which are significant against the table value 7.82 for 3 d.f at 5% level (Table 43-20 and Appendix II), thereby indicating that genes controlling these characters do not follow independent assortment.

However, estimation of linkage on minimum discrepancy on F_2/M_2F_2 of crosses 1-4 gave X^2 for linkage = 95.95, 100.97, 37.34, and 19.71 with P values 1.86, 1.84, 1.81 and 2.67, respetively thereby indicating that leaf blade and leaf margin are not linked.

However, joint segregation on the assumption of pleiotropy of these characters in F_2/M_2F_2 of crosses 1, 3 & 4 gave X^2 = 4.79, 2.28 and 0.62, suggesting pleiotropic association of these characters (Table 43-20). Pleiotropic gene is identified as $\underline{Pl}_{\underline{a/b}}$ or $\underline{Pl}_{\underline{a/b/c}}$ for leaf blade and $\underline{Plm}$ or $\underline{Plm}_{\underline{a/b}}$ for leaf margin pigmentation. Cross 2 did not show either independent assortment/linkage or pleiotropy (Appendix II).

27. Leaf blade pigmentation vs leaf tip pigmentation

Leaf blade showed 9:7 in crosses 1 & 2 and 27:37 in 3 & 4, while leaf tip showed 3:1 in crosses 1 & 3 and 9:7 in 2 & 4 (Table 39&3,5).

Joint segregation of these characters in F_2/M_2F_2 of crosses 1-4 gave X^2 = 82.75, 99.68, 40.67 and 20.36 respectively, which are significant against the table value 7.82 for 3 d.f at 5% level (Table 43-21), thereby indicating that genes controlling these characters do not follow independent assortment. However, estimation of linkage on minimum discrepancy on F_2/M_2F_2 of crosses 1-4 gave X^2 for linkage = 72.43, 91.71, 36.01 and 17.31 with P = 1.86, 1.77, 2.93 and 2.67 respectively, indicating that leaf blade and lef tip are not linked.

However, joint segregation on the assumption of pleiotropy of these characters in F_2/M_2F_2 of crosses 1,3 & 4 gave X^2 = 2.04, 5.90 and 1.71 suggesting pleiotropic association of these characters (Table 43-21). Pleiotropic gene is identified as $\underline{Pl_{a/b}}$ or $\underline{Pl_{a/b/c}}$ for leaf blade and $\underline{Pla}$ or $\underline{Pla_{a/b}}$ for leaf tip pigmentation. Cross 2 did not show either independent assortment/ linkage or pleiotropy (Appendix II).

28. Leaf blade pigmentation vs junctura proper pigmentation

Leaf blade showed 9:7 in crosses 1&2 and 27:37 in 3&4, while junctura proper showed 9:7 in cross 1 and 27:37 in crosses 2-4 (Table 39&3,6).

Joint segregation of these characters in F_2/M_2F_2 of crosses 1-4 gave X^2 =86.21, 140.04, 42.73 and 14.09 respectively, which are

significant against the table value 7.82 for 3 d.f at 5% level (Table 43-22 & Appendix II), thereby indicating that genes controlling these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy on F_2/M_2F_2 of crosses 1-4 gave X^2 for linkage = 82.80, 132.04, 40.75 and 135.22 with P = 1.59, 1.99, 2.44, 1.97 respectively showing that leaf blade is not linked with junctura proper.

However, joint segregation on the assumption of pleiotropy of these characters in M_2F_2 of crosses 2,3 & 4 gave X^2 = 7.24, 4.64, and 0.56 (Table 43-22) suggesting pleiotropic association of these characters. Gene $\underline{Pl_{a/b}}$ or $\underline{Pl_{a/b/c}}$ of leaf blade is pleiotropic to $\underline{Pjp_{a/b/c}}$ of leaf margin or vice-versa. Cross 1 did not show either independent assortment/linkage/pleiotropy (Appendix II).

29. Leaf blade pigmentation vs ligule pigmentation

Leaf blade pigmentation showed 9:7 in crosses 1&2 and 27:37 in 3&4, while ligule showed 9:7 in crosses 1-3 and 27:37 in cross 4 (Table 39a-3,7).

Joint segregation of these characters in F_2/M_2F_2 of crosses 1-4 gave X^2 = 61.59, 130.58, 17.41 and 19.43 respectively, which are significant against the table value 7.82 for 3 d.f at 5% level (Table

43-23 & Appendix II), thereby indicating that genes controlling these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy on F_2/M_2F_2 of crosses 1-4 gave X^2 for linkage = 59.67, 125.02, 67.03 and 20.36 with P = 1.39, 1.72, 2.86, 1.93 respectively, indicating that leaf blde is not linked with ligule.

However, joint segregation on the assumption of pleiotropy of these characters in M_2F_2 of crosses 3 & 4 gave X^2 = 6.65 and 1.77 suggesting pleiotropic association of these characters (Table 43-22). Gene $\underline{Pl_{a/b/c}}$ for leaf blade is pleiotropic to $\underline{Plg_{a/b}}$ or $\underline{Plg_{a/b/c}}$ for ligule or vice versa, while crosses 1&2 showed neither independent assortment/ linkage nor pleiotropy (Appendix II).

30. Leaf blade pigmentation vs auricle pigmentation

Leaf blade pigmentation showed 9:7 in crosses 1&2 and 27:37 in 3&4, while auricle showed 9:7 in cross 1 and 27:37 in crosses 2-4 (Table 39-3,8).

Joint segregation of these characters in F_2/M_2F_2 of crosses 1-4 gave X^2 = 87.69, 139.55 48.96 and 17.06 respectively,which are significant against the table value 7.82 for 3 d.f. at 5% level (Table 43-24 & Appendix II), thereby indicating that genes controlling these characters do not follow independent assortment.

Estimation of linkage on minimum discrepancy on F_2/M_2F_2 of the crosses 1-4 gave X^2 for linkage = 87.11, 2.32, 46.98 and 14.85 with P = 1.61, 1.61, 2.63, 2.13 respectively, indicating that leaf blade and auricle are not linked.

However, joint segregation on the assumption of pleiotropy of these characters in M_2F_2 of crosses 3 & 4 gave X^2 = 6.66 and 2.26 suggesting pleiotropic association of these characters (Table 43-24). Gene $\underline{Pl_{a/b}}$ or $\underline{Pl_{a/b/c}}$ for leaf blade is pleiotropic to $\underline{Pau_{a/b/c}}$ for auricle pigmentation or vice-versa, while crosses 1 & 2 showed neither independent assortment/ linkage nor pleiotropy (Appendix1).

31. Leaf blade pigmentation vs nodal pigmentation

Leaf blade pigmentation showed 9:7 in crosses 1 & 2 and 27:37 in 3 & 4, while nodal pigmentation showed 3:13 in 1-4 crosses (Table 39a-3,9).

Joint segregation of these characters in M_2F_2 of cross 4 gave X^2 = 4.62 ,which is not significant against the table value 7.82 for 3 d.f at 5% level (Table 41-17), thereby showing independent assortment of these characters.

However, joint segregation of these characters in F_2/M_2F_2 of

crosses 1-3 gave X^2 = 15.23, 18.19 and 8.23 which are significant against the table value suggesting that these characters do not follow independent assortment (Table 43-25). Estimation of linkage on minimum discrepancy on M_2F_2 of crosses 1-3 gave X^2 for linkage = 17.16, 13.63, 6.05 with P = 1.42, 1.45, -0.73 respectively, thereby indicating that leaf blade and node are not linked.

However, joint segregation on the assumption of pleiotropy of these characters in F_2/M_2F_2 of crosses 1-3 gave X^2 = 3.18, 7.27 and 3.30 suggesting pleiotropic association of these characters (Table 43-25). Gene $\underline{Pl_{a/b}}$ or $\underline{Pl_{a/b/c}}$ for leaf blade is pleiotropic to $\underline{Pn}$ for nodal pigmentation.

32. Leaf blade pigmentation vs internode pigmentation

Leaf blade pigmentation showed 9:7 in crosses 1 & 2 and 27:37 in 3 & 4, while internode pigmentation showed 3:1 in 1-3 and 9:7 in cross 4 (Table 39-3,10).

Joint segregation of these characters in F_2/M_2F_2 of the crosses 1-4 gave X^2 = 70.56, 103.38, 35.89 and 15.53 respectively, which are not significant against the table value 7.82 for 3 d.f at 5% level (Table 43-26), thereby indicating that genes controlling these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy on F_2/M_2F_2 of crosses 1-4 gave X^2

for linkage = 55.84, 100.59, 30.78 and 15.06 with P = 1.87, 1.89, 2.93, 2.67 respectively, indicating that leaf blade and inter node are not linked.

However, joint segregation on the assumption of pleiotropy of these characters in F_2/M_2F_2 of crosses 1-4 gave X^2 = 0.46, 2.89, 4.78, and 1.61, suggesting pleiotropic association of these characters (Table 43-26). Pleiotropic gene is identified as $\underline{Pl_{a/b}}$ or $\underline{Pl_{a/b/c}}$ for leaf blade and $\underline{Pin}$ or $\underline{Pin_{a/b}}$ for internode pigmentation.

33. Leaf blade pigmentation vs stigma pigmentation

Leaf blade pigmentation showed 9:7 in crosses 1 & 2 and 27:37 in 3 & 4, while stigma pigmentation showed 3:1 in crosses 1-3 and 9:7 in cross 4 (Table 39a-3,11).

Joint segregation of these characters in F_2/M_2F_2 of crosses 1-4 gave X^2 = 60.27, 59.75, 35.89 and 15.53 respectively, which are significant against the table value 7.82 for 3 d.f at 5% level (Table 43-27), thereby indicating that genes controlling these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy on F_2/M_2F_2 of crosses 1-4 gave X^2 for linkage = 66.14, 58.13, 30.78 and 9.30 with P = 1.86, 1.89, 2.93, 2.75 respectively, indicating that leaf blade and stigma are not linked.

However, joint segregation on the assumption of pleiotropy of these characters in F_2/M_2F_2 of crosses 1-4 gave X^2 = 0.08, 4.48, 4.78, and 1.61 respectively (Table 43-27), suggesting pleiotropic association of these characters. Gene $\underline{Pl_{a/b}}$ or $\underline{Pl_{a/b/c}}$ for leaf blade is pleiotropic to $\underline{Ps}$ or $\underline{Ps_{a/b}}$ for stigma or vice-versa.

34. Leaf blade pigmentation vs apiculus pigmentation

Leaf blade pigmentation showed 9:7 in crosses 1 & 2 and 27:37 in 3 & 4, while apiculus pigmentation showed 3:1 in crosses 1-3 and 9:7 in cross 4 (Table 39&3,12).

Joint segregation of these characters in F_2/M_2F_2 of the crosses 1-4 gave X^2 = 70.56, 99.00, 32.98 and 15.55 respectively, which are significant against the table value 7.82 for 3 d.f. at 5% level (Table 43-28), thereby indicating that genes controlling these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy on F_2/M_2F_2 of the crosses 1-4 gave X^2 for linkage = 61.33, 100.59, 30.78, and 11.04 with P = 1.86, 1.89, 2.93 and 2.65 respectively, thereby indicating that leaf blade is not linked with apiculus.

However, joint segregation on the assumption of pleiotropy of these characters in F_2/M_2F_2 of crosses 1-4 gave X^2 = 0.46, 2.41 3.84

and 1.61 respectively, suggesting pleiotropic association of these characters (Table 43-28). Pleiotropic gene is identified as gene $\underline{Pl}_{\underline{a/b}}$ or $\underline{Pl}_{\underline{a/b/c}}$ for leaf blade being pleiotropic to $\underline{Pa}$ or $\underline{Pa}_{\underline{a/b}}$ for apiculus pigmentation showing non-differential pleiotropy.

35. Leaf blade pigmentation vs awning

Leaf blade showed 9:7 in crosses 1 & 2 and 27:37 in 3 & 4 and awning showed 3:1 in crosses 1 - 4 (Table 39a-3,13).

Joint segregation of these characters in F_2/M_2F_2 of the above crosses gave X^2 = 0.07, 4.32 3.97 and 2.76 which are not significant against the table value 7.82 for 3 d.f. at 5% level (Table 41-18), thereby showing independent assortment of these characters.

36. Leaf blade pigmentation vs tip-sterility

Leaf blade showed 9:7 in crosses 1 & 2 and 27:37 in 3 & 4, while tip-sterility showed 3:1 in 1-4 crosses (Table 39a-3,14).

Joint segregation of these characters in F_2/M_2F_2 of the above crosses gave X^2 = 0.24, 4.06, 1.97 and 4.41 which are not significant against the table value 7.82 for 3 d.f at 5% level (Table 41-19), thereby showing independent assortment of these characters.

37. Leaf margin pigmentation vs leaf tip pigmentation

Leaf margin showed 3:1 in cross 1 and 9:7 in 2-4, while leaf tip showed 3:1 in crosses 1 & 3 and 9:7 in crosses 2 & 4 (Table 39 & 4,5).

Joint segregation of these characters in F_2/M_2F_2 of crosses 1-4 gave X^2 = 144.64, 175.51 61.05 and 22.62 respectively, which are significant against the table value 7.82 for 3 d.f. at 5% level (Table 43-29 Appendix II), thereby indicating that genes controlling these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy on F_2 of the cross 1 gave X^2 for linkage = 141.35 with c.o. = 5.13 (Table 42-10), while other crosses 2-4 on estimation of linkage gave X^2 for linkage = 168.83, 53.36 and 40.45 with P = 1.96, 1.83, 1.83 respectively thereby indicating no linkage between leaf margin and leaf tip.

However, joint segregation on the assumption of pleiotropy of these characters in M_2F_2 of crosses 2 & 4 gave X^2 = 3.34 and 5 15 suggesting pleiotropic association of these characters (Table 43-29). Gene Plm or $Plm_{a/b}$ for leaf margin is pleiotropic to Pla or $Pla_{a/b}$ for leaf tip or vice-versa, while cross 3 showed neither independent assortment/ linkage nor pleiotropy (Appendix II).

38. Leaf margin pigmentation vs junctura proper pigmentation

Leaf margin showed 3:1 in cross 1 and 9:7 in 2-4, while junctura proper showed 3:1 in cross 1 and 27:37 in crosses 2-4 (Table 39-4,6).

Joint segregation of these characters in F_2/M_2F_2 of crosses 1-4 gave X^2 = 84.74, 85.71, 32.65 and 25.80 respectively, which are significant against the table value 7.82 for 3 d.f. at 5% level (Tables 42-11 and 43-30), thereby indicating that genes controlling these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy on F_2 of cross 1 gave X^2 for linkage = 81.35 with c.o. = 1.51 (Table 42-11). The gene Plm for leaf margin is linked with $\underline{Pjp_{a/b}}$ for junctura proper pigmentation, while other crosses 2-4 on estimation of linkage gave X^2 for linkage = 84.49, 30.53 and 22.27 respectively with P = 1.93, 1.83 and 1.80 indicating no linkage between leaf margin and junctura proper.

However, joint segregation on the assumption of pleiotropy of these characters in M_2F_2 of crosses 2-4 gave X^2 = 4.92, 3.84 and 0.74, suggesting pleiotropic association of these characters (Table 43-30). Gene $\underline{Plm_{a/b}}$ for leaf margin is pleiotropic to $\underline{Pjp_{a/b/c}}$ for junctura proper pigmentation or vice-versa.

39. Leaf margin pigmentation vs ligule pigmentation

Leaf margin showed 3:1 in cross 1 and 9:7 in 2-4, while ligule showed 9:7 in crosses 1-3 and 27:37 in cross 4 (Table 39a-4,7).

Joint segregation of these characters in F_2/M_2F_2 of crosses 1-4 gave X^2 =102.61, 101.20, 42.69 and 25.80 respectively, which are significant against the table value 7.82 for 3 d.f at 5% level (Tables 42-12, 43-31 & Appendix II), thereby indicating that genes controlling these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy on F_2 of the cross 1 gave X^2 for linkage = 99.79 with c.o. =15.74 (Table 42-12). The gene Plm for leaf margin is closely linked with $Plg_{a/b}$ for ligule. Other crosses 2-4 on estimation of linkage gave X^2 for linkage = 97.96,39.87 and 22.27 with P = 1.64, 1.82 and 1.80 respectively, thereby indicating that leaf margin and ligule are not linked.

However, joint segregation on the assumption of pleiotropy of these characters in M_2F_2 of cross 4 gave X^2 = 0.74 suggesting pleiotropic association of these characters (Table 43-31). Pleiotropic gene is identified as $\underline{Plm_{a/b}}$ for leaf margin and as $\underline{Plg_{a/b}}$ for ligule pigmentation, while crosses 2 & 3 showed neither independent assortment/ linkage nor pleiotropy (Appendix II).

40. Leaf margin pigmentation vs auricle pigmentation

Leaf margin showed 3:1 in cross 1 and 9:7 in 2-4, while auricle showed 9:7 in cross 1 and 27:37 in crosses 2-4 (Table 39-4,8).

Joint segregation of these characters in F_2/M_2F_2 of crosses 1-4 gave X^2 = 87.71, 112.10, 32.66 and 33.54 respectively, which are significant against the table value 7.82 for 3 d.f at 5% level (Table 42-13 and 43-32), thereby indicating that genes controlling these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy on F_2 of cross 1 gave X^2 for linkage = 84.87 with c. o. = 1.01 (Table 42-13). The gene Plm for leaf margin is linked with $\underline{Pau}_{a/b}$ for auricle, while other crosses 2-4 on estimation of linkage gave X^2 for linkage = 99.76, 30.53 and 14.82 with P = 1.94, 1.83 and 1.68 respectively, indicating that leaf margin and auricle are not linked.

However, joint segregation on the assumption of pleiotropy of these characters in M_2F_2 of crosses 2-4 gave X^2 = 3.98, 3.84 and 1.87 respectively, suggesting pleiotropic association of these characters (Table 43-32). Pleiotropic gene is identified as $\underline{Plm}_{a/b}$ for leaf margin and $\underline{Pau}_{a/b/c}$ for auricle pigmentation.

41. Leaf margin vs nodal pigmentation

Leaf margin showed 3:1 in cross 1 and 9:7 in crosses 2-4, while nodal pigmentation showed 3:13 in all the above crosses (Table 39a-4,9).

Joint segregation of these characters in M_2F_2 of cross 4 gave X^2 = 5.03 which is not significant against the table value 7.82 for 3 d.f at 5% level (Table 41-20), thereby showing independent assortment of these characters.

However, joint segregation of these characters in F_2/M_2F_2 of the crosses 1 - 3 gave X^2 =22.47, 8.64 and 14.26respectively which are significant against the table value, thereby indicating that the genes controlling these characters do not follow independent assortment (Tables 42-14 and 43-33). Estimation of linkage on minimum discrepancy gave X^2 for linkage = 19.69 with c.o. = 3.04 (Table 42-14), thereby indicating that the gene Plm is linked with Pn for nodal pigmentation, while the crosses 2&3 gave X^2 for linkage = 3.83 and 8.61 with P = 1.13 and 1.67, thereby indicating that they are not linked.

However, joint segregation on the assumption of pleiotropy of these characters in M_2F_2 of the crosses 2&3 gave X^2 =7.62 and 2.47

respectively, suggesting pleiotropic association of these characters (Table 43-33). $\underline{Plm}_{a/b}$ for leaf margin is pleiotropic to Pn for nodal pigmentation or vice-versa.

42. Leaf margin vs internode pigmentation

Leaf margin showed 3:1 in cross 1 and 9:7 in crosses 2-4, while internode pigmentation showed 3:1 in crosses 1-3 and 9:7 in cross 4 (Table 39&4,10).

Joint segregation of these characters in F_2/M_2F_2 of the above crosses gave X^2 = 121.09, 144.40, 53.40 and 22.63 , which are significant against the table value 7.82 for 3 d.f at 5% level (Tables 42-15 and 43-34), thereby indicating that genes controlling these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy on M_2F_2 of cross 1 gave X^2 for linkage = 118.57 with c.o. = 6.19 (Table 42-15), thereby showing that gene Plm for leaf margin is linked with gene Pin for internode, while the crosses 2,3&4 gave X^2 for linkage = 147.06, 51.08, 40.45 with P = 1.86, 1.73 and 1.83 respectively, thereby indicating that they are not linked.

However, joint segregation on the assumption of pleiotropic association of these characters in M_2F_2 of the crosses 3&4 gave X^2

= 5.90 and 5.15 respectively (Table 43-34), suggesting pleiotropic association of these characters. Gene $\underline{Plm_{a/b}}$ for leaf margin is pleiotropic to Pin or $\underline{Pin_{a/b}}$ for internode pigmentation or vice-versa, while cross 2 did not show either independent assortment/linkage/pleiotropy (Appendix II).

43. Leaf margin vs stigma pigmentation

Leaf margin showed 3:1 in cross 1 and 9:7 in crosses 2-4, while stigma pigmentation showed 3:1 in crosses 1-3 and 9:7 in cross 4 (Table 39a-4,11).

Joint segregation of these characters in F_2/M_2F_2 of the above crosses gave X^2 = 97.98, 92.28, 53.40 and 19.34 , which are significant against the table value 7.82 for 3 d.f at 5% level (Table 42-16 and 43-35), thereby indicating that genes controlling these characters do not follow independent assortment.

However, joint segregation of these characters in M_2F_2 of cross 1 on estimation of linkage on minimum discrepancy gave X^2 for linkage = 95.60 with c.o. = 8.35 (Table 42-16), thereby indicating that gene Plm is linked with Ps for stigma. The crosses 2,3&4 gave X^2 for linkage = 88.81,46.81 and 40.45 with P = 1.88, 1.73 and 1.83 thereby indicating that they are not linked.

However, joint segregation on the assumption of pleiotropy in M_2F_2 of crosses 2-4 gave X^2 = 4.48, 5.90 and 5.15 respectively, suggesting pleiotropic association of these characters (Table 43-35). Gene Plm or $Plm_{a/b}$ for leaf margin is pleiotropic to gene Ps or $Ps_{a/b}$ for stigma pigmentation or vice-versa.

44. Leaf margin vs apiculus pigmentation

Leaf margin showed 3:1 in cross 1 and 9:7 in crosses 2-4, while apiculus showed 3:1 in crosses 1-3 and 9:7 in cross 4 (Table 39a-4,12).

Joint segregation of these characters in F_2/M_2F_2 of the above crosses gave X^2 = 111.60, 138.31, 53.40 and 16.08 which are significant against the table value 7.82 for 3 d.f at 5% level (Tables, 42-17 and 43-36), thereby indicating that genes controlling these characters do not follow independent assortment. Estimation of linkage by minimum discrepancy on M_2F_2 of the cross 1 gave X^2 for linkage = 109.09 with c.o. = 7.80 (Table 42-17), thereby indicating that gene Plm for leaf margin and Pa for apiculus are linked. The crosses 2,3&4 gave X^2 for linkage = 40.45,17.72 and 46.81 with P = 1.85, 1.73 and 1.83 respectively, thereby indicating that they are not linked.

However, joint segregation on the assumption of pleiotropy of these characters in M_2F_2 of crosses 3&4 gave X^2 = 5.90 and 2.82 respectively, suggesting pleiotropic association of these characters (Table 43-36). Gene Plm or $Plm_{a/b}$ is pleiotropic to Pa or $Pa_{a/b}$ for apiculus pigmentation or vice-versa, while cross 2 showed neither independent assortment/linkage nor pleiotropy (Appendix II).

45. Leaf margin vs awning

Leaf margin showed 3:1 in cross 1 and 9:7 in crosses 2-4, while awning showed 3:1 in all the above crosses (Table 39a-4,13).

Joint segregation of these characters in F_2/M_2F_2 of the above crosses gave X^2 = 2.42, 5.26, 3.50 and 1.01 respectively, which are not significant against the table value 7.82 for 3 d.f at 5% level (Table 41-21), thereby showing independent assortment of these characters.

46. Leaf margin vs tip-sterility

Leaf margin showed 3:1 in cross 1 and 9:7 in crosses 2-4, while tip-sterility showed 3:1 in all the above crosses (Table 39a-4,14).

Joint segregation of these characters in F_2/M_2F_2 of the above crosses gave X^2 = 7.12, 6.32, 2.56 and 3.79 which are not significant

against the table value 7.82 for 3 d.f at 5% level (Table 41-22), thereby showing independent assortment of these characters.

47. Leaf tip vs junctura proper pigmentation

Leaf tip showed 3:1 in cross 1 & 3 and 9:7 in crosses 2 & 4, while junctura proper showed 9:7 in cross 1 and 27:37 in crosses 2-4 (Table 39a-5,6).

Joint segregation of these characters in F_2/M_2F_2 of the above crosses gave X^2 = 77.91, 87.46, 29.58 and 22.68, which are significant against the table value 7.82 for 3 d.f at 5% level (Table 42-18 and 43-37), thereby indicating that genes controlling these characters do not follow independent assortment. Estimation of linkage by minimum discrepancy on F_2/M_2F_2 of the crosses 1 & 3 gave X^2 for linkage = 69.97 and 28.41 with c.o. = 1.51 and 2.53 respectively (Table 42-18), thereby indicating that gene Pla or $Pla_{a/b}$ for leaf tip is linked with $Pjp_{a/b}$ or $Pjp_{a/b/c}$ for junctura proper pigmentation. The crosses 2 & 4 gave X^2 for linkage = 82.15 and 19.71 with P = 1.93 and 1.77, thereby indicating that they are not linked.

However, joint segregation on the assumption of pleiotropy of these characters in M_2F_2 of the crosses 2&4 gave X^2 = 4.92 and 0.20

respectively, suggesting pleiotropic association of these characters (Table 43-37). The gene $\underline{Pla_{a/b}}$ for leaf tip is pleiotropic to $\underline{Pjp_{a/b/c}}$ for junctura proper or vice-versa.

48. Leaf tip vs ligule pigmentation

Leaf tip showed 3:1 in crosses 1 & 3 and 9:7 in crosses 2 & 4. Ligule showed 9:7 in crosses 1-3 and 27:37 in cross 4 (Table 39-5,7).

Joint segregation of these characters in F_2/M_2F_2 of the above crosses gave X^2 = 63.31, 89.81, 42.94 and 22.68, which are significant against the table value 7.82 for 3 d.f at 5% level (Tables 42-19 and 43-38), thereby indicating that genes controlling these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy on F_2/M_2F_2 of the crosses 1 & 3 gave X^2 for linkage = 60.58 and 39.98 with c.o. = 4.08 and 3.05 respectively (Table 42-19), thereby indicating that gene $\underline{Pla}$ or $\underline{Pla_{a/b}}$ for leaf tip is linked with $\underline{Plg_{a/b}}$ for ligule pigmentation. The crosses 2 & 3 gave X^2 for linkage = 89.56 and 19.71 with P = 1.58 and 1.77, thereby indicating that they are not linked.

However, joint segregation on the assumption of pleiotropy in M_2F_2 of cross 4 gave X^2 = 0.20 suggesting pleiotropic association of these characters (Table 43-38). Gene $\underline{Pla_{a/b}}$ for leaf tip is

pleiotropic to $\underline{Plg}_{a/b/c}$ for ligule pigmentation or vice-versa, while cross 2 showed either linkage or pleiotropy (Appendix II).

49. Leaf tip pigmentation vs auricle pigmentation

Leaf tip showed 3:1 in cross 1 & 3 and 9:7 in crosses 2 & 4. Auricle showed 9:7 in cross 1 and 27:37 in crosses 2-4 (Tables 39-5,8).

Joint segregation of these characters in F_2/M_2F_2 of the above crosses gave X^2 = 50.48, 94.26, 29.58 and 16.85 respectively, which are significant against the table value 7.82 for 3 d.f at 5% level (Tables 42-20 and 43-39), thereby indicating that genes controlling these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy on F_2/M_2F_2 of crosses 1&3 on gave X^2 for linkage = 49.10 and 28.41 with c.o. = 8.35 and 2.53 respectively (Table 42-20), thereby indicating that the gene $\underline{Pla}$ for leaf tip is linked with $\underline{Pau}_{a/b}$ or $\underline{Pau}_{a/b/c}$ for auricle pigmentation, while crosses 2&3 gave X^2 for linkage = 90.52, and 12.75 with P = 1.84 and 1.67, thereby indicating that they are not linked.

However, joint segregation on the assumption of pleiotropy of these characters in M_2F_2 of cross 4 gave X^2 = 2.45 suggesting pleiotropic association of these characters (Table 43-39).

Pleiotropic gene could be identified as $\underline{Plm}_{\underline{a/b}}$ for leaf margin and $\underline{Pau}_{\underline{a/b/c}}$ for auricle pigmentation, while cross 2 showed neither independent assortment/linkage nor pleiotropy (Appendix II).

50. Leaf tip pigmentation vs nodal pigmentation

Leaf tip showed 3:1 in cross 1 and 9:7 in crosses 2-4 and nodal pigmentation showed 3:13 in 1-4 crosses (Table 39-5,9).

Joint segregation of these characters in M_2F_2 of cross 4 gave X^2 = 3.99 which is not significant against the table value 7.82 for 3 d.f at 5% level (Table 41-23), thereby indicating independent assortment of genes controlling these characters.

However, joint segregation of these characters in F_2/M_2F_2 of crosses 1-3 gave X^2= 11.41, 11.08, 11.78 which are significant (Tables 42-21 and 43-40), thereby indicating that these genes do not follow independent assortment. Estimation of linkage on minimum discrepancy in 1 & 3 crosses gave X^2 for linkage = 9.42 and 10.08 with c.o. = 11.12 and 4.61 respectively (Table 42-21), thereby indicating that gene $\underline{Pla}$ or $\underline{Pla}_{\underline{a/b}}$ for leaf tip is linked with $\underline{Pn}$ for nodal pigmentation.

However, joint segregation on the assumption of pleiotropy in

M_2F_2 of the cross 2 gave X^2 = 7.61 suggesting the pleiotropic association of these characters (Table 43-40). Gene $\underline{Pla_{a/b}}$ for leaf tip is pleiotropic to Pn for the nodal pigmentation or vice-versa.

51. Leaf tip pigmentation vs internode pigmentation

Leaf tip showed 3:1 in crosses 1 & 3 and 9:7 in crosses 2 & 4. Internode showed 3:1 in crosses 1-3 and 9:7 in cross 4 (Table 39 & 5,10).

Joint segregation of these characters in M_2F_2 of the above crosses gave X^2 = 82.07, 144.40, 69.96 and 18.03 which are significant against the table value 7.82 for 3 d.f at 5% level (Table 42-22, 43-41 & Appendix 1), thereby indicating that genes controlling these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy on F_2/M_2F_2 of the crosses 1 & 3 gave X^2 for linkage = 31.00 and 68.21 with c.o. = 12.82 and 8.90 respectively (Table 42-22), thereby indicating that gene Pla for leaf tip is linked with Pin for internode pigmentation. The crosses 2 & 4 gave X^2 for linkage = 144.98 and 9.07 with P = 1.85 and 1.45 respectively, thereby indicating that they are not linked.

However joint segregation on the assumption of pleiotropy in M_2F_2 of cross 4 gave X^2 = 3.32 suggesting pleiotropic association

of these characters (Table 43-41) Gene Pla $_{a/b}$ for leaf tip is pleiotropic to Pin$_{a/b}$ for internode or vice-versa, while cross 2 did not show either independent assortment/ linkage or pleiotropy (Appendix II).

52. Leaf tip pigmentation vs stigma pigmentation

Leaf tip showed 3:1 in crosses 1 & 3 and 9:7 in crosses 2 & 4. Stigma showed 3:1 in crosses 1-3 and 9:7 in cross 4 (Table 39a-5,11).

Joint segregation of these characters in F_2/M_2F_2 of the above crosses gave X^2 = 94.37, 92.28 , 60.23 and 18.03 which are significant against the table value 7.82 for 3 d.f at 5% level (Table 42-23 and 43-42), thereby indicating that genes controlling these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy on F_2/M_2F_2 of the crosses 1 & 3 gave X^2 for linkage = 93.44 and 58.48 with c.o. = 9.45 and 11.12 respectively (Table 42-23), thereby indicating that the gene Pla for leaf tip is linked with Ps for stigma. The crosses 2 & 4 gave X^2 for linkage = 98.00 and 20.31 with P = 1.85 and 1.50, thereby indicating that they are not linked.

However, joint segregation on the assumption of pleiotropy in

M_2F_2 of crosses 2 & 4 gave X^2 = 4.48 and 3.32 (Table 43-42) suggesting pleiotropic association of these characters with the gene $\underline{Pla_{a/b}}$ for leaf tip being pleiotropic to $\underline{Ps}$ or $\underline{Ps_{a/b}}$ for stigma or vice-versa.

53. Leaf tip pigmentation vs apiculus pigmentation

Leaf tip showed 3:1 in crosses 1 & 3 and 9:7 in crosses 2 & 4 and apiculus showed 3:1 in crosses 1-3 and 9:7in cross 4 (Table 39 & 5,12).

Joint segregation of these characters in F_2/M_2F_2 of the above crosses gave X^2 = 82.07, 144.40 62.25 and 24.03 respectively, which are significant against the table value 7.82 for 3 d.f at 5% level (Tables 42-24 and 43-43), thereby indicating that genes controlling these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy on F_2/M_2F_2 of the crosses 1 & 3 gave X^2 for linkage : 81.00 and 60.84 with c.o. = 12.82 and 9.45 respectively (Table 42-24), thereby indicating that the gene $\underline{Pla}$ for leaf tip is linked with $\underline{Pa}$ for apiculus. The crosses 2 & 3 gave X^2 for linkage = 144.98 and 20.31 with P values 1.85 and 1.50, thereby indicating that they are not linked.

However, joint segregation on the assumption of pleiotropy in M_2F_2 of cross 4 gave X^2 = 6.32 suggesting pleiotropic association

of these characters (Table 43-43). Gene $\underline{Pla_{a/b}}$ for leaf tip is pleiotropic to $\underline{Pa_{a/b}}$ for apiculus or vice-versa, while cross 2 showed neither independent assortment, linkage nor pleiotropy (Appendix II).

54. Leaf tip pigmentation vs awning

Leaf tip showed 3:1 in crosses 1 & 3 and 9:7 in crosses 2 & 4, while awning showed 3:1 in all crosses (Table 39-5,13). Joint segregation of these characters in F_2/M_2F_2 of 1-4 crosses gave X^2 = 1.01, 4.90, 2.80and 0.53 respectively, which are not significant against the table value 7.82 for 3 d.f at 5% level (Table 41-24), thereby showing independent assortment of these characters.

55. Leaf tip pigmentation vs tip-sterility

Leaf tip showed 3:1 in crosses 1 & 3 and 9:7 in crosses 2 & 4, while tip-sterility showed 3:1 in all the crosses (Table 39-5,14). Joint segregation of these characters in F_2/M_2F_2 of the above crosses gave X^2 = 6.93, 6.52, 1.33 and 3.21 which are not significant against the table value 7.82 for 3 d.f at 5% level (Table 41-25), thereby showing independent assortment of these characters.

56. Junctura proper pigmentation vs ligule pigmentation

Junctura proper showed 9:7 in cross 1 and 27:37 in crosses 2-4, while ligule showed 9:7 in crosses 1-3 and 27:37 in cross 4 (Table 3~~&~~6,7).

Joint segregation of these characters in F_2/M_2F_2 of 1-4 crosses gave X^2 = 65.14, 112.15, 57.05 and 11.78 respectively, which are significant against the table value 7.82 for 3 d.f at 5% level (Table 43-44), thereby indicating that genes controlling these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy on F_2/M_2F_2 of crosses 1-4 gave X^2 for linkage = 62.31, 112.45, 50.49, and 11.54 with P = 1.53, 3.15, 2.94 and 1.81 respectively, thereby indicating that genes controlling these characters are not linked.

However, joint segregation on the assumption of pleiotropy in M_2F_2 of crosses 2-4 gave X^2 = 0.64, 2.78 and 0.15, suggesting pleiotropic association of these characters (Table 43-44). Gene $\underline{Pjp}_{\underline{a/b/c}}$ for junctura proper pigmentation is pleiotropic to $\underline{Plg}_{\underline{a/b}}$ or $\underline{Plg}_{\underline{a/b/c}}$ for ligule pigmentation or vice-versa, while cross 1 showed neither independent assortment, linkage nor pleiotropy (Appendix II).

57. Junctura proper pigmentation vs auricle pigmentation

Junctura proper showed 9:7 in cross 1 and 27:37 in crosses 2-4, while auricle showed 9:7 in cross 1 and 27:37 in crosses 2-4 (Table 39a-6,8).

Joint segregation of these characters in F_2/M_2F_2 of 1-4 crosses gave X^2 = 79.37, 139.01, 47.78 and 19.84 respectively, which are significant against the table value 7.82 for 3 d.f at 5% level (Table 43-45 and Appendix 1), thereby indicating that genes controlling these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy on F_2/M_2F_2 of crosses 1-4 gave X^2 for linkage = 81.29, 137.63, 47.69 and 17.97 with P = 1.50, 2.86, 2.55 and 2.41 respectively, thereby indicating that genes controlling these characters are not linked.

However, joint segregation on the assumption of pleiotropy in M_2F_2 of cross 4 gave X^2 = 3.25 suggesting pleiotropic association of these characters (Table 43-45). The pleiotropic gene could be identified as $\underline{Pjp}_{a/b/c}$ for junctura proper pigmentation and $\underline{Pau}_{a/b/c}$ for auricle pigmentation, while crosses 1-3 showed neither independent assortment / linkage nor pleiotropy (Appendix II).

58. Junctura proper pigmentation vs nodal pigmentation

Junctura proper showed 9:7 in cross 1 and 27:37 in crosses 2-4, while nodal pigmentation showed 3:13 in 1-4 crosses (Table 39-6,9).

Joint segregation of these characters in M_2F_2 of cross 4 gave X^2 = 3.67, which is not significant against the table value 7.82 for 3 d.f at 5% level (Table 41-26), thereby showing independent assortment of genes controlling these characters.

However, joint segregation of these characters in F_2/M_2F_2 of crosses 1-3 gave X^2 = 22.89, 14.63 and 9.44 respectively, which are significant against the table value, indicating that the genes controlling these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy gave X^2 for linkage = 23.44, 14.33 and 9.43 with P = 1.63, 1.34 and 4.81 respectively, thereby indicating that genes controlling these characters are not linked.

However, joint segregation on the assumption of pleiotropy in M_2F_2 of crosses 2 & 3 gave X^2 = 7.14 and 3.21, suggesting pleiotropic association of these characters (Table 43-46). Gene $\underline{Pjp_{a/b/c}}$ for junctura proper pigmentation is pleiotropic to $\underline{Pn}$ for nodal pigmentation or vice-versa, while cross 1 showed neither independent assortment/ linkage nor pleiotropy (Appendix II).

59. Junctura proper pigmentation vs internode pigmentation

Junctura proper showed 9:7 in cross 1 and 27:37 in crosses 2-4, while internode showed 3:1 in crosses 1-3 and 9:7 in cross 4 (Table 39&6,10).

Joint segregation of these characters in M_2F_2 of 1-4 crosses gave X^2 = 60.61, 73.35, 26.74 and 17.55 respectively, which are significant against the table value 7.82 for 3 d.f at 5% level (Table 43-47), thereby indicating that genes controlling these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy on F_2/M_2F_2 of crosses 1-4 gave X^2 for linkage = 52.91, 70.68, 61.30 and 17.40 with P-values 1.86, 3.15, 2.94 and 2.67 respectively, thereby indicating that genes controlling these characters are not linked.

However, joint segregation on the assumption of pleiotropy in F_2/M_2F_2 of crosses 1-4 gave X^2 = 1.15, 5.29, 1.12 and 0.61 suggesting pleiotropic association of these characters (Table 43-47). Gene $\underline{Pjp_{a/b}}$ or $\underline{Pjp_{a/b/c}}$ for junctura proper pigmentation is pleiotropic to $\underline{Pin}$ or $\underline{Pin_{a/b}}$ for internodal pigmentation.

60. Junctura proper pigmentation vs stigma pigmentation

Junctura proper showed 9:7 in cross 1 and 27:37 in crosses 2-4,

while stigma showed 3:1 in crosses 1-3 and 9:7 in cross 4 (Table 39a 6,11).

Joint segregation of these characters in F_2/M_2F_2 of 1-4 crosses gave X^2 = 53.93, 44.30, 26.74 and 17.55 respectively, which are significant against the table value 7.82 for 3 d.f at 5% level (Table 43-48), thereby indicating that genes controlling these characters do not follow independent assortment. Estimation of linkage by minimum discrepancy on F_2/M_2F_2 of crosses 1-4 gave X^2 for linkage=52.91, 46.17, 26.05 and 17.39 with P = 1.86,3.14, 2.94 and 2.67 respectively, thereby indicating that genes controlling these characters are not linked.

However, joint segregation on the assumption of pleiotropy in F_2/M_2F_2 of crosses 1-4 gave X^2 = 1.78, 0.32, 1.12 and 0.61 suggesting pleiotropic association of these characters (Table 43-48). Gene $\underline{Pjp_{a/b}}$ or $\underline{Pjp_{a/b/c}}$ for junctura proper pigmentation is pleiotropic to $\underline{Ps}$ or $\underline{Ps_{a/b}}$ for stigma pigmentation.

61. Junctura proper pigmentation vs apiculus pigmentation

Junctura proper showed 9:7 in cross 1 and 27:37 in crosses 2-4, while apiculus showed 3:1 in crosses 1-3 and 9:7 in cross 4 (Table 39a 6,12).

Joint segregation of these characters in F_2/M_2F_2 of crosses 1-4 gave X^2 = 60.61, 70.37, 24.13 and 17.55 respectively, which are significant against the table value 7.82 for 3 d.f at 5% level (Table 43-49), thereby indicating that genes controlling these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy on F_2/M_2F_2 of crosses 1-4 gave X^2 for linkage = 52.91, 73.19, 23.79 and 17.39 with P = 1.86, 3.15, 2.94 and 2.67 respectively, thereby indicating that genes controlling these characters are not linked.

However, joint segregation on the assumption of pleiotropy in F_2/M_2F_2 of crosses 1-4 gave X^2 = 1.15, 4.44, 0.58 and 0.61 suggesting pleiotropic association of these characters (Table 43-49). Gene $\underline{Pjp_{a/b}}$ or $\underline{Pjp_{a/b/c}}$ for junctura proper pigmentation is pleiotropic to $\underline{Pa}$ or $\underline{Pa_{a/b}}$ for apiculus pigmentation or vice-versa.

62. Junctura proper pigmentation vs awning

Junctura proper showed 9:7 in cross 1 and 27:37 in crosses 2-4, while awning showed 3:1 in 1-4 crosses (Table 39&6,13).

Joint segregation of these characters in F_2/M_2F_2 of the crosses gave X^2 = 2.70, 2.97, 1.22 and 0.52 respectively, which are not significant against the table value 7.82 for 3 d.f at 5% level (Table

41-27), thereby showing independent assortment of the genes controlling these characters.

63. Junctura proper pigmentation vs tip-sterility

Junctura proper pigmentation showed 9:7 in cross 1 and 27:37 in crosses 2-4, while tip-sterility showed 3:1 in all the above crosses (Table 39a-6,14).

Joint segregation of these characters in F_2/M_2F_2 of the crosses gave X^2 = 1.86, 2.84, 3.09 and 3.71 which are not significant against the table value 7.82 for 3 d.f at 5% level (Table 41-28), thereby showing independent assortment of these characters.

64. Ligule pigmentation vs auricle pigmentation

Ligule showed 9:7 in crosses 1-3 and 27:37 in cross 4, while auricle showed 9:7 in cross 1 and 27:37 in crosses 2-4 (Table 39a-7,8).

Joint segregation of these characters in F_2/M_2F_2 of 1-4 crosses gave X^2 = 53.74, 142.12, 57.06 and 14.65 respectively, which are significant against the table value 7.82 for 3 d.f at 5% level (Table 43-50 and Appendix II), thereby indicating that genes controlling these characters do not follow independent assortment. Estimation of

linkage on minimum discrepancy on F_2/M_2F_2 of crosses 1-4 gave X^2 for linkage = 51.47, 132.80, 56.26 and 12.77 with P = 1.37, 1.99, 1.97 and 1.07 respectively, thereby indicating that genes controlling these characters are not linked.

However, joint segregation on the assumption of pleiotropy in F_2/M_2F_2 of crosses 3-4 gave X^2 = 2.51 and 2.11 suggesting pleiotropic association of these characters (Table 43-50). Gene $\underline{Plg}_{\underline{a/b}}$ or $\underline{Plg}_{\underline{a/b/c}}$ for ligule pigmentation is pleiotropic to $\underline{Pau}_{\underline{a/b}}$ or $\underline{Pau}_{\underline{a/b/c}}$ for auricle pigmentation, while crosses 1 & 2 showed neither independent assortment/linkage nor pleiotropy (Appendix II).

65. Ligule pigmentation vs nodal pigmentation

Ligule showed 9:7 in crosses 1-3 and 27:37 in cross 4, while nodal pigmentation showed 3:13 in 1-4 crosses (Table 39a-7,9).

Joint segregation of these characters in M_2F_2 of crosses 3 & 4 gave X^2 = 5.82 and 7.54 respectively, which are not significant against the table value 7.82 for 3 d.f at 5% level (Table 41-29), thereby showing independent assortment of genes controlling these characters.

However, joint segregation of these characters in F_2/M_2F_2 of

crosses 1 & 2 gave X^2=18.92 and 10.23 which are significant against the table value suggesting that the genes controlling these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy gave X^2 for linkage = 7.83 and 9.20 with P = 1.03 and 1.38 respectively, indicating that genes controlling these characters are not linked.

However, joint segregation on the assumption of pleiotropy in F_2/M_2F_2 of crosses 1 & 2 gave X^2 = 5.13 and 3.29 suggesting pleiotropic association of these characters (Table 43-51). Gene $\underline{Plg_{a/b}}$ for ligule pigmentation is pleiotropic to Pn for nodal pigmentation or vice-versa.

66. Ligule pigmentation vs internode pigmentation

Ligule showed 9:7 in crosses 1-3 and 27:37 in cross 4, while internode showed 3:1 in crosses 1-3 and 9:7 in cross 4 (Table 39~~&~~7, 10).

Joint segregation of these characters in F_2/M_2F_2 of 1-4 crosses gave X^2 = 84.19, 96.55, 38.71 and 17.52 respectively, which are significant against the table value 7.82 for 3 d.f at 5% level (Table 43-52 & Appendix II), thereby indicating that genes controlling these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy on F_2/M_2F_2 of above crosses gave X^2

for linkage = 78.32, 97.57, 37.30 and 17.40 with P = 1.85, 1.62, 1.77 and 2.67 respectively, thereby indicating that genes controlling these characters are not linked.

However, joint segregation on the assumption of pleiotropy in F_2/M_2F_2 of crosses 1, 3 & 4 gave X^2 = 4.61, 0.84 and 0.61 suggesting pleiotropic association of these characters (Table 43-52). Gene $\underline{Plg}_{a/b}$ or $\underline{Plg}_{a/b/c}$ for ligule pigmentation is pleiotropic to Pin or $\underline{Pin}_{a/b}$ for internode pigmentation or vice-versa, while cross 2 showed neither independent assortment/linkage nor pleiotropy (Appendix II).

67. Ligule pigmentation vs stigma pigmentation

Ligule showed 9:7 in crosses 1-3 and 27:37 in the cross 4, while stigma showed 3:1 in crosses 1-3 and 9:7 in cross 4 (Table 39a-7,11).

Joint segregation of these characters in F_2/M_2F_2 of 1-4 crosses gave X^2 = 76.32, 70.46, 38.71 and 17.52 respectively, which are significant against the table value 7.82 for 3 d.f. at 5% level (Table 45-53), thereby indicating that genes regulating these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy on F_2/M_2F_2 of above crosses gave X^2 for linkage = 74.51, 67.09, 37.72 and 17.40 with P = 1.86, 1.89, 1.77, and 2.67 respectively, thereby indicating that genes controlling these characters are not linked.

However, joint segregation on the assumption of pleiotropy in F_2/M_2F_2 of 1-4 crosses gave X^2 = 3.16,0.24, 0.84 and 0.61 (Table 43-53) suggesting pleiotropic association of these characters. Gene $\underline{Plg_{a/b}}$ or $\underline{Plg_{a/b/c}}$ for ligule pigmentation is pleiotropic to $\underline{Ps}$ or $\underline{Ps_{a/b}}$ for stigma pigmentation or vice-versa.

68. Ligule pigmentation vs apiculus pigmentation

Ligule showed 9:7 in crosses 1-3 and 27:37 in the cross 4, while stigma pigmentation showed 3:1 in crosses 1-3 and 9:7 in cross 4 (Table 39a-7,12).

Joint segregation of these characters in F_2/M_2F_2 of 1-4 crosses gave X^2 = 84.19, 113.05, 34.78 and 17.52 respectively, which are significant against the table value 7.82 for 3 d.f at 5% level (Table 43-54), thereby indicating that genes controlling these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy on F_2/M_2F_2 of 1-4 crosses gave X^2 for linkage = 70.79, 110.81, 32.43 and 15.14 with P = 1.86, 1.89, 1.77, and 2.67 respectively, thereby indicating that genes controlling these characters are not linked.

However, joint segregation on the assumption of pleiotropy in F_2/M_2F_2 of 1-4 crosses gave X^2 = 4.61, 4.05, 0.75 and 1.55

suggesting pleiotropic association of these characters (Table 43-54). Gene $\underline{Plg_{a/b}}$ or $\underline{Plg_{a/b/c}}$ for ligule pigmentation is pleiotropic to $\underline{Pa}$ or $\underline{Pa_{a/b}}$ for apiculus pigmentation or vice-versa.

69. Ligule pigmentation vs awning

Ligule showed 9:7 in crosses 1-3 and 27:37 in the cross 4, while awning showed 3:1 in 1-4 crosses (Table 39&7,13).

Joint segregation of these characters in F_2/M_2F_2 of 1-4 crosses gave X^2 = 1.84, 2.95, 2.52 and 1.93 respectively, which are not significant against the table value 7.82 for 3 d.f at 5% level (Table 41-30), thereby showing independent assortment of these characters.

70. Ligule pigmentation vs tip-sterility

Ligule showed 9:7 in crosses 1-3 and 27:37 in cross 4, while tip-sterility showed 3:1 in 1-4 crosses (Table 39&7,14).

Joint segregation of these characters in F_2/M_2F_2 of 1-4 crosses gave X^2 = 4.03, 2.42, 0.77 and 3.71 respectively, which are not significant against the table value 7.82 for 3 d.f at 5% level (Table 41-31), thereby showing independent assortment of these characters.

71. Auricle pigmentation vs nodal pigmentation

Auricle showed 9:7 in cross 1 and 27:37 in crosses 2-4, while nodal pigmentation showed 3:13 in 1-4 crosses (Table 39-8,9).

Joint segregation of these characters in M_2F_2 of cross 4 gave X^2 = 7.32, which is not significant against the table value 7.82 for 3 d.f at 5% level (Table 41-32), thereby showing independent assortment of these genes.

However, joint segregation of these characters in F_2/M_2F_2 of crosses 1-3 gave X^2 =18.92, 66.85 and 9.55 which are significant against the table value 7.82, thereby indicating that genes controlling these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy gave X^2 for linkage = 14.18, 12.78 and 9.20 with P = 1.33, 2.09 and 1.27 respectively, thereby indicating that genes controlling these characters are not linked.

However, joint segregation on the assumption of pleiotropy in F_2/M_2F_2 of crosses 1 & 3 gave X^2 = 5.13 and 3.20 suggesting pleiotropic association of these characters (Table 43-55). The pleiotropic gene could be identified as $\underline{Pau}_{a/b}$ or $\underline{Pau}_{a/b/c}$ for auricle pigmentation and Pn for nodal pigmentation. Cross 2 showed neither independent assortment/linkage nor pleiotropy (Appendix II).

72. Auricle pigmentation vs internode pigmentation

Auricle showed 9:7 in cross 1 and 27:37 in crosses 2-4, while internode showed 3:1 in 1-3 crosses and 9:7 in cross 4 (Table 39a 8,10).

Joint segregation of these characters in F_2/M_2F_2 of 1-4 crosses gave X^2 = 53.08, 60.69, 26.74 and 12.67 respectively, which are significant against the table value 7.82 for 3 d.f at 5% level (Table 43-56), thereby indicating that genes controlling these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy on F_2/M_2F_2 of 1-4 crosses gave X^2 for linkage = 55.77, 79.74, 26.05 and 10.91 with P = 1.86, 3.15, 2.93 and 2.64 respectively, thereby indicating that genes controlling these characters are not linked.

However, joint segregation on the assumption of pleiotropy in F_2/M_2F_2 of crosses 1, 3 & 4 gave X^2 = 0.46, 1.12 and 5.10 suggesting pleiotropic association of these characters (Table 43-56). Gene $\underline{Pau}_{a/b}$ or $\underline{Pau}_{a/b/c}$ for auricle pigmentation is pleiotropic to $\underline{Pin}$ or $\underline{Pin}_{a/b}$ for internode pigmentation, while cross 2 showed neither independent assortment/linkage nor pleiotropy (Appendix II).

73. Auricle pigmentation vs stigma pigmentation

Auricle showed 9:7 in cross 1 and 27:37 in crosses 2-4, while stigma showed 3:1 in 1-3 crosses and 9:7 in cross 4 (Table 39-8,11).

Joint segregation of these characters in F_2/M_2F_2 of 1-4 crosses gave X^2 = 56.22. 58.58, 26.74, and 12.67 respectively, which are significant against the table value 7.82 for 3 d.f at 5% level (Table 43-57), thereby indicating that genes controlling these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy on F_2/M_2F_2 of 1-4 crosses gave X^2 for linkage = 55.77, 53.54, 26.05, and 10.90 with P = 1.86, 3.14, 2.94 and 2.64 respectively, thereby indicating that genes controlling these characters are not linked.

However, joint segregation on the assumption of pleiotropy in F_2/M_2F_2 of crosses 1-4 gave X^2 = 0.79, 4.02. 1.12 and 5.10 suggesting pleiotropic association of these characters (Table 43-57). Gene $\underline{Pau}_{\underline{a/b}}$ or $\underline{Pau}_{\underline{a/b/c}}$ for auricle pigmentation is pleiotropic to $\underline{Ps}$ or $\underline{Ps}_{\underline{a/b}}$ for stigma pigmentation.

74. Auricle pigmentation vs apiculus pigmentation

Auricle showed 9:7 in cross 1 and 27:37 in crosses 2-4, while

apiculus showed 3:1 in 1-3 crosses and 9:7 in cross 4 (Table 39a-8,12).

Joint segregation of these characters in F_2/M_2F_2 of 1-4 crosses gave X^2 = 63.08, 81.03, 24.13 and 11.15 respectively, which are significant against the table value 7.82 for 3 d.f at 5% level (Table 43-58), thereby indicating that genes controlling these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy on F_2/M_2F_2 of 1-4 crosses gave X^2 for linkage = 52.56, 79.74, 23.80 and 9.15 with P = 1.86, 3.15, 2.94 and 2.63 respectively, thereby indicating that genes controlling these characters are not linked.

However, joint segregation on the assumption of pleiotropy in F_2/M_2F_2 of crosses 1, 3 & 4 gave X^2 = 0.46, 0.58 and 7.17 suggesting pleiotropic association of these characters (Table 43-58). Gene $\underline{Pau}_{a/b}$ or $\underline{Pau}_{a/b/c}$ for auricle pigmentation is pleiotropic to $\underline{Pa}$ or $\underline{Pa}_{a/b}$ for apiculus pigmentation or vice-versa, while cross 2 showed neither independent assortment/linkage nor pleiotropy (Appendix II).

75. Auricle pigmentation vs awning

Auricle showed 9:7 in cross 1 and 27:37 in the crosses 2-4, while awning showed 3:1 in 1-4 crosses (Table 39a-8,13).

Joint segregation of these characters in F_2/M_2F_2 of the above crosses gave X^2 = 5.56, 7.26, 1.87 and 2.15 respectively, which are not significant against the table value 7.82 for 3 d.f at 5% level (Table 41-33), thereby showing independent assortment of these characters.

76. Auricle pigmentation vs tip-sterility

Auricle showed 9:7 in cross 1 and 27:37 in the crosses 2-4, while tip-sterility showed 3:1 in 1-4 crosses (Table 39a-8,14).

Joint segregation of these characters in F_2/M_2F_2 of the above crosses gave X^2 = 7.04, 5.70, 1.51 and 6.57 respectively, which are not significant against the table value 7.82 for 3 d.f. at 5% level (Table 41-34), thereby showing independent assortment of these characters.

77. Nodal pigmentation vs internode pigmentation

Nodal pigmentation showed 3:13 in 1-4 crosses, while internode showed 3:1 in the crosses 1-3 and 9:7 in cross 4 (Table 39a-9,10).

Joint segregation of these characters in M_2F_2 of crosses 3 & 4 gave X^2 = 6.74 and 6.15 which are not significant against the table

value 7.82 for 3 d.f at 5% level (Table 41-35), thereby showing independent assortment of these genes.

However, joint segregation of these characters in F_2/M_2F_2 of the crosses 1 & 2 gave X^2 =15.64 and 18.31 which are significant (Table 43-59), thereby indicating that these genes are not following independent assortment. Estimation of linkage on minimum discrepancy gave X^2 for linkage = 17.05, 12.04 with P = 2.75 and 9.25 respectively, thereby indicating that genes controlling these characters are not linked.

However joint segregation on the assumption of pleiotropy in F_2/M_2F_2 of crosses 1 & 2 gave X^2 = 1.64 and 4.46 respectively, suggesting pleiotropic association of these characters (Table 43-59). The basic gene Pn for nodal pigmentation is pleiotropic to Pin for the internode pigmentation.

78. Nodal pigmentation vs stigma pigmentation

Nodal pigmentation showed 3:13 in 1-4 crosses, while stigma showed 3:1 in crosses 1-3 and 9:7 in cross 4 (Table 39-9,11).

Joint segregation of these characters in M_2F_2 of crosses 2 & 4 gave X^2 = 5.87 and 6.15 which are not significant against the table

value 7.82 for 3 d.f at 5% level (Table 41-36), thereby showing independent assortment of these genes.

However, joint segregation of these characters in F_2/M_2F_2 of crosses 1 & 3 gave X^2 = 14.09 and 10.58 which are significant, thereby indicating that these genes are not following independent assortment. Estimation of linkage on minimum discripency gave X^2 for linkage = 13.90, 9.594 with P = -2.56 and 8.62 respectively, thereby indicating that genes controlling these characters are not linked.

However, joint segregation on the assumption of pleiotropy in F_2/M_2F_2 of crosses 1 & 3 gave X^2 = 0.73 and 1.22, suggesting pleiotropic association of these characters (Table 43-60). The basic gene Pn for nodal pigmentation is pleiotropic to the gene Ps for stigma pigmentation or vice-versa.

79. Nodal pigmentation vs apiculus pigmentation

Nodal pigmentation showed 3:13 in 1-4 crosses, while apiculus showed 3:1 in crosses 1-3 and 9:7 in cross 4 (Table 39-9,12).

Joint segregation of these characters in M_2F_2 of cross 4 gave X^2 = 6.15 which is not significant against the table value 7.82 for 3 d.f at 5% level (Table 41-37), thereby showing independent assortment of these genes.

However, joint segregation of these characters in F_2/M_2F_2 of crosses 1-3 gave X^2 = 15.64, 17.15 and 9.36 which are significant, thereby indicating that these genes do not follow independent assortment. Estimation of linkage on minimum discrepancy gave X^2 for linkage = 16.30, 12.62, and 8.82 with P = -2.94, 9.21; -2.76 respectively, thereby indicating that genes controlling these characters are not linked.

However, joint segregation on the assumption of pleiotropy in F_2/M_2F_2 of crosses 1-3 gave X^2 = 0.75, 3.56 and 0.75 respectively, suggesting pleiotropic association of these characters (Table 43-61). The basic gene Pn for nodal pigmentation is identified to be pleiotropic to Pa for apiculus pigmentation or vice-versa.

80. Nodal pigmentation vs awning

Nodal pigmentation showed 3:13 in 1-4 crosses, while awning showed 3:1 in all the crosses (Table 39-9,13).

Joint segregation of these characters in F_2/M_2F_2 of the above crosses gave X^2 = 0.82, 6.07, 3.71 and 0.32 respectively, which are not significant against the table value 7.82 for 3 d.f. at 5% level (Table 41-38), thereby showing independent assortment of these characters.

81. Nodal pigmentation vs tip-sterility

Nodal pigmentation showed 3:13 in crosses 1-4, while tip-sterility showed 3:1 in all the crosses (Table 39~~a~~9,14).

Joint segregation of these characters in F_2/M_2F_2 of the above crosses gave X^2 = 1.14, ~~4~~.44, 7.63 and 3.95 respectively, which are not significant against the table value 7.82 for 3 d.f. at 5% level (Table 41-39), thereby showing independent assortment of these characters.

82. Internode pigmentation vs stigma pigmentation

Both internode and stigma pigmentation showed 3:1 in crosses 1-3 and 9:7 in cross 4 (Table 39~~a~~10,11).

Joint segregation of these characters in F_2/M_2F_2 of crosses 1-4 gave X^2 = 118.72, 124.55, 72.02 and 37.45 which are significant against the table value 7.82 for 3 d.f. at 5% level (Table 42-25), thereby indicating that genes controlling these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy on F_2/M_2F_2 of crosses 1&3 gave X^2 for linkage = 118.57 and 70.76 with c.o c.o. = 5.13 and 7.27 respectively (Table 42-25), thereby indicating that gene Pin for internode

pigmentation is linked with Ps for stigma pigmentation, while cross 4 gave X^2 for linkage = 37.37 with P = 1.83, thereby indicating that genes controlling these characters are not linked .

However, joint segregation on the assumption of pleiotropy in M_2F_2 of cross 4 gave X^2 = 0.04 suggesting pleiotropic association of these characters (Table 43-62). Gene $Pin_{a/b}$ for internode pigmentation is identified to be pleiotropic to $Ps_{a/b}$ for stigma pigmentation or vice-versa. while cross 2 neither showed independent assortment/ linkage or pleiotropy (Appendix II).

83. Internode pigmentation vs apiculus pigmentation

Both Internode and apiculus pigmentation showed 3:1 in crosses 1-3 and 9:7 in cross 4 (Table 39-10,12).

Joint segregation of these characters in F_2/M_2F_2 of crosses 1-4 gave X^2 = 104.79, 187.39, 64.16 and 37.45 which are significant against the table value 7.82 for 3 d.f. at 5% level (Table 42-26 & 43-63), thereby indicating that genes controlling these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy on F_2/M_2F_2 of crosses 1-3 gave X^2 for linkage = 104.49, 182.34 and 63.25 with c.o. = 8.35, 5.66 and 8.35 respectively (Table 42-26), thereby indicating that genes controlling these characters are linked. Gene $Pin_{a/b}$ for internode is pigmentation linked with the basic gene Pa for apiculus pigmentation, while cross 4 gave X^2

for linkage = 37.37 with P = 1.83, thereby indicating that genes controlling these characters are not linked.

However, joint segregation on the assumption of pleiotropy in M_2F_2 of cross 4 gave X^2 = 0.04 suggesting pleiotropic association of these characters (Table 43-63). Gene $\underline{Pin}_{a/b}$ for internode pigmentation is identified to be pleiotropic to $\underline{Pa}_{a/b}$ for apiculus pigmentation or vice-versa.

84. Internode pigmentation vs awning

Internode pigmentation showed 3:1 in crosses 1-3 and 9:7 in cross 4 , while awning showed 3:1 in all the crosses (Tables 39-10,13).

Joint segregation of these characters in F_2/M_2F_2 of 1-4 crosses gave X^2 = 0.29, 4.84, 2.60 and 0.42 respectively, which are not significant against the table value 7.82 for 3 d.f. at 5% level (Table 41-40), thereby showing independent assortment of these characters.

85. Internode pigmentation vs tip-sterility

Internode pigmentation showed 3:1 in crosses 1-3 and 9:7 in the

cross 4 while tip-sterility showed 3:1 in 1-4 crosses (Table 39& 10,14).

Joint segregation of these characters in F_2/M_2F_2 of the above crosses gave X^2 = 5.63, 4.79, 0.73 and 2.88 respectively, which are not significant against the table value 7.82 for 3 d.f. at 5% level (Table 41-41), thereby showing independent assortment of these characters.

86. Stigma pigmentation vs apiculus pigmentation

Both stigma and apiculus pigmentation showed 3:1 in crosses 1-3 and 9:7 in cross 4 (Table 39& 11,12).

Joint segregation of these characters in F_2/M_2F_2 of crosses 1-3 gave X^2 = 128.59, 124.55, and 85.12 which are significant against the table value 7.82 for 3 d.f at 5% level (Tables 42-47 and 43-64), thereby indicating that genes controlling these characters do not follow independent assortment. Estimation of linkage on minimum discrepancy on F_2/M_2F_2 of crosses 1 & 3 gave X^2 for linkage = 128.44 and 84.21 with c.o. = 3.56 and 3.05 respectively (Table 42-27), thereby indicating that genes of these characters are linked. Gene Ps for stigma pigmentation is linked with Pa for apiculus pigmentation, while cross 2 and 4 gave X^2 for linkage = 128.21 and

37.37 with P = 2.23 and 1.83, thereby indicating that these characters are not linked.

However, joint segregation on the assumption of pleiotropy in M_2F_2 of cross 4 gave X^2 = 0.04 suggesting pleiotropic association of these characters (Table 43-64). Gene $\underline{Ps_{a/b}}$ for stigma pigmentation is identified to be pleiotropic to $\underline{Pa_{a/b}}$ for apiculus pigmentation. While cross 2 showed neither independent assortment/linkage/ pleiotropy (Appendix II).

87. Stigma pigmentation vs awning

Stigma showed 3:1 in crosses 1-3 and 9:7 in cross 4, while awning showed 3:1 in 1-4 crosses (Table 39a-11,13).

Joint segregation of these characters in F_2/M_2F_2 of 1-4 crosses gave X^2 = 2.12, 2.19, 1.84 and 0.42, respectively, which are not significant against the table value 7.82 for 3 d.f. at 5% level (Table 41-42), thereby showing independent assortment of these characters.

88. Stigma pigmentation vs tip-sterility

Stigma showed 3:1 in crosses 1-3 and 9:7 in cross 4, while tip-sterility showed 3:1 in 1-4 crosses (Table 39a-11,14).

Joint segregation of these characters in F_2/M_2F_2 of 1-4 crosses gave X^2 = 6.57, 2.17, 1.47, and 0.80 respectively, which are not significant against the table value 7.82 for 3 d.f at 5% level (Table 41-43), thereby showing independent assortment of these characters.

89. Apiculus pigmentation vs awning

Apiculus showed 3:1 in crosses 1-3 and 9:7 in cross 4, while awning showed 3:1 in 1-4 crosses (Table 39&12,13).

Joint segregation of these characters in F_2/M_2F_2 of the above crosses gave X^2 = 0.79, 4.08, 1.61 and 1.38 respectively, which are not significant against the table value 7.82 for 3 d.f. at 5% level (Table 41-44), thereby showing independent assortment of these characters.

90. Apiculus pigmentation vs tip-sterility

Apiculus showed 3:1 in crosses 1-3 and 9:7 in cross 4, while tip-sterility showed 3:1 in 1-4 crosses (Table 39&12,14).

Joint segregation of these characters in F_2/M_2F_2 of 1-4 crosses gave X^2 = 3.75, 4.29, 0.73 and 3.15 respectively, which are not significant against the table value 7.82 for 3 d.f at 5% level (Table 41-45), thereby showing independent assortment of these characters.

91. Awning vs tip-sterility

Awning and tip-sterility showed 3:1 in 1-4 crosses (Table 39& 13,14).

Joint segregation of these characters in F_2/M_2F_2 of the above crosses gave X^2 = 0.12, 4.02, 2.27 and 7.37 respectively, which are not significant against the table value 7.82 for 3 d.f at 5% level (Table 41-47), thereby showing independent assortment of these characters.

F. QUANTITATIVE MORPHOLOGICAL CHARACTERS

EMS induced variations in quantitative morphological characters were studied in M_1, M_2 and M_3 generations of Japan violet under different pre-treatment + treatments: DW + 0.5% EMS, Vit.C + 0.5% EMS, DW + 0.75% EMS, Vit.C + 0.75% EMS, DW + 1% EMS, and Vit.C + 1% EMS for 12h + 12h respectively. The characters considered in the present study are plant height, culm length (main culm), ear bearing tillers, percentage of ear bearing tillers, days to flower, panicle length, spikelets/panicle, grains/panicle, spikelet sterility and panicle density. Frequency distributions of the variants in M_1,M_2 and M_3 populations in respect of the above characters are presented in Figs. 20-29. Data on range, mean and analysis of variance of the

Fig 20. Frequency distribution for plant height in M_1-M_3 of EMS treated Japan violet

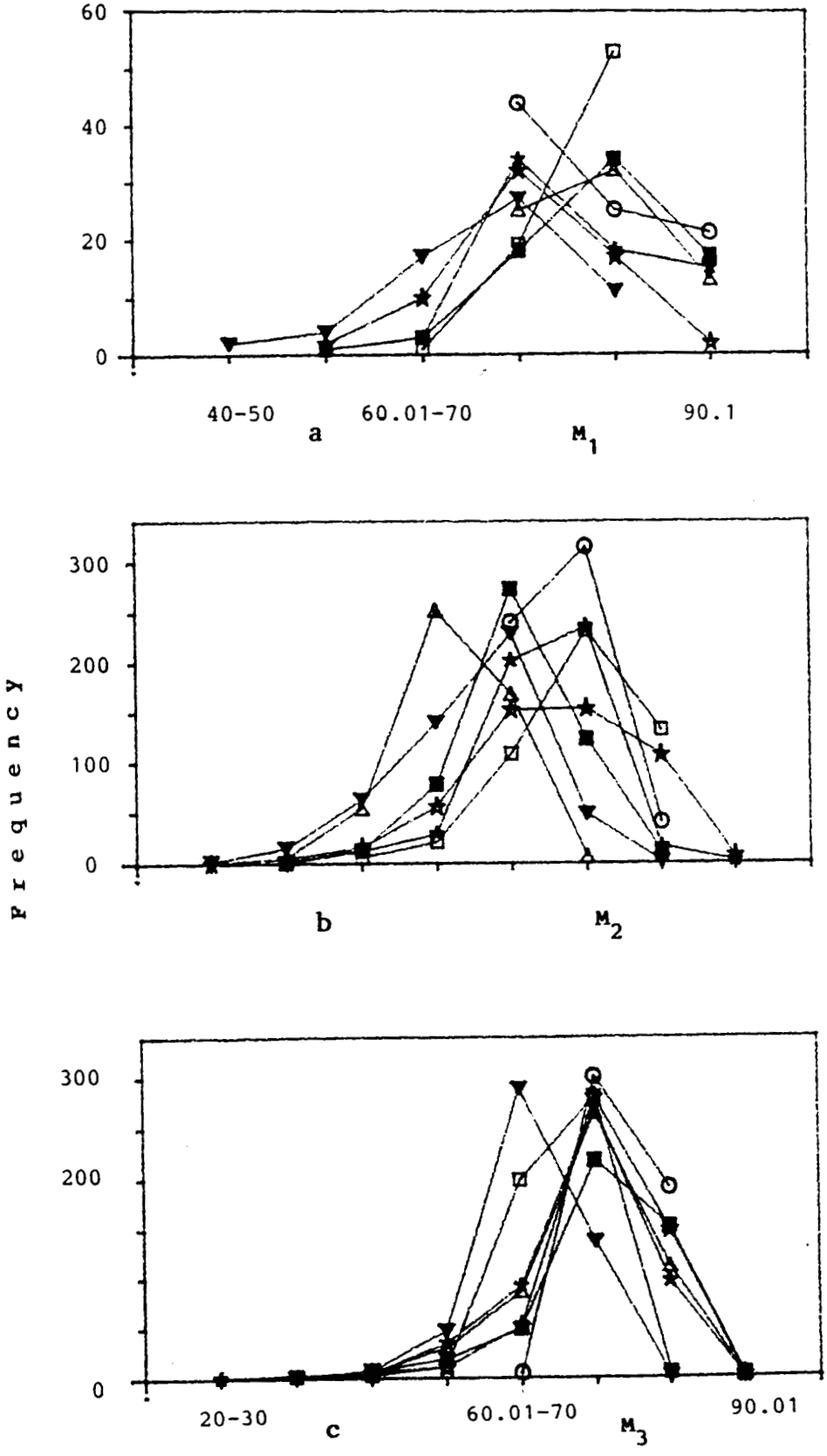

Fig. 20

□ DW+0.5% EMS

■ Vit.C+0.5% EMS

▽ DW+0.75% EMS

▲ Vit.C+0.75% EMS

✲ DW+1% EMS

✱ Vit.C+1% EMS

○ Control

Fig 21. Frequency distribution for main culm length in M_1-M_3 of EMS treated Japan violet

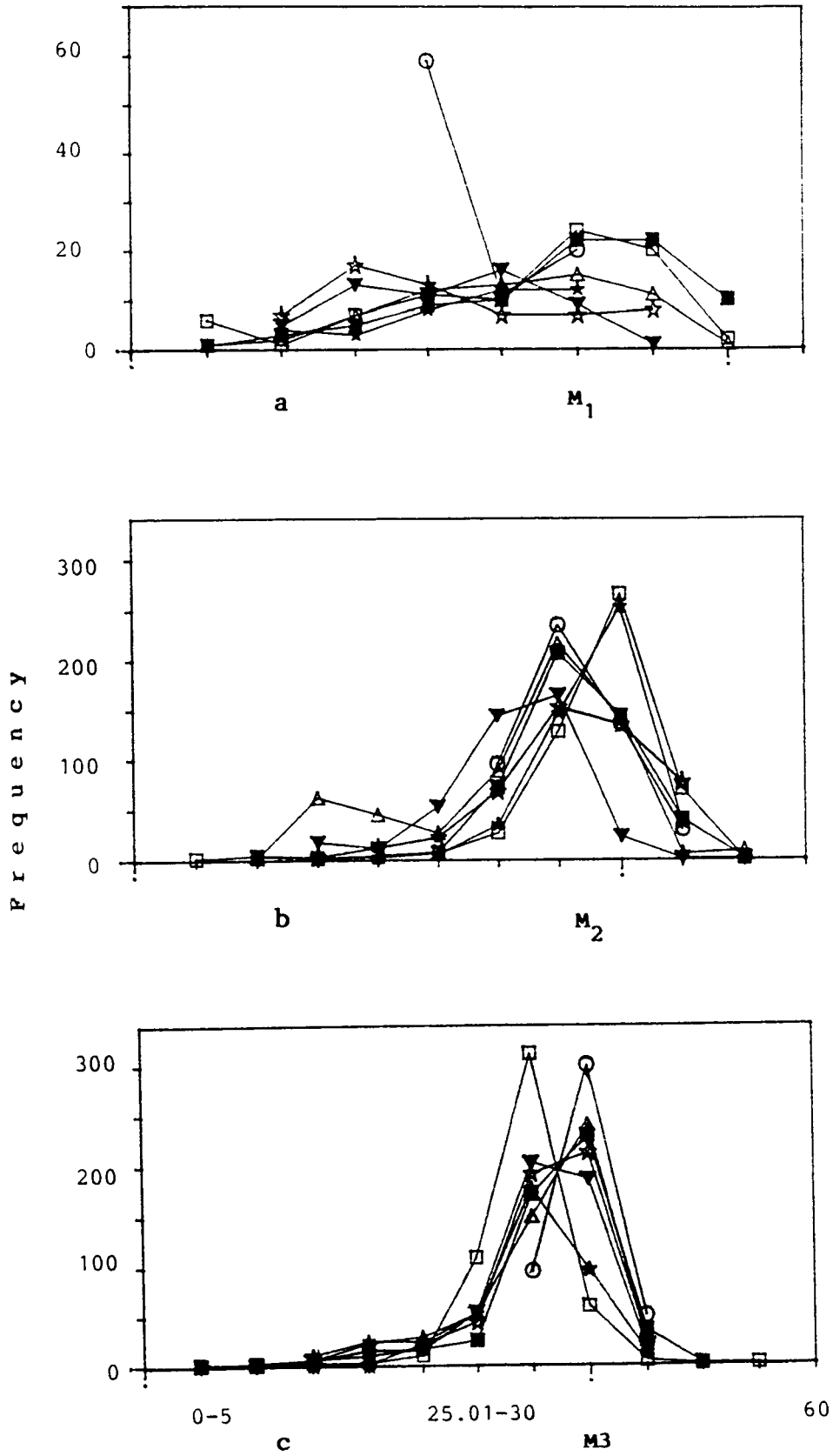

Fig. 21

□ DW+0.5% EMS
☆ DW+1% EMS
■ Vit.C+0.5% EMS
✱ Vit.C+1% EMS
▽ DW+0.75% EMS
○ Control
▲ Vit.C+0.75% EMS

Fig 22. Frequency distribution for EBT number in M_1-M_3 of EMS treated Japan violet

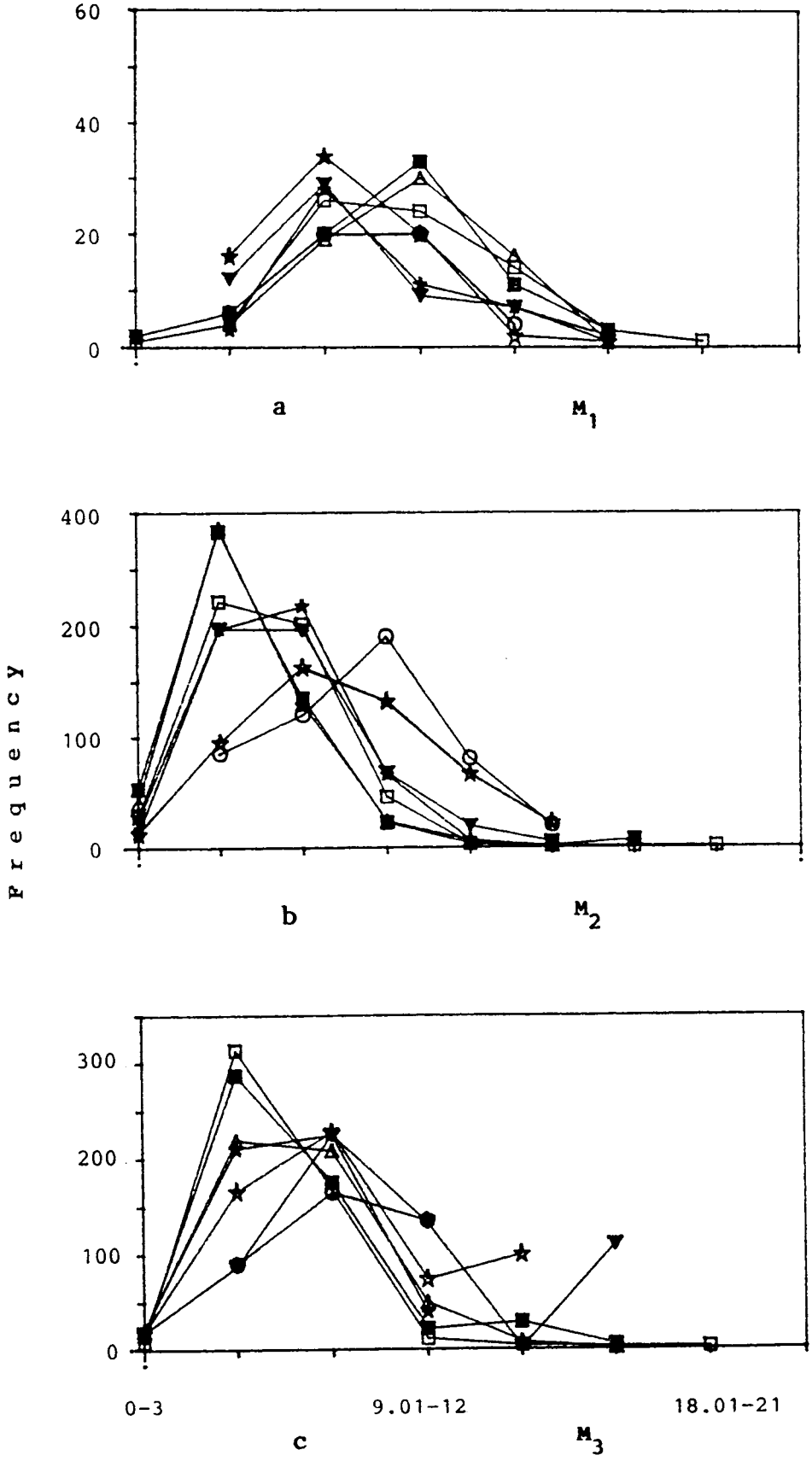

Fig. 22

□ DW+0.5% EMS

■ Vit.C+0.5% EMS

▽ DW+0.75% EMS

▲ Vit.C+0.75% EMS

✡ DW+1% EMS

✱ Vit.C+1% EMS

○ Control

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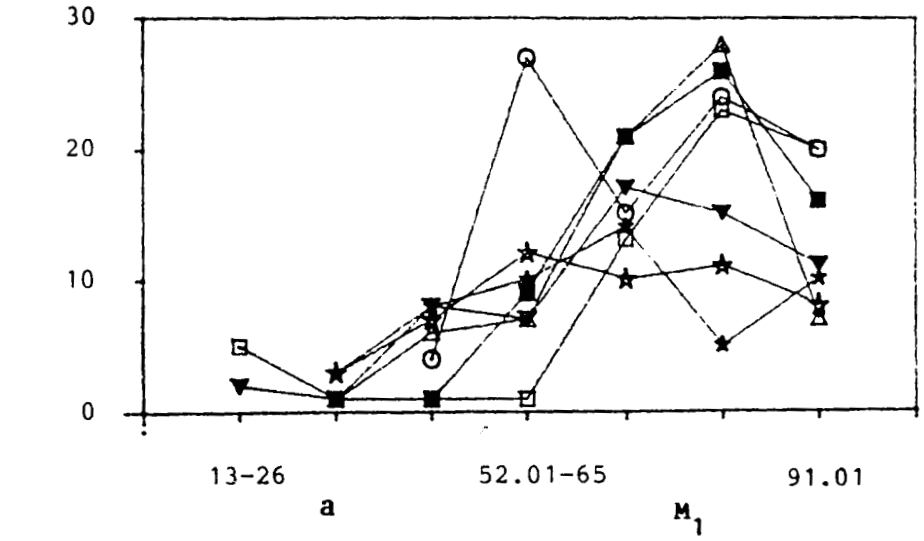
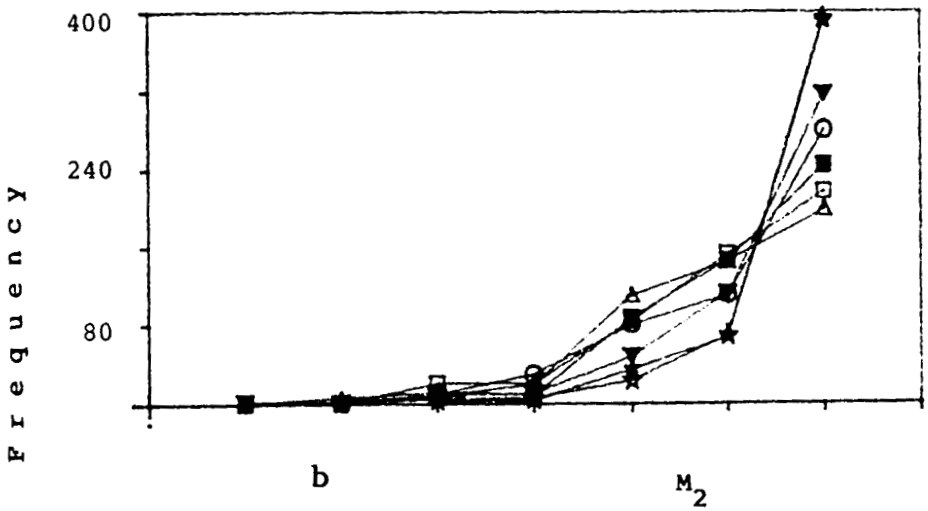
Fig 23. Frequency distribution for EBT % in M_1-M_3 of EMS treated Japan violet





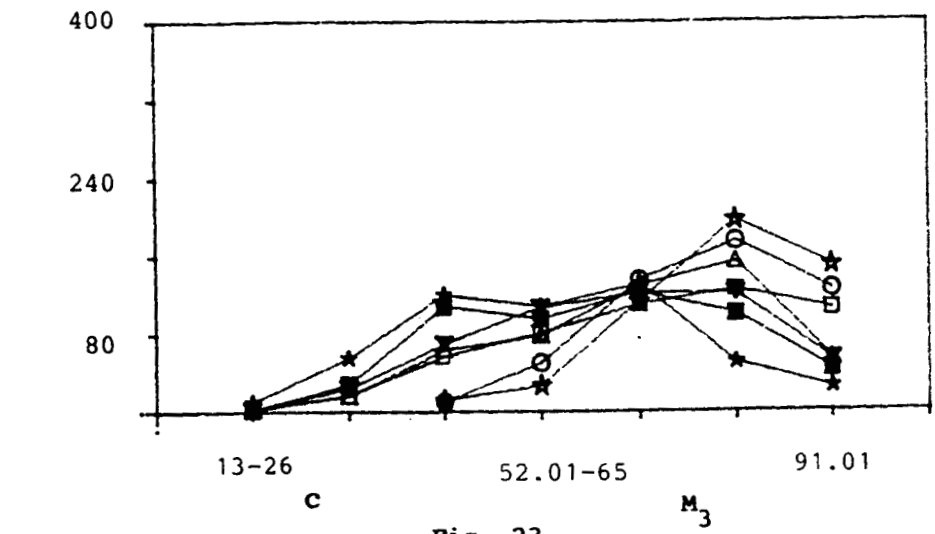
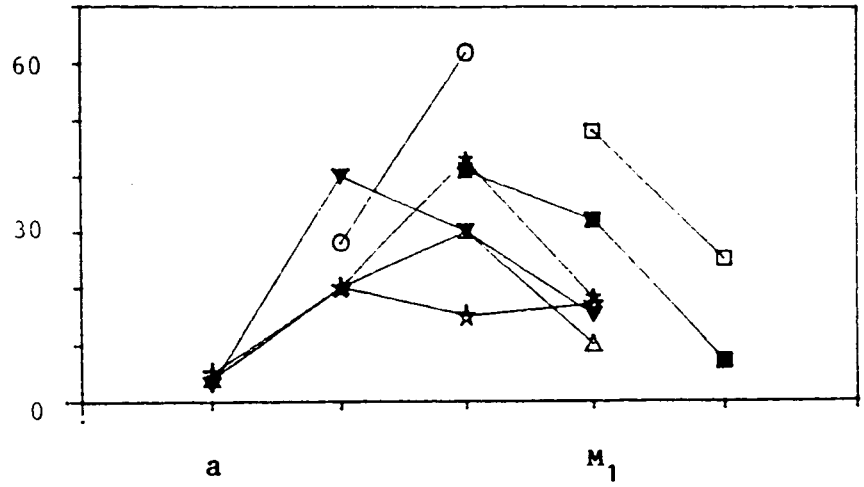
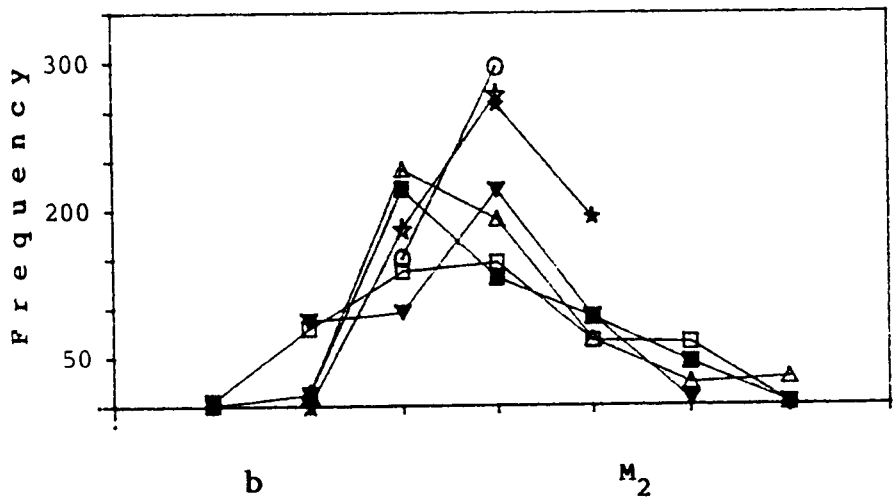


Fig. 23

- DW+0.5% EMS
- Vit.C+0.5% EMS
- DW+0.75% EMS
- Vit.C+0.75% EMS
- DW+1% EMS
- Vit.C+1% EMS
- Control

Fig 24. Frequency distribution for days to flower in M_1-M_3 of EMS treated Japan violet





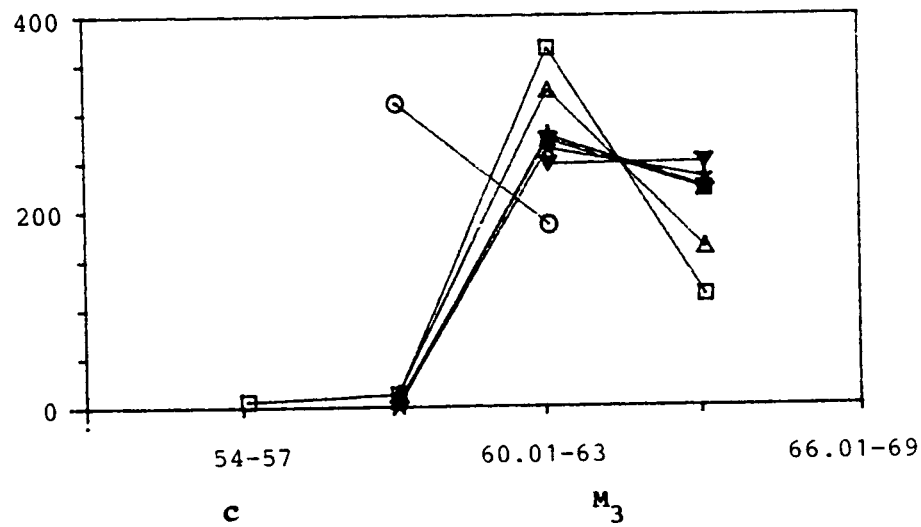
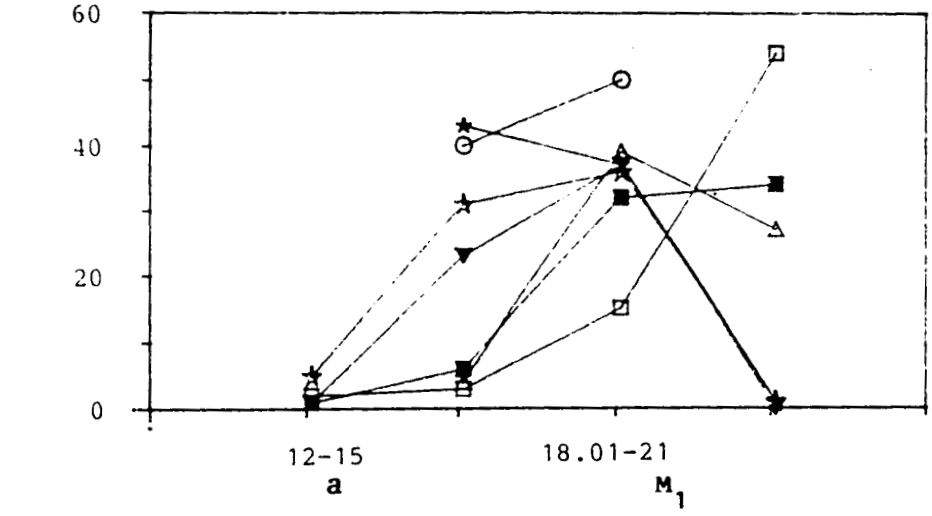
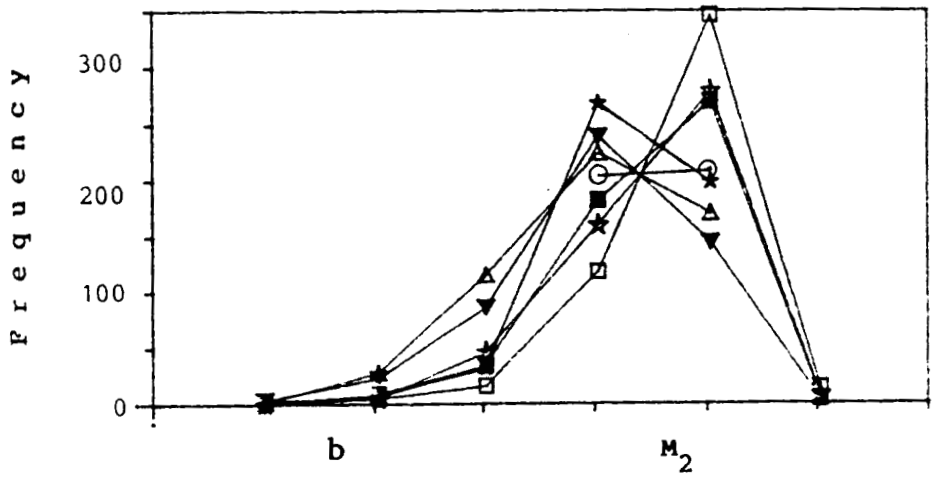


Fig. 24

- DW+0.5% EMS
- DW+1% EMS
- Vit.C+0.5% EMS
- Vit.C+1% EMS
- DW+0.75% EMS
- Control
- Vit.C+0.75% EMS

Fig 25. Frequency distribution for panicle length in M_1-M_3 of EMS treated Japan violet





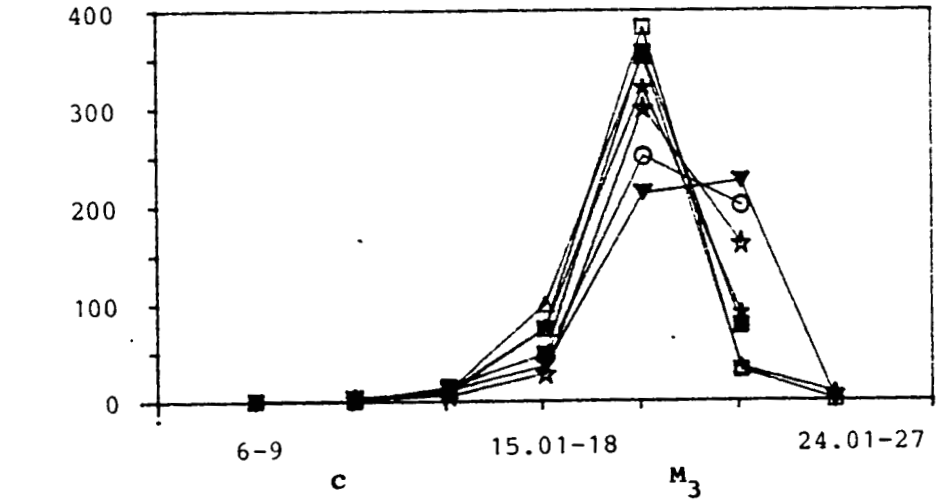


Fig. 25

- DW+0.5% EMS
- DW+1% EMS
- Vit.C+0.5% EMS
- Vit.C+1% EMS
- DW+0.75% EMS
- Control
- Vit.C+0.75% EMS

Fig 26. Frequency distribution for spikelets/ panicle in M_1-M_3 of EMS treated Japan violet

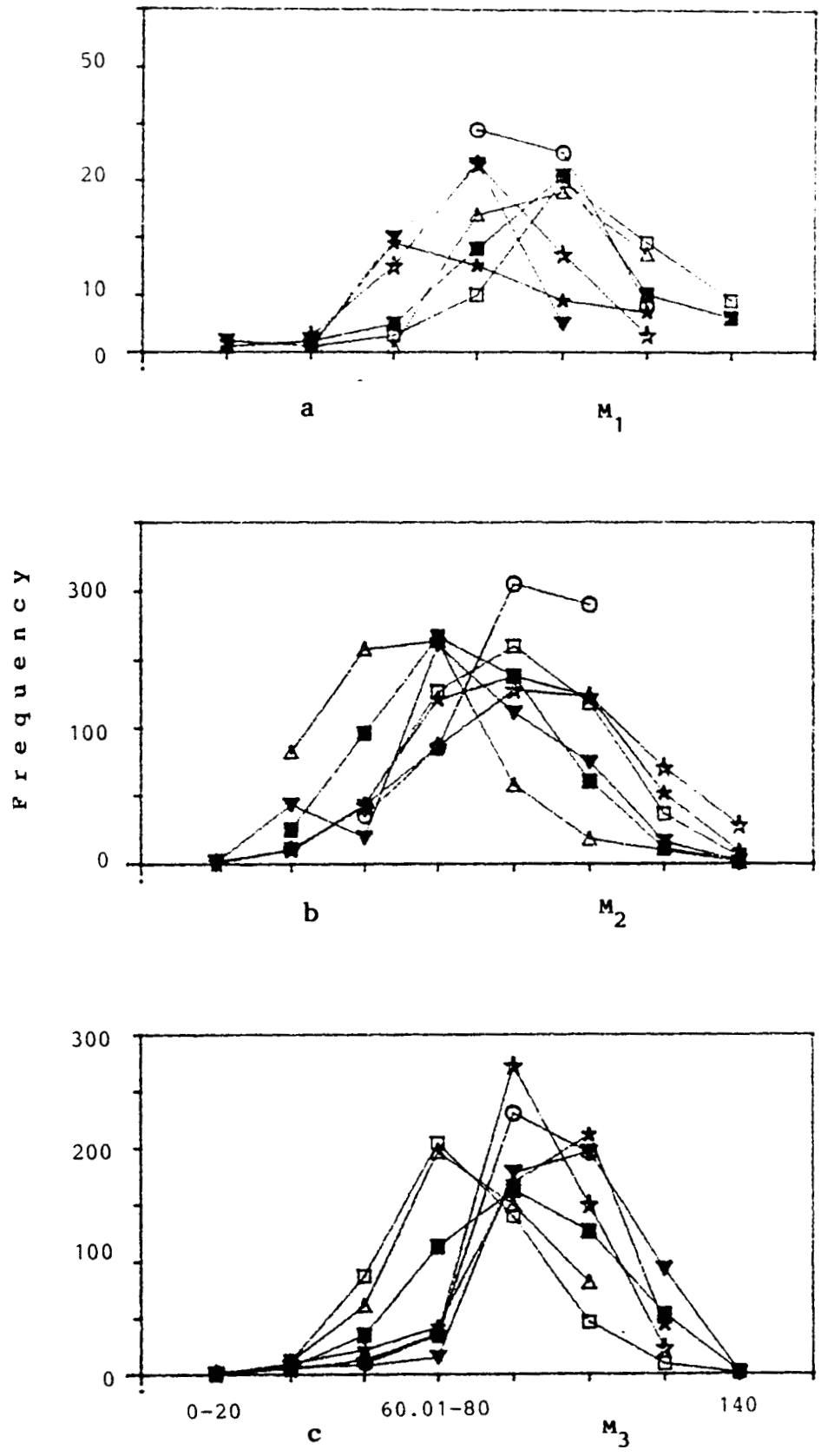


Fig. 26

□ DW+0.5% EMS
■ Vit.C+0.5% EMS
▽ DW+0.75% EMS
▲ Vit.C+0.75% EMS
☆ DW+1% EMS
✶ Vit.C+1% EMS
○ Control

Fig 27. Frequency distribution for grains/ panicle in M_1-M_3 of EMS treated Japan violet

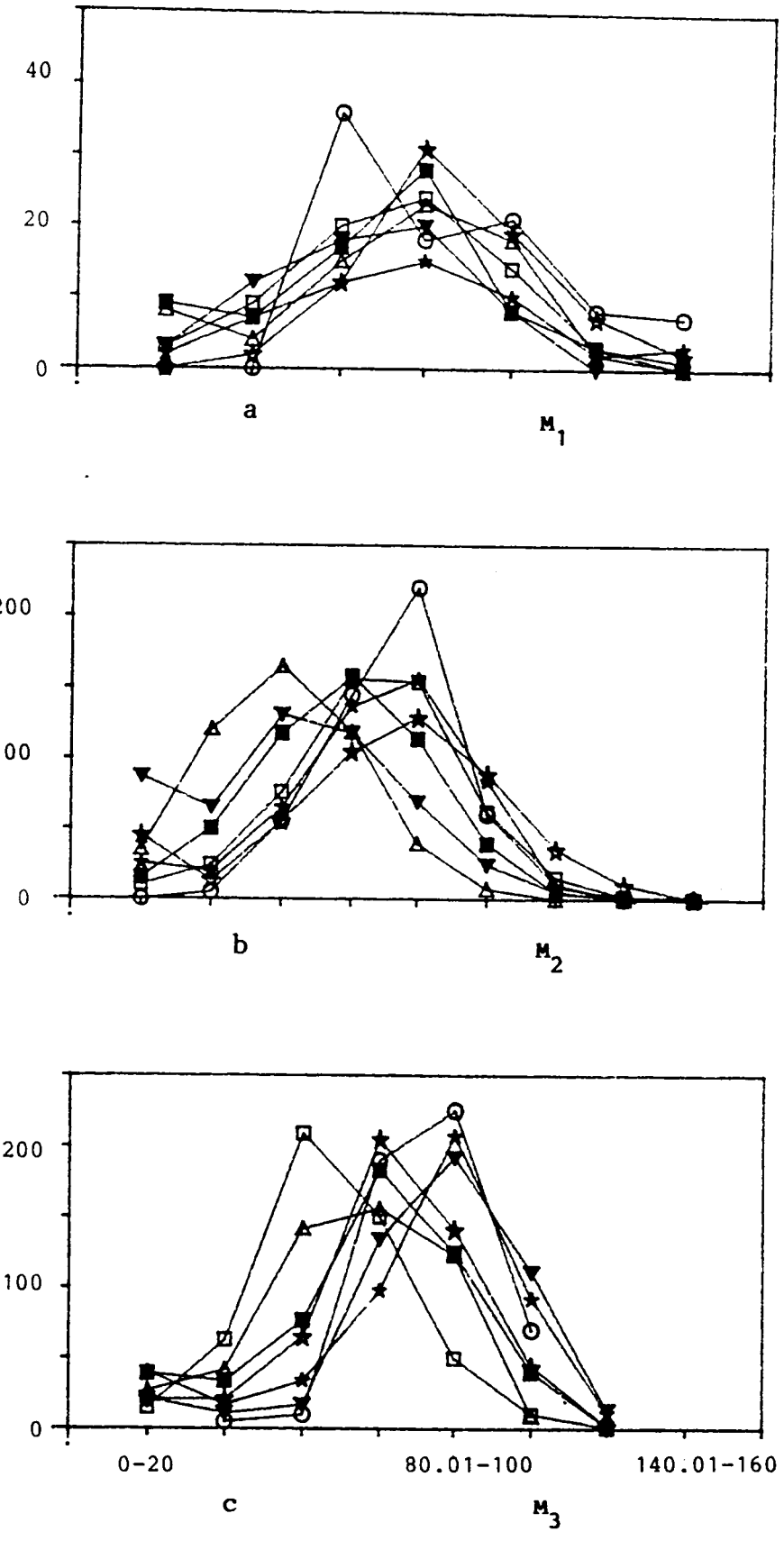


Fig. 27

□ DW+0.5% EMS
■ Vit.C+0.5% EMS
▽ DW+0.75% EMS
▲ Vit.C+0.75% EMS
✡ DW+1% EMS
✱ Vit.C+1% EMS
○ Control

Fig 28. Frequency distribution for percentage of spikelet sterility in M_1-M_3 of EMS treated Japan violet

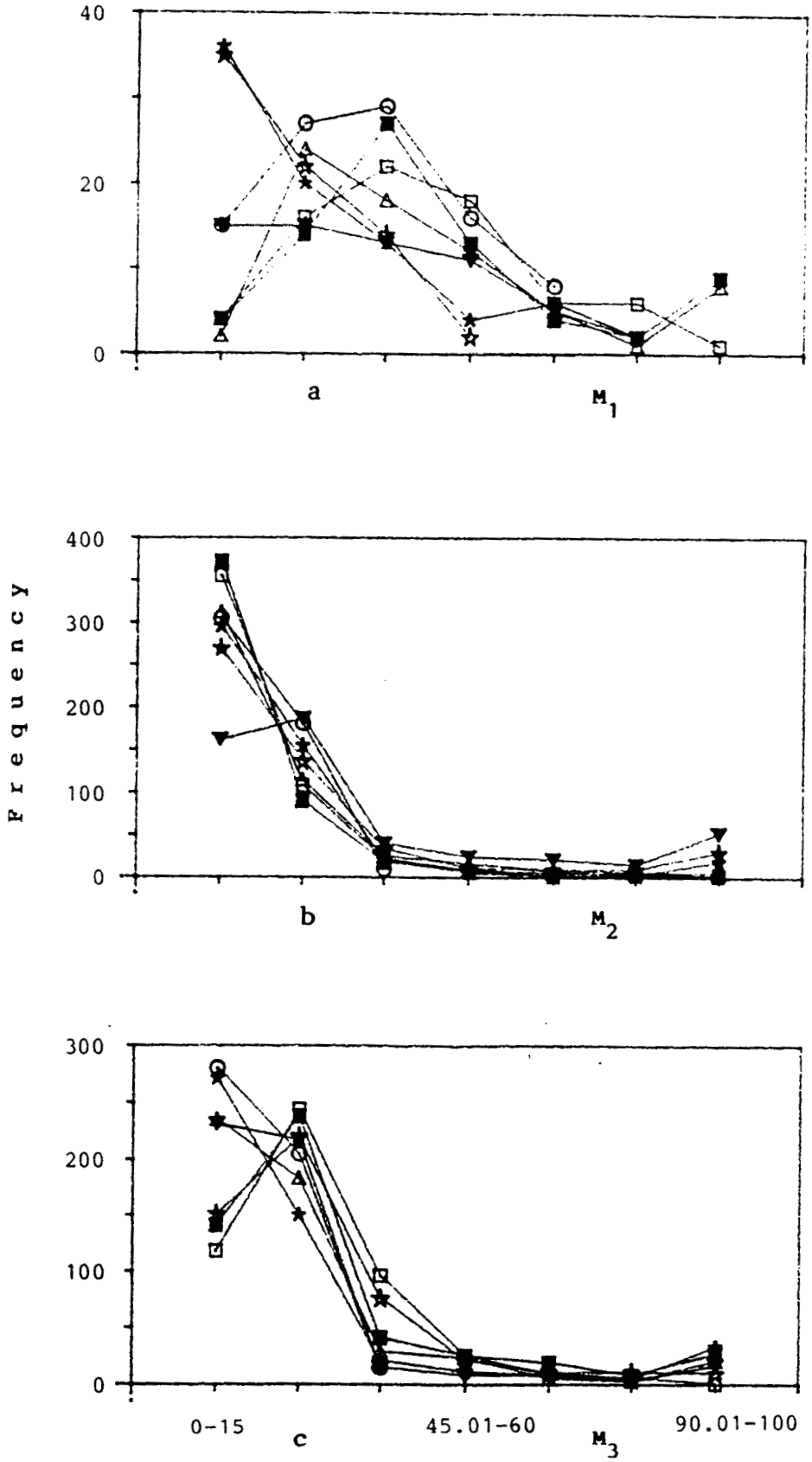


Fig. 28

□ DW+0.5% EMS
■ Vit.C+0.5% EMS
▽ DW+0.75% EMS
▲ Vit.C+0.75% EMS
✲ DW+1% EMS
✱ Vit.C+1% EMS
○ Control

Fig 29. Frequency distribution for panicle density in M_1-M_3 of EMS treated Japan violet

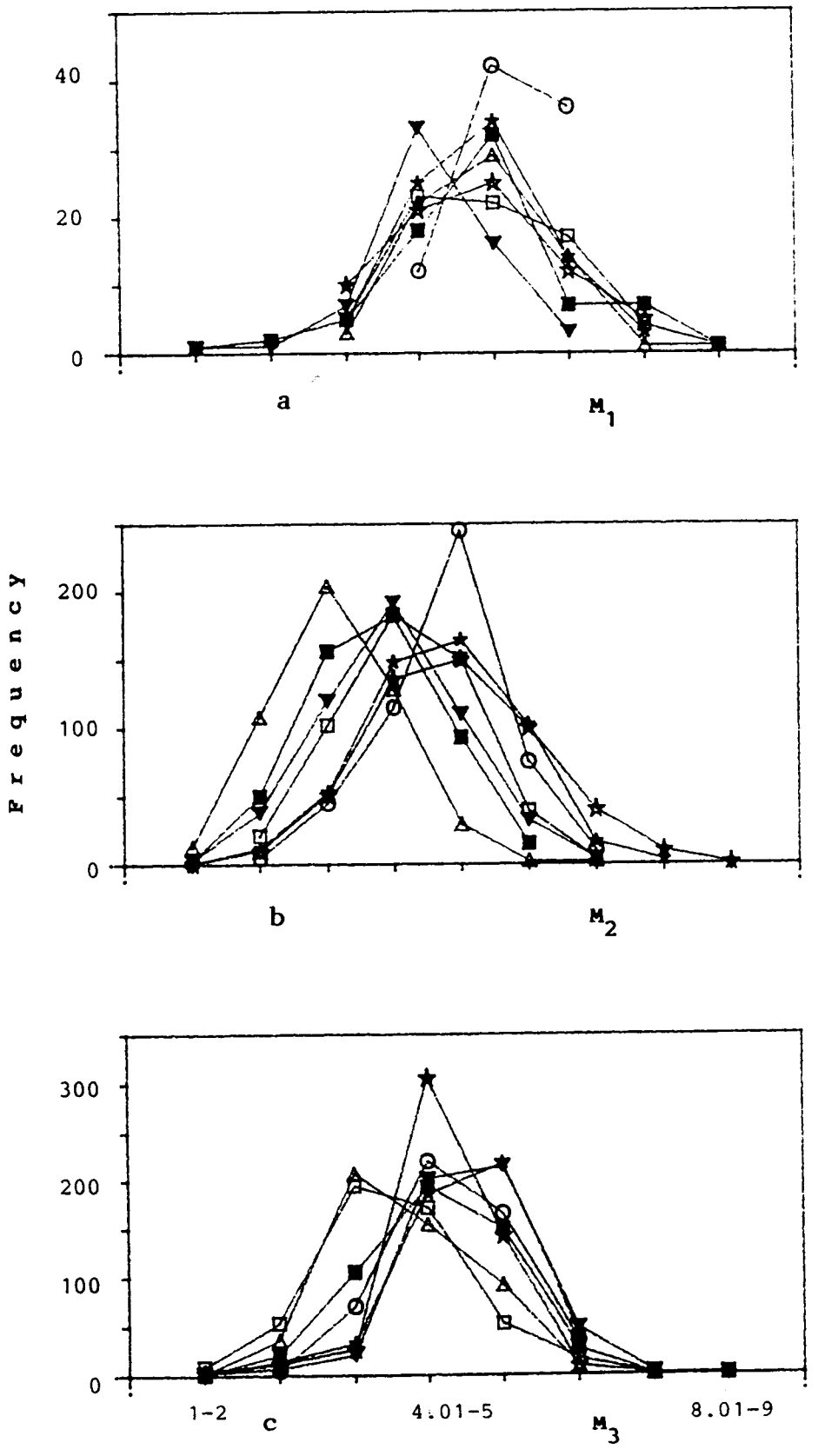


Fig. 29

□ DW+0.5% EMS
■ Vit.C+0.5% EMS
▽ DW+0.75% EMS
▲ Vit.C+0.75% EMS
✡ DW+1% EMS
✱ Vit.C+1% EMS
○ Control

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above characters in M_1, M_2 and M_3 generation are presented in Table 46. The degree of variance and co-efficient of dispersion of variation relative to control (variance index) of these characters in M_1,M_2 and M_3 populations of Japan violet are presented in Table 47. Details of results obtained are presented below under appropriate heads.

1. Plant height

Frequency distribution of M_1 plants of Japan violet treated with EMS showed considerable variation in plant height, exceeding the range of control with maximum plants falling within the range of 80-90 cm (Fig. 20a). M_2 plants also showed variation beyond the limits of control range, most plants falling within 60-70 cm (Fig. 20b). Frequency distribution of plant height of M_3 plants also showed variation exceeding the limits of control, most plants falling within 70-80 cm (Fig. 20c).

Analysis of variance of plant height showed significant variation induced by various treatments in M_1 with F value = 10.43 (Table 46-1). The range of variation in plant height exceeded both limits of control, 72-91 cm. Among M_1 plants shortest plant was observed in the treatment Vit.C + 0.75% EMS (49cm) and the tallest in the treatment DW + 0.75% EMS (99 cm)(Table 46). M_2 plants also

TABLE 46. Observation recorded on range and mean of morphological characters and their respective F values and CD for analysis of variance in M_1, M_2 and M_3 of JapanViolet treated with EMS

Sl. No.	Treatment	M_1			M_2			M_3		
		Range	Mean	F value/CD	Range	Mean	F value/CD	Range	Mean	F value
1	2	3	4	5	6	7	8	9	10	11	12
1. Plant height	Control	72.00 - 91.00	81.47		60.50 - 87.00	72.59		66.00 - 88.0	78.520	
	DW + 0.5% EMS	69.00 - 89.00	82.18		45.00 - 87.00	73.80		30.00 - 85.0	69.970	
	Vit.C + 0.5% EMS	59.00 - 96.00	83.16		31.00 - 91.00	65.14		40.00 - 94.0	43.800	
	DW + 0.75% EMS	71.00 - 99.00	82.45		40.00 - 87.00	66.73		40.00 - 93.0	73.580	
	Vit.C + 0.75% EMS	49.00 - 89.00	71.51		28.00 -105.00	59.33		35.00 - 96.0	75.240	
	DW + 1% EMS	57.00 - 95.00	74.47		26.00 - 97.00	70.27		41.00 - 90.0	75.770	
	Vit.C + 1% EMS	50.00 - 98.00	80.76	10.43* **3.40**	39.00 - 95.00	68.87	10.18* **4.19**	24.50 - 94.0	73.110	14.90* **1.66**
2. Culm length	Control	35.00 - 47.00	40.43		36.50 - 51.50	43.23		40.00 - 53.0	45.880	
	DW + 0.5% EMS	29.50 - 54.50	44.51		30.00 - 57.00	45.63		18.00 - 54.5	41.190	
	Vit.C + 0.5% EMS	25.00 - 56.00	44.97		22.00 - 55.00	43.45		13.50 - 69.5	43.800	
	DW + 0.75% EMS	28.00 - 58.00	44.41		22.00 - 54.00	41.75		14.00 - 53.0	42.650	
	Vit.C + 0.75% EMS	21.00 - 50.00	37.02		18.00 - 82.00	35.95		20.50 - 64.0	42.650	
	DW + 1% EMS	21.00 - 49.00	38.23		17.00 - 56.50	42.83		22.00 - 54.0	43.720	
	Vit.C + 1% EMS	21.00 - 55.00	41.62	11.14* **2.31**	13.00 - 66.00	44.33	8.78* **2.48**	19.00 -56.0	42.050	9.86* **1.17**

1	2	3	4	5	6	7	8	9	10	11	12
3.	EBT	Control	5.00 - 13.00	8.47		4.00 - 15.00	8.98		5.00 - 15.0	8.540	
		DW + 0.5% EMS	2.00 - 19.00	9.51		1.00 - 17.00	5.63		2.00 - 11.0	5.100	
		Vit.C + 0.5% EMS	4.00 - 15.00	9.31		1.00 - 20.00	4.93		2.00 - 11.0	5.250	
		DW + 0.75% EMS	3.00 - 16.00	9.33		1.00 - 15.00	4.98		2.00 - 13.0	5.780	
		Vit.C + 0.75% EMS	1.00 - 19.00	7.58		1.00 - 16.00	6.25		2.00 - 60.0	7.590	
		DW + 1% EMS	3.00 - 16.00	7.51		2.00 - 23.00	8.51		1.00 - 17.0	6.520	
		Vit.C + 1% EMS	4.00 - 15.00	8.12	3.13* **1.17**	2.00 - 16.00	6.12	17.48* **0.89**	1.00 - 16.0	5.730	7.99* **1.10**
4.	EBT%	Control	42.86 -100.00	76.90		41.67 -100.00	88.75		50.00 -100.0	82.02	
		DW + 0.5% EMS	18.00 -100.00	80.74		13.33 -100.00	86.32		25.00 -100.0	74.430	
		Vit.C + 0.5% EMS	45.45 -100.00	80.80		14.29 -100.00	88.17		18.75 -100.0	66.300	
		DW + 0.75% EMS	37.50 -100.00	75.60		33.33 -100.00	85.72		15.38 -100.0	72.090	
		Vit.C + 0.75% EMS	20.00 -100.00	73.02		25.00 -100.00	91.48		25.00 -100.0	69.700	
		DW + 1% EMS	35.29 -100.00	68.95		28.57 -100.00	94.85		40.00 -100.0	84.280	
		Vit.C + 1% EMS	30.00 -100.00	72.22	3.27* **5.97**	44.40 -100.00	95.26	6.98* **1.37**	13.33 -100.0	60.180	14.01* **5.47**

1	2	3	4	5	6	7	8	9	10	11	12
5.	Days to flowering	Control	58.00 - 62.00	60.06		63.00 - 68.00	66.23		64.00 - 67.0	65.550	
		DW + 0.5% EMS	63.00 - 68.00	65.49		57.00 - 72.00	63.35		59.00 - 69.0	64.450	
		Vit.C + 0.5% EMS	60.00 - 65.00	62.30		57.00 - 73.00	63.88		62.00 - 69.0	65.420	
		DW + 0.75% EMS	60.00 - 65.00	61.66		57.00 - 70.00	63.76		62.00 - 69.0	65.820	
		Vit.C + 0.75% EMS	60.00 - 65.00	61.95		57.00 - 70.00	63.03		60.00 - 69.0	65.410	
		DW + 1% EMS	57.00 - 65.00	61.04		59.00 - 68.00	65.75		63.00 - 69.0	65.160	
		Vit.C + 1% EMS	58.00 - 65.00	61.24	23.62* **0.85**	63.00 - 68.00	65.12	4.85* **0.94**	62.00 - 69.0	65.250	3.84* 0.44
6.	Panicle length	Control	15.50 - 20.00	21.24		16.00 - 20.75	17.87		12.75 - 22.7	20.095	
		DW + 0.5% EMS	12.00 - 23.50	20.05		10.10 - 22.00	18.51		11.50 - 22.5	19.050	
		Vit.C + 0.5% EMS	13.50 - 23.00	20.02		10.35 - 22.25	17.78		8.75 - 24.0	19.490	
		DW + 0.75% EMS	17.00 - 23.50	17.74		7.50 - 20.85	16.08		11.50 - 22.5	18.850	
		Vit.C + 0.75% EMS	13.50 - 20.00	17.62		7.50 - 23.50	16.38		10.50 - 23.5	20.350	
		DW + 1% EMS	14.00 - 22.00	17.74		8.50 - 22.00	17.78		9.50 - 24.5	20.090	
		Vit.C + 1% EMS	15.00 - 21.00	21.25	18.92* **0.83**	7.25 - 20.75	17.18	12.29* **3.62**	10.50 - 22.5	19.320	3.84* 0.72

1	2	3	4	5	6	7	8	9	10	11	12
7.	spikelets/ panicle	Control	88.00 -145.00	103.62		45.50 -141.00	94.61		50.00 -122.5	95.850	
		DW + 0.5% EMS	56.00 -191.00	114.73		22.50 -158.00	87.91		24.00 -164.0	76.010	
		Vit.C + 0.5% EMS	53.00 -156.00	105.82		22.00 -145.50	75.89		18.50 -152.5	90.570	
		DW + 0.75% EMS	79.00 -136.00	105.67		14.00 -156.00	59.81		25.50 -116.0	78.610	
		Vit.C + 0.75% EMS	28.00 -118.00	82.31		12.55 -144.00	74.78		19.50 -143.0	102.420	
		DW + 1% EMS	54.00 -141.50	91.90		13.50 -203.50	97.43		17.50 -139.5	94.100	
		Vit.C + 1% EMS	53.00 -139.00	93.81	11.68* **7.65**	14.50 -174.50	90.73	14.20* **8.61**	11.00 -139.0	96.920	9.98* **7.50**
8.	grains/ panicle	Control	45.00 -128.50	81.52		36.50 -129.00	79.50		33.50 -109.5	82.220	
		DW + 0.5% EMS	11.00 -119.00	63.51		0.50 -149.50	76.73		1.50 -149.5	57.550	
		Vit.C + 0.5% EMS	0.00 -128.00	56.98		0.00 -140.00	67.49		0.00 -136.5	67.080	
		DW + 0.75% EMS	0.00 -128.00	57.55		0.00 -115.50	50.88		0.00 -108.5	62.980	
		Vit.C + 0.75% EMS	11.50 - 99.00	56.11		0.00 -128.00	53.67		0.00 -131.5	83.500	
		DW + 1% EMS	34.00 -136.50	66.31		0.00 -190.50	78.05		0.00 -120.5	72.100	
		Vit.C + 1% EMS	11.50 -134.00	72.70	2.72* **13.83**	0.00 -169.50	76.35	13.60* **7.89**	0.00 -129.5	78.160	7.58* **8.67**

1	2	3	4	5	6	7	8	9	10	11	12
9.	Percentage of sterility	Control	2.22 - 33.75	20.39		4.44 - 31.11	14.57		3.97 - 44.7	14.475	
		DW + 0.5% EMS	1.86 - 91.06	43.48		0.87 - 98.76	13.54		2.99 - 97.3	25.160	
		Vit.C + 0.5% EMS	10.00 -100.00	47.20		0.52 -100.00	12.48		2.10 -100.0	27.950	
		DW + 0.75% EMS	0.00 - 84.56	45.97		0.66 -100.00	16.74		4.85 -100.0	22.280	
		Vit.C + 0.75% EMS	0.00 - 80.99	35.91		1.95 -100.00	31.85		2.67 -100.0	20.040	
		DW + 1% EMS	0.00 - 54.36	28.70		0.00 -100.00	22.39		4.52 -100.0	25.090	
		Vit.C + 1% EMS	0.00 - 83.69	23.57	6.11* **10.75**	1.48 -100.00	18.05	13.63* **4.45**	2.23 -100.0	22.060	3.28* **5.81**
10.	Panicle density	Control	4.56 - 7.84	5.83		2.68 - 7.42	5.27		3.97 - 6.4	4.477	
		DW + 0.5% EMS	3.16 - 9.67	5.44		2.19 - 7.71	4.69		1.33 - 8.1	4.007	
		Vit.C + 0.5% EMS	1.83 - 8.33	5.31		1.80 - 6.93	4.20		1.55 - 8.2	4.612	
		DW + 0.75% EMS	3.90 - 8.00	5.29		1.09 - 7.41	3.64		1.70 - 7.4	4.162	
		Vit.C + 0.75% EMS	1.56 - 6.38	4.63		1.47 - 7.12	4.45		1.44 - 7.2	5.041	
		DW + 1% EMS	3.16 - 7.62	5.21		1.59 - 9.69	5.39		1.84 - 6.7	4.704	
		Vit.C + 1% EMS	3.00 - 7.72	5.28	6.04* **0.35**	1.96 - 8.73	5.20	18.69* **0.36**	1.05 - 7.6	4.998	6.58* **0.36**

* significant at 5% level.

Table 47. Observation on the degree of variance (%) in morphological characters in M_1, M_2 and M_3 populations of Japan violet treated with EMS

Sl. No.	Character	Variety/ treatment	M_1		M_2		M_3	
			-	+	-	+	-	+
1	2	3	4	5	6	7	8	9
1.	Plant height	Japan violet						
		Control	11.62	11.70	16.66	19.85	15.94	12.07
		DW + 0.5% EMS	15.31	9.24	38.01	22.61	61.79	8.25
		Vit C + 0.5% EMS	34.94	17.83	57.29	25.36	49.06	19.71
		DW + 0.75% EMS	12.85	17.53	44.90	19.85	49.06	18.44
		Vit C + 0.75% EMS	39.86	9.24	61.43	44.65	55.43	22.26
		DW + 1% EMS	30.04	16.61	64.18	33.63	47.78	14.62
		Vit C + 1% EMS	38.63	20.29	46.27	30.87	68.80	19.71
		Mean (Treatments)	28.61	15.12	52.01	29.50	55.32	17.17
		Variance index	1.46	0.29	2.12	0.49	2.47	0.42
2.	Culm length							
		Control	13.43	16.25	15.57	19.13	12.82	15.52
		DW + 0.5% EMS	27.03	34.80	30.60	31.85	60.77	18.79
		Vit C + 0.5% EMS	38.16	38.51	49.11	27.23	70.58	51.48
		DW + 0.75% EMS	30.74	43.46	49.11	24.91	69.49	15.52

1	2	3	4	5	6	7	8	9
		Vit C + 0.75% EMS	48.06	23.67	58.36	89.68	55.32	39.49
		DW + 1% EMS	48.06	21.20	60.68	30.70	52.32	17.07
		Vit C + 1% EMS	48.06	36.04	69.93	52.67	58.59	22.06
		Mean (Treatments)	40.02	32.95	52.97	42.84	61.18	27.40
		Variance index	1.98	1.03	2.40	1.24	3.77	0.77
3.	No. of EBT							
		Control	40.97	53.48	55.46	67.04	41.45	75.64
		DW + 0.5% EMS	76.39	124.32	88.86	89.31	76.58	28.80
		Vit C + 0.5% EMS	52.77	77.10	88.86	122.72	76.58	28.80
		DW + 0.75% EMS	64.58	88.90	88.86	67.04	76.58	52.27
		Vit C + 0.75% EMS	88.19	124.32	88.86	78.17	76.58	602.57
		DW + 1% EMS	64.58	88.90	77.73	156.12	88.29	101.42
		Vit C + 1% EMS	52.77	77.10	77.73	78.17	88.29	87.37
		Mean (Treatments)	66.55	96.77	88.15	98.59	80.48	150.20
		Variance index	0.29	0.81	0.54	0.47	0.94	0.99
4.	EBT %							
		Control	44.27	30.04	53.05	12.68	38.76	22.49
		DW + 0.5% EMS	76.59	30.04	84.98	12.68	69.38	22.49
		Vit C + 0.5% EMS						

1	2	3	4	5	6	7	8	9
		DW + 0.75% EMS	51.24	30.04	62.45	12.68	81.16	22.49
		Vit C + 0.75% EMS	73.99	30.04	71.83	12.68	69.38	22.49
		DW + 1% EMS	54.11	30.04	67.81	12.68	51.00	22.49
		Vit C + 1% EMS	60.99	30.04	49.97	12.68	83.67	22.49
		Mean (Treatments)	59.64	30.04	70.16	12.68	71.94	22.49
		Variance index	0.99	0.00	0.32	0.00	0.86	0.00
5.	Days to flower	Control	3.43	3.23	4.88	2.67	1.77	2.21
		DW + 0.5% EMS	4.90	13.22	13.94	8.71	9.44	5.26
		Vit C + 0.5% EMS	0.001	8.23	13.94	10.22	4.83	5.26
		DW + 0.75% EMS	0.001	8.23	13.94	5.69	4.83	5.26
		Vit C + 0.75% EMS	0.001	8.23	13.94	5.69	7.90	5.26
		DW + 1% EMS	5.09	8.23	10.92	2.67	3.30	5.26
		Vit C + 1% EMS	3.43	8.23	4.88	2.67	4.83	4.37
		Mean (Treatments)	2.24	9.06	11.93	5.94	5.86	5.26
		Variance index	-0.35	1.82	1.44	1.22	2.31	1.38
6.	Panicle length	Control	27.02	5.84	10.46	16.12	6.67	13.24
		DW + 0.5% EMS	43.50	10.64	43.48	23.11	42.76	12.00
		Vit C + 0.5% EMS	36.44	8.23	42.08	24.51	56.45	19.46

1	2	3	4	5	6	7	8	9
		DW + 0.75% EMS	19.96	10.64	58.03	16.68	42.76	12.00
		Vit C + 0.75% EMS	36.44	5.83	58.03	31.51	52.26	166.30
		DW + 1% EMS	34.09	3.58	52.43	23.11	52.71	21.95
		Vit C + 1% EMS	29.38	1.13	59.43	16.12	47.74	12.00
		Mean (Treatments)	33.30	6.69	52.25	22.51	49.11	40.62
		Variance index	0.57	0.69	4.00	0.40	6.36	2.07
7.	Spikelets/ panicle							
		Control	15.07	39.93	51.91	49.03	47.84	27.80
		DW + 0.5% EMS	45.96	84.33	76.22	67.00	74.96	71.10
		Vit C + 0.5% EMS	48.85	50.55	76.75	53.79	80.70	59.10
		DW + 0.75% EMS	23.76	31.25	85.20	64.89	73.40	21.02
		Vit C + 0.75% EMS	72.98	13.88	86.74	52.20	79.66	49.19
		DW + 1% EMS	47.89	36.56	85.73	62.25	81.74	45.53
		Vit C + 1% EMS	48.85	34.14	84.67	84.44	88.52	45.02
		Mean (Treatments)	48.05	41.75	82.55	64.10	79.83	48.49
		Variance index	1.25	0.05	0.68	0.31	0.67	0.74

1	2	3	4	5	6	7	8	9
8.	Grains/ panicle							
		Control	36.46	60.08	54.08	62.26	58.18	36.69
		DW + 0.5% EMS	86.50	45.97	99.37	88.05	98.18	86.62
		Vit C + 0.5% EMS	0.00	57.01	0.00	76.10	0.00	70.39
		DW + 0.75% EMS	0.00	57.01	0.00	45.28	0.00	35.44
		Vit C + 0.75% EMS	85.89	21.44	0.00	61.00	0.00	64.15
		DW + 1% EMS	58.29	67.44	0.00	139.62	0.00	50.42
		Vit C + 1% EMS	85.89	64.37	0.00	113.21	0.00	61.65
		Mean (Treatments)	52.76	52.20	16.56	87.21	16.36	61.45
		Variance index	0.45	-0.13	-0.71	0.40	-0.72	0.67
9.	Percent of spikelet sterility							
		Control	65.09	65.52	69.52	113.52	52.00	167.16
		DW + 0.5% EMS	91.00	346.59	94.02	577.76	82.15	480.90
		Vit C + 0.5% EMS	49.70	403.02	96.43	586.34	87.46	497.01
		DW + 0.75% EMS	0.00	325.05	95.47	586.34	71.04	497.01
		Vit C + 0.75% EMS	0.00	307.39	87.00	586.34	84.06	497.01
		DW + 1% EMS	40.44	173.44	0.00	586.34	73.01	497.01
		Vit C + 1% EMS	0.00	320.98	89.84	586.34	86.69	497.01
		Mean (Treatments)	30.19	314.66	77.12	584.91	80.74	494.33
		Variance index	-0.54	3.80	0.11	4.15	0.36	1.96

1	2	3	4	5	6	7	8	9
10.	Panicle density	Control	21.78	34.48	49.15	40.80	11.18	43.17
		DW + 0.5% EMS	47.80	65.87	58.44	46.30	70.24	81.20
		Vit C + 0.5% EMS	68.61	42.88	65.84	31.50	65.32	83.45
		DW + 0.75% EMS	33.10	37.22	79.32	40.61	61.97	65.55
		Vit C + 0.75% EMS	73.24	9.43	72.11	35.10	67.79	61.07
		DW + 1% EMS	45.80	30.70	69.83	83.87	58.83	49.89
		Vit C + 1% EMS	48.54	32.42	62.81	65.65	58.84	70.02
		Mean (Treatments)	52.85	36.42	68.06	50.42	63.83	68.53
		Variance index	1.43	0.06	0.38	0.24	4.70	0.33

- = decrease, + = increase

showed wider range of variation in plant height than the control range of 60.5-87 (Table 46). The shortest plant with 26 cm and the tallest plant with 105 cm were observed in the treatments DW + 1% EMS and Vit.C + 0.75% EMS respectively. Significant variation was observed in M_2 with F value = 10.18 and in M_3 with F value = 14.90 (Table 46-1). The range of variation in all treatments exceeded the limits of control, indicating probable induction of transgressive polygenic variation in plant height.

Variance index of plant height based on the mean expression relative to control showed linear reduction in plant height (negative effect) in M_1, M_2 and M_3 of Japan violet, while the positive effect was comparatively higher in M_2 (0.49) and more reduction was observed in M_3 (2.47). However, the response of Japan violet to the mutagen appeared to be bidirectional (Table 47-1).

2. Main culm length

Frequency distribution of M_1 plants of Japan violet treated with EMS showed variation in main culm length, exceeding both limits of control with maximum plants falling within 45-50 cm and showing wider variation with DW+0.75% EMS treatment (Fig. 21a). Frequency distribution of M_2 plants also showed variation in main culm length exceeding both limits of control with majority of plants falling

within the control range, with widest variation observed with DW + 1% EMS treatment (Fig. 21b). M_3 plants showed a trend of deviation towards reduction in main culm length, with most plants falling within the range of the control in all the six treatments (Fig. 21c).

Analysis of variance of culm length showed significant variation in M_1. It varied from 21-58 cm among the treatments when compared to the control range of 35-47 cm. In M_1 lowest culm length was shown by three treatments such as Vit.C + 0.75% EMS, DW + 1% EMS and Vit. C + 1% EMS with 21 cm, and highest culm length was shown by plants in the treatment of DW + 0.75% EMS with 58 cm. M_2 and M_3 also showed significant variations in culm length compared to the respective controls. Analysis of variance of culm length in M_1, M_2 and M_3 gave F values = 11.14, 8.78 and 9.86 respectively (Table 46-2), which are highly significant against the table value 2.42, the range of variation exceeding both limits of control in almost all cases. This represents a situation of induction of polygenic variation in culm length due to EMS treatment.

Varince index or the co-efficient of dispersion of variation relative to control in main culm length showed an increasing trend of reduction of main culm length from M_1-M_3, 1.98, 2.40 and 3.77. However, the positive effect was comparatively higher in M_2 (1.24) than in M_1 and M_3 (1.03 and 0.77) populations (Table 47-2).

3. Number of EBT

Frequency distribution of number of EBT in M_1 of Japan violet treated with EMS showed variation exceeding both limits of control, with most plants falling within the control range (Fig. 22a). M_2 plants also transgressed the limits of control, with a maximum variation observed in DW+1% EMS treatment (Fig. 22b). However, maximum number of M_2 plants fell within the range of the control. M_3 populations of all treatments showed reduction in EBT than the control with more number of plants with reduced EBT number being observed in the treatment Vit.C + 1% EMS (Fig. 22c). A few plants in the treatment of Vit. C + 0.75% showed higher number of EBT (Fig. 22c).

Analysis of variance for number of EBT in M_1, M_2 and M_3 showed significant variation with F values = 3.13, 17.45, and 7.99 respectively (Table 46-3) with range of variations exceeding the control limits. The high range of variation in EBT in treated plants in comparison to their respective control may be due to the possible induction of polygenic variation.

Variance index or co-efficient of dispersion of variation relative to control in number of EBT in treated populations showed bidirectional induction of variation in M_1-M_3 population. However

there is an increasing trend to the positive direction in M_1 and M_3 with 0.81 and 0.99, while in M_2 the trend was towards reduction (0.54) in EBT (Table 47-3).

4. Percentage of EBT

Frequency distribution of M_1 plants of Japan violet treated with EMS showed more variation in percentage of EBT exceeding the lower limit of the control range with most plants falling within the range of control (Fig. 23a). M_2 plants also showed variation in EBT percentage exceeding the lower limit of control range with most plants falling within the control range of 91-100 (Fig. 23b). Frequency distribution of the M_3 plants showed similar trend exceeding the lower limit of control with maximum plants falling within the range of control (Fig. 23c).

Analysis of variance of EBT% in M_1, M_2 and M_3 showed significant variation with F values = 3.27, 6.98 and 14.01 respectively with the range of variation exceeding the lower limits of the control (Table 46-4).

Variance index or co-efficient of dispersion of variation relative to control of EBT % showed reduction in M, M_2 and M_3 populations with values 0.99, 0.32 and 0.86. However, the decreasing

trend is comparatively low in M_2 (0.32) and highest in M_1 (0.99) Table 47-4.

5. Days to flower

Frequency distribution of plants in M_1 of Japan violet treated with EMS showed significant variation towards delay in flowering (increase in days to flower) exceeding the limit of control range particularly in respect of three treatments, DW + 0.5% EMS , Vit.C + 0.5% EMS, and Vit.C + 1% EMS (Fig. 24a). However, M_2 population of Japan violet also showed considerable variation in days to flowering by exceeding the limits of control, the variation being bidirectional with maximum plants falling within the control range (Fig. 24b). The days to flower showed increase in M_2 of the treatment DW + 0.75% EMS, while in treatment Vit.C + 0.75% EMS gave maximum number of early flowering plants. However, the M_2 of different treatment showed a positive trend towards delay in flowering when compared to early flowering. M_3 population of all the treatments also reflected delay in flowering when compared to the control and the transgression was more towards the increase in days to flower (Fig. 24c).

Analysis of variance of days to flower in M_1, M_2 and M_3 generations of Japan violet showed significant variation in days to flower with F-values = 23.62, 4.85, and 3.84 respectively (Table 46-5).

The variance index or co-efficient of dispersion of variation relative to control for days to flower showed bidirectional induction of variation in M_1 to M_3 populations. However, a trend in increasing the days to flower was observed with variance index 1.82, 1.22 and 1.38 in M_1, M_2 and M_3 respectively with maximum in M_1 (Table 47-5).

6. Panicle length

Frequency distribution for panicle length in M_1 population of Japan violet treated with EMS showed variation in panicle length exceeding the limits of the control (Fig. 25a). Panicle length showed increase with the treatments of DW +0.5% EMS, Vit. C + 0.5% EMS and DW _ 0.75% EMS treatments (Fig. 25a). However, few plants had reduced panicle length than the control range of 15-21 cm, while most plants fell within the control range. M_2 population of all six treatments showed reduction in panicle length than the control, while maximum number of plants falling within the range of 15-18 to 18-21 and also towards lower range of 12-15 beyond the control (Fig. 25b), probably indicating recessive polygenic mutation. In M_3 also frequency distribution of plants for panicle length showed much variation, mostly exceeding the lower limits of the control. Very few plants fell beyond the upper range of the control in the treatment of Vit. C + 0.5%, Vit. C + 0.75% and DW + 1% treatments. However, panicle length in most of the plants in the treated populations remained within the control range (Fig. 25c).

Analysis of variance of panicle length in Japan violet treated with EMS showed significant variation in M_1, M_2 and M_3 with F values = 18.92, 12.29 and 3.84 respectively (Table 46-6). The range of variation exceeded both limits of the control in most of the treatments. In M_2 lower range of mean values in treated populations showed comparatively low values than the control except DW + 0.5% EMS. In M_3 Vit C + 0.75% EMS showed slightly high mean value than the control, while in DW + 1% EMS mean value was equal to the control and in other treatments means were less than the control (Table 46-6). The results indicated induction of polygenic variation due to mutagenesis. Mean values for ranges of expression of panicle length showed bidirectional induction of variation in M_1 to M_3 (Table 46-6). However, the expression showed a diminishing trend to positive side when compared to the negative side.

The co-efficient of dispersion of variation or variance index showed a linear reduction in panicle length from M_1 to M_3 generations with variance index 0.57, 4.00 and 6.36 respectively (Table 47-6).

7. Spikelets/Panicle

Frequency distribution for spikelets/panicle in M_1 population of Japan violet treated with EMS showed wider range of variation exceeding the limits of the control 80-120, most plants in all

treatments falling within the control range (Fig. 26a) M_2 plants also showed variation in spikelets per panicle exceeding both limits of control range. The treatments Vit. C + 0.5%, DW + 0.75% and Vit. C + 0.75% EMS caused reduction in total spikelets/panicle, while DW + 1% and Vit. C + 1% EMS showed variation exceeding the control limits with higher frequency towards increasing spikelet number/panicle (Fig. 26b). M_3 plants of DW + 0.5%, Vit. C + 0.5%, Vit.C 0.75%, DW + 1% and Vit.C + 1% EMS treatments also showed variations transgressing the limits of control, with most plants in all treatments falling within the control range, 60-140 (Fig. 26c).

Analysis of variance of spikelets/panicle in Japan violet treated with EMS showed significant variation in M_1, M_2 and M_3 with F values = 11.68, 14.20 and 9.98 respectively (Table 46-7). In most of the treatments range of variation exceeded the both limits of control in M_1, M_2 and M_3 (Table 46-7). In M_1, DW + 0.5% EMS, Vit.C +0.5% EMS and DW +0.75% EMS showed higher mean values than the control. M_2 population of all the treatments showed reduction in mean values than the control except DW + 1% EMS treatment. In M_3 Vit C + 0.75% EMS and Vit C + 1% EMS treatments showed greater mean values than the control but M_3 of other treatments had comparatively low values than control. In M_3 range of variation exceeded both limits of the control in most of the cases exhibiting polygenic variation induced by mutagenic treatment (Table 46-7).

Variance index or co-efficient of dispersion of variants in spikelets/panicle showed a gradual increase in M_2 and M_3 (0.31 and 0.74) with bidirectional trend in variation, while M_1 showed higher reduction in spikelets/panicle (1.25) (Table 47-7).

8. Grains/Panicle

Frequency distribution for grains/panicle in M_1 of Japan violet treated with EMS showed more variation by exceeding the limit of control range 40-140 with maximum plants falling within the range of control (Fig. 47a). M_2 populations of the six treatments showed wider range of variation in grains/panicle. In the treatments DW+ 0.5%, Vit.C + 0.5%, DW + 1% and Vit.C + 1% showed transgression towards increase in grains/panicle and a decreasing trend beyond the lower range limit of the control 40-60, (Fig. 27b). M_3 population also showed variation in grains/panicle beyond the control range of 40-120 with maximum plants falling within the range of control (Fig. 27c).

Analysis of variance of grains/panicle in Japan violet treated with EMS showed significant variation in M_2 and M_3 generations with F values = 13.60 and 7.58, but not in M_1 (Table 46-8). Mean values showed a reductional trend in M_2 of all treatments when compared to the control, while range of variation exceeded both limits of

control in most of the cases of M_1,M_2 and M_3. In M_3, except the treatment Vit C + 0.75% EMS, all other treatments showed very low mean values than the control (Table 46-8). The results again presented polygenic transgressive variation, probably, induced by the mutagenic effect.

Variance index or co-efficient of dispersion of variants relative to control showed highly significant reduction in grains per panicles in M_1 generation (0.45). However M_2 and M_3 populations showed an increase in grains per panicle (0.40 and 0.67) (Table 47-8).

9. Percentage of spikelet sterility

Frequency distribution of plants with spikelet sterility in M_1 of Japan violet treated with EMS showed an increasing trend in spikelet sterility beyond the upper limit of the range of control (Table 46-9) with maximum plants falling within the control range (Fig. 28a). M_2 also showed unidirectional trend towards increase of spikelet sterility beyond the upper limit of control range. Maximum plants with high sterility (75-100) were induced by DW + 1%EMS treatment. Most of the plants of other treatments were with the frequency of control range (Fig. 28b). M_3 also showed unidirectional trend in variation in sterility, more increase than the control range with maximum plants falling within control range in respect of all treatments (Fig. 28c).

Analysis of variance of spikelet sterility in M_1, M_2 and M_3 showed significant variation with F values = 6.11, 13.63 and 3.28 respectively (Table 46-9). Range of variation exceeded both limits of control in all treatments. Highest range of sterility, 100% was shown by all the treatments except DW + 0.5% EMS in M_1, M_2 and M_3 populations (Table 46-9). The results showed polygenic control of variation in sterility induced by the mutagen.

Variance index or co-efficient of dispersion of variations relative to control for percentage of spikelet sterility showed considerable increase of sterility in M_1 and M_2 (3.80 and 4.15) and reduction in percentage of sterility in M_3 (Table 47-9).

10. Panicle density

Frequency distribution for panicle density in M_1 of Japan violet treated with EMS showed variation exceeding the ranges of control with maximum plants falling within the control range 4-7 (Fig. 29a). M_2 plants also showed similar trend in variation in panicle density. However, the treatment DW + 1% EMS showed more increasing trend in variation in panicle density with most plants falling within the control limits (Fig. 29b). M_3 population of Japan violet showed bidirectional trend in variation exceeding the limits of control with most plants falling within the mid-range of control (Fig. 29c).

Analysis of variance of panicle density also showed significant variation in M_1, M_2 and M_3 with the F values = 6.04, 18.69, and 6.58 respectively (Table 46-10) with range of variations exceeding the limits of the control (Table 46-10). In all cases of M_1 the mean values are less than control, while in M_2, DW + 1% EMS treatment and in M_3, Vit.C + 0.5% EMS, Vit.C + 0.75% EMS, DW + 1% EMS, and Vit .C + 1% EMS showed slightly high values than the control (Table 46-10) thereby exhibiting induction of polygenic variation by the mutagenic effects.

Variance index or co-efficient of dispersion of variations relative to control for panicle density showed bidirectional induction of variation, while more reduction observed in M_1 (1.43) than in M_2 and M_3. However , it showed a gradual increase from M_1 - M_3 (0.06, 0.24 and 0.33) populations (Table 47-10).

DISCUSSION

D I S C U S S I O N

The present investigation refers to studies on (a) mutagenesis of Japan Violet treated with different concentrations of EMS with or without pre-soaking in Vitamin C solution, (b) hybridization experiments using the cross Cherumodan X Japan Violet as control and the same cross combintion wherein 1500, 2000 and 5000 rads X-ray irradiated pollen grains of Japan Violet were used for pollination and studied upto F_3/M_3F_3 generations and (c) 14 crosses between Japan Violet and EMS-induced mutants studied upto F_3 generations as detailed elsewhere (Tables 19 & 20). Observations enabled the study of 23 macromutations and 10 genomutations. Macromutants/morphomutants are visually identifiable morphological mutations, while genomutants are not morphologically identifiable, but elicited through altered or transformed genetical ratios. Further, inheritance and interrelationship of 14 morphological characters, biological effects, mutagenic effectiveness and efficiency of the mutagen were also studied. The study includes analysis of micromutations also on the basis of variance index. As such, the present investigation has broughtforth considerable new information on macromutations, micromutations, transformation of genetical ratios, inheritance and interrelationships of morphological characters in rice as discussed below under appropriate heads.

A. GENERAL CONSIDERATIONS ON MUTAGENICITY

The problem of mutation and mutagenicity has been considered by many in the past ever since Hugo de-Vries asserted its existence by 1901. Series of studies reported in the current century have elucidated the phenomenon of mutagenicity in relation to several physical and chemical mutagens. However, Neutrons and EMS have been reported to be the most efficient physical and chemical mutagens respectively (Loveless and Howarth, 1959; Gaul, 1958, 1964; Konzak et al., 1965; Siddiq, 1967; Soriano, 1968; Swaminathan, 1969a; Kaul and Bhan, 1977). The effectiveness and efficiency of the mutagen depends on many intrinsic and extrinsic factors as reviewed elsewhere (Cervigini and Belli, 1962; Konzak et al., 1964; Nilan et al., 1964; Heslot, 1964). However, relative biological effectiveness of mutagens has been evaluated with rate of germination, seedling growth, survival, fertility, mutation frequency and so on (Matsmura, 1964; Siddiq, 1967; Kawai, 1968). Further, dosages, duration of treatments, pre-treatments and post-treatment processing are also important as deterministic factors of mutagenicity and its efficiency. Observations presently recorded refer to mutagenic effectiveness and efficiency of EMS in relation to various treatments, particularly with reference to pre-treatments with distilled water/Vit. C solution. This approach has yielded certain new information on the mutagenicity of EMS as discussed below.

1. Studies on germination

Rate of seed germination observed in M_1, M_2 and M_3 generations of Japan Violet pre-treated with distilled water/0.01% Vit. C solution and treated with 0.5, 0.75 and 1.0% aqueous solution of EMS showed that percentage of germination was comparatively lowest in M_2 generation in all treatments with maximum reduction occurred in 1% EMS treatment (Table 7) in conformity with certain early reports Shobha (1993). The mean percentage of germination for M_1 of all treatments showed 88.7%, for M_2 of all treatments 68.8% and for M_3 of all treatments 80.2%. Early studies on germination were mostly confined to M_1 level only (Myttenare et. al., 1965; Siddiq, 1967, Ganashan, 1970; Kumar and Mallick, 1986). However, the effect of Vit. C was better shown with 1% EMS where percentage of germination was significantly higher (Table 7).

However, in one of the treatments (0.5% EMS) 5 plants out of 74 in M_1 generation showed abnormal type of germination in the rainy season which was termed as **cleisto-viviparous germination** (Pavithran and Santhosh lal, 1992). Out of 43 seeds which showed vivipary, 43.8% showed cleiso-viviparous germination. Cleistoparous germination has been reported earlier in rice (Pavithran and Shobha, 1989). Cliestopary refers to penetration of the plumule through the lemmar space leading to its anterior emergence. In the present situation

cleistopary is coupled with viviparous germination and exhibited three types of situations. The first type is cleistovivipary with anterior emergence of the plumule during viviparous germination and 11.63% of germinated seeds showed this type of germination. In the second case both plumule and radicle emerged through the anterior end of the grain or through the side of the grain by break-opening the side of the lemma-palea and nearly 32.65% germinated this way. In the third type the dynamic plumular growth pushed the radicle and endosperm out of the lemma-palea leading to eject out the young seedling and this situation occurred with 4.65% of the seeds germinated (Fig 3).

Grass seed germination exhibits certain primary outward manifestation of coleorhizal and coleoptile enlargement followed by elongation of the primary root and embryonic shoot, thereby both root and shoot grow through the micropylar region (Gould, 1968). Emergence of shoot or root through the anti-micropylar region or the anterior region of the grain is an abnormal situation and such a situation causing cleistoparous germination has been reported in rice (Pavithran and Shobha, 1989) from this laboratory. However, the present study has furnished more information, as the seeds of Japan Violet showed cleistoparous germination, of which some of the 0.5% EMS treated plants expressed three types in different proportions as described earlier. The abnormal germination pattern has been

attributed to a shift in the plumular growth of subsequent axillary development followed, probably, by decapitation due to mutagenic injury (Pavithran and Shobha, 1989). However, none of the population of the later generation expressed cleistoparous condition and, hence, it may not have any genetic control, but may be physiological.

The third type of cleisto-viviparous germination described above exhibits certain dynamic features of the germination force. It seems under viviparous condition the micropylar expansion does not appear to be normal. The dynamic growing point of the plumule develops a push-back force upon the endosperm which gets ejected through anti-micropylar end. The root growth is found to be comparatively slow, probably, due to the energetic growth pattern of plumule. Further, the direction of the plumular growth also appears to be straight towards the anterior side rather than at the 45° as seen in normal cases. The dynamic plumular elongation must be the primary cause for the poor root growth and its push-back force to eject out the endosperm as seen in the Fig. 3e. The vivipary further accelerates the haste in germination due to lack of dormancy.

Further, observation on radicle/plumule and radicle-plumule inhibition occurred with different concentrations of EMS showed that Vit. C + 1% EMS caused maximum plumule inhibition as shown in Table

- 9. On the whole a linear increase in radicle, plumule inhibition could be observed with the optimum reaching between 0.75 % and 1 % EMS treatments (Table 9).

2. Root/shoot growth

Studies on root-shoot growth and root/shoot ratio showed that plumule growth was more affected by the treatment than the radicle growth which is in conformity with the early reports (Ganashan, 1970; Ramesh, 1984; Mohanan, 1988; Shobha, 1993). Maximum inhibition of plumule was observed with DW + 1 % EMS and radicle inhibition with Vit. C + 0.75% EMS. However, root-shoot ratio showed significant variation in M_2 of DW + 0.75%, DW + 1 % EMS and in M_3 of Vit. C + 0.75 % EMS and in M_1, M_2 and M_3 of Vit. C + 1% EMS. This indicated recovery of root-shoot growth in later generations and the influence of pre-treatment with Vit. C followed by 1% EMS treatment, as it was found to be affecting M_1, M_2 and M_3 generations (Table 7). However, the results are in conformity with the postulate on bio-synthesis activities by ascorbic acid and stimulation of growth and development processes in plants (Chinoy, 1962, 1969; Chinoy and Mansuri, 1969, Asthana and Srivastava, 1978). However, no work seems to be on record regarding the use of Vit. C as a pretreatment agent in mutagenic studies.

3. Seedling Survival

Data on survival value of Japan Violet treated with different concentrations of EMS with/without Vit. C are presented in Table - 7. Observations showed that in all treatments the M_1 generation showed an increasing trend in the reduction of survival of seedlings from 7.61 with DW + 0.5% EMS to a maximum reduction of 25.30 % with DW + 1 % EMS, while slight increase in survival was seen in M_1 of the treatment in Vit. C + 1 % EMS. However, survival was higher in M_2 and M_3 generation in almost all treatments. It may be surmised that survival recovery is higher in later generations than in M_1, where the initial and direct mutagenic effects are more responsile for lower survival of the seedlings. These observations are in conformity with the earlier reports. Seedling survival has been found to have better recovery with EMS treatment (Swaminathan et al., 1970; Shobha, 1993). However, in the present experiment in respect of the treatments wherein Vit. C was used, percentage of reduction in seedling survival showed linear increase upto the treatment of Vit. C + 0.75% EMS. [illegible] Vit. C + 1% EMS showed less percentage of reduction in seedling survival in comparison to the treatment DW + 1 % EMS in M_1, thereby showing enhancement of mutagenic effect, probably, by the stimulation of growth and development due to Vit. C (Chinoy and Mansuri, 1969; and Asthana and Srivastava, 1975) upto certain level and the optimum being

Vit. C + 0.75% EMS.

4. Mutegenic effectiveness and efficiency

The data on Mutagenic effectiveness and efficiency of EMS, in relation to pre-treatment with DW/0.01 % Vit. C in Japan Violet are detailed in Table 10. Seeds received pre-treatment with 0.01% Vit. C before EMS treatment were affected more by EMS treatment than those presoaked in distilled water. Vit. C + 1% EMS treatment showed maximum effectiveness, on the basis of mutated panicle (0.0064) and seedlings (0.0015). Further, it also showed maximum efficiency for mutated spikelets (0.39 and 0.26) or mutated seedlings (0.09 and 0.063) on the basis of M_1 lethality and sterility (Table 10). The high mutagenic potency of EMS in comparison with certain ionizing radiation has been reported (Kawai and Sato, 1965, Swaminathan 1966, Matsuo and Yamaguchi, 1967). There exists certain contrary reports on chemical mutagen like EMS. This refers to its higher mutagenic efficiency than radiatons (Loveless and Howarth 1959; Gaul, 1962, 1963; Kaul and Bhan, 1977) and also its less advantage or effectiveness over ionizing radiation (Siddiq and Swaminathan 1968, Swaminathan et al., 1970). However, studies on mutagenic effectiveness and efficiency of chemical mutagen in comparison with physical mutagen has been reviewed elsewhere. There is no study referring to the use of Vit. C pre-treatment for chemical mutagenesis with EMS or other mutagens. The fact that Vit. C has some role in enhancing growth and development

by activating the biosynthesis of total nucleic acid and protein (Chinoy, 1962, 1969) prompted to use Vit. C as a pre-treatment agent in order to promote mutagenic efficacy of EMS. The present experiment, as a preliminary approach, has yielded positive results in most cases of treatments with Vit. C as indicated in Table 10. However, further studies are desirable in this line.

B. EMS INDUCED MACROMUTATIONS

An attempt was made for the study of mutation in the rice variety Japan Violet by treating with different concentrations of EMS after pre-treatment with distilled water or Vit. C aqueous solution in order to find out if there is any role for Vit. C pre-treatment in enhancing the mutagenicity of EMS. Studies on the frequency of occurrence, types of mutation, number of mutation and their inheritance followed upto M_3, and on their respective crosses with the original source parent followed upto F_3 generation, broughtforth several new information including the role of Vit. C in enhancing the mutagenicity of EMS in rice for the first time, as discussed in detail below under appropriate heads.

1. Frequency of occurrence and inheritance of different types of macromutations

The morphological mutations elicited from M_2 and M_3 populations

of Japan Violet treated with different concentrations of EMS after pre-treating with or without Vit. C solution are finished in Table 11. Data on their frequency of occurrence are presented in Table 38. Description of various mutants have been furnished under Chapter IV. The pattern of inheritance of mutants studied in their respective M_1 to M_3 populations are presented in Tables 17,18, 19 and 20. These aspects are discussed below under separate heads. The frequency of occurrence of the mutants ranged from 0.07 (striped mutant) to 0.093 % albinos (Table 38).

Albinos were produced by all the six treatments. The frequency of occurrence was found to be increased in relation to Vit.C pre-treatment from 0.10 to 0.95%, with 0.07, 0.19 and 0.81 respectively in the case of the treatments with 0.5%, 0.75% and 1% EMS with Vit. C pre-treatment. However DW + 1% EMS with Vit. C showed only 0.12% mutation in comparison to 0.92% in respect of 0.75% EMS. Albinos induced by chemical mutagens have been reported earlier (Swaminathan, 1966; Siddiq and Swaminathan, 1968; Viado, 1968; Ismail, 1969; Singh 1970; Vimala and Reddy 1972; Choudhary et al., 1986, 1987; Shobha, 1993). Mostly monogenic ratio has been reported (Ramiah and Parthasarathy, 1938; Ramiah and Rao, 1953 Iwata and Omura, 1978; Pavithran, 1986 and Shobha, 1993). The present observations are in conformity with the earlier reports. However, albinos obtained from the purple plant Japan Violet also showed

fading expression of anthocyanin pigmentation (Fig. 3) which gradually disappeared within a few days, probably, as a result of the inability of the albinos for pigment production and/or photosynthetic function. This shows an indirect evidence that production and/or sustenance of anthocyanin pigmentation may require substantial functional interaction with chloroplast and that such information appears to be meagre, but requires more comprehensive analysis.

The mutant, **lethal yellow** described elsewhere was obtained from the three treatments of DW + EMS which showed a linear increase in relation to dosage, ranging from 0.07 % to 0.59% frequency. Only 0.5% EMS with Vit. C pre-treatment showed lethal yellow mutants with 0.35% frequency, which is 0.28 % increase over the corresponding DW + EMS treatment (Table 38). The non-appearance of lethal yellow mutants in Vit. C + 0.75% and Vit. C + 1% EMS may be attributed to the comparatively smaller population size. Inheritance of lethal yellow showed 3 : 1 or 15 : 1 in M_2 which was not confirmed at M_3 level, probably, due to genotypic elimination. However, lethal yellow reported here and its inheritance are in conformity with earlier reports (Imai, 1935; Ramiah and Parthasarathy, 1938; Kadam, 1941; Pavithran, 1986; Shobha, 1993).

The mutant, **chlorina** described elsewhere occurred only in M_2 of the treatments 0.5% EMS with DW pre-treatment and in 1% EMS pre-

treated with Vit. C solution. In all other treatments chlorina could not be obtained, the probable reason seems not traceable in view of the complex factors affecting mutagenicity of various tissues (Cervigini and Belli, 1962; Hainer, 1963) and others reviewed elsewhere. Chlorina in rice has been reported earlier (Ramiah and Rao, 1953; Iwata and Omura, 1971a, b; Iwata et al., 1978; Omura et al., 1978; Majumdar, 1980) with monogenic inheritance at M_2. In the present study 3 : 1 was obtained in M_2 which could be confirmed by the M_3 and F_3 generations (Table 18 & 20). The present chlorina also expressed slight pigmentation especially on the leaf sheath.

The **striped mutant** described elsewhere was induced by EMS, only in the case of three treatments wherein Vit. C was used for pretreatment. The results showed a linear increase in the frequency of occurrence from 0.07 % with Vit. C + 0.5% EMS to 0.10 with Vit. C + 0.75 % EMS and 0.37 % with Vit. C + 1% EMS, thereby showing a relative increase of frequency of occurrence of 0.03 % and 0.30 % over 0.07 % of the Vit. C + 0.5 % EMS treatment (Table 38). Striped mutant has been reported earlier in rice (Morinaga, 1932; Ramiah and Ramanujam, 1935; Ramiah and Parthasarathy, 1938; Pal and Ramanujam, 1941; Maekawa et al., 1990; Shobha, 1993). However, the role of Vit. C pretreatment in the mutagenic activity of EMS is reported for the first time. In this case particularly, the influence of Vit. C causing enhancement of striped mutation is evident from the fact

that only Vit. C + EMS treatments alone produced mutation, while EMS alone did not produce any effect in this regard. Similarly, early reported striped mutations referred to yellow or white stripes. In the present case the striped mutant showed green and 'white stripes' shaded with anthocyanin, thereby giving the impression of green and light purple stripes (Fig. 4). Probably, the sectorial mutation must have affected only the production of chlorophyll in the striped region, leaving chromoplasts mostly undisturbed.

The **deformed or palealess mutant** described to have occurred with Vit. C. + 0.75 % EMS showed 0.20% frequency of occurrence and no other treatment induced this mutation. Its inheritance showed recessive monogenic control (Table 32). Reports on deformed or palealess mutants are very rare (Jachuck and Sampath, 1969). However, the mutant was induced in Japan Violet when treated with EMS with Vit. C pre-treatment and it appears to be a new report for EMS induced deformed palea mutation.

Mutants such as **beaked lemma-depressed palea, long sterile glume, procumbent plant type, broad seed, and grassy rhizomatous plant type** described elsewhere occurred only with 1% EMS treatment in the absence of Vit. C with the respective frequencies of occurrence, 0.24, 0.12, 0.24, 0.30, and 0.36 % (Table 38). Among these mutants the grassy rhizomatous condition, procumbent plant type and broad

seeded condition appeared to be new reports with EMS. The mutants showed recessive monogenic control except long sterile glume which gave 15:1 in M_2 and confirmed by M_3/F_3 generations (Tables 18 & 20).

The **grassy rhizomatous mutant** exhibited characteristics resembling trailing, rhizomatous, grassy habit capable of rooting from nodal regions and producing axillary miniature inflorescence bearing less than 10 spikelets. The mutant propagates itself vegetatively. Normally the plant could be obtained only from segregating populations. As there is no seed setting, the plant could not be produced from seeds. However, at the seedling stage of 3-4 leaves the seedlings appear indistinguishable from the normal ones. Subsequently,the mutant plant produces similar small types of leaves with distinctive characteristic grassy habit and become distinguishable from the normal plant. The rhizomatous grassy mutant differs in almost all characteristics except pigmentation from its original source plant, Japan Violet. It shows resemblance with certain characteristics of Leersia hexandra L., eventhough it doesn't appear to be Leersia-like. However, the mutant exhibits characteristics of evolutionary significance as a situation of reversion, expressing certain grassy ancestral characteristics of the rice plant (Nayar, 1973; Pavithran, 1978). This requires further studies of the material or such mutants of evolutionary significance in order to unravel the mistery behind origin of the

rice plant.

The mutant **anther sterile** obtained from DW + 0.50 % EMS and from Vit. C + 1% EMS treatments described elsewhere showed a frequency of occurrence of 0.10% and 0.15 % respectively with the relative increase in mutants with Vit. C + EMS treatment by about 0.05%, thereby indicating that the mutagenicity of EMS oculd be slightly enhanced with Vit. C pre-treatment. Anther sterility has been reported earlier (Misra and Sastry, 1969; Pavithran and Mohandas, 1976a; Singh and Ikehashi, 1981; Pavithran 1983; Lu and Rutger, 1984; Lu and Zhang, 1986; Shobha, 1993). However, the non-dehiscent anthers caused complete spikelet sterility, eventhough nearly 8% pollen fertility existed with the plant. The M_2 and M_3 populations and F_1 to F_3 populations (Tables 17,18,19 & 20) confirmed the recessive monogenic control of this mutant. The mutant resembled Japan Violet in all characteristics except anther sterility or functional male sterility.

Another mutant which showed **abnorphic spikelets** (Fig. 8) was obtained only with Vit. C + 1% EMS treatment with a frequency of occurrence of o.45% (Table 38). This also showed recessive monogenic control in M_2 and was confirmed in M_3 (Table 17,18). The morphological variations included open spikelet, multiple pistils, multihusk, extra long glume, multiple kernels, beaked lemma, depressed palea and long

lemma-palea (Table 28, Fig. 8). It may surmised that normally a major gene is responsible for the overall development of the spikelet. The mutant gene, being recessive to the normal, might have caused the abnormality of the morphological parts in the spikelets. However, an extensive developmental and genetical analysis of the mutant might give a better perspective of the situation.

The **tall recessive mutant** elicited from Vit. C + 0.75 % EMS described elsewhere, differs in several respects from the earlier types. The mutant induced by EMS after pretreatment with Vit. C gave 0.13 % frequency of occurrence. The mutant showed elongation of first four nodes with 77.36, 141.96, 60.71 and 66.67% increase respectively over the corresponding internodes of the parent plant (Table 35), in contrast to the early reports on elongation of the upper most internode (Rutger and Carnahan, 1981; Mackill et al., 1992a) and last or lower most internode (Okuno and Kawai, 1978a,b). The present mutant is a new report. Both M_3 and F_3 populations confirmed the monogenic recessive control of the mutant (Tables 18 & 20). The gene is designated as ein (elongated internode) in the present case unlike the early reported "eui" gene (Rutger and Carnahan, 1981). The early reported "eui" gene has been incorporated in the cms plant to increase panicle exsertion (Shen and He, 1989; He and Shen 1991). However, tall recessive is an appropriate plant type for hybrid rice technology, as it helps to enhance the out-

crossing potential of the male parent. Besides cms maintainers and restorers which are known to be essential for hybrid seed production, tall recessive male plant resulting from incorporation of eui/ein (presently identified) gene into the pollen (fertility maintaining) parents, would be of great advantage in the production of semidwarf F_1 plants for hybrid seed production in rice and also in such other cereals. Tall recessive gene can also be used to replace indiscriminate application of gibberellic acid in the hybrid seed production programmes which is likely to cause ecological hazards.

The six mutants obtained from M_3 generation of the treatments wherein Vit. C pre-soaking was adopted are discussed below.

The **multiple pistil mutant** described elsewhere resembled Japan Violet but for the mutant character. It showed 0.16% frequency of occurrence with one of the M_3 families of Vit. C + 0.5% EMS treatment (Table 38). The monogenic ratio 3:1 obtained in M_3 was confirmed in M_4 by the breeding behaviour of 20 M_4 families studied (Tables 23 & 24). This is in conformity with earlier reports (Parthasarathy, 1935; Jodon, 1955; Pavithran et al., 1989; Kinoshita and Takahashi, 1991). An astaminate multipistilate recessive mutant (female sterile) has also been reported in rice (Shobha, 1993). The multipistilate or polypistilate condition might have originated from a reducing rachilla of the panicle partially preserving certain floral entities

(Pavithran et al., 1989). However, the mutant exhibited differential expressivity within the panicles and it might be used as a good material for studying expressivity of the mutant character under different conditions.

The mutant **high tillering dwarf** of Japan Violet described elsewhere (Table 36) under chapter IV occurred in M_3 of Vit. C + 0.75% EMS with a frequency of 0.41% (Table 38). The high tillering dwarf showed higher tillering index of 8.63 and still higher EBT index of 9.30, with higher percentage of EBT than the control (Table 36). The disadvantage with the mutant is short panicle length and low number of spikelets and panicle density. The dwarf mutant has an added advantage of continuous tillering habit unlike the ancestral perennial tillering habit. Exploitation of continuous tillering habit could ensure periodical productivity, provided the above mentioned disadvantages are substituted with appropriate genetic changes necessary to increase production. As such this appears to be a promising mutant for crop improvement. Earlier reports on high tillering dwarf (Bose, 1968; Miah and Bhatti, 1968; Misra et al., 1971; Nair and Ninan, 1973; Rao and Siddiq, 1977) appear to be different from the present type of high tillering dwerf which exhibited continuous tillering habit. The high tillering dwarf character appeared to have monogenic recessive control (Tables 23 & 24).

The **partial green** mutant described elsewhere (Table 25) was elicited from 1% EMS treatment and it showed 0.48 % frequency of occurrence. It expressed characteristic recessive mutation changing into green leaf margin, leaf blade, ligule and auricle and all other plant parts remaining purple resembling Japan Violet. Both in M_3 of 1% EMS treatment and in F_2 of the cross (Tables 23 & 19) distinct segregation for green colour of leaf margin, leaf blade, ligule and auricle could be obtained. It showed a monogenic recessive control (Tables 23 & 19). No recombination appeared to have occurred with regard to this set of characters, thereby showing pleiotropic control of pigmentation in these plant parts (Table 26). Consequently, it could mean that the mutation might have occurred to a pleiotropic localisation gene for these characteristics. This situation appears to be the first report in rice.

On the contrary, another mutant which showed **complete green** in all plant parts except kernel colour, described elsewhere, was obtained from Vit. C + 1% EMS treatment. This mutant showed 0.69 % frequency of occurrence and increase in spikelet number, EBT % and total tillers (Table 27). Its inheritance showed monogenic recessive control of complete green phenotype, both in M_3, M_4 and also in the F_2, F_3 progenies of the cross studied (Tables 23 & 24). The complete green mutant might have occurred as a result of a recessive mutation occurred in respect of the CAP system for pigment production (Nagao,

1951; Nagao and Takahashi, 1956; Takahashi, 1957; Misro, 1981; Pavithran, 1986), preferably either to the gene 'C' (Chromogen) or to the gene 'A' (Activator) which are basically responsible for the production of anthocyanin pigmentation in plant parts in rice. This indicates the possibility of mutating a complete purple plant into a complete green plant. It enhances the productivity with increase in photosynthetic efficiency. This is substantiated, to some extent, by the increase in tillers, spikelets, grain number and panicle density in comparison to its control (Table 27). Segregation for purple and green plants did not give any recombinant class, suggesting pleiotropy of the gene concerned (Table 26).

The treatment Vit. C + 1% EMS induced two other mutants: **brittle culm** and **spotted leaf**. The mutant brittle culm resembled Japan Violet in all other characters and so also the mutant spotted leaf, described elsewhere. **Brittle culm** showed 0.50% and **spotted leaf** 1.60% frequency of occurrence and monogenic recessive control like other mutants, and were confirmed by respective M_4 and F_3 families (Tables 24 & 20). There are certain early reports on brittle culm mutant in rice (Jones, 1933; Nagao and Takahashi, 1963; Takahashi et al., 1968; Iwata and Omura, 1977). Present observation of monogenic recessive control for brittle culm is in conformity with the early reports. However, the present observation on spotted leaf is in conformity with the spotted leaf reported earlier (Iwata and Omura,

1975, 1977; Iwata et al., 1978; Yoshimura et al., 1982).

On the whole, Vit. C + EMS treatments produced more number of mutants, some of which like male sterile, tall recessive, high tillering dwarf and complete green mutant are agronomically significant indirectly/directly. The common mutations occurred with or without Vit. C pre-treatments are albinos, lethal yellows and chlorina (Table 38). Altogether treatments with EMS alone produced 9 mutations. The combined action of Vit. C and EMS together with same complement of dosages, produced 13 mutations in the present study (Table 38). This could, probably, indicate the possibility of 'synergistic' interaction between Vit. C and EMS in increasing mutations in rice. Ascorbic acid (Vit. C) which is responsible for enhanced cell division (Chinoy, 1962, 1969, Chinoy and Mansuri, 1966; Mehta and Chinoy, 1978) might have facilitated better alkylational interaction of EMS with DNA, thereby enhancing mutational changes. However, the exact nature of interaction between Vit. C pretreatment and EMS treatment in this situation requires more studies for confirmation.

C. INHERITANCE AND INTERRELATIONSHIP OF GENES

Studies on inheritance of 14 morphological characters and their genic interrelationships were confined to 1-4 crosses described elsewhere (Tables 39a, 40, 41, 42, 43). The crosses were studied upto F_3/M_3F_3 generations for genetic analysis in relation to

inheritance and interrelationships of morphological characters. These were also studied for transformation of genetic controls of morphological characters occurred due to X-ray treated pollen grains of the male parent. The results on these aspects are discussed below.

1. Inheritance of morphological characters and transformation of genetic controls

Inheritance of 14 morphological characters showed duplicate factors for leaf axil, inhibitory factor control for nodal pigmentation and monogenic recessive control for awning and tipsterility invariably in all the above crosses studied, as detailed elsewhere (Tables 39a,b). On the other hand, leaf sheath, leaf margin, leaf tip, internode, stigma and apiculus showed dominant monogenic control of pigmentation in the normal cross, while digenic complementary interaction in the cross Cherumodan X Japan Violet - 5000 rad (Tables 39a,b). Further, leaf margin gave 9:7 in 1500 rad and 2000 rad treatments and leaf tip gave 9:7 in 1500 rad treatment, not in 2000 rad treatment (Table 39a,b). However, in the case of leaf sheath, internode, apiculus and stigma, only 5000 rad treatment gave digenic complementary ratio against 3:1 in normal or other crosses. This, probably, demonstrates the fact that only one of the loci remained heterozygous in the normal cross, while X-ray must have

transformed the other locus also heterozygous by mutating (either of) the complementary gene(s) into recessive in the treated pollen grains of Japan Violet. Further, observation recorded in respect of leaf blade, junctura proper, ligule and auricle showed genetic control by two complementary genes, as elicited in the normal cross. However, in the case of junctura proper and auricle, 1500 rads, 2000 rads and 5000 rads treated pollen grains gave trigenic complementary ratio of 27:37 in all the three treated crosses. In the case of leaf blade, both 2000 and 5000 rad treated pllen grains gave trigenic complementary ratio 27:37, and in the case of ligule only 5000 rads gave 27:37 (Table 39b). This demonstrates, probably, the differential response of these characters in relation to the dosages of the X-ray treatments (Table 39a,b). This situation of differential genetical ratios (transformed ratios) for the same character obtained in the crosses of the same parents under the influence of pollination with irradiated pollen grains appears to be a new report. The presently, available literature on similar studies do not furnish any information on such results except information on gene transfer through pollen (Pandey, 1975, 1976, 1980a,b, 1983). However, the situation commands reasonable genetical explanation. Early workers do not seem to have considered genetical transformations in terms of inheritance of morphological characters.

The above situation could be explained on the basis of three

complementary loci instead of the two in the normal cross for the control of these characters. The additional heterozygous condition (third locus) might have arisen as a result of recessive mutation, as in the case of earlier instances described above. It may be surmised that response to X-radiation in rice appears to be varietal/ character related. The situations thus observed with characters presently studied are discussed in view of the present status of inheritance and character associations in rice in the light of literature reviewed elsewhere.

a. Inheritance of leaf axil pigmentation

Leaf axil pigmentation has been reported to have monogenic control by several authors (Ghose et al., 1960; Nair, 1958; Butany et al., 1959; Panda 1962; Setty and Misro, 1973; Annie, 1986; Nadaf, 1989; Sukeskumar, 1990). Complementary genic control of two (Nair, 1958; Ghose et al., 1960; Panda, 1962; Misro, 1963; Annie, 1986; Shobha 1993), three giving 27:37 (Ghose et al., 1960; Singh et al., 1989b) or 54:10 (ghose et al., 1960; Misro and Sastry, 1962; Dhulappanavar, 1973a; Manjunath, 1973; Dhulappanavar and Hiremath, 1974a,b) or 45:19 (Panda, 1962) and four complementary genes giving 81:175 (Ghose et al., 1960), 162:94 (Ghose et al., 1960; Misro, 1963; Dhulappanavar, 1973b, 1975a, 1976b; Dhulappanavar et al., 1973b) have been reported. Further inhibitory ratios 117:139

(Sadananda, 1981), and 27:229 (Dhulappanavar, 1981) have also been reported earlier. The present study of the cross Cherumodan x Japan Violet and of the crosses with irradiated pollen grains of Japan Violet gave duplicate recessive ratio 15:1 in contrast to the early reports of 1-4 complementary genes and an inhibitory gene for the inheritance of leaf axil pigmentation. The present observation on duplicate factors (Table 39a-1) may be a new report. The duplicate factors $px_{1/2}$ also appeared to be stable against X-ray treatments given, as evidenced by the lack of genetic transformation by X-ray treatment of pollen grains (Table 44).

b. Inheritance of leaf sheath pigmentation

Leaf sheath gave monogenic ratio in all the crosses except in the cross Cherumodan x Japan Violet - 5000 rads X-rays. This treatment showed a complementary interaction of two genes giving 9:7 (Table 39a-2). The change in the ratio from monogenic to digenic complementary indicated the possible recessive mutation occurred to either of the complementary genes existed in the purple parent, thereby giving rise to the said ratio in M_2F_2. Both the monogenic and digenic complementary ratios obtained presently are in conformity with early reports since Parnell et al (1917) to Kashikar and Kulkarni (1990) for 3:1 and from Hector (1916) to Shobha (1993) for 9:7 as reviewed elsewhere. In addition to these ratios 27:37 (Hector,

1922, Ghose et al., 1960; Kadam and D'Cruz, 1960; Ramirez et al., 1960; Tripathi and Rao, 1979; Ramesh, 1984; Prasad et al., 1987), 54:10 (Richharia et al., 1960; Dhulappanavar and Mensinkai, 1970; Dhulappanavar, 1973c,d; Manjunath, 1973; Setty and Misro, 1973; Prasad et al., 1987; Shyla and Pavithran, 1989), 162:94 (Ghose et al., 1960; Panda, 1962; Saran and Srivastava, 1969; Setty et al., 1973; Singh et al., 1989a), 81:175 (Ghose et al., 1960; Ramirez et al., 1960) 45:19 (Ghose et al., 1960; Setty and Misro, 1973; Setty et al., 1973; Hedagal, 1980; Pavithran and Annie, 1980; Shyla, 1984; Annie, 1986), 3:13 (Kuang 1951; Shastry and Patnaik, 1962), 9:55 (Shastry and Patnaik, 1962), 3:253 (Dhulappanavar et al., 1975b), 63:193 (Dhulappanavar, 1981), 117:139 (Richharia et al., 1960), 15:1 (Hector, 1922, Kuang, 1951), (9:3:4 (Kadam, 1936; Dave, 1948), 12:3:1 (Julka, 1967) and 255:1 (Thimmappiah, 1975), have also been reported for leaf sheath pigmentation in rice.

As such 1 to 4 genes have been found to be responsible for leaf sheath pigmentation, where basic, 2-4 complementary and inhibitory genes, anti-inhibitory or 1-4 duplicate factors or recessive epistatic factors are involved for the control of pigmentation in leaf sheath.

c. Inheritance of leaf blade pigmentation

Leaf blade also showed a genetical transformation from 9:7 into 27:37 in the later two crosses wherein 2000 and 5000 rad X-

rayed pollen grains were used for pollination (Table 39a-43). The genetic transformation here could also be explained on the same basis of recessive mutation occurred in one of the three loci concerned, in the purple parent, Japan Violet, due to X-ray induction. However, the ratios 9:7 and 27:37 are in conformity with the early reports on leaf blade pigmentation. Many authors have reported 9:7 (Parnell et al., 1917; Jones, 1930; Butany et al., 1959; Hsieh, 1960; Ghose et al., 1963) and 27:37 (Ramiah and Rao, 1953; Panda, 1962; Ghose et al.,1963; Saran and Srivastave, 1969; Singh et al., 1989a; Shobha, 1993) which are in conformity with the present report. Further, 3:1, 15:1, 3:13, 9:55, 39:25, 117:139, 27:229, 15:241 and 39:12:13 have also been reported for the genetic control of leaf blade pigmentation in rice as reviewed elsewhere in detail. As such basic, 3 complementary genes, one or two inhibitories and anti-inhibitory or duplicate and inhibitory-duplicate genes are known to have genetic control for leaf blade pigmentation in rice.

d. Inheritance of leaf margin pigmentation

Leaf margin gave 3:1 in the normal cross of Cherumodan x Japan Violet, while the crosses with 1500, 2000 and 5000 rad X-ray treated pollen grains used for pollination showed 9:7 (two complementary genes) for leaf margin pigmentation (Table 39a-4). This indicates that pollen irradiation might have caused one of the concerned loci also to be heterozygous due to the probable recessive mutation occurred

in the pigmented parent. Early reports on leaf margin pigmentation have recorded monogenic (Mitra and Ganguli, 1932; Kuang, 1951; Nadaf, 1989), digenic complementary (Kuang, 1951; Nadaf, 1989; Shobha, 1993), trigenic complementary giving 27:37 (Panda, 1962; Singh et al., 1989a), 45:19 (Setty and Misro, 1973; Setty et al., 1973; Hedagal, 1980; Hedagal et al., 1981; Rao and Misra, 1986) and tetragenic complementary ratio of 162:94 (Panda, 1962, Saran and Srivastava, 1989; Setty and Misra, 1973; Setty et al., 1973; Singh et al., 1989a) for leaf margin pigmentation. Besides, duplicate and triplicate recessives have also been reported (Kuang, 1951 and Thimmappiah, 1975). The present reports of 3:1 and the transformed ratio 9:7 are in conformity with early reports. Transformation of the monogenic ratio into a digenic complementary ratio, 9:7, is the first report on X-ray induction, probably, due to the fact that genetical studies are seldom carried forward with such mutation studies for analysing the genetic system operating upon character heritage or its inheritance.

e. Leaf tip pigmentation

Leaf tip showed 3:1 in normal cross and in the cross wherein 2000 rad X-ray irradiated pollen grains were used for crossing. On the other hand, the crosses wherein 1500 and 5000 rad X-rayed pollen grains were used, showed digenic complementary ratio 9:7 (Table 39a-

5). Only in the case of leaf tip the linearity in response was not observed. However, both 3:1 and 9:7 have been reported earlier and the present observations are in conformity with several early reports for 3:1 (Mitra and Ganguli, 1932; Ghose et al., 1960; Nadaf, 1989) and 9:7 (Ghose et al., 1960; Manjunath, 1973; Pavithran, 1977; Shobha, 1993). Several complementary ratios such as 27:37, 54:10, 45:19, 162:94, 81:175, 189:67 and 702:322 involving to 2-5 complementary genes have been reported by different authors as reviewed elsewhere for this character. Further 1:15, 255:1 have also been reported (Dhulappanavar et al., 1975c; Thimmappiah, 1975) for the control of this character and such complementaries up to 5 genes and duplicate/ triplicate/tetraplicate recessives were also knwon for the genetic control of these character.

f. Junctura proper and auricle pigmentation

Junctura proper pigmentation showed 9:7 in the normal cross that was transformed into 27:37 in 1500 rad, 2000 and 5000 rad X-radiated pollen grains of Japan Violet used for crossing (Table 39a-6). This indicates transformation of one of the concerned dominant complementary genes for pigmentation in Japan Violet into its recessive, so as to accomplish 3 heterozygous complementary loci in F_1 giving rise to 27:37 in M_2F_2 (Table 39a-6). The same situation was also observed with auricle pigmentation in all the three treatments

(Table 39a-8). The ratio 27:37 obtained presently as an altered ratio of 9:7 is in conformity with early reports, both in the case of junctura proper (Parnell et al., 1917; Jones 1930; Butany et al 1959; Iyer, 1959; Panda, 1962; Setty and Misro, 1973; Singh et al, 1989a) and auricle (Jones, 1930; Butany and Bhattacharya, 1962; Dhulappanavar et al., 1973b; Shyla, 1984; Ahmad and Das, 1990; Sukeskumar, 1990; Shobha, 1993). The survey of literature on the genetics of junctura proper showed that 3:1 and complementary ratios 9:7, 45:19, 81:175 and 243:781 and inhibitory ratio 1:3, 3:13 and 3:61 have been reported earlier along with duplicate recessive, as reviewed elsewhere. It shows, juncture proper pigmentation is controlled in rice with a basic gene with 2-5 complementaries and also by the involvement of one or two inhibitory genes or by duplicate genes.

In the case of auricle also monogenic, digenic or tri/tetra genic complementary ratios given by 2-4 complementaries, 1-2 inhibitories and inhibitory complementary genes or duplicate genes have been reported earlier, as reviewed elsewhere.

g. Ligule pigmentation

Ligule pigmentation gave 9:7 in the normal cross and in the crosses where 1500, 2000 rad X-rayed pollen grains were used for crossing. In the case of 5000 rads treatment trigenic complementary

ratio of 27:37 was obtained (Table 39a-7), indicating genic transformation or mutation of a dominant complementary gene of Japan Violet into its recessive form, so as to elicit the ratio 27:37 in M_2F_2 of the concerned cross (Table 39a-7). It seems the genetic constitution for ligule pigmentation could be changed only by higher dosage of X-rays (Table 39b-7).

The present reports of both 9:7 and 27:37 are in conformity with certain early reports (Hector, 1922; Mitra et al., 1928; Chao, 1928; Jones, 1930; Mitra and Ganguli, 1932; Kuang, 1951; Ghose et al., 1960; Pavithran and Mohandas, 1976 a,b; Pavithran, 1977; Prasad et al., 1987; Singh et al., 1989a, Ahmad and Das, 1990; Shobha, 1993). Additional ratios like 3:1; 1:3; 15:1; 63:1; 45:19; 54:10; 81:175; 117:139; 162:94; 189:67; 27:229; 243:13 have also been reported as reviewed elsewhere. As such ligule pigmentation is controlled by basic and 2-3 complementary genes interacting often with inhibitory and duplicate factors.

h. Nodal pigmentation, awning and tip-sterility

Node, awning and tip-sterility like leaf axil did not show any variation in the genetic control due to any of the X-ray treatments (Table 39a-9, 13,14), thereby showing that these characters exhibited more genetic stability against X-radiation. However, present genetical study of nodal pigmentation gave an inhibitory ratio 3:13 which is

in conformity wih certain early report (Sadananda, 1981). Other early reports on nodal pigmentation have shown 3:1; 9:7; 27:37 and 162:94 indicating 2-4 complementary genes and the inhibitory ratios such as 21:43; 9:55; 117:139 and 111:145 and also duplicate recessive for the control of nodal pigmentation, as reviewed elsewhere.

In the case of awning the present ratio 3:1, obtained in all the treatments and in the control is in conformity with certain early reports (Ramiah, 1935; Kuang, 1951; Ghose et al., 1960; Sastry, 1977; Pavithran, 1986; Shobha, 1993). Several other ratios have also been reported for awning which include both complementary and inhibitory, duplicate recessive and triple recessive for awning. These are 9:7, 27:37, 54:10, 81:175, 243:781, 3:13, 9:55, 117:139, 9:247, 9:3:4, 15:1 and 63:1, as reviewed elsewhere.

Tip-sterility also did not show any genetic transformation with regard to inheritance with any of the X-ray treatments and showed 3:1 in all cases, which is in conformity with early observations (Ramiah, 1931; Ramiah and Parthasarathy, 1938). Perhaps, tip-sterility may be one of the least understood genetical characters in rice as seen evident from the literature. However, tip-sterility appears to be an important morphological character, as it could affect the spikelet fertility and grain production in rice and, as such, it deserves further consideration of researchers, as has been

exhaustively studied in wheat by Dr. O.H. Frankel.

Internode, apiculus and **stigma** showed similar response in transformation of 3:1 in normal cross into 9:7 only in the cross wherein 5000 rads X-rayed pollen grains were used for crossing. The genetics of these characters is discussed below.

i. Internode pigmentation

The ratio 3:1 obtained in the normal cross of Cherumodan X Japan Violet (Table 39a-10) is in conformity with several early reports (Hector, 1922; Mitra et al., 1928; Bhattacharya, 1957; Hsieh, 1960; Uzir, 1961; Misro, 1963; Kondo, 1963; Mori et al., 1981). The changed ratio of 9:7 obtained from the cross wherein 5000 rad X-rayed pollen grains were used for pollination is also in conformity with certain early reports (Hector, 1922; Mitra et al., 1928; Kuang, 1951; Iyer, 1959; Ghose et al., 1960; Nagai et al., 1962; ; Dhulappanavar, 1975a,b; Dhulappanavar et al., 1974; Ahmad and Das, 1990; Singh et al 1989b). Transformation of ratio may be due to recessive mutation occurred in Japan Violet with either of the complementary genes for pigmentation as explained earlier. Further 9:6:1 and various other ratios such as 27:37, 45:19, 54:10, 81:175, 162:94, 3:13, 117:139 and 147:109 have also been reported by early workers as reviewed elsewhere. It seems that pigmentation in internode in rice is controlled by 2-4 complementary genes often

interfered with inhibitory and anti-inhibitory genes.

j. Apiculus and stigma pigmentation

Stigma and apiculus gave 3:1 in the first three crosses including the normal cross (Table 39a-11,12). The cross wherein 5000 rads X-rayed pollen grains were used for pollination both gave 9:7 due to the probable gene transformation or recessive mutation as explained elsewhere. However, both 3:1 and 9:7 are in conformity with many early reports from Hector (1916, 1922) to Shobha (1993) as reviewed elsewhere.

Nagai et al. (1962) reported two dominant complementary genes for green stigma colour giving 9 G : 7 P. Further, duplicate recessive genes for stigma pigmentation have been reported by Mitra et al. (1928) giving 15G : 1P. Other ratios reported for stigma and apiculus pigmentation are 27:37, 45:19, 54:10, 81:175, 162:94, 189:67, 15:1, 27:9:28, 171:85, 135:121, 39:25, 9:55 and 9:3:4 as reviewed elsewhere. It can be surmised that pigmentation in stigma and apiculus is controlled by basic and 2-4 complementary genes, often interfered with inhibitory, anti-inhibitory or epistatic genes as reviewed elsewhere.

2. Interrelationship of genes governing morphological characters

A total of 91 character combinations of 14 morphological

characters were studied for tranformation, if any, in the interrelationship of genes in the treated crosses in comparison with the control (Tables 41, 42 & 43). Consolidation and extrapolation of the data (Table 44) led to the following inferences.

The morphological character-combinations of Px-Pn, Px-an, Px-tst, Psh-an, Psh-tst, Pl-an, Pl-tst, Pa-an, an-tst, Plm-an, Plm-tst, Pla-an, Pla-tst, Pjp-an, Pjp-tst, Plg-an, Pa-tst, Plg-tst, Pau-an, Pau-tst, Pn-an, Pn-tst, Pin-an, Pin-tSt, Ps-an and Ps-tst showed independent assortment both in the control and the treated crosses (Tables 41 & 44). All other characters, except leaf axil, node, awning and tip sterility, showed transformation of genetical ratios in contrast to the control due to X-radiation (Table 39b), as discussed earlier.

The relative frequencies of incidence of independent assortment, linkage relationships and pleiotropy in the control and treated crosses presented elsewhere (Table 45) showed linear increases due to treatments in respect of independent assortment and pleiotropy. In the case of linkage, considerable reduction in number of linkages occurred with 1500 rad and 2000 rad treatments culminating in 0.00 linkage relation in 5000 rad treatment (Table 45). However, in certain cases interrelationships did not appear to be specific,

eventhough high X^2 values were elicited in each case (Appendix -1). Probably, X-radiation of pollen grains might have caused chromosomal aberrations like translocation and/or inversions, breaking the linkage associations in the treated cross/es unlike the control (Table 44,45).

In the control cross 30 instances of pleiotropic relation of morphological characters were recorded (Table 44). Among these the pleiotropic relations between Pl-Pin, Pl-Ps, Pl-pa, Pjp-Pin, Pjp-Ps, Pjp-Pa, Plg-Ps, Plg-pa and Pau-Ps remained unchanged with all the treatments (Table 44). This indicated that pleiotropy in these cases appeared confirmed or more stable (fig. 31a-d). However, in respect of Pl-Plm, Pl-Pla, Plg-Pin, Pau-Pin and Pau-Pa, pleiotropic relations were not found broken with 2000 and 5000 rad treatments. With 1500 rads, neither pleiotropy nor linkage nor independent assortment could be obtained. This situation might be due to genotypic elimination (Oka, 1980) as evident from the low recombinent class/es obtained. However, this situation required further research. The relationship of these characters, hence, could be surmised to be pleiotropic relations without any probable change with the treatments, thereby showing confirmation of the pleiotropic condition or instances of tight/close linkages in respect of these character combinations.

The instances of pleiotropic association changed under different treatments could be categorised as follows. In the case of the

pleiotropic relations of Px-Psh, Px-Plm, Px-Pla, Px-Pin, Px-Ps, Px-Pa, Pl-Pn, and Pn-pa, pleiotrpy was changed into independent assortment with 5000 rad treatment only (Table 44, Fig. 31-d). This would suggest that pleiotropic relation recorded in the control and in other treatments might be only instances of tight linkage. It is extremely difficult to differentiate close or tight linkage of characters from their pleiotropic relations due to complete absence of recombinant classes, as has been observed by earlier workers (Misro, 1968, 1981). Misro (1968) suggested effect of radiation as a test for pleiotropy or tight linkage. In the present situation the above mentioned instances of tight linkages might have been converted into independent assortment, possibly, by inter-chromosomal alterations.

Pleiotropic relation of the character combinations Px-Pjp and Px-Pau were changed into independent assortment invariably with all the treatments (Table 44-5,7). In respect of pleiotropy between Px-Pl, Px-Plg, Plg-Pn and Pn-Pin pleiotropy was transformed into independent assortment with 2000 and 5000 rads treatments (Table 44-2,6,65,77). The above situations indicate differential response of morphological characters to X-radiation indicating that response to radiation might be genotypic or gene specific, as observed in the present instances. Similarly in respect of pleiotropy between Pau-Pn and Pn-Ps, 2000 rads treatment did not affect the pleiotropic

relations (Table 44-71,78), while 5000 rad treatment caused change into independent assortment (Table 44-78, Fig. 31b&d). However, this discrepancy in radiation response to its linearity needs further analysis. It may also be noted that with 1500 rad treatment, the characters Pau-Pn showed neither independent assortment nor linkage or pleiotropy (Table 44-71). However, the specific situations of induced genic transformations that led to differential (inheritance and) interrelationships of genes are discussed below.

a. Independent assortment

Out of 91 character combinations, 26 showed instances of independent assortment. The combinations Px-Pn, Px-an, Px-tst, Pn-an, Pn-tst and an-tst did not exhibit any transformation of genetical ratios while showing independent assortment (Table 44-8, 12,13,80,81,91).

On the other hand in certain other instances of independent assortment, the genetical ratios of characters other than Pn, tst and Px showed transformation of genetical ratios due to X-radiation either in 2,3 or all the treatments (Table 41). Such character combinations are Psh-an, Psh-tst, Pl-an, Pl-tst, Plm-an, Plm-tst, Pla-an, Pla-tst, Pjp-an, Pjp-tst, Plg-an, Plg-tst, Pau-an, Pau-tst, Pin-an, Pin-tst, Ps-an, Ps-tst, Pa-an and Pa-tst. In other words Psh, Pl, Plm, Pla, Pjp, Plg, Pau, Pin, Ps, Pa, Pn and Px are

independent of an and tst in their inheritance (Table 41).

However, awning (An) has been reported to be independent of Psh, Px, Plg, Pau, Pa, Ps and certain other characters earlier (Annie, 1989, Sukeskumar, 1990; Shobha, 1993; Anitha, 1994). In the present study recessive awning (an) was observed to be independent of Psh, Pl, Plm, Pla, Pjp, Plg, Pau, Pin, Ps and Pa and hence may be considered a new report.

Similarly, Px has been reported to be independent of An, Pj and Pau and certain other characters (Saran and Srivastava, 1969; Annie, 1986, Sukeskumar, 1990) rather, in conformity with the present report, in spite of the fact that the awning reported here is a recessive character.

Leaf sheath has been reported independent of Cl, An (Saran and Srivastava, 1969; Misro, 1981; Shyla, 1984; Annie, 1986; Sukeskumar, 1990; Shobha, 1993) and of Plg, Pau, Pl, Pjb, Plm and Pg (Anitha, 1994) in conformity with the present report, except that awning is a recessive character here. Anitha (1994) has further reported independent assortment of Plg with Px, Psh, Pau, Pjf, Pjb and Pg.

Junctura proper has been reported to be independent of An, Px and Pin (Sukeskumar, 1990 and Shobha, 1993) and with Psh (Anitha, 1994).

However, the present observation on independent assortment of recessive awning and tip-sterility with the above 12 anthocyanin pigmentation characters appears to be a new report.

Ten out of twelve anthocyanin pigmentation characters at 5000 rads treatment, wherein the degree of pleiotropy was considerably increased, showed a single gene common for all the characters (Fig. 32d). The gene $Px_{1/2}$ which exhibited pleiotropy with 10 characters and Pn which exhibited pleiotropy with 6 other characters in the control cross turned out to be independent of all other characters in 5000 rad treatment (Tables 44 and 45 and Fig. 31a-d).

On the contrary the gene Psh which showed pleiotropy with $Px_{1/2}$ in the control cross and 1500 rads cross, showed pleiotropic relation of $Psh_{a/b}$ with 9 other anthocyanin pigmentation characters in 5000 rad treatment (Fig. 31a-d).

b. Linkage relationships

Linkage relationships of genes controlling eleven morphological characters analysed in the cross Cherumodan x Japan Violet are discussed below in relation to their association, location of gene and transformation in interrelationships occurred in the different X-ray treated populations of the said cross. Nine out of eleven genes identified to have control over different morphological

characters have been located in the linkage group III (Misro, 1981; Kinoshita, 1984b; Khush;; 1984 and Pavithran et al., 1991). Hence the linkages observed mostly could be assigned to the same linkage group as detailed below.

i. Linkage association

The linkage between Psh and $Pl_{a/b}$ with c.o. = 2.02 in the control gave 1.01 in 1500 and 2000 rad X-ray treatments (Table 42-1). In the cross with 5000 rad X-ray treatment transformation of linkage occurred to give pleiotropic relationship of the above characters in the context of change of ratio 9:7 into 27:37 (Table 39b). The situation reveals allelic transformation and chromosomal alterations leading to alteration of genetic ratios and interrelationships. Preferably, an appropriate inversion could transform their linkage relationship into a pleiotropic situation due to tight linkage. Almost similar situation has occurred with Psh-Pjp with c.o. = 2.53, Psh-Plg with c.o = 3.05 , Psh - Pin with c.o. 12.82, Psh-Ps with c.o. = 10.55, Psh-Pa with c.o. = 11.12, Pin-Ps with c.o. = 5.13; Pin-Pa with c.o. = 8.35, Psh-Pau with c.o = 2.53, Pla-Plg with c.o. = 4.08, Pla-Pau with c.o. = 8.35, Pla-Pin with c.o. = 12.25, Pla-Pa with c.o. = 12.82 and Ps-Pa with c.o. = 3.56 in the control and with the variations in their crossover values in 1500 and/2000 rad X-ray treated crosses, showing genetic

alterations also in certain cases. However, all these 14 linkage relations showed transformation into pleiotropic relationships with 5000 rad X-ray treatment (Tables 42,44). This suggests that there is no one-to-one correspondence between transformation of genetic ratios and changes in interrelationships of genes. However, the present conversion of distinct linkage associations of genes into pleiotropic relationships might be instances of transformed tight linkages. However, the consequential elimination of recombinant genotypes in a lower population might also have contributed to the pleiotropic situation caused by X-ray treatment with 5000 rads.

Three instances of linkage association between Psh-Pn, Plm-Pn and Pla-Pn with c.o. values = 10.56, 3.04 and 11.12 respectively showed indepdnent assortment with 5000 rad treatment, which probably, indicates inter-chromosomal exchanges in relation to these characters. In the case of these characters node gave 3:13, in all the treatments like the control, while all other characters showed genetic alterations especially with 5000 rads treatment, and Plm showed genetic conversion in all the treatments (Table 44). This also indicated the assumption that the transformation of genetic ratios is independent of transformation of genic interrelationships.

Another 10 instances of linkages observed in the control are Psh-Pla with c.o. = 6.74, Plm-Pla with c.o. = 5.13; Plm-$Pjp_{a/b}$ with

c.o. = 1.51, Plm-$Plg_{a/b}$ with c.o. = 15.74, Plm-$Pau_{a/b}$ with c.o. = 1.01, Plm-Pin with c.o. = 6.19, Plm-Ps with c.o. 8.35, Plm-pa with c.o. = 7.80 Pla-Ps with c.o. = 9.45 and Pla-$Pjp_{a/b}$ with c.o. = 1.51 (Tables 42, 44). These characters invariablly showed genetic transformaton into complementary ratios, almost in both cases, mostly with 5000 rads treatment (Tables 39a,b). These characters also showed transformation of their interrelationships into pleiotropic associations. This could only be explained on the basis of tight linkage that did not yield any recombinant classes, probably, due to lower populaton. However, the results further indicated that no correspondence normally existed between allelic tranformation and transformation of interrelationships due to chromosomal aberrations.

In the present context two distinct strategies are recognised to have existed with the present results. The first one is that 26 character combinations observed for independent assortment in the control remained stable with X-ray treatments. Similarly, 9 pleiotrpic association (Pl-Pin, Pl-Ps, Pl-Pa, Pjp-Pin, Pjp-Ps, Pjp-Pn, Plg-Ps, Plg-Pa and Pau-Ps) observed in the control did not show any alteration with X-ray treatments (Table 44). The second strategy is expressed by the alterations of all the 27 linkage associations (mostly) into "pleiotropy". Majority of the pleiotropic associations of the control were transformed into situations of independent assortment (Table 44). This situation, though intricate and complex, leads to

the conclusion that all the pleiotropic conditions being observed experimentally need not necessarily be pleiotropic at the functional genic level, while certain situations do exist unaltered with X-ray treatments as pleiotropy (Table 44). The linkage associations observed in the control cross were altered in the X-ray treated crosses in terms of cross over values or transformed into situations of pleiotropy, the later pointing rather towards tight linkages than real pleiotropy.

ii. Location and assignment of genes

Present study of the cross Cherumodan x Japan violet (normal cross) facilitated linkage analysis of eleven genes (Pn, Plm, $Plg_{a/b}$, Pla, $Pl_{a/b}$, Psh, Ps, Pa, Pin, $Pau_{a/b}$, $Pjp_{a/b}$) of which nine genes are seen tohave been already assigned/located in the linkage group III by earlier workers (Misro, 1981, Kinoshita, 1984; Khush, 1984 and Pavithran et al., 1991) as furnished in Fig. 30. As the additional genes $Pl_{a/b}$ showed close linkage with Psh and $Plg_{a/b}$ with Pla and Psh, these genes could also be included in linkage group III (Fig. 30). The linkage associations between Psh and $Pau_{a/b}$ with c.o. = 2.53, Psh and $Pjp_{a/b}$ with c.o. = 2.53, Pla and $Pjp_{a/b}$ with c.o. = 1.51, Plm and $Pjp_{a/b}$ with c.o. = 1.51, Plm and $Plg_{a/b}$ with c.o. = 15.74, Plm and $Pau_{a/b}$ with c.o. = 1.01 and between Pla and $Pau_{a/b}$ with 8.35 could not be properly integrated into the map of the linkage group III due to considerable variation observed with cross over values and with

Fig. 30. Presently reported additions to linkage relationship of genes assigned to group III.

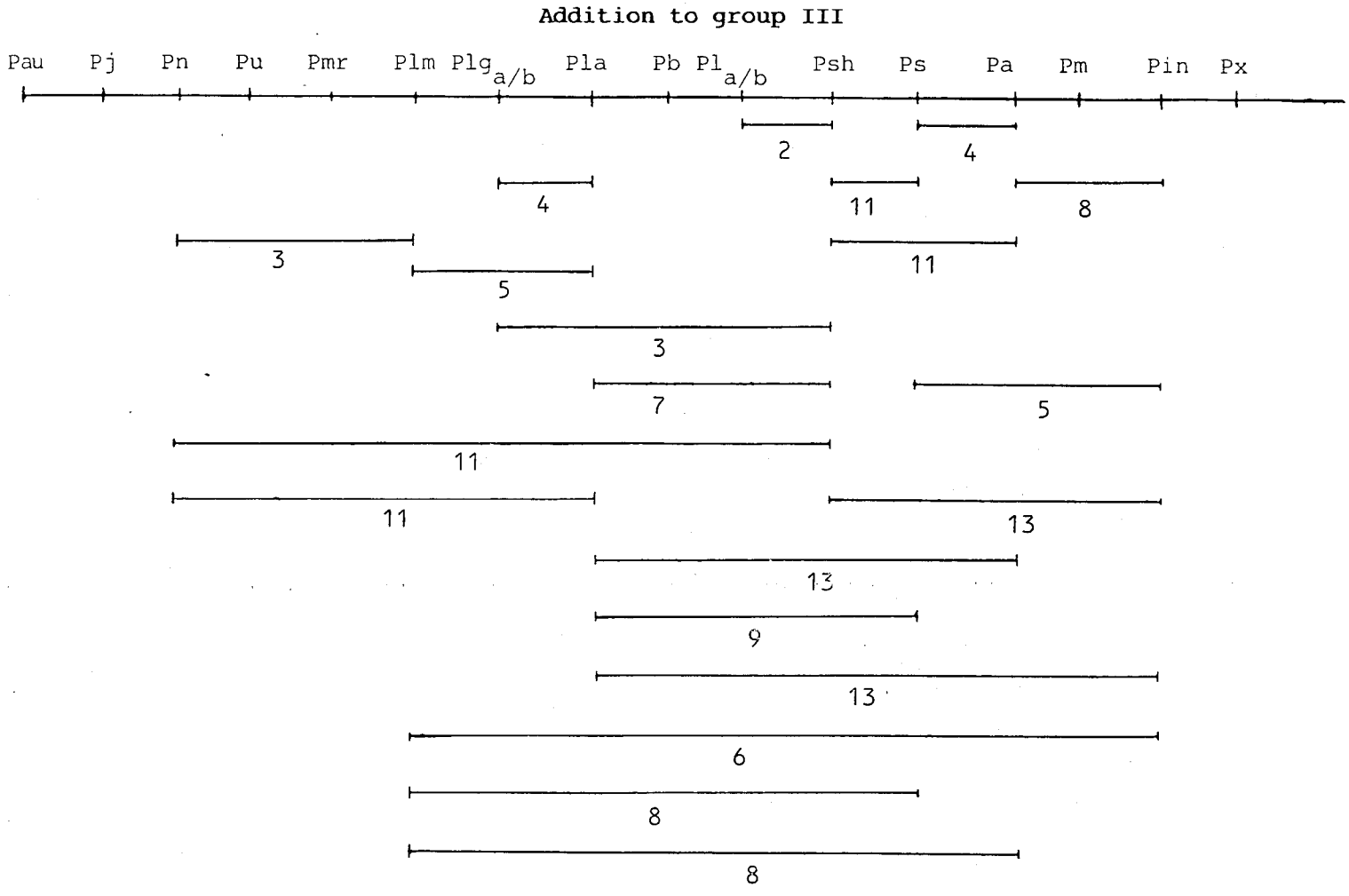

Addition to group III
Pau
Pj
Pn
Pu
Pmr
Plm
Plg a/b
Pla
Pb
Pl a/b
Psh
Ps
Pa
Pm
Pin
Px
2
4
4
11
8
3
11
5
3
7
5
11
11
13
13
9
13
6
8
8

Fig. 30

distance among the genes as located in the map. However, these are listed among the unassigned genes in the group III.

The linkage between Psh and $Pl_{a/b}$ with c.o. = 2.02 appears to be a new report as an addition to the linkage group III in spite of the fact that Psh - Pl with c.o. = 10.0 has been included in the linkage group II of indica (Misro, 1981). Pl is also linked with lg in group II of both indica and japonica and also with wh in indica II and is linked also with Pin in japonica II. In view of the linkage relation of Psh with more than seven genes in group III, including that with Pl a/b, it could be included in the same group as represented in the map (Fig. 30).

Linkage between Psh and Pjp a/b with c.o. = 2.53 also appears to be a new report, though tentatively assigned to group III.

Misro (1981),Kinoshita (1984) Annie (1986), Sukeskumar (1990) and Pavithran (1991) located Psh and Pjp a/b in the linkage group III, showing linkage relation also with Pb, Pin, Pla and with bd respectively. The above linkage between Psh and Pjp a/b is an addition to group III as a new report.

The linkage between Psh and $Plg_{a/b}$ with c.o. = 3.05 also appears to be a new report. $Plg_{a/b}$ has not been located so far,

though Plg (basic gene) has been located in the linkage group II being linked with Pla and Pg. However, Plg_a is seen located in group X being linked with Pin_a and Er_a (Fig. 1). However, the linkages between Psh and Plg a/b could be tentatively assigned to the linkage group III along with other associations of the gene Psh. $Plg_{a/b}$ is also seen linked with Pla in group III (Fig. 3).

Linkages between Psh and Pin with c.o. = 12.82 has been reported earlier but with varying c.o. values 2-14, assigned to group III (Kinoshita, 1984, Pavithran et al., 1991). The present report is in conformity with early reports. Pin is also seen linked with Pb and Pa/Ps and Px, Pc, Pla, Plm and Pmr, Sp, Pg, Pn, Pau and others assigned to group III already (Fig. 1).

The linkages between Psh and Ps with c.o. = 10.00 and between Psh and Pa with c.o = 11.12 have been established earlier and located in group III with varying c.o. values 6-18, as reviewed elsewhere. The present report is in conformity with early reports. Even though Pa/Ps has been found to be pleiotropic in several instances (Hector, 1922; Chao, 1928; Dhulappanavar, 1975; Shyla and Pavithran, 1989 and Shobha, 1993), they are found linked here with c.o. = 3.56, as a new report.

Linkages between Pin and Ps/Pa with c.o. = 12 has been reported earlier (Fig. 1). Pin has shown linkages with Px and Pc, Pla, Plm,

Pmr and Pg and Pau (Fig. 1). The present reports on linkages between Pin and Ps/Pa with the respective c.o. values 5.13 and 8.35 are in conformity with early reports, but with different c.o. values for both Ps and Pa (Fig. 30).

The linkage association between Psh and Pn is in conformity with early reports (Fig. 1) where Pn is linked with Pau, Pc, Psh, Pin, Pg and Sp. However, the present c.o. value 10.56 for Psh - Pn is new. Linkage between Psh and Pla with c.o. = 6.74 is in conformity with certain early reports (Dhulappanavar, 1973a; Hedàgal et al., 1981; Misro, 1981; Pavithran et al., 1991). Pla is also linked with Plm and Pmr in group III. The linkage between Plm and Pla with c.o. = 5.13 is also assigned to group III in conformity with early reports (Fig. 30).

The linkage obtained presently for Plm and PjPa/b appears to be reported for the first time. However, both genes have already been located in group III. The genes fall apart, but the present c.o. = 1.51 does not permit its proper location and it is tentatively considered under group III, subject to further analysis.

The linkages between $Plg_{a/b}$ and Pla with c.o. = 4.08 is a new report and is located in linkage group III, as the gene $plg_{a/b}$ is also linked with Psh with c.o. = 3.05 and it also located in the same group III as presented in Fig. 30.

Both Plm and $Pau_{a/b}$ have already been located in linkage group III earlier (Dhulappanavar, 1973a, Misro, 1981). The linkages between Plm and $Pau_{a/b}$ with c.o. = 1.01 is a new report. However, appropriate incorporation appeared to be difficult due to cross over value differences.

Similarly, the linkage between Plm and Pn with c.o. = 3.04 also appears to be a new report, in spite of the fact that these two genes are already located in III linkage group. On the other hand the linkage between Plm and Pin with c.o. = 6.19 (Fig. 30) is in conformity with early reports with c.o. = 8 (Fig. 1).

The linkages between Plm and Pa/Ps with c.o. = 7.86 and 8.35 are new reports. The genes are located to group III as they had been already assigned to group III (Fig. 30).

The linkage between Psh and $Pau_{a/b}$ with c.o. = 2.53 is in conformity with early reports, though the c.o. value reported is higher (14.0). These genes have been located in group III earlier (Dhulappanavar, 1973a, Misro 1981; Pavithran et al., 1991).

The linkage between Pla and $Plg_{a/b}$ is also a new report eventhough the genes have already been located in the linkage group II earlier(Fig. 1), but presently they are added to the group III on

the basis of other associations. Similarly the linkages between Pla and $Pau_{a/b}$ with c.o. = 8.35 is also a new report, though the genes have already been located in the group III earlier (Dhulappanavar, 1973b; Hedagal et al., 1981; Misro, 1981; Pavithran et al., 1991). However, due to discrepancy in c.o. values, the linkages could not be appropriately incorporated into the present map.

The linkage between Pla and Pin with c.o. = 12.82 is in conformity with early reports and the genes have been located already in group III with c.o. = 6 (Hedegal et al.,1981).

The linkage between Pla and Pa/Ps with c.o. = 12.82 and 9.45 respectively also appear to be new, though the genes have been already located in group III (Pavithran et al., 1991 and Anitha,1994). The genes Pa and Ps are not having pleiotropic relations here.

The interrelationship of Pa and Ps is often referred to as pleiotropic by earlier workers (Chao, 1928; Nagao, 1951; Takahashi, 1957; Annie, 1986; Shyla and Pavithran, 1989; Shobha, 1993), but in the present cross they are linked with c.o. = 3.56 and are included in the group III as the genes have already been located therein. This is reported for the first time.

Pla and Pn are linked with c.o. = 11.12 in the present study

and it is a new report, though the genes have been located in group III earlier as they are linked with other genes (Dhulappanavar, 1973a; Hedegal et al.,1981; Pavithran et al., 1991).

Similarly, the linkage between Pla and $Pjp_{a/b}$ with c.o. = 1.51 appears a new report included in group III, as the genes have already been located therein by early workers (Dhulappanavar, 1973a; Hedegal et al.,1981; Pavithran, 1991).

The linkage associations observed in the control cross were transformed mostly into pleiotropy/independent assortment in the treated crosses, especially with 5000 rad treatment, as discussed elsewhere, it is not further discussed here. However, breakage of linkage is better accomplished with irradiation of pollen grains prior to using them for pollination.

c. Pleiotropy

Out of 91 combinations for 14 morphological characters studied, 30 showed pleiotropic associations in the control cross, of which ultimately 17 combinations involving 9 morphological characters showed pleiotropy for a common complementary gene in the 5000 rad treatment (Table 43, Fig. 31d). All these 17 combinations showed no change in their pleiotropic association from control to 5000 rad

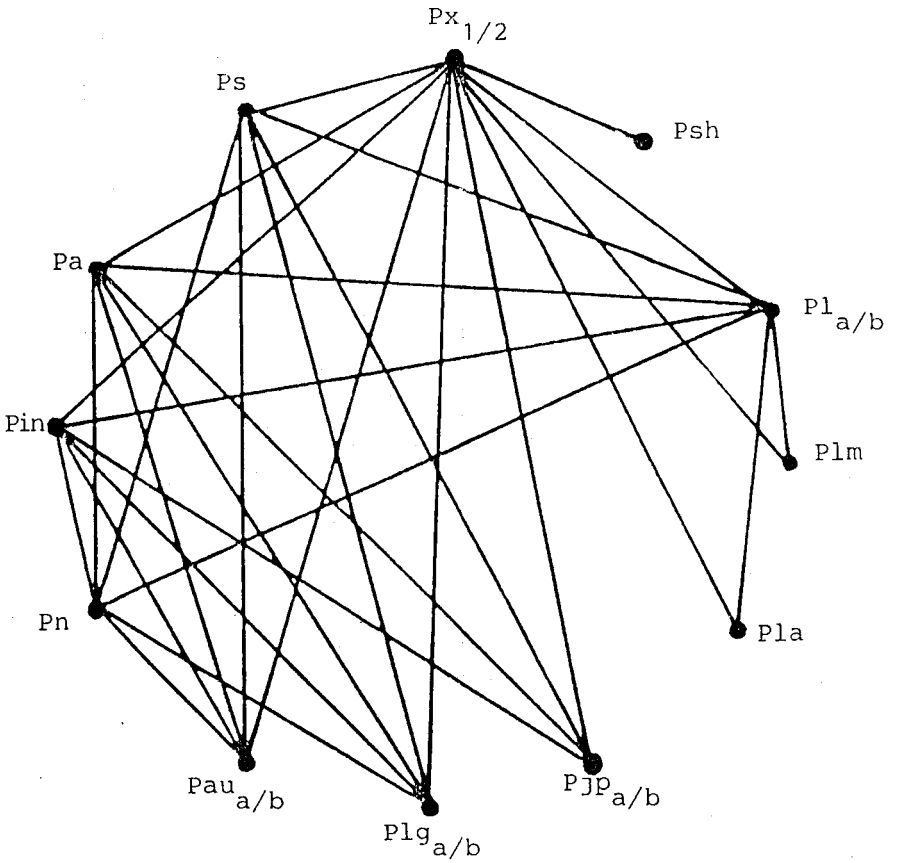
treatment, in spite of the fact that all the characters showed transformation of genetical ratios from the control, ie, either from 3:1 to 9:7 or from 9:7 to 27:37 (Table 39b). The pleiotropic relations of characters in 1-4 crosses are discussed below.

Leaf sheath (Psh) showed pleiotropic relation with $Px_{1/2}$ (differential) in the control, 1500 and 2000 rad (crosses 1-3) and also with $Plm_{a/b}$ in 2000 rad and $Psh_{a/b}$ with 9 characters as a common gene for $Pl_{a/b/c}$, $Pa_{a/b}$, $Plm_{a/b}$, $Pjp_{a/b/c}$ $Plg_{a/b/c}$, $Pau_{a/b/c}$, $Pin_{a/b}$ and $Ps_{a/b}$ in 5000 rad treatment (cross 4) (Table 43, Figs. 31a-d and 32).

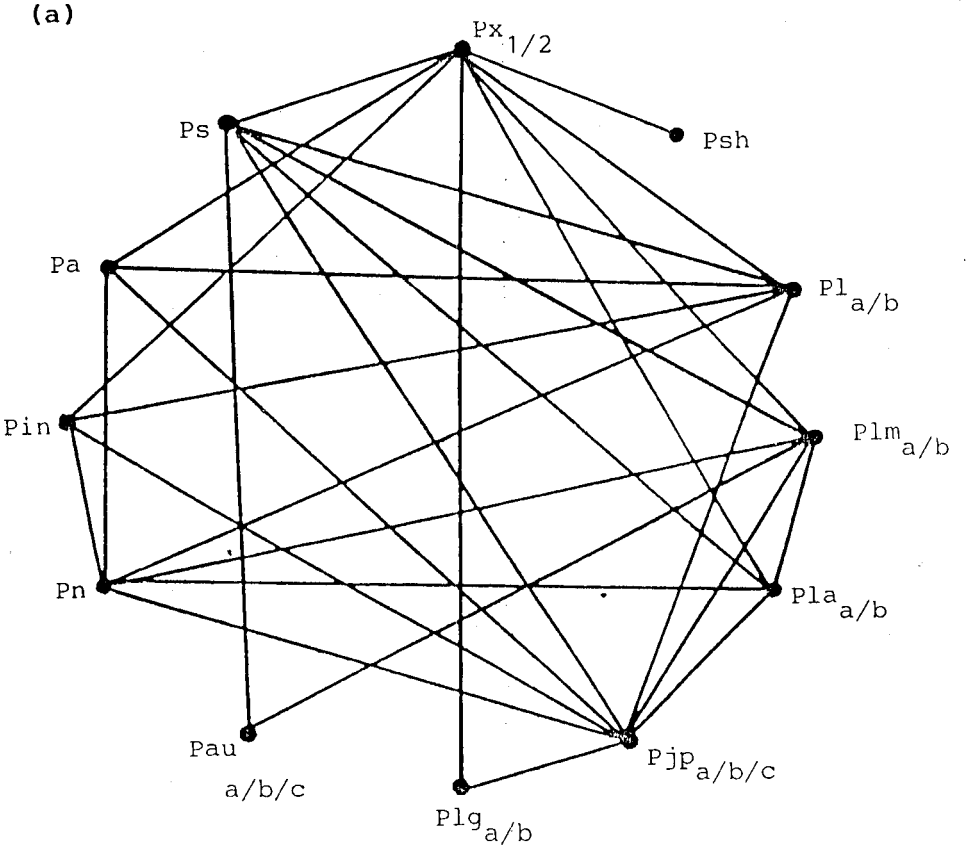
The pleiotropic association of Psh with these 9 anthocyanin characters is in conformity with early reports in respect of the characters (specific genes have been identified in the present study), while a total of 18 morphological characters including Psh have been estimated to have common gene/s for their control earlier (Setty and Misro, 1971; Dhulappanavar, 1973b, 1979; Sukeskumar, 1990; Shobha, 1993; Anitha, 1994), as represented in the pleiogenic Chart (Fig. 33). It may be concluded that the gene Psh might be complementary in nature including in the control cross (though elicited only 3:1) and is common for all other characters as one of the complementary genes in 5000 rad treatment (Fig. 31d) but showing differential action as a duplicate factor for the leaf axil (Table 39b) in the control, 1500 and 2000 rad (crosses 1-3). However, it

Fig. 31. Pleiotropic relationship of genes governing morphological characters in the normal cross (a) of Cherumodan x Japan violet and in the crosses (b, c & d) wherein 1500, 2000 and 5000 rad X-rayed pollen grains were used for pollination.

Pleiotropic genic relationship presently obtained in 1-4 crosses

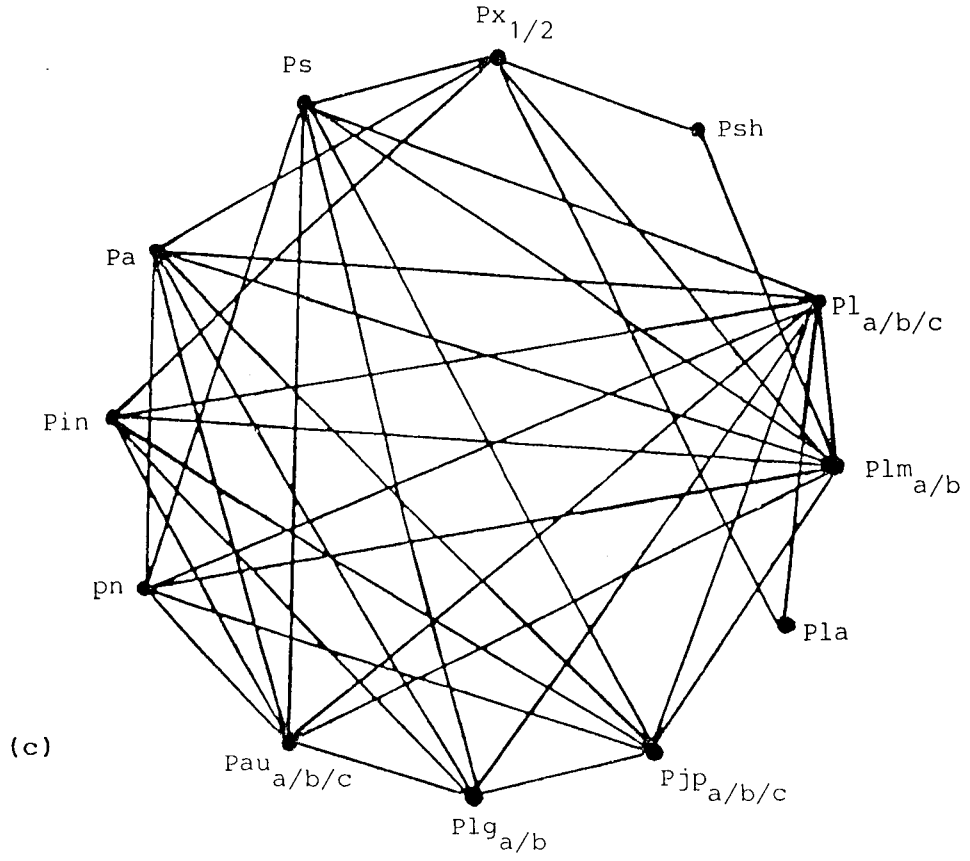


(a)

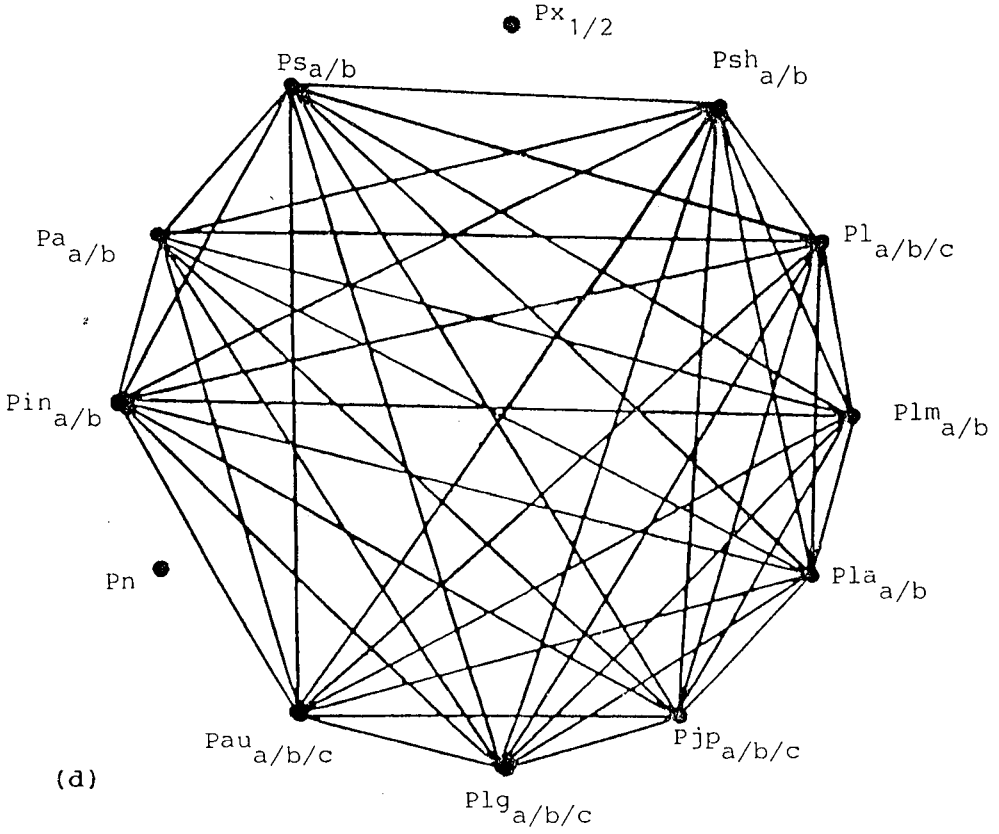


(b)

Fig. 31


Px 1/2
Ps
Psh
Pa
Pl a/b/c
Pin
Plm a/b
pn
Pla
Pau a/b/c
Pjp a/b/c
Plg a/b

(c)


Px 1/2
Ps a/b
Psh a/b
Pa a/b
Pl a/b/c
Pin a/b
Plm a/b
Pn
Pla a/b
Pau a/b/c
Pjp a/b/c
Plg a/b/c

(d)

Fig. 32. Instances of differential pleiotropic genic control of characters obtained in the crosses presently studied

DIFFERENTIAL PLEIOTROPIC GENIC RELATIONSHIP

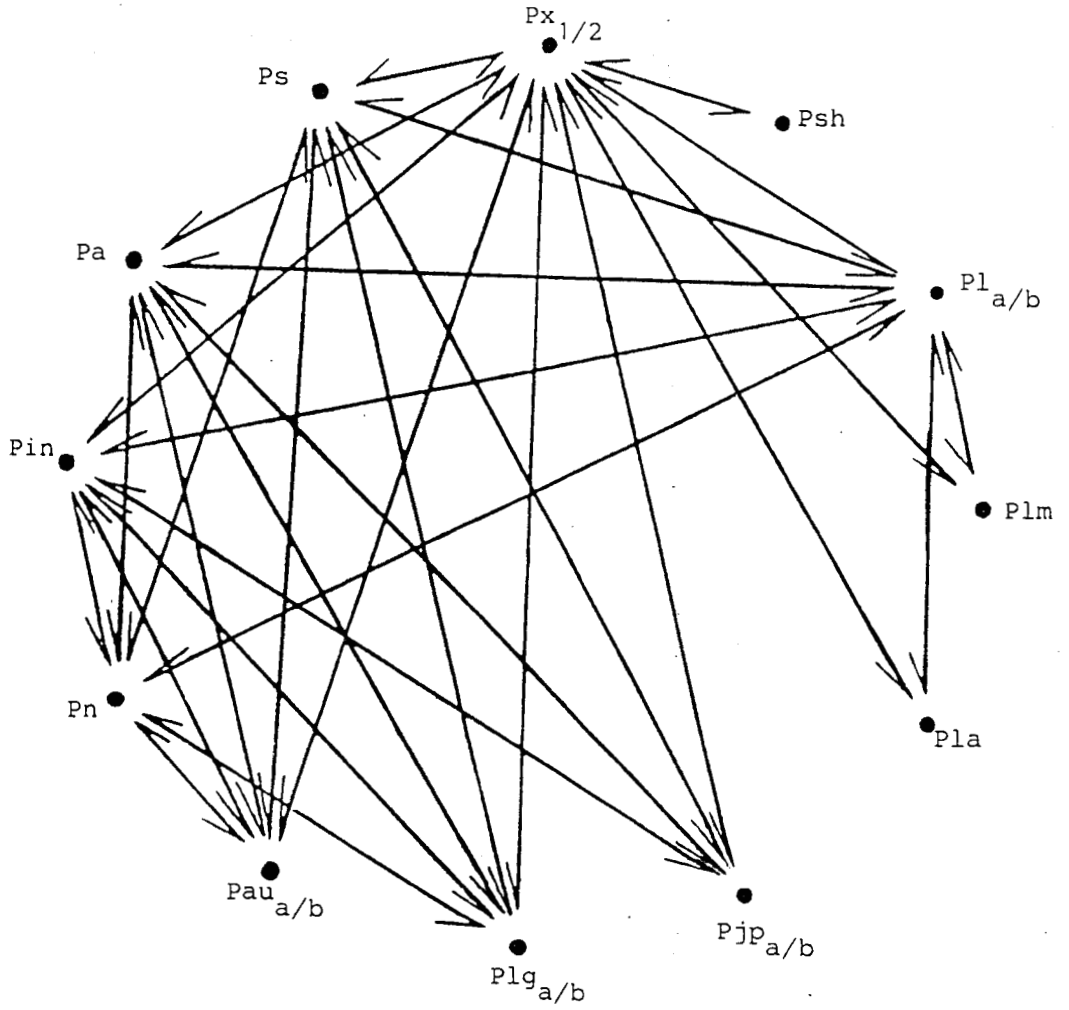
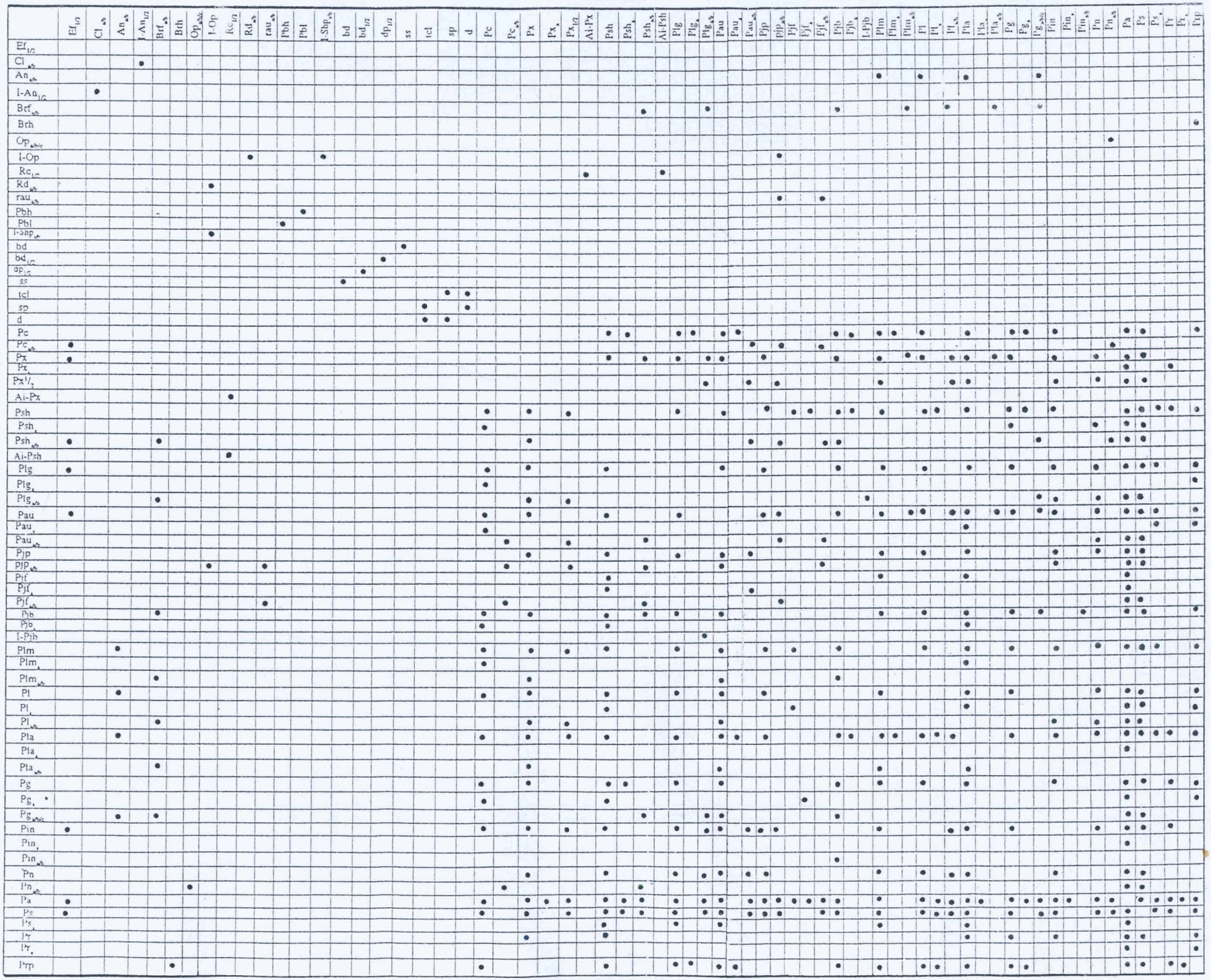


Fig. 32

Fig 33. Pleiogenic chart of rice showing the extent of pleiotropy of genes concerned - Present status

PLEIOGENIC CHART OF RICE



may also be noted that the linkage relations of Psh with $Pl_{a/b}$, $Pjp_{a/b}$, $Plg_{a/b}$, Pin, Pla, $Pau_{a/b}$, Ps, Pa and Pn were broken in 5000 rad treatment alone, giving rise to their pleiotropic association with Psh except for Pn which broke into independent assortment in 2000 and 5000 rad treatments, as discussed earlier. In this context $Psh_{a/b}$ functioned as a complementary common gene for all these characters (Fig. 31a-d).

Leaf blade ($Pl_{a/b}$) showed pleiotropy for Pa in 1-4 crosses, for Pn in 1-3, for Plm in 1, 3 & 4, for Pla in 1, 3&4 for Pin in 1-4, for Ps in 1-4 and for Px in 1 & 2. In addition, in 1500 rad (cross 2) $Pl_{a/b}$ is pleiotropic for $Pjp_{a/b/c}$, while 2000 rad (cross 3) treatment showed pleiotropy of $Pl_{a/b/c}$ with 9 characters to include further $Plg_{a/b}$ and $Pau_{a/b/c}$ (Fig. 31c). However, in 5000 rad (cross 4) the number of pleiotropy was increased to have one common gene for 10 morphological characters ie. $Pl_{a/b/c}$ being pleiotropic for $Pa_{a/b}$, $Plm_{a/b}$, $Pla_{a/b}$, $Pjp_{a/b/c}$, $Plg_{a/b/c}$, $Pau_{a/b/c}$, $Pin_{a/b}$, $Ps_{a/b}$, $Psh_{a/b}$ (Fig. 31d). According to early reports leaf blade has been estimated to have pleiotropic association with 15 morphological characters (Yamaguti, 1927; Dhulappanavar and Menzinkai, 1970; Dhulappanavar, 1973b; Sukeskumar, 1990; Shobha, 1993; Anitha, 1994). However, pleiotropy of leaf blade with Pin or $Pin_{a/b}$ is a new report (Fig. 31a-d). The specificity of genes in the present study in relation to the pleiotropic association of leaf blade, is

reported for the first time. However, the predominant action of the pleiotropic gene appears to be complementary in nature. The increase in number of pleiotropic association of genes in 5000 rad treatment is, probably, due to the change/breakage of linkage relations due to inversions as discussed earlier.

Apiculus (Pa) showed pleiotropy with $Pl_{a/b/c}$, $Pjp_{a/b/c}$, $Plg_{a/b/c}$, $Pau_{a/b/c}$ in 1-4 crosses and with Pn in 1-3 crosses and $Px_{1/2}$ in 1-3 crosses. In addition to this in 5000 rad, apiculus ($Pa_{a/b}$) also showed pleiotropic association with $Pla_{a/b}$, $Pin_{a/b}$, $Ps_{a/b}$ and $Psh_{a/b}$ thereby increasing its pleiotropic relations with more characters in 5000 rad treatment (cross 4) (Fig. 31-d). However, maximum extent of pleiotropic relationships with 17 morphological characters have been estimated earlier for apiculus (Anitha, 1994) and the present reports in 1-4 crosses are in conformity with early reports (Dhulappanavar and Menzinkai, 1970; Setty and Misro, 1970; Dhulappanavar, 1973a; Dhulappanavar et al., 1975a; Sukeskumar, 1990; Shobha, 1993) but for the specificity of $Pa_{a/b}$ and certain other genes (Fig. 31a-d). However, the pleiotropic relations of Pa-$Px_{1/2}$ in crosses 1 & 2, Pa-$Plm_{a/b}$ in cross 3 and of $Pa_{a/b}$ with $Pl_{a/b/c}$, $Psh_{a/b}$, $Ps_{a/b}$, $Pin_{a/b}$, $Pau_{a/b/c}$, $Plg_{a/b/c}$, $Pjp_{a/b/c}$, $Pl_{a/b}$ and $Plm_{a/b}$ are new reports for the specificity of the pleiotropic gene identified (Fig. 31a-d).

Pa is also linked with Psh, Pin, Plm, Pla and Ps in 1-3 crosses,

except Plm which showed pleiotropy in cross-3, while in the 4th cross these characters showed pleiotropic relations as discussed earlier.

Node (Pn) showed pleiotropy with Pa, $Pl_{a/b}$, Ps, Pin, $Pau_{a/b}$ and $Plg_{a/b}$ in control (cross 1). In 1500 rad (cross 2) Pn showed pleiotropy with Pa, $Plm_{a/b}$, $Pl_{a/b}$, $Pjp_{a/b/c}$, Pin, $Pla_{a/b}$, and $Plg_{a/b}$ and in 2000 rad, again with Pa, $Plm_{a/b}$, $Pjp_{a/b/c}$, $Pl_{a/b/c}$, Ps and $Pau_{a/b/c}$ and in the case of 5000 rad treatment no pleiotropic relationship could be observed for Pn with any of the morphological characters (Fig. 31a-d). It may also be noted that the linkage relation of Pn with Psh, Plm and Pla discussed elsewhere were transformed into independent assortment in cross 4, while Pn-Plm and Pn-Pla showed pleiotropy in crosses 2 and 3 and Pn-Psh gave independent assortment in cross 3 (Fig. 31a-d). These situations might be responsible for the total elimination of its pleiotropic relations in 5000 rad. However, the pleiotropy for Pn-$Pl_{a/b}$, Pn-$Pau_{a/b/c}$, Pn-$Plg_{a/b}$, Pn-$Pjp_{a/b/c}$, Pn-$Pla_{a/b}$, Pn-$Plm_{a/b}$ may be considered new reports. Pn has been reported to have pleiotropic relation with 11 morphological characters including Pl, Pla, Plm, Pjp, Pau and Plg earlier (Dhulappanavar, 1973a, 1975a; Shobha, 1993), but for the specific complementary gene being common among those characters, as seen from the estimated account of Anitha (1994) and as represented in the pleiogenic Chart (Fig. 33).

Leaf margin (Plm) showed pleiotropy with $Pl_{a/b}$ and $Px_{1/2}$ (differential) in control (Fig. 32), while $Plm_{a/b}$ showed pleiotropy for $Px_{1/2}$, Ps, $Pau_{a/b/c}$, $Pjp_{a/b/c}$, $Pl_{a/b}$ and Pn in cross 2 and, additionally, to Pa, $Pl_{a/b/c}$, Psh and Pin in cross 3. In 5000 rad a common complementary gene appeared pleiotropic for 10 morphological characters being $Plm_{a/b}$, $Pa_{a/b}$, $Pl_{a/b/c}$, $Psh_{a/b}$, $Ps_{a/b}$, $Pin_{a/b}$, $Pau_{a/b/c}$, $Plg_{a/b/c}$, $Pjp_{a/b/c}$ and $Pla_{a/b}$ (Fig. 31d) as described elsewhere. The pleiotropic relations of Plm, with $Pl_{a/b}$, $Px_{1/2}$ in crosses 1 & 2 and with $Pau_{a/b/c}$, $Pjp_{a/b/c}$ and $Pla_{a/b}$ in cross 2 and of $Plm_{a/b}$ with Pn, Pa, $Pl_{a/b/c}$, $Px_{1/2}$, Ps, Pin, $Pau_{a/b/c}$ and $Pjp_{a/b/c}$ in cross 3 and with $Pa_{a/b}$, $Pl_{a/b/c}$, $Psh_{a/b}$, $Ps_{a/b}$, $Pin_{a/b}$, $Pau_{a/b/c}$, $Plg_{a/b/c}$, $Pjp_{a/b/c}$ and $Pla_{a/b}$ in cross 4, appear to be reports for the first time in respect of the specificity of the pleiotropic gene concerned. However, the pleiotropic association of Plm with 17 morphological characters including Px, Psh,Plg, Pau, Pjp,Pl, Pla, Pin, Pa and Ps has been reported earlier (Setty and Misro, 1971; Shyla and Pavithran, 1989; Annie, 1986; Sukeskumar, 1990; Shobha, 1993; Anitha, 1994), as represented in the pleiogenic Chart (Fig. 33). A common pleiotropic complementary gene could be identified to have control over 10 morphological characters in cross 4, in spite of the fact that Plm showed pleiotropy only with Pl and Px in the control. It may be noted that the linkage relations of Plm, with Pla, Pjp, $Pjp_{a/b}$, $Plg_{a/b}$, $Pau_{a/b}$, Pn, Pin, Ps and Pa were broken in crosses 2, 3 & 4 in most cases except the linkage between Plm and

Pn that changed into independent assortment in cross 4, while into pleiotropy in cross 2 & 3. This, probably, suggests the reason for the linear increase of pleiotropic association of Plm with other morphological characters due to the treatment.

Leaf tip (Pla) showed pleiotropy with $Pl_{a/b}$ and $Px_{1/2}$ in control, $Pla_{a/b}$ with $Plm_{a/b}$, Pn, $Px_{1/2}$, Ps and $Pjp_{a/b/c}$ in cross 2 and Pla with $pl_{a/b/c}$, Psh, and $Px_{1/2}$ in cross 3 while in cross 4 a common complementary gene could be observed for 10 morphological characters being $Pla_{a/b}$, $Plm_{a/b}$, $Pa_{a/b}$, $Pl_{a/b/c}$, $Psh_{a/b}$, $Ps_{a/b}$, $Pin_{a/b}$, $Pau_{a/b/c}$, $Plg_{a/b/c}$ and $Pjp_{a/b/c}$, thereby showing almost linear increase in pleiotropic association in the treated crosses (Fig. 31a-d). Pla has been reported to have pleiotropic association with 18 morphological characters including Plm, Pa, Pl, Psh, Ps, pin, Pau, Plg and Pjp (Setty and Misro, 1971; Dhulappanavar, 1975a; Annie, 1986; Sukeskumar, 1990; Shobha, 1993; Anitha, 1994) in conformity with the present report but for the specificity of the pleiotropic gene identified here as one of the complementary factors. However, Pla has shown differential pleiotropy by being duplicate factor for the control of leaf axil pigmentation. The pleiotropic relations Pla-$Pl_{a/b}$, Pla-$Px_{1/2}$, $Pla_{a/b}$ with $Plm_{a/b}$, Pn, $Px_{1/2}$, Ps and $Pjp_{a/b/c}$, Pla-$Pl_{a/b/c}$ and with $Px_{1/2}$, $Pla_{a/b}$ being pleiotropic for $Plm_{a/b}$, $Pa_{a/b}$, $Pl_{a/b/c}$, $Psh_{a/b}$, $Ps_{a/b}$, $Pin_{a/b}$, $Pau_{a/b/c}$, $Plg_{a/b/c}$ and $Pjp_{a/b/c}$, as discussed above, may be considered as new reports on

the basis of specificity of the identified pleiotropic gene for these characters.

Junctura proper $Pjp_{a/b}$ showed pleiotropy with Pa, $Px_{1/2}$, Ps and Pn in cross 1, $Pjp_{a/b/c}$ with $Pl_{a/b}$, $Plm_{a/b}$, Pn, Pa, $Pl_{a/b}$, Ps, Pin and $Plg_{a/b}$ in cross 2, with $Plm_{a/b}$, Pn, Pa, $Pl_{a/b/c}$, Ps, Pin and $Plg_{a/b}$ in cross 3 and $Pla_{a/b}$ with $Plm_{a/b}$, $Pa_{a/b}$, $Pl_{a/b/c}$, $Psh_{a/b}$, $Ps_{a/b}$, $Pin_{a/b}$, $Pau_{a/b/c}$ and $Plg_{a/b/c}$, where a common complementary gene appeared pleiotropic for these characters. The situation represents a linear increase in the pleiotropic association of Pjp from crosses 1-4, it may also be noted $Pjp_{a/b}$ showed linkage with Psh, Plm and Pla, which gave rise to pleiotropy in cross 4 and in crosses 2&3 (Fig. 31b-d).

Junctura proper (Pjp) has been reported to have pleiotropic association with 11 morphological characters including Psh, Pau, Pa, Ps, Pn, Pla, Pin, Pn, Plm and Plg, as reported and estimated earlier (Setty and Misro, 1970; Dhulappanavar, 1973b, 1979; Shobha, 1993; Anitha, 1994) and represented in the pleiogenic Chart (Fig. 33). However, the pleiotropic relations $Pjp_{a/b}$--$Px_{1/2}$ (differential), $Pjp_{a/b}$--Pin (differential) in cross 1 and $Pjp_{a/b}$--Pla, Plm, Pn, Pl, Pin and Plg in cross 2 (differential), $Pjp_{a/b/c}$ with $Plm_{a/b}$, Pn, $Pl_{a/b/c}$, Pin and $Plg_{a/b}$ in cross 3 and sharing a common gene for $Pla_{a/b}$, $Plm_{a/b}$, $Pa_{a/b}$, $Pl_{a/b/c}$, $Ps_{a/b}$, $Pin_{a/b}$, $Pau_{a/b}$ and $Plg_{a/b/c}$

and $Psh_{a/b}$ are probably new reports in respect of the specificity of the common complementary gene responsible for the pleiotropic association of these characters (Fig. 31b-d).

Ligule ($Plg_{a/b}$) showed pleiotropic association with Pn, Pa, $Px_{1/2}$, Ps and Pin in cross 1, for $PjP_{a/b/c}$, Pn, $Px_{1/2}$ and Ps in cross 2 and for $Pjp_{a/b}$, Pa, $Pl_{a/b/c}$, Ps, Pin, $Pau_{a/b/c}$ in cross 3, while $Plg_{a/b/c}$ showing one of the genes pleiotropic for $Pjp_{a/b/c}$, $Pla_{a/b}$ $Plm_{a/b}$, $Pa_{a/b}$ $Pl_{a/b/c}$, $Psh_{a/b}$, $Ps_{a/b}$, $Pin_{a/b}$ and $Pau_{a/b/c}$ in cross 4 (Fig. 31d). Plg also showed linkage with Psh, Plm and Pla in cross 1-3 (Fig. 30) and changed its system of interrelationship in cross 4, probably due to pollen irradiation. However, a linear increase in pleiotropic association could be observed in the treatments with maximising it in the cross 4 (Fig. 31d). Ligule pigmentation has been reported to have pleiotropic association with 15 morphological characters including Pn, Plm, Pa, Ps and Plg (Dhulappanavar and Menzinkai, 1970; Dhulappanavar, 1973a; Dhulappanavar et al., 1975a; Sukeskumar, 1990; Shobha, 1993; Anitha, 1994). The pleiotropic relation of $Plg_{a/b}$ with Pn, $Px_{1/2}$, Pin, Pjp, $Pjp_{a/b/c}$, $Pl_{a/b/c}$, $Pau_{a/b/c}$ and between $Plg_{a/b/c}$ and $Pjp_{a/b/c}$, $Pla_{a/b}$, $Plm_{a/b}$, $Pa_{a/b}$, $Pl_{a/b/c}$, $Psh_{a/b}$, $Ps_{a/b}$, $Pin_{a/b}$ and $Pau_{a/b/c}$ may be considered as reports for the first time in respect of the specificity of the gene identified.

Auricle (Pau) pigmentation showed pleiotropic association with

Pn, Pa, $Px_{1/2}$, Ps and Pn in cross 1. In cross 2 $Pau_{a/b/c}$ showed pleiotropic relation with $Plg_{a/b}$, $Plm_{a/b}$ and Ps, and in cross 3 with Pn, $Plg_{a/b}$, Pa, $Plm_{a/b}$, $Pl_{a/b/c}$, Ps and Pn. In cross 4 one of the complementaries for auricle pigmentation ($Pau_{a/b/c}$) showed pleiotropic association with $Pjp_{a/b/c}$, $Plm_{a/b}$, $Pl_{a/b/c}$, $Ps_{a/b}$, $Plg_{a/b/c}$, $Pla_{a/b}$, $Pa_{a/b}$, $Psh_{a/b}$ and $Pin_{a/b}$, thereby showing linear increase in pleiotropic association of $Pau_{a/b}$ due to pollen irradiation. It may also be noted that $Pau_{a/b}$ showed linkage with Plm, Psh and Pla which was broken to yield their pleiotropic association in crosses 2-4 due to the use of irradiated pollen.

Auricle pigmentation has been reported to have pleiotropic control over 15 plant parts including $Psh_{a/b}$, $Pjp_{a/b}$, Pa and Ps, Pla and others as reported earlier (Hector, 1922; Dhulappanavar, 1973a, 1975a, 1979; Shobha, 1993) and as consolidated earlier (Anitha, 1994) and represented in the pleiogenic Chart (Fig. 33). As such the pleiotropic relations of auricle: $Pau_{a/b}$--Pn/$Px_{1/2}$/Pin, $Pau_{a/b/c}$ -- $Plg_{a/b}$, $Plm_{a/b}$, $Plg_{a/b}$, $Plm_{a/b}$, Pn, $Pl_{a/b/c}$ and $Pau_{a/b/c}$ -- $Plg_{a/b/c}$, $Pjp_{a/b/c}$, $Pla_{a/b}$, $Plm_{a/b}$, $Pa_{a/b}$,$Pl_{a/b/c}$, $Psh_{a/b}$, $Ps_{a/b}$ and $Pin_{a/b}$ may be considered reports for first time especially in respect of the identity of the pleiotropic gene (Fig. 31d).

Internode (Pin) showed pleiotropic association with $Pau_{a/b}$, $Plg_{a/b}$, $PjP_{a/b}$, Pn, $Pl_{a/b}$ and $Px_{1/2}$ in cross 1, with $Pjp_{a/b/c}$,

$Plm_{a/b}$, Pn, $Pl_{a/b}$ and $Px_{1/2}$ in cross 2, with $Pau_{a/b/c}$, $Plg_{a/b}$, $Pjp_{a/b/c}$, $Plm_{a/b}$, $Pl_{a/b/c}$ and $Px_{1/2}$ in cross 3. In cross 4 purple internode ($pin_{a/b}$) showed pleiotropy with $Pau_{a/b/c}$, $Plg_{a/b/c}$, $Pjp_{a/b/c}$, $Pla_{a/b}$, $Plm_{a/b}$, $Pa_{a/b}$, $Pl_{a/b/c}$, $Psh_{a/b/c}$ and $Ps_{a/b}$ sharing one of the complementary genes being pleiotropic for all the character (Fig. 31d). The situation represents a linear increase in the pleiotropic association of internode. It may be noted that Pin showed linkage with Psh, Plm and Pla which turned out to be pleiotropic mostly in cross 3 and 4 by breaking the linkage associations.

The pleiotropic relations of Pin in view of the literature in relation to $Pau_{a/b}$, $Plg_{a/b}$, $Pjp_{a/b}$, $Pl_{a/b}$, $Px_{1/2}$, $Pjp_{a/b/c}$, $Plm_{a/b}$, $Pau_{a/b/c}$, $Pjp_{a/b/c}$, $Pl_{a/b/c}$ in 1-3 crosses and the pleiotropic association of $Pin_{a/b}$ with $Pau_{a/b/c}$, $Plg_{a/b/c}$, $Pjp_{a/b/c}$, $Pla_{a/b}$, $Plm_{a/b}$, $Pa_{a/b}$, $Pl_{a/b/c}$, $Psh_{a/b}$ and $Ps_{a/b}$ showing a common complementary pleiotropic gene for all the above characters may be considered report for first time. However, Pin has been reported to have pleiotropic association with 13 morphological characters (Anitha, 1994) including Px, Psh, Plg, Pau, Pjp, Plm, Pla, Pa and Ps which is in conformity with present observation, but for the specific identity of the complementary gene reported herein (Fig. 31a-d).

Stigma (Ps) showed pleiotropic association with $Pau_{a/b}$, $Plg_{a/b}$, $Pjp_{a/b}$, Pn, $Pl_{a/b}$ and $Px_{1/2}$ in cross 1, with $Pau_{a/b/c}$, $Plg_{a/b}$,

$Pjp_{a/b/c}$, $Pla_{a/b}$, $Plm_{a/b}$, $Pl_{a/b}$ and $Px_{1/2}$ in cross 2 and with $Pau_{a/b/c}$, $Plg_{a/b}$, $Pjp_{a/b/c}$, $Plm_{a/b}$, Pn, $Pl_{a/b/c}$ and $Px_{1/2}$ in cross 3. Stigma in cross 4 ($Ps_{a/b}$) shared a common complementary pleiotropic gene with $Pin_{a/b}$, $Pau_{a/b/c}$, $Plg_{a/b/c}$, $Pjp_{a/b/c}$, $Pl_{a/b}$, $Plm_{a/b}$, $Pa_{a/b}$, $Pl_{a/b/c}$ and $Psh_{a/b}$, thereby indicating the existence of a common complementary pleiotropic gene for all these characters which functioned as one of the duplicate factors for leaf axil. The situation expresses a linear increase in pleiotropic associations of stigma with the other characters maximising in cross 4, probably, due to the breakage of its linkage associations as discussed earlier. It may also be noted that Ps showed linkages with Pa, Psh, Pin, Plm and Pla which ultimately broke into pleiotropic association especially in cross 4 (Table 44).

Stigma pigmentation has been reported to have pleiotropic association with 19 morphological characters including Px, Psh, Plg, Pau, Pjp, Plm, Pl, Pla, Pin and Pa and several others as consolidated by Anitha (1994) and reported by Dhulappanavar and Menzinkai (1970), Setty and Misro (1970), Dhulappanavar et al. (1975c), Annie (1986), Shyla and Pavithran (1989), Sukeskumar (1990) and Shobha (1993) and represented in the pleiogenic Chart (Fig. 33). However, the following pleiotropic relations of Ps with $Pl_{a/b}$, $Px_{1/2}$, $Pla_{a/b}$, $Plm_{a/b}$, $Pl_{a/b/c}$ and that of $Ps_{a/b}$ with $Pin_{a/b}$, $Pau_{a/b/c}$, $Plg_{a/b/c}$, $Pjp_{a/b/c}$, $Pla_{a/b}$, $Plm_{a/b}$, $Pa_{a/b}$, $Pl_{a/b/c}$ and $Psh_{a/b}$ may be considered

reports for first time, particularly in respect of the specific common complementary pleiotropic gene which showed control over these morphological characters.

Leaf axil ($Px_{1/2}$) showed pleiotropic association with Psh, $Pl_{a/b}$, Pa, Plm, Pla, $Pjp_{a/b}$, $Plg_{a/b}$, $Pau_{a/b}$, Pin and Ps in cross 1, with Psh, $Pl_{a/b}$, Pa, $Plm_{a/b}$, $Pla_{a/b}$, $Plg_{a/b}$, Pin and Ps in cross 2, and with Psh, Pa, $Plm_{a/b}$, Pla, Pin and Ps in cross 3, leaving no pleiotropic association in cross 4 as discussed earlier (Fig. 31a-d). It may also be noted that leaf axil did not have any linkage relation with any of these characters studied in 1-4 crosses irrespective of pollen irradiation treatments (Fig. 30). However, leaf axil has been reported to have pleiotropic association with 14 morphological characters including Psh, Plg, Pau, Pjp, Plm, Pl, Pla, Pin, Pa and Ps (Setty and Misro, 1970; Dhulappanavar, 1975a; Anitha, 1994), but no pleiotropic association has been reported with duplicate factors responsible for leaf axil pigmentation. As such the pleiotropic associations, of $Px_{1/2}$ mentioned above may be considered reports for the first time.

i. Pleiotropism in rice

It has been approximately estimated that nearly 650 genes have been identified in rice to have control over 200 unit characters so far recognised in rice (Pavithran, 1986,1991; Kinoshitha

1991, 1992, 1993) and also that over 10% of those genes have been estimated to have pleiotropic control over different morphological characters (Anitha, 1994). The Extent of pleiotropy varies from 2-30/36 morphological characters. Rice genetics exhibits enormous complexity in the patterns of inheritance and also in pleiotropic control of genes by being either non-differential or differential (Dhulappanavar, 1973a, b, c, 1975a, b; 1977; Pavithran and Annie, 1980; Shyla, 1984; Pavithran, 1985a, b; Pavithran and Shyla, 1989; Sukeskumar, 1990; Pavithran et al., 1991; Shobha, 1993 and Anitha, 1994). As the pleiotropic genes are not properly represented in the linkage maps, it is difficult to survey the extent of pleiotropic genes discovered so far in rice (Dhulappanavar, 1973-1977, Pavithran, 1985b).

Pleiotropy has been recorded in rice since 1910, first by Plate, who put forward the theory of pleiotropism in plants and animals. Pleiotropic gene action has been regarded as a reflection of highly integrated state of cellular metabolism that leads to manyfold phenotypic effects of a single gene or its polyphenetic expressions. It may also be asserted that the exact mechanism of pleiotropism during developmental cycle is not fully known at its biochemical or molecular level. However, several hypotheses have been put forward to explain the phenomenon of pleiotropism on the basis of

post-translational regulation of functional enzymes or proteins that finally make up the characters concerned (Postelthwait and Schneederman, 1973 and Scandalios, 1980). Goud and Kullaiswamy (1984) postulated the role of a common enzyme for different bio-chemical pathways operated by several other genes. Similarly presence of two or more common genes may mediate different stages in the two synthetic pathways of anthocyanin, thereby expressing morphographic similarities of different characters being controlled by pleiotropic genes. However, the possible origin of pleiotropism through mutation in higher organisms has been already emphasised by Stebbins (1954, 1974). No experimental follow up is seen to have been made in this direction so far, as revealed by the available literature. Pleiotropism in rice, especially in respect of anthocyanin pigmentation, has been consistently substantiated with genetical analysis. The present study carried out with irradiated pollen grains for crossing in contrast to normal pollination has resulted in certain salient observations on the phenomenon of transformation of genetical ratios and interrelationships of genes, as discussed earlier.

However, it may be concluded that 1/2/3 complementary genes or inhibitory gene/duplicate genes or complementary duplicate genes have been reported functioning as pleiotropic gene/s for the phenotypic

control of several characters in rice in general. Further, 1/2/3 genes being complementary in certain characters function as duplicate/inhibitory/ or basic in certain other characters, thereby showing differential pleiotropy with the extent of pleiotropy reaching upto 14 to 15 characters or even more as has been consolidated in the pleiogenic chart (Fig. 33).

D. QUANTITATIVE MORPHOLOGICAL CHARACTERS

Japan Violet treated with different dosages of EMS was also studied for variations induced in certain quantitative morphological characters. The results are presented elsewhere under chapter IV. Critical analysis was done on micromutations in respect of the morphological characters: plant height, culm length, EBT, % of EBT, days to flower, panicle length, total spikelets/panicle, grains/panicle, panicle density and % of sterility. Their frequency distribution (Figs. 20-29), range, mean and analysis of variance (Table 46) and variance index (Table 47) indicated that plant height, culm length, EBT, days to flower, panicle length, grains/panicle, panicle density and % of sterility showed significant variation in M_1-M_3 with similar trend of bidirectional variation. EBT% showed significant variation with reductional or negatively unidirectional trend of variation in M_1, M_2 and M_3 populations (Tables 46 & 47). Total

spikelets/panicle also showed significant variation with negatively unidirectional trend in M_1 and with bidirectional trends of variation in M_2 and M_3 (Tables 46 & 47). All the above quantitative morphological characters showed significant variation, as indicated by the F values for analysis of variance in M_1, M_2 and M_3 populations of various treatments. The values ranged from 3.13 for EBT to 23.62 for days to flower in M_1, from 4.85 for days to flower to 18.69 for panicle density in M_2 and from 3.84 for days to flower and panicle length to 14.90 for plant height in M_3 (Table 46).

The range of variations in morphological characters that showed bidirectional trend in M1, M2 and M3 populations exceeded both the limits of the respective control. This indicates the probable polygenic transgressive segregation due to mutation to polygenes that might have occurred as a result of EMS treatment. The extent of transgression might be dependent upon the number of negative/positive polygenic mutations occurred to the total polygenic loci in the control.

Unlike the above morphological characters, EBT % showed significant variation with negatively unidirectional trend towards reduction in EBT % in M1, M2 and M3 relative to the respective control. This situation might, probably, indicate the degree of recessive polygenic mutation occurred for the control of EBT production. On the other hand total spikelets/panicle,

variation showed negatively unidirectional trend towards reduction in spikelet number/panicle in M_1 and bidirectional variation showing transgressive segregation in M_2 and M_3. This indicated the probable polygenic mutations towards negative and positive directions as in the case of certain other characters described above. The negatively unidirectional trend of variation in spikelets/panicle was observed only with certain treatments, while a bidirectional variation could be observed with 0.5% EMS treatment. However almost all characters followed bidirectional induction of polygenic mutation except in the case of % of EBT, probably, due to differential response of genetic systems controlling various morphological characters to the mutagen used in the present context.

The above observations on the positive and negative effects of the mutagens on quantitative morphological characters are in conformity with earlier observations (Oka et al., 1958; Tanaka, 1968; Shobha, 1993). Early studies on quantitative morphological characters and their inheritance have been reviewed elsewhere (Johanson, 1903; Yule, 1906; Nilson Ehle, 1908; East, 1916; Mather, 1941). However, Batesman (1959) observed symmetrical induction of positive and negative mutation in respect of heading date which varied unidirectionaly towards lateness, and plant height towards positive direction.

Studies have shown that induced variability of quantitative morphological characters, especially of agronomically significant morphological characters, have shown the possibility of genetic manipulation in mutation breeding (Gaul, 1958, 1964; Narahari, 1969; Basu and Basu, 1970; Nair, 1972; Nayar and Ninan, 1974; Roy and Jena, 1975; Gangadharan and Mishra, 1976; Sreedharan, 1979; Mather and Jinks, 1982; Hajra et al., 1986; Mohanan, 1988 and Shobha, 1993). It has been reported earlier that longer duration of EMS treatment (16 & 20 h) has increased panicle length, fertile spikelets/panicle and length/breadth ratio and certain other characters (Sarawgi and Soni, 1993). Isozyme loci have been suggested to be used as markers for quantitative traits, when they are linked with certain quantitative morphological characters in rice (Lopez and Virmani, 1990). Induced micromutations or quantitative variations have been attributed to mutations of the 'controlling elements' rather than of the structural genes (Mukai and Cockerham, 1977). However, it seems relevant to assume that polygenes are no exception to mutations or recombinations and also that polygenes also follow Mendelian principles in their inheritance like major genes (Mather and Jinks, 1982). The authors have further pointed out that intragenic mutation and intergenic recombination will not be directly distinguishable when genes (polygenes) have physiologically small, similar and supplementary actions. It may be further concluded that induction of polygenic mutations might produce negative or positive unidirectional variation or symmetrical or asymmetrical bidirectional variations in relation

to the nature of response of genes for morphological characters concerned and for their genetic background to the mutagen. However, informations on the subtle aspects of polygenic inheritance are relatively very scantly in crop plants like rice or wheat. It seems imperative that attention be focussed on the genetic manipulation of quantitative morphological characters for evolving effective plant breeding strategies in crop plants.

SUMMARY AND CONCLUSION

SUMMARY AND CONCLUSION

1. The present investigation on 'Genetical and transformation studies in rice, Oryza sativa Linn.,' comprises of (a) mutagenesis in an indica rice, Japan Violet, treated with different concentrations of Ethylmethane sulfonate, (b) hybridization experiment using the cross Cherumodan x Japan Violet as control and the same cross combination using irradiated pollen grains of Japan Violet for pollination and (c) 14 crosses between the source parent, Japan Violet, and the mutants isolated from the mutagenic studies.

2. The studies were carried out with M_1 - M_3 or F_1/M_1F_1 to F_3/M_3F_3 generations in respect of mutagenic studies and the hybridization experiments respectively in the Botanical Garden, University of Calicut, Kerala, India, during 1990-1994. The materials used and methodology adopted for the studies are detailed elsewhere under Chapter III.

3. Literature relevant to mutagenesis in rice, inheritance and interrelationships of genes governing morphological characters have been reviewed elsewhere under Chapter II and considered while interpreting the results.

4. The morphological characters studied upto M_3/M_4 generation in mutagenesis with EMS on Japan Violet included 19 macromutants mentioned elsewhere. Of these, 14 mutants were crossed with the source parent Japan Violet and their inheritance was confirmed with the respective F_3 generations.

5. The morphological characters analysed in hybridization experiments with Cherumodan x Japan Violet (Normal cross and crosses wherein irradiated pollen grains of Japan Violet with different dosages of X-rays were used for pollination) are leaf axil, leaf sheath, leaf margin, leaf tip,ligule, auricle, node, internode, apiculus and stigma pigmentation and two non-anthocyanin characters, tip-sterility and awning.

6. Appropriate statistical techniques such as Chi-square test for goodness of fit of individual genetical ratios/joint segregation of characters, specific formulae for estimation of linkage on minimum discrepancy, and frequency distribution, range, mean, analysis of variance and variance index for analysis of quantitative morphological characters were used as described in detail under Chapter III. For estimation of mutagenic effectiveness and efficiency, technique used by Konzak and others was followed.

7. Results and conclusions on the studies of mutagenicity,

morphology, inheritance and interrelationships of EMS induced mutant morphological characters,(2)inheritance of anthocyanin and non-anthocyanin morphological characters in the crosses of Cherumodan x Japan Violet wherein irradiated pollen grains were used for pollination,(3)interrelationships of morphological characters in these crosses and(4)analysis of quantitative morphological characters in M1-M3 of Japan Violet treated with EMS are summarised below. These studies have broughtforth considerable new information on mutagenicity, genetic transformation, pattern of inheritance and interrelationships of morphological characters presently studied.

(a) **Mutagenic effectiveness and efficiency of EMS** in relation to various dosages and pre-treatment with Vit. C solution exhibited better effects with Vit. C pre-treatment with 1% EMS, where percentage of germination was significantly higher. Further, a situation of cleistopary coupled with viviparous germination presently obtained exhibited 3 types of situations, ie.,(1)cleisto-vivipary with anterior emergence of the plumule,(2) cleisto-vivipary with anterior/ sub-anterior emergence of both plumule and radicle and(3) cleisto-vivipary with dynamic plumular growth leading to ejection of the young seedling through the anterior micropylar end. However, observation on radicle/plumule and radicle-plumule inhibition occurred with different concentrations of EMS showed that Vit. C + 1% EMS caused maximum plumule inhibition. On the whole a linear increase

in radicle, plumule inhibition could be observed with the optimum reaching between 0.75% and 1% EMS treatment.

(b) **Seedling survival** showed that survival was higher in M2 and M3 generations in almost all treatments. It could be surmised that survival recovery is higher in later generations than in M_1, where the initial and direct mutagenic effects are responsible for lower survival of seedlings. This is in conformity with early reports. With regard to the use of Vit. C pre-treatment, percentage of reduction in seedling survival showed linear increase upto the treatment Vit. C + 0.75% EMS, With Vit. C + 1% EMS, it showed less percentage of reduction in seedling survival in comparison to the treatment DW + 1% EMS in M_1, thereby, showing Vit. C + 0.75% EMS as the optimum level of enhancement of the mutagenic effects.

(c) Studies on **root-shoot growth** and **root/shoot ratio** showed that plumule growth was more affected by the treatment than the radicle growth. Maximum inhibition of plumule was observed with DW + 1% EMS and radicle inhibition with Vit. C + 0.75% EMS. However, root-shoot ratio showed significant variation in M_2 of DW + 0.75% EMS, DW + 1% EMS and in M3 of Vit. C + 0.75% EMS and in M_1, M_2 and M_3 of Vit. C + 1% EMS. This indicated recovery of root-shoot growth in later generations and the influence of pre-treatment with Vit. C followed by 1% EMS treatment, as it was found to be affecting M_1, M_2 and M_3 generations.

(d) The use of **Vit. C solution** as a pre-treatment agent preceding EMS treatment is a novel attempt in the present study to discover if there is any synergistic interaction between Vit. C and EMS in relation to its mutagenicity. The study has broughtforth several informations including the role of Vit. C in enhancing the mutagenicity of the EMS in rice for the first time.

(e) The **mutants** elicited from various treatments of EMS and the patterns of inheritance and genic interrelationships are described in detail under Chapter IV. The mutants so described and discussed are albinos, lethal yellow, chlorina, striped mutant (with rose-purple stripes), deformed palea or palealess mutant (new for EMS induction), beaked lemma-depressed palea, long sterile glume, grassy rhizomatous purple plant (new report), procumbent (new report), broad seed (new with EMS), anther sterile, abnomorphic spikelets, recessive tall (new report), multipistil, continuous high tillering dwarf (new with EMS), partial green (new report), complete green (new report), brittle culm and spotted leaf. All these mutants showed recessive monogenic control except long sterile glume which showed duplicate recessive. On the whole treatments of EMS with Vit. C pre-treatment seems to produce more number of mutants (some of which like male sterile, tall recessive, high tillering dwarfs and complete green) appeared to be directly/indirectly significant agronomically. The common mutants occurred with or without Vit. C treatment are albinos, lethal yellow

and chlorina. Altogether EMS alone produced 10 mutations, but the synergistic action of Vit. C and EMS with the same dosages produced 12 mutations, thereby indicating the possibility of enhancement of mutagenicity of EMS with Vit. C pretreatment.

(f) Studies on the **inheritance** of 14 morphological characters in the crosses Cherumodan x Japan Violet as control and Cherumodan x Japan Violet pollen grains irradiated with 1500 rad/2000 rad/5000 rad X-rays showed duplicate factors for leaf axil, inhibitory factor control for nodal pigmentation and monogenic recessive control for awning and tip sterility invariably in all the above crosses.

On the other hand leaf sheath, leaf margin, leaf tip, internode, stigma and apiculus showed dominant monogenic control of pigmentation in the normal cross but digenic complementary interaction invariably in the cross Cherumodan x Japan Violet pollen treated with 5000 rad. Further, in the case of leaf margin, the treatment with 1500 and 2000 rad X-ray gave 9:7 and leaf tip gave 9:7 with 1500 rad X-ray, not with 2000 rad X-ray treatment. However, in the case of leaf sheath, internode, apiculus and stigma only 5000 rad X-ray treatment gave digenic complementary ratio instead of 3:1 in the normal cross or other crosses.

This **transformation of genetical ratios** in the treated crosses demonstrates the fact that one of the loci remained heterozygous in

the original cross, while X-ray might have transformed the other locus (loci) also heterozygous by mutating (either of the complementary genes) into recessive in the treated pollen grains of Japan violet leading to the change of genetical ratio concerned.

Further, in the case of leaf blade, junctura proper, ligule and auricle, digenic complementary ratio was obtained in normal cross. However, junctura proper and auricle, gave trigenic complementary ratio 27:37 in all the three treated crosses (1500, 2000 and 5000 rad X-ray), while leaf blade gave 27:37 in 2000 and 5000 rad treatments and ligule gave 27:37 in 5000 rad treatment. These results demonstrated the probable differential response of these characters in relation to the dosages of the X-ray treatments. This situation of differential transformation of genetical ratios for morphological characters under the influence of hybridization with irradiated pollen grains appears to be a new report.

(g) Interrelationships of genes governing 14 morphological characters were studied in 91 combinations both in control and treated crosses referred to earlier, in terms of independent assortment, linkage relations and pleiotropic association of characters.

The character combinations of Px-Pn, Px-an, Px-tst, Psh-an, Psh-

tst, Pl-an, Pl-tst, Plm-tst, Pa-an, an-tst, Plm-an, Pla-an, Pla-tst, Pjp-an, Pjp-tst, Plg-an, Pa-tst, Plg-tst, Pau-an, Pau-tst, Pn-an, Pn-tst, Pin-an, Pin-tst, Ps-an and Ps-tst showed **independent assortment** of genes controlling these characters both in control and treated crosses, in spite of the fact that except leaf axil, node, awning and tip-sterility all other characters showed transformation of genetical ratios in the treated crosses, as discussed elsewhere.

The present observation on independent assortment of recessive awning and tip-sterility to the above 12 anthocyanin pigmentation characters appears to be a new report.

Analysis of **linkage relationships** of eleven morphological characters studied presently showed that 9 out of 11 genes identified to have control over these characters were found to have been located already in the linkage group III by early workers. Hence the linkage associations Psh-Pl, Psh-Pjp, Psh-Plg, Psh-Pin, Psh-Ps, Psh-Pa, Pin-Ps, Pin-Pa, Psh-Pn, Psh-Pla, Plm-Pla, Plm-Pjp, Plm-Plg, Plm-Pau, Plm-Pn, Plm-Pin, Plm-Ps, Plm-Pa, Psh-Pau, Pla-Plg, Pla-Pin, Pla-Pau, Pla-Pa, Ps-Pa, Pla-Pn, Pla-Ps and Pla-Pjp, presently obtained could also be assigned to the same linkage group tentatively, as discussed elsewhere. However, transformation of interrelationships of genes occurred in the treated crosses due to X-ray irradiation of pollen grains of Japan violet indicated that no correspondence normally

existed between allelic transformation and transformation of genic interrelationships.

The present experimental results on the phenomenon of **transformation** exhibited two distinct strategies, The first one is that 26 character combination which showed independent assortment in the control remained stable with X-ray treatment. Similarly 9 pleiotropic associations observed in the control cross also did not show any alterations with X-ray treatments. The second strategy may be referred to the alterations that occurred in all the 27 linkage associations mostly into "pleiotropy" (probably tight linkage) and majority of the pleiotropic associations in the control were transformed into situations of independent assortment. This complex and intricate observation may lead to the conclusion that all pleiotropic conditions need not be real pleiotropy, but may be tight linkages leading to absence of recombinant classes during segregation in a normal population size. The results also indicate that breakage of linkage appears to the better accomplished with the irradiation of pollen grains prior to pollination rather than seed irradiation.

Out of 91 combinations for 14 morphological characters studied, 30 showed **pleiotropic associations** in the control cross. Of these 17 combinations involving 9 morphological characters showed pleiotropy for a common complementary gene in 5000 rad treatment

All these 17 combinations showed no change in their pleiotropic association from control to 5000 rad treatment, in spite of the fact that all the characters showed transformation of genetical ratios from the control, ie, either from 3:1 to 9:7 or from 9:7 to 27:37. It appears that induction of pleiotropic association by radiation would be possible due to tight linkage or congregation of genes or by reassociation of genes in close proximity resulting in apparent pleiotropic conditions. It is also possible to break the linkage system into independent assortment as observed in the case of linkage between Psh and Pn.

8. Study on mutagenesis of Japan violet also enabled analysis of certain **quantitative morphological characters** such as plant height, culm length, EBT, percentage of EBT, days to flower, panicle length, total spikelets/panicle, grains/panicle, percentage of sterility and panicle density. Observations on frequency distribution, range, mean, analysis of variance and variance index showed significant variation in M_1-M_3, and similar trends of bidirectional variation towards reduction and increase in plant height, culm length, EBT, days to flower, panicle length, grains/panicle, panicle density and percentage of sterility, thereby expressing transgressive segregation of polygenes due to negative and positive mutations. This might have occurred as a result of EMS treatment. On the other hand EBT% showed significant variation with negatively unidirectional trend towards reduction in EBT% in M_1, M_2 and M_3 relative to the respective

control, thereby indicating recessive polygenic mutation controlling EBT production. It is concluded that induction of polygenic mutation might produce negative or positive unidirectional variation and/or symmetrical or asymmetrical bidirectional variation in relation to the nature and response of the morphological character.

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APPENDIX

Appendix I. Description of mutant characters in comparison to the cross Cherumodan x Japan violet pollen irradiated with 2000/ 5000 rad X-rays

Parents/mutants		Parents		Mutants/ treatments/ generations			
Characters/		Cherumodan	Japan violet	Green awnless 5000 M_3F_3	Green awnned 5000 M_3F_3	Terminal twin 2000 M_3F_3	Clustered 2000 M_4F_4
1		2	3	4	5	6	7
1. Plant height (cm)	R	92-116	71-89	71-97	104-118	56-75	96-116
	M	102.72	82.20	90.76	111.26	60.76	108.32
2. Culm length (cm)	R	55-75	38.50-51.50	55-60	68.00-74.50	30-43	73-90
	M	64.80	44.82	53.46	68.98	38.08	81.66
3. Total tiller	R	6-12	7-16	8-18	6-15	15-25	8-16
	M	9.32	10.92	10.40	9.72	21.48	12.12
4. No of EBT	R	5-10	6-12	5-15	6-13	17-22	7-14
	M	7.84	9.32	8.28	7.64	19.08	9.80
5. % of EBT	R	60.00-100.00	62.50-100.00	70.00 100.00	71.43-81.82	73.91 100.00	70.00 90.99
	M	75.78	82.67	79.31	78.65	89.13	80.16

1		2	3	4	5	6	7
6. Panicle length	R	14.75-18.25	18-22	22.50-27.00	21.00-28.50	17.75-20.33	15.50-19.25
	M	17.21	20.71	24.34	24.79	19.33	17.71
7. Total spikelets	R	56-76	75.50-120.50	89.52-116.00	51.50-95.00	21.50-93.36	81-117
	M	61.72	105.02	102.60	67.96	93.36	100.28
8. No. of ster. spikelets	R	5.50-14.00	9.50-32.00	18.50-30.00	8.00-21.50	57.52-113.00	33.00-57.00
	M	8.52	21.16	22.45	16.02	63.58	49.32
9. % of sterility	R	7.04-19.20	8.44-35.56	15.08 29.32	15.08 28.85	38.00 92.50	31.13 58.74
	M	13.30	20.48	21.81	21.80	68.46	49.62
10. Panicle density	R	3.11-4.41	3.78-6.19	3.54-4.69	3.54-4.78	3.42-4.55	4.39-6.48
	M	3.62	5.08	4.17	4.17	4.82	5.67
11. 1000 grain wt.	R	22.13-23.65	24.97-26.85	30.51-32.18	27.11-30.81	14.56-15.34	13.14-14.04
	M	22.87	25.90	30.96	29.65	14.89	13.63

R = Range , M = Mean.

Appendix II. Instances with high chi-square values that yielded neither independent assortment, linkage nor pleiotropy

Sl.No	Character Combination / F_2 ratio /s			Cross /es	Population O/E	F_2 Frequency AB	Ab	aB	ab	Total	X^2
1	2			3	4	5	6	7	8	9	10
1.	Psh		Plm	Cherumodan X	O	99.00	12.00	0.00	33.00	144	
	(3:1)	Vs	(3:1)	Japan Violet	E on indep	81.00	27.00	27.00	9.00	144	42.81
				(Normal)	E on pleio	108.00	10.00	0.00	9.00	144	14.14
	Psh		Plm	Cherumodan X	O	112.00	14.00	2.00	53.00	181	
	(3:1)	Vs	(9:7)	Japan Violet	E on indep	76.36	59.39	25.45	19.80	181	128.60
				(1500 rad)	E on pleio	101.81	33.94	0.00	45.25	181	
2.	Psh		Pla	Cherumodan X	O	114.00	12.00	0.00	55.00	181	
	(3:1)	Vs	(9:7)	Japan Violet	E on indep	76.36	59.39	25.45	19.80	181	149.62
				(1500 rad)	E on pleio	101.81	33.94	0.00	45.25	181	19.52
	Psh		Pla	Cherumodan X	O	53.00	2.00	0.00	21.00	76	
	(3:1)	Vs	(3:1)	Japan Violet	E on indep	42.75	14.25	4.75	4.75	76	82.83
				(1500 rad)	E on pleio	57.00	0.00	19.00	19.00	76	
3.	Psh		Pau	Cherumodan X	O	86.00	40.00	3.00	52.00	181	
	(3:1)	Vs	(27:37)	Japan Violet	E on indep	57.27	78.48	19.09	26.16	181	73.37
				(2500 rad)	E on pleio	76.36	59.39	0.00	45.25	181	
4.	Psh		Ps	Cherumodan X	O	123.00	3.00	15.00	40.00	181	
	(3:1)	Vs	(3:1)	Japan Violet	E on indep	101.81	33.94	33.94	11.31	181	115.98
				(1500 rad)	E on pleio	135.75	0	0	45.25	181	
5.	Pl		Plm	Cherumodan X	O	93.00	0.00	21.00	67.00	181	
	(9:7)	Vs	(9:7)	Japan Violet	E on indep	57.27	44.54	44.54	34.65	181	105.66
				(1500 rad)	E on pleio	101.81	0.00	0.00	79.19	181	

1	2		3	4	5	6	7	8	9	10
6.	Pl	Pla	Cherumodan X	O	92.00	1.00	22.00	66.00	181	
	(9:7) Vs	(9:7)	Japan Violet	E on indep	75.94	59.06	5.06	3.94	181	99.68
			(1500 rad)	E on pleio	101.81	0.00	0.00	79.19	181	
7.	Pl	Pjp	Cherumodan X	O	70.00	12.00	5.00	57.00	144	
	(9:7) Vs	(9:7)	Japan Violet	E on indep	45.56	35.44	35.44	27.56	144	86.21
			(Normal)	E on pleio	40.50	31.50	40.50	31.50	144	85.32
8.	Pl	Plg	Cherumodan X	O	74.00	8.00	15.00	47.00	144	
	(9:7) Vs.	(9:7)	Japan Violet	E on indep	45.56	35.44	35.44	27.56	144	61.59
			(Normal)	E on pleio	60.75	20.25	20.25	42.75	144	10.75
			Cherumodan X	O	90.00	3.00	12.00	76.00	181	
	(9:7)	(9:7)	Japan Violet	E on indep	57.26	44.54	44.54	34.65	181	130.58
			(1500 rad)	E on pleio	101.81	0.00	0.00	79.19	181	38.58
9.	Pl	Pau	Cherumodan X	O	72.00	10.00	5.00	57.00	144	
	(9:7) Vs.	(9:7)	Japan Violet	E on indep	45.56	35.44	35.44	27.56	144	87.69
			(Normal)	E on pleio	60.75	20.25	20.25	42.75	144	23.51
			Cherumodan X	O	85.00	8.00	4.00	84.00	181	
	(9:7)	(27:37)	Japan Violet	E on indep	42.95	58.86	33.41	45.78	181	139.55
			(1500 rad)	E on pleio	76.36	25.45	0.00	42.75	144	
10.	Plm	Pla	Cherumodan X	O	49.00	0.00	4.00	23.00	76	
	(9:7) Vs.	(3:1)	Japan Violet	E on indep	24.05	18.70	18.70	14.55	76	61.05
			(2000 rad)	E on pleio	42.75	0.00	14.25	19.00	76	9.13
11.	Plm	Plg	Cherumodan X	O	97.00	17.00	5.00	62.00	181	
	(9:7) Vs.	(9:7)	Japan Violet	E on indep	57.26	44.54	44.54	34.66	181	101.28
			(1500 rad)	E on pleio	101.81	0.00	0.00	79.19	181	
			Cherumodan X	O	39.00	10.00	0.00	27.00	76	
	(9:7)	(9:7)	Japan Violet	E on indep	24.05	18.70	18.70	14.55	76	42.69
			(1500 rad)	E on pleio	32.06	10.69	10.69	22.56	76	16.68

1	2			3	4	5	6	7	8	9	10
12.	Plm		Pin	Cherumodan X	O	114.00	0.00	12.00	55.00	181	
	(9:7)	Vs.	(9:7)	Japan Violet	E on indep	76.36	25.45	59.39	19.80	181	144.40
				(1500 rad)	E on pleio	101.81	0.00	33.94	45.25	181	23.84
13.	Plm		Pa	Cherumodan X	O	114.00	0.00	13.00	54.00	181	
	(9:7)	Vs.	(3:1)	Japan Violet	E on indep	76.36	24.45	59.39	19.80	181	138.31
				(1500 rad)	E on pleio	101.81	0.00	33.94	45.25	181	17.74
14.	Pla		Plg	Cherumodan X	O	95.00	19.00	6.00	61.00	181	
	(9:7)	Vs.	(9:7)	Japan Violet	E on indep	57.27	44.54	44.54	34.64	181	89.81
				(1500 rad)	E on pleio	76.36	25.45	25.45	53.73	181	31.11
15.	Pla		Pau	Cherumodan X	O	86.00	28.00	3.00	64.00	181	
	(9:7)	Vs.	(27:37)	Japan Violet	E on indep	42.95	58.86	33.41	45.78	181	94.26
				(1500 rad)	E on pleio	76.36	25.45	0.00	79.19	181	
16.	Pla		Pin	Cherumodan X	O	114.00	0.00	12.00	55.00	181	
	(9:7)	Vs.	(3:1)	Japan Violet	E on indep	76.36	25.45	59.39	19.80	181	144.40
				(1500 rad)	E on pleio	101.81	0.00	33.94	45.25	181	17.74
17.	Pla		Pa	Cherumodan X	O	114.00	0.00	13.00	54.00	181	
	(9:7)	Vs.	(3:1)	Japan Violet	E on indep	76.36	25.45	59.39	19.80	181	144.40
				(1500 rad)	E on pleio	101.81	0.00	33.94	45.25	181	17.74
18.	Pjp		Pau	Cherumodan X	O	66.00	9.00	11.00	58.00	144	
	(27:37)	Vs.	(27:37)	Japan Violet	E on indep	45.56	35.44	35.44	27.56	144	79.37
				(Normal)	E on pleio	60.75	20.25	20.25	42.75	144	16.37
	Pjp		Pau	Cherumodan X	O	78.00	2.00	11.00	90.00	181	
	(27:37)	Vs.	(27:37)	Japan Violet	E on indep	32.21	44.15	44.15	60.49	181	139.01
				(1500 rad)	E on pleio	57.27	19.09	19.09	85.55	181	22.93

1	2			3	4	5	6	7	8	9	10
19.	Pjp		Plg	Cherumodan X	O	70.00	5.00	19.00	50.00	144	
	(9:7)	Vs.	(9:7)	Japan Violet	E on indep	45.56	35.44	35.44	27.56	144	65.14
				(Normal)	E on pleio	60.75	20.25	20.25	42.75	144	14.15
20.	Pjp		Pn	Cherumodan X	O	27.00	48.00	3.00	66.00	144	
	(9:7)	Vs.	(3:13)	Japan Violet	E on indep	15.19	65.81	11.81	51.19	144	22.89
				(Normal)	E on pleio	20.25	60.75	6.75	56.25	144	8.70
21.	Plg		Pau	Cherumodan X	O	69.00	20.00	8.00	47.00	144	
	(9:7)	Vs.	(9:7)	Japan Violet	E on indep	45.56	35.44	35.44	27.56	144	53.74
				(Normal)	E on pleio	60.75	20.25	20.25	42.75	144	8.96
	Plg		Pau	Cherumodan X	O	89.00	13.00	0.00	79.00	181	
	(9:7)	Vs.	(27:37)	Japan Violet	E on indep	42.95	58.86	33.41	45.78	181	142.12
				(1500 rad)	E on pleio	76.36	25.45	0.00	79.19	181	8.18
22.	Plg		Pin	Cherumodan X	O	99.00	3.00	27.00	52.00	181	
	(9:7)	Vs.	(3:1)	Japan Violet	E on indep	76.36	25.45	59.39	19.80	181	96.55
				(1500 rad)	E on pleio	101.81	0.00	33.94	45.25	181	
23.	Pau		Pn	Cherumodan X	O	22.00	67.00	3.00	89.00	181	
	(27:37)	Vs.	(3:13)	Japan Violet	E on indep	32.21	44.15	44.15	60.49	181	66.85
				(1500 rad)	E on pleio	19.09	57.27	18.85	89.79	181	15.43
24.	Pau		Pin	Cherumodan X	O	89.00	0.00	37.50	55.00	181	
	(27:37)	Vs.	(3:1)	Japan Violet	E on indep	57.27	19.09	78.48	26.16	181	60.69
				(1500 rad)	E on pleio	76.36	0.00	59.39	45.25	181	12.63
25.	Pau		Pa	Cherumodan X	O	88.00	1.00	39.00	53.00	181	
	(27:37)	Vs.	(3:1)	Japan Violet	E on indep	57.27	19.09	78.48	26.16	181	81.03
				(1500 rad)	E on pleio	76.36	0.00	59.39	45.25	181	
26.	Pin		Ps	Cherumodan X	O	124.00	2.00	14.00	41.00	181	
	(3:1)	Vs.	(3:1)	Japan Violet	E on indep	101.81	33.94	33.94	11.31	181	124.55
				(1500 rad)	E on pleio	135.75	0.00	0.00	45.25	181	
27.	Ps		Pa	Cherumodan X	O	124.00	2.00	14.00	2.00	181	
	(3:1)	Vs.	(3:1)	Japan Violet	E on indep	101.81	33.94	33.94	11.31	181	124.55
				(1500 rad)	E on pleio	135.75	0.00	0.00	45.25	181	