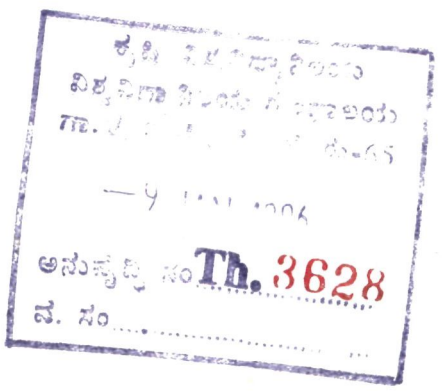
# CHROMOSOMAL LOCATION OF GENES THROUGH PRIMARY TRISOMICS OF *indica* RICE (*Oryza sativa* L.)

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AUGUST, 1992.


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# CHROMOSOMAL LOCATION OF GENES THROUGH PRIMARY TRISOMICS OF *indica* RICE (*Oryza sativa* L.)

**Thesis Submitted to the**
**University of Agricultural Sciences, Dharwad**
**in partial fulfilment of the requirements for the**
**Degree of**

## Doctor of Philosophy

**in**

**GENETICS AND PLANT BREEDING**

*By*

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**AUGUST, 1992.**

DIVISION OF GENETICS AND PLANT BREEDING
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## CERTIFICATE

This is to certify that the thesis entitled "CHROMOSOMAL LOCATION OF GENES THROUGH PRIMARY TRISOMICS OF *indica* RICE (*Oryza sativa* L.)" submitted by Mr. ATUL MAHENDRABHAI MEHTA, for the degree of DOCTOR OF PHILOSOPHY IN GENETICS AND PLANT BREEDING, of the University of Agricultural Sciences, Dharwad, is a record of research work done by him during the period of his study in this University, under my guidance and supervision and the thesis has not previously formed the basis of the award of any degree, diploma, associateship, fellowship and other similar titles.

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At the Lotus Feet
of my Master
Bhagavan
SRI RAMANA MAHARSHI

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## CHAPTER - I

# INTRODUCTION

# I. INTRODUCTION

Needless to say, rice is the most important staple food for mankind. No other crop plant is more adaptive to diversified environments than rice. Among crop plants, rice has genetic diversity unparalleled by others.

In order to upgrade the genotypes without upsetting their original desirable qualities, a complete and systematic knowledge of the genetic system of any crop plant is the foremost fundamental necessity and pre-requisite. The knowledge of the genetic system of rice plant so far achived is not commensurate with its pre-eminent position as a food crop.

Genetic studies leading to linkage information, are important from theoretical and applied view points. Cross over values indicate the distance between the loci and would help the breeders to devise breeding experiments in such a way that they raise adequate populations in the segregating generations to isolate the desired recombinants. Close linkages between the genes controlling undesirable characters like susceptibility to diseases with those controlling economic characters, if found, may have to be broken by resorting to induced mutagenesis. Yields can be boosted by eliminating the undesirable characters and by bringing together the desirable genes. This sort of genetic manipulation can be achieved efficiently only when the genetic architecture of the plant is thoroughly understood.

In rice, linkage studies have been carried out by many workers. Over 500 genes have been found to control different morphological and pigmentation characters in rice by 1987. Of these, about 206 genes have been allotted to the 12 linkage groups and 142 marker genes have been positioned on chromosomes in japonica rice (Kinoshita and Takahashi, 1991). In indica rice 133 genes (Goud and Nadaf, 1990a) have been actually allotted to different linkage groups owing to the availability of linkage relationships among them.

Jodon (1948) summarised the information on linked genes available up to that time and identified eight linkage groups in Oryza sativa L. Nagao and Takahashi (1963) updated the information and recognized 12 linkage groups corresponding with the haploid number of chromosomes of Oryza sativa L. The cross-over values and linkage realized by the Japanese workers in japonica rices, however, could not be confirmed by the Indian workers in indica rices (Sharma, 1986). Further the sterility observed in the japonica x indica hybrids made it difficult to check and compare the data on linkage. This led Misro et al. (1966), Misro (1981) and Goud and Nadaf (1985) to propose different linkage maps for indica rices. Misro et al. (1966) even suggested that separate linkage groups should be established for indica, japonica and javanica rices. Khush et al. (1984), however, felt that the linkage maps for the two ecotypes cannot be so much different. They

have found complete correspondence between the linkage groups of *japonica* and *indica* rices. The differences could be resolved by establishing the identity of chromosomes, developing translocation stocks and primary trisomics in *japonica* as well as *indica* rices and locating the genes on the chromosomes with the help of these stocks (Sharma, 1986).

Kinoshita and Takahashi (1991) have presented the current linkage maps (Anonymous, 1990a) with the up-to-date information on the recombination frequencies which include the unlocated genes belonging to each group. They admitted that, it is difficult to consolidate the Indian linkage groups in *indica* rice published by Misro (1981) in the maps because of the differences in the genetic scheme for anthocyanin coloration and scarcity of identical genes involved in the two series of linkage groups at that time. Thus it has been difficult to establish 12 groups common to *indica* and *japonica* types.

Kinoshita (1984, 1986a, 1986b) has reviewed the discrepancy of the colour producing genes postulated in *japonica* and *indica* rices stressing the need for further efforts for the identification of genic system used in pigmentation characters.

The variability in *indica* rices is very high compared with that in the *japonica* rices. The anthocyanin pigmentation in various plant parts is one of the most fascinating and complicated group of characters, the genes

governing which, nevertheless, form the bulk of the genes analysed, constituting different linkage groups.

In Japanese rice, Nagao and Takahashi carried out extensive genetic analysis on anthocyanin colouration and established a scheme in which gene C produces chromogen and gene A activates C and turns the chromogen into anthocyanin (Nagao, 1951; Takahashi, 1957). Later the third gene P, which controls the spreading of chromogen over the entire apiculus, was found necessary for the basic pigmentation (Nagao and Takahashi, 1956). In these three loci, there was a multiple differentiation of alleles to account for the intensity of pigmentation and colour hues, depending on the various combinations of the alleles of the three genes. The colouration of parts other than the apiculus can be partly explained by a pleiotropic action of the basic genes. However, majority of colour patterns is the end product of the manifold gene effect of distribution of localizing genes into respective parts in co-existence with the basic colour-producing genes.

The C-A-P gene system for anthocyanin pigmentation in japonica rice is also applicable to indica except for the nature and number of localized genes (Setty and Misro, 1973; Pavithran, 1986).

By studying the inheritance patterns of pigmentation characters a wealth of data has been accumulated. Of

the total 133 genes assigned to different linkage groups of *indica* rice, 87 genes are associated with pigmentation alone in different parts of the rice plant. Eighty-seven pigmentation genes are distributed in first ten linkage groups with three genes in I group, 26 in II, 23 in III, 16 in IV, 3 in V, 1 in VI, 3 in VII, 1 in VIII, 3 in IX and 8 in X group (Goud and Nadaf, 1990a). This clearly indicates that pigmentation genes are mostly concentrated in II, III, IV and X groups in order of their magnitude. In view of such distribution of genes responsible for the same character in different linkage groups, identifying gene chromosome relationship appears to be a very difficult task. A detailed cytogenetic analysis using trisomics and translocation stocks would certainly facilitate the task.

The trisomic series in primary, secondary or tertiary forms are being produced in an increasing number of crop plants to locate genes on specific chromosomes, to identify chromosomes with their respective linkage groups and to determine phenotypic effects of individual chromosomes.

The trisomic method of identifying individual chromosomes with their respective genes as a result of a modification of the genetic ratios by the presence of extra chromosomes has been successfully applied to a number of crop plants and fruitful results have been obtained in the following cases.

i) where a complete set of trisomics corresponding to the haploid number of a species are available,

ii) where the trisomics are morphologically distinguishable from each other and

iii) where the extra chromosomes are easily identifiable either at meiosis or mitosis.

In case of rice, a complete set of primary trisomics has been developed in Taiwanese and Japanese rices (Hu, 1968; Iwata et al., 1970; Watanabe et al., 1969). This has enabled the Japanese rice workers to assign genes to specific chromosomes in japonica rice (Watanabe and Koga, 1975; Iwata and Omura, 1975, 1976).

Since the two sub-species, japonica and indica are known to have non-identical linkage groups (Nagao and Takahashi, 1963; Misro et al., 1966), a complete set of primary trisomics in indica rice has also been developed for use in genetic studies. Development of 12 primary trisomics in two different varieties of rice independently at IRRI, Manila, Philippines and CRRI, Cuttack, India (Khush and Misra, 1983) stimulated research on the use of trisomics in genetic studies in indica rice.

The trisomic analysis is well suited for assigning genes for qualitative characters like those governing plant pigmentation. However, only a single report (Siddiq et al.,

1986); dealing with trisomic analysis for pigmentation characters is available.

In view of the above considerations and with realization of basic and applied significance of trisomic analysis in relation to crop improvement, the endeavour of the present investigation is -

i) to study the inheritance pattern of the anthocyanin pigmentation and other characters,

ii) to identify and locate these genes to specific chromosomes utilizing the primary trisomics of *indica* rice so as to establish gene-chromosome relationship in rice, and

iii) to study the transmission rate of the extra chromosome of each trisomic through hybridization and selfing.

# CHAPTER - II

# REVIEW OF LITERATURE

# II. REVIEW OF LITERATURE

As early as 1898, artificial hybridization of rice under controlled conditions was successfully undertaken by Takahashi who compared the characteristics of the hybrid with those of its parents (Morinaga, 1957). The first recorded instance of Mendelian segregation in rice was by Van Der Stok (1908) in Indonesia. In India, Parnell et al. (1917, 1922) were first to publish instances of linkage and cross-over mainly with anthocyanin colour. Anthocyanin colouration in different plant parts is a striking feature and the genetical behaviour of this character has received considerable attention since the initiation of genetical research on rice in India.

The present investigation deals with the inheritance pattern of 29 different anthocyanin pigmentation characters and one morphological character as well as establishment of gene-chromosome relationships by using a complete primary trisomic series of indica rice. The literature pertaining to the present investigation is reviewed and presented in two headings:

1. Inheritance studies and
2. Primary trisomic analysis.

## 2.1 Inheritance studies

### 2.1.1 Anthocyanin pigmentation characters

Pigmentation of the various parts of the rice plant

involves a most fascinating and complicated system of gene interaction. Anthocyanin pigmentation varies in intensity and distribution. It is found in all the vegetative and several floral parts but not in the embryo or endosperm (Chang, 1964). The inheritance studies of anthocyanin pigmentation in rice plant organs conducted in India have been summarized by Ramiah and Rao (1953) and Ghose et al. (1960). Anthocyanin colour characters in japonica rice have also been studied extensively (Nagai, 1958; Nagao and Takahashi, 1956).

The inheritance pattern of the anthocyanin pigmentation characters is observed to be simple in some genotypes for certain characters, while in others, it is reported to be complex with an involvement of interacting genes of varying actions (Ghose et al., 1960; Ramiah and Rao, 1953; Khush, 1974). Various genetic ratios in $F_2$ have been reported in the past for several characters in different crosses which indicate the involvement of not a single locus but several loci of interacting genes viz., complementary, duplicate, complementary duplicate, inhibitory complementary, inhibitory duplicate, anti-inhibitory complementary, anti-inhibitory duplicate etc. As a result, different $F_2$ ratios for the same characters in different cross combination have been reported as two or more genes are involved in the expression of characters. The genetic explanation for a particular ratio would be the same regardless of the character for which

it is reported. The genetic explanation for different $F_2$ ratios (mono-di-tri-tetra and pentagenic) is presented in Appendix I.

Although there appears to be no advantage in possessing pigmentation in both cultivated and wild varieties (Ramiah and Rao, 1953), genetic association observed between pigmentation and other important characters like spikelet sterility, vigour and yield (Ramiah, 1945), tillering and panicle length (Dhulappanavar, 1962), resistance to gall-midge (Sastry and Prakash Rao, 1973) resistance to brown plant hopper (Krishna, 1977; Prasad et al., 1987) and quality of rice (Das and Ahmed, 1982) indicates its possible utility in selection of the appropriate genotypes. Further, wide compatibility genes of indica and japonica have also been located between the chromogenic gene "C" for apiculus colour and "Wx" for waxy endosperm (Araki et al., 1986; Ikehashi and Araki, 1986; and Kumar and Virmani, 1989).

Varieties were classified based on the pattern of pigmentation of various plant parts (Takezaki, 1921 and 1923; Hector, 1922; Misro et al., 1960 and Dhulappanavar and Menshinkai, 1965). The classification based on pigment was found to be more scientific than by the mere morphological characters. In most of the inheritance studies only the presence or absence of pigment was considered. However, a very few have attempted to study the intensity of pigment also.

2.1.1.1 Chemistry of anthocyanins

Colour production in rice plant was shown to be due to three kinds of pigments viz., 1) anthocyanins, 2) reddish brown and brown pigment and 3) chlorophylls and related pigments. Anthocyanin pigment is clearly visible when the crop is at fully grown stage and it fades as the maturity advances. Non-anthocyanin pigments like reddish brown and brown pigments start appearing only in the latter stages of maturity (Nagai, 1958).

Chromogenic substances namely, flavones and flavanols give rise to anthocyanins on reduction while the oxidation of the same result in brown pigments (Nagai, 1958). Anthocyanins constitute four kinds: viz., Cyanin (Cyanidin 3, 5 glucoside), chrysanthemin (cyanidin 3-glucoside), keracyanin (cyanidin-3-rhamno-glucoside) and ubiginosin (malvidin-3-galactoside). These are distributed widely in both cultivated and wild species (Nagai et al., 1960 and 1962 and Mizushima et al., 1963). The first three pigments are more frequent. The specific action of various genes and their alleles in the production of the above pigments was discussed by Mizushima et al. (1963).

2.1.1.2 Histological position of anthocyanins

The pigment in the cell sap is referred to as purple, but its actual appearance to the naked eye varies

from various grades of purple through scarlet red, rosy red, bluish red, blue, dark blue to black. Various factors like 1) the position of the pigment in the plant anatomy, 2) intensity of the pigment and 3) depth of the chlorophyll in which it is present, will influence the perception of the pigment. For instance, stigma appears as reddish under low concentration of pigment while the same looks black when the pigment is highly concentrated.

In some varieties, the colour in different parts may look alike, but on close examination, it may be found to be situated in different tissues of plant histologically. Thus, there are varieties having pigment only in parenchymatous cells, others only in vascular bundles and a few in both the tissues for sheath colour (Ramiah and Rao, 1953). Purple colour in the leaf blade is due to dense distribution of pigment in the epidermal cells, motor (bulliform) cells and sclerenchymatous cells located outside the phloem. The presence of pigment in single layer of parenchymatous cells adjacent to vascular bundles gives purple lines to the midrib. The presence of pigments in the cell sap of motor and epidermal cells produce the purple lines on leaf tip and margin. The pigment in internode is found to be located in parenchymatous cells.

2.1.1.3 Inheritance of apiculus pigmentation

The extending tip of the lemma or palea is

referred to as apiculus. The two apiculi may be distinguished as lemmal apiculus and paleal apiculus (Chang and Bardenas, 1965). The colour of the apiculus is useful in analysing the inheritance of pigmentation, not only in the apiculus itself but also in other vegetative organs. Thus, the genes for apiculus colour are also basic for anthocyanin formation in other organs (Nagao, 1951). The main evidence for this hypothesis was that, when only one part was pigmented, that part invariably happened to be apiculus and the pigmentation in other plant parts was rather rare, without the pigmentation in apiculus. However, several exceptions have been noted by Jones (1929), Ramiah (1945), Misro et al. (1960) and Dhulappanavar and Mensinkai (1965).

The production of anthocyanin pigment in apiculus or in any other part, requires the presence of two basic complementary genes C and Sp (C and A). C produces a chromogen base, while the Sp (A) gene produces an enzyme which reduces the chromogen into anthocyanin (Parnell et al., 1917; Yamaguchi, 1932; Nakayama, 1935; Nagao, 1951; Takahashi, 1957). Apart from these two genes, a third gene P which localises the pigment in the entire apiculus part is also found to be basic for pigmentation in other parts of the plant (Nagao and Takahashi, 1956). The C-A-P complementary genic system suggested first for the purple apiculus in japonica rice was subsequently established in indica rice as well (Kondo, 1963a; Ghose et al., 1963; Setty and Misro, 1973;

Hadagal et al., 1980 and Pavithran, 1986).

Setty and Misro (1973) made a systematic study of the genetics of pigmentation in *indica* rice, by studying six crosses involving two common parents. They obtained ratios of 15:1 and 54:10 for apiculus pigmentation. Accordingly, they hypothesized the involvement of three more complementary duplicate genes $P_a$, $P_b$ and $P_c$, in addition to the two basic genes C and A. Thus, a modified C - A - P complementary genic system was proposed by them. In accordance with their postulation, the complementary genic scheme for *indica* group of rice is C - A - $P_a$, $P_b$ or C - A - $P_a$, $P_c$ or C - A - $P_b$, $P_c$. This scheme was claimed to explain almost all the ratios (3:1, 15:1, 9:7, 45:19, 54:10, 162:94 etc.) reported for the presence or absence of anthocyanin pigment in the apiculus of intra and inter-racial crosses involving *indica* and *japonica* rices. Thus they also proved the applicability of C - A - P genic system for *indica* rices.

According to Nagao and Takahashi (1956) and Kinoshita (1984) the intensity of pigmentation is due to the multiple differentiation of alleles at these three loci. The rank of dominance of alleles in these loci is schematically represented in the Figure 1.

2.1.1.4 Localization genes

The occurrence of genetic ratios like 15:1, 27:37, 81:175, 162:94 etc., for the presence or absence of pigment

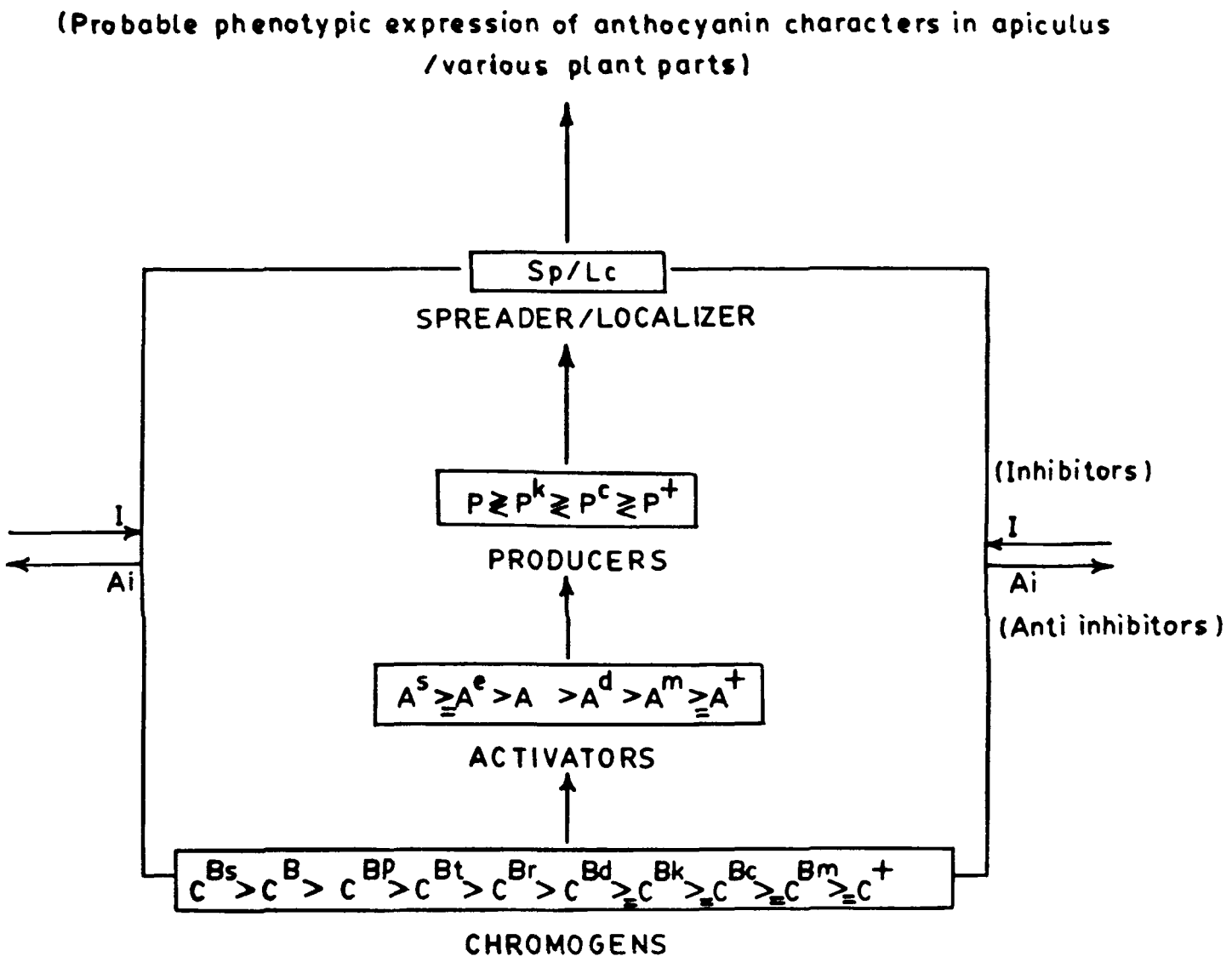



Figure: 1-Schematic representation of the genetic system of anthocyanin colouration in rice.

in particular plant organs, indicates that besides the basic genes C - A - P, there are other genes involved in the production of anthocyanin pigmentation. These are called localization genes whose main function is to localize the pigment in the respective parts of the plant (Nagao and Takahashi, 1956). Besides these genes, there are intensifying, diluting, inhibitory and anti-inhibitory genes also (Ramiah and Rao, 1953).

Several localization genes differing in their action have been recognized. In many cases more than one localization genes have been reported for one and the same plant part. In such cases, for complementary genes, letter subscripts and for duplicate and polymeric genes, numerical subscripts are used along with the gene symbols. An internationally acceptable nomenclature of genes in rice was evolved (Anonymous, 1959 and 1963 and Chang and Jodon, 1963), which has been recently revised (Anonymous, 1986). Some of the localization genes concerned with the present investigation are given in Table 1 wherein only one gene is represented against each part. The standardized description of plant parts in rice was first presented by Hutchinson et al. (1938) and subsequently by Chang and Bardenas (1965). The diagrams indicating the position of various plant parts is shown in Fig.2A, 2B, 2C, 2D and 2E.

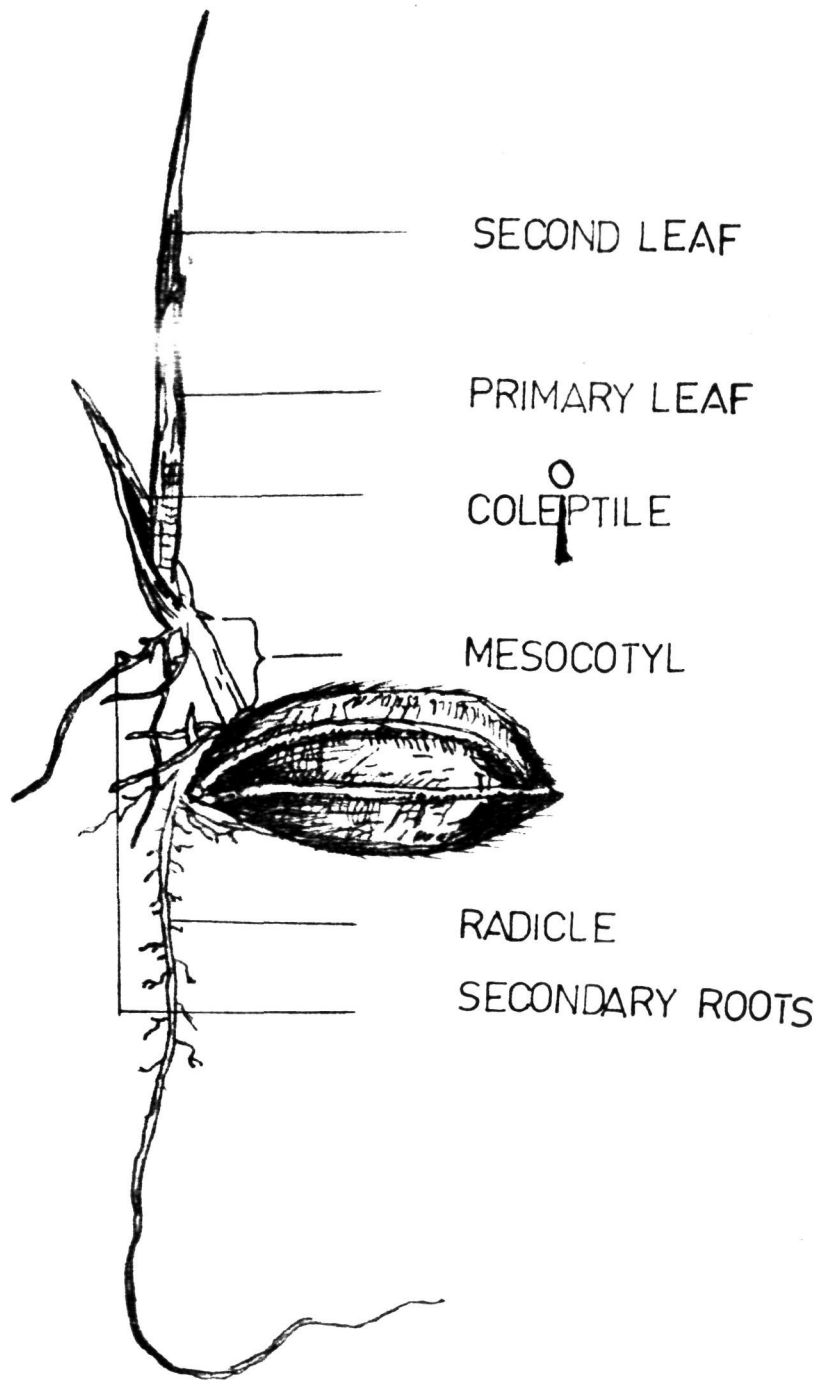
Localization gene(s) reported for pigmentation in one part is also found to be responsible for localizing pigment in some other parts, thus indicating the presence of pleiotropy.

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Table 1. Localization genes for pigmentation in different parts of rice plant

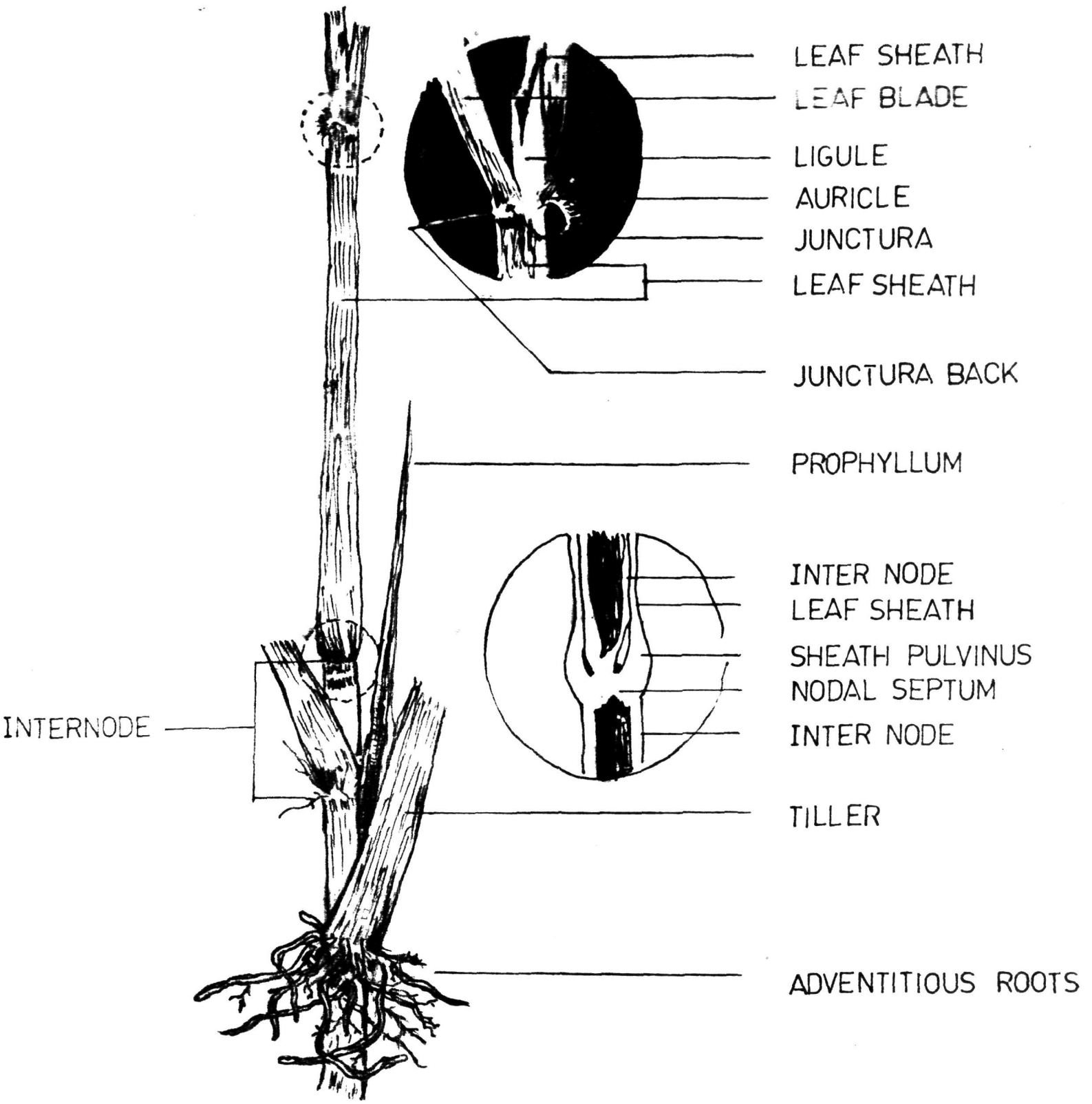
| Sl. no. | Plant part | Gene symbol | Sl. No. | Plant part | Gene symbol |
|---|---|---|---|---|---|
| 1 | Culm base | Pcb | 13 | Node | Pn |
| 2 | Leaf sheath and leaf sheath (external) | Psh | 14 | Sheath pulvinus | Pu |
| 3 | Leaf sheath (internal) | Psh(Int) | 15 | Pulvinus ring | Pur |
| 4 | Prophyllum | Ppr | 16 | Glumes (empty) | Pg |
| 5 a(i) | Leaf blade (seedling stage) | Pl(S) | 17 | Lemma/palea | Pr |
| (ii) | Leaf tip (seedling stage) | Pla(S) | 18 | Apiculus | Pa |
| (iii) | Leaf margin (Seedling stage) | Plm(S) | 19 | Stigma | Ps |
| 5 b(i) | Leaf blade (flowering stage) | Pl | 20 | i) Panicle base | Ppb |
| (ii) | Leaf tip (flowering stage) | Pla | | ii) Panicle pulvinus | Ppu |
| (iii) | Leaf margin (flowering stage) | Plm | | iii) Panicle axis | Ppa |
| 6 | Leaf midrib | Pmr | | | |
| 7 | Leaf axil | Px | 21 | Nodal septum | Pm |
| 8 | Ligule | Plg | 22 | Nodal ring | Pnr |
| 9 | Auricle | Pau | | | |
| 10 | Juncture | Pj | 23 | Awn colour (Red awn) | Ran |
| 11 | Juncture back | Pjb | 24 | Awning(presence/ absence of awns) | An |
| 12 | Internode | Pin | | | |



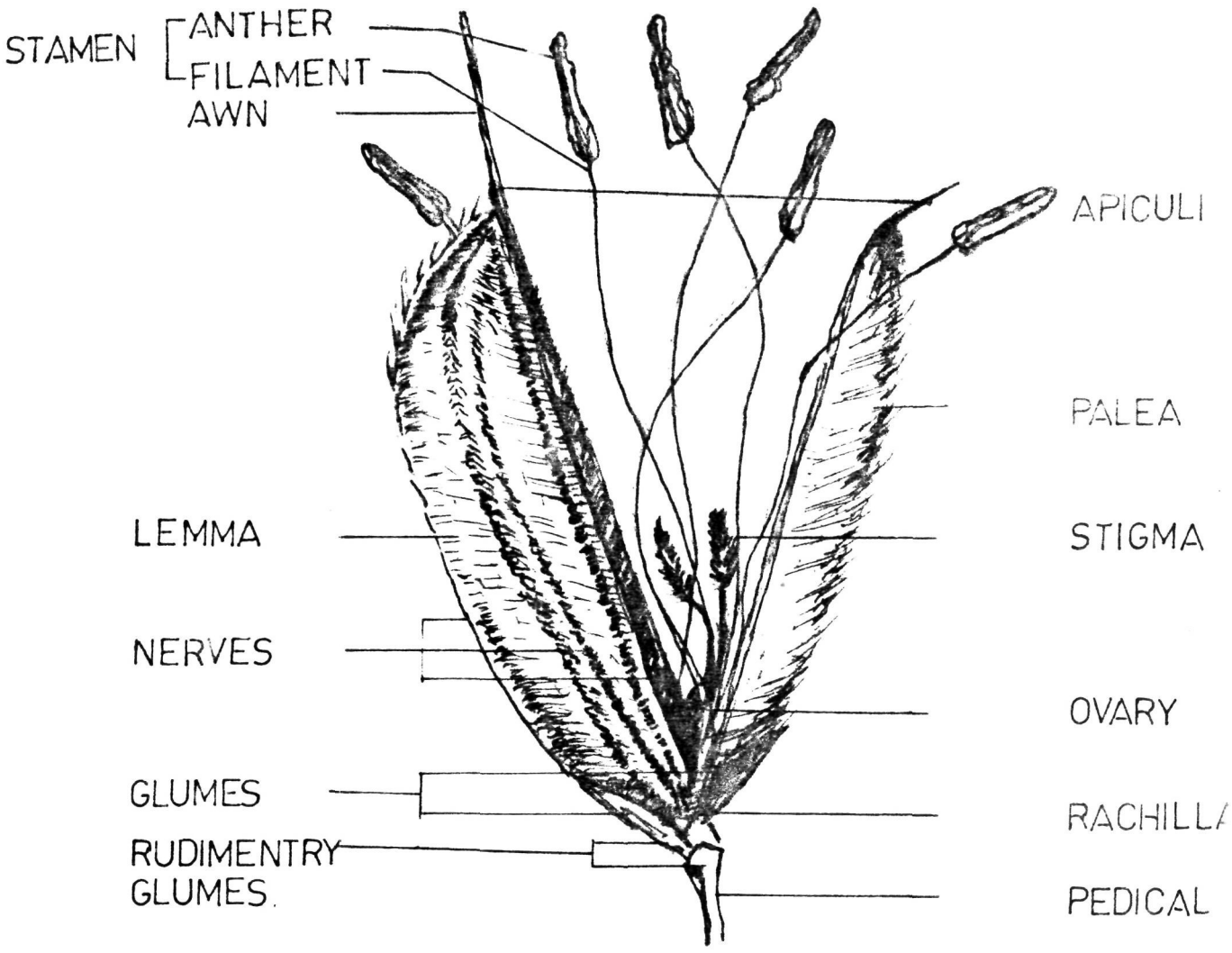

A PARTS OF A YOUNG SEEDLING

# DIFFERENT PARTS OF A RICE PLANT

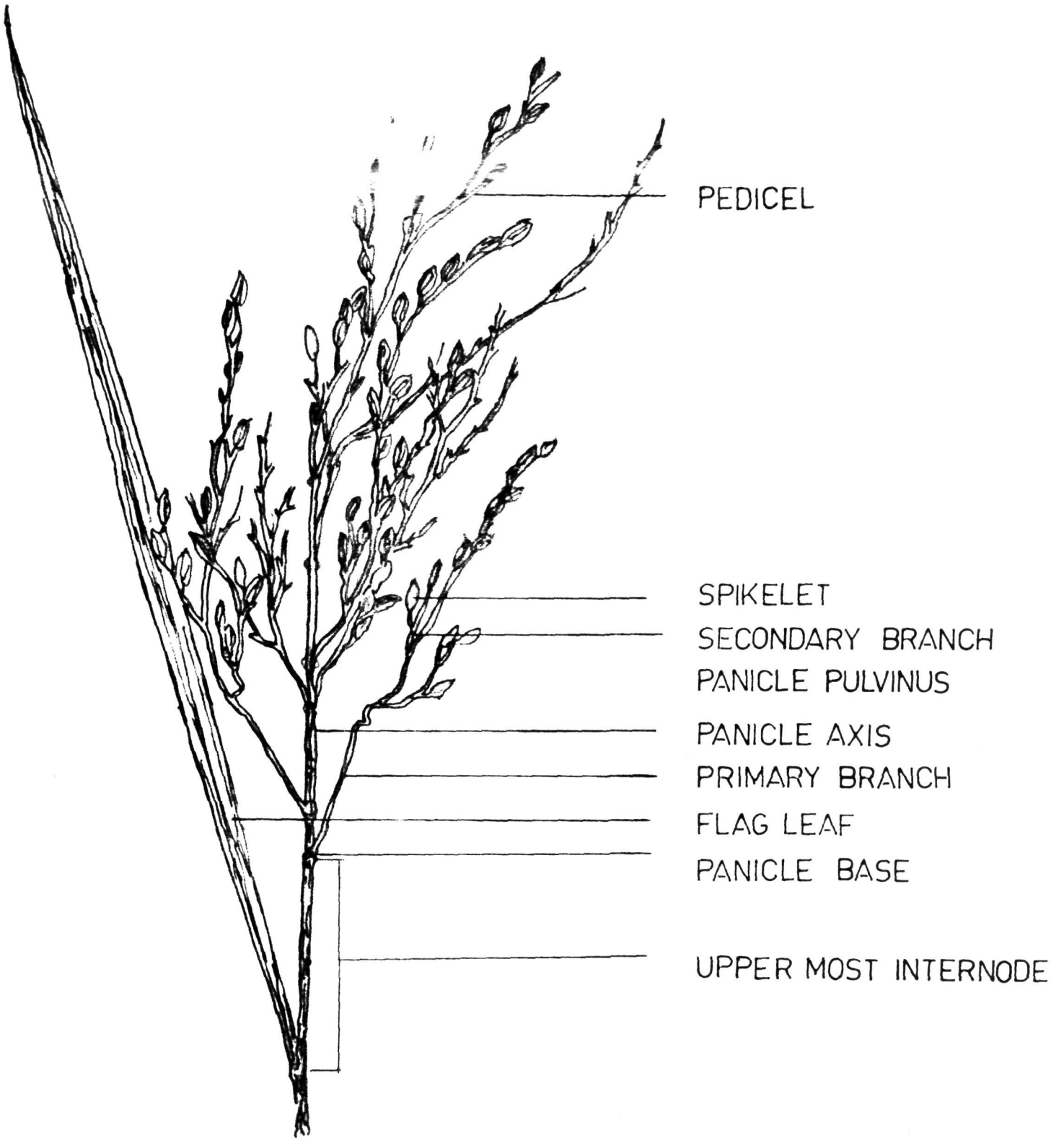
FIG 2.



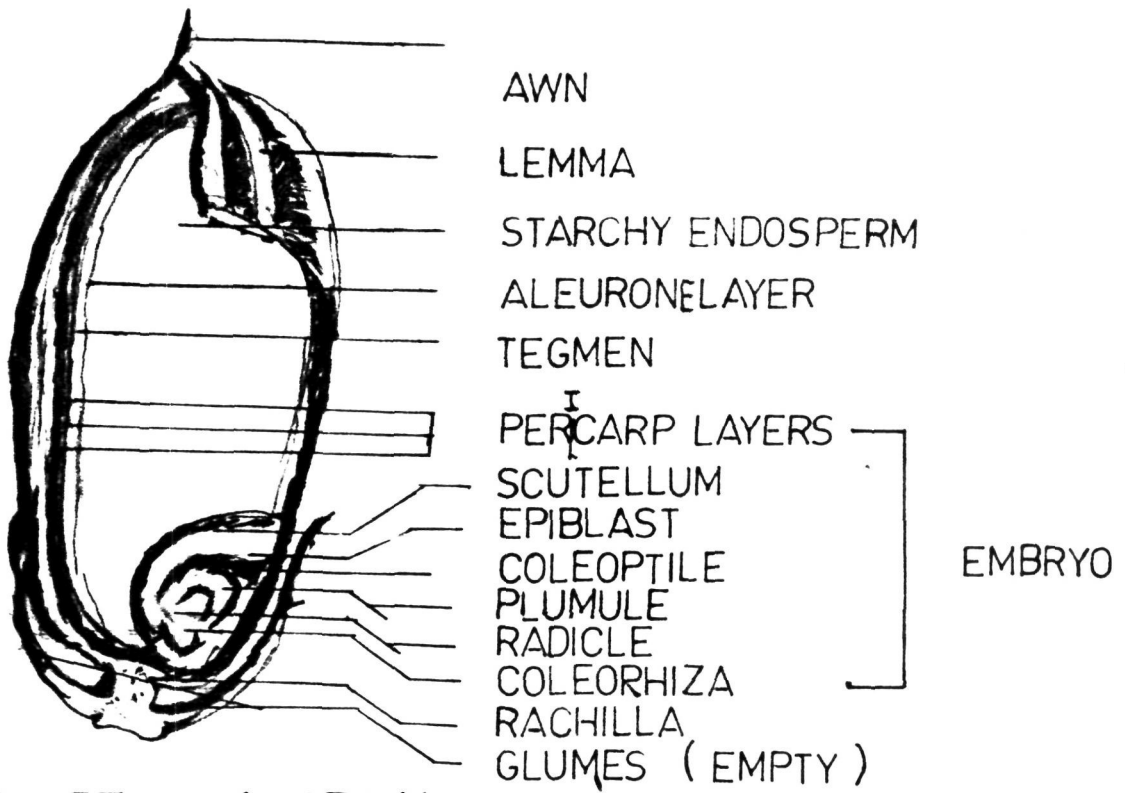

B PARTS OF A PRIMARY TILLER




C PARTS OF A SPIKELET




D COMPONENT PARTS OF A PANICLE




E STRUCTURE OF A GRAIN

For instance, the gene Pl for purple leaf blade also affects pigmentation in leaf sheath, pulvinus, auricle, ligule etc. (Nagao, 1951). Pleiotropic localization genes have been reported to act either similarly or differentially for localising pigment in different parts (Setty and Misro, 1973; Dhulappanavar, 1973a; Goud and Kullaiswamy, 1984; Shyla, 1985; Goud and Madaf, 1990b and Sukeshkumar, 1990).

Multiple alleles, have also been reported for the loci of localization genes such as Pl and Pla (Nagao et al., 1962, 1968 and Kondo, 1963b). But multiple allelism has not been clearly established in case of gene Pla.

### 2.1.1.5 Inhibitory and anti-inhibitory genes

Inhibitory and anti-inhibitory genes for some of the pigmentation characters have been reported in rice. Through the interaction of the above genes, various colour expressions and intensities are possible (Chang, 1964). The inhibitory genes may be one or more, when present in dominant condition inhibit the expression of genes conditioning the characters. The inhibitory genes are designated as 'I' followed by a notation of gene for particular part. For example, inhibitory gene for juncture pigmentation is designated as I-Pj. Dhulappanavar et al. (1975a) reported a ratio of 1 purple : 15 white for leaf tip pigmentation indicating the action of two inhibitory duplicate genes.

Anti-inhibitory genes are those which when present in dominant condition inhibit the expression of inhibitory genes. The anti-inhibitory genes are designated as "Ai" followed with a notation of gene for particular part, for example, anti-inhibitory gene for juncture pigmentation is designated as Ai-Pj. Dhulappanavar (1979) obtained a ratio of 117 purple : 139 white for pigmentation in ligule. This indicated that pigmentation in this part was governed by two complementary, one inhibitory and one anti-inhibitory genes.

2.1.1.6 Inheritance pattern of anthocyanin pigmentation in different plant parts

2.1.1.6.1 Culm base

This refers to the base of lower most internode, which some times retain the shades of purple in seedling stage only.

$F_2$ segregation ratios indicating a single dominant gene has been reported by -

| | | |
|---|---|---|
| Majumdar | (1985) | 3 : 1 |
| Nadaf | (1989) | 3 : 1 |

2.1.1.6.2 Leaf sheath (Syn.sheath, vagina)

This is the lowermost part of the leaf, originating from a node and enclosing the internode above it, and some times the leaf sheaths and blades of the succeeding internodes.

2.1.1.6.2.1 Leaf sheath (External)

This refers to the external portion of the leaf sheath. In rice, purple leaf sheath has been reported to be dominant over green. However, complementary factors are involved for pigmentation and its expression is suppressed often by inhibitory factors. Different genetic ratios have been reported for inheritance of leaf sheath pigmentation by different workers as under.

| Authority | Genetic ratios |
|---|---|
| Hector (1916) | 9:7 |
| Parnell et al.(1917), Roy(1921) | 3:1 |
| Hector (1922) | 3:1, 9:7, 15:1 and 27:37 |
| Mitra et al. (1928) | 3:1 and 9:7 |
| Kadam (1936) | 9:3:4 |
| Houraye and deLA (1942) | 3:1 |
| Dave (1948) | 9:7 and 9:3:4 |
| Kuang (1951) and other | 3:1, 9:7, 15:1, 3:13,63:1 |
| Chinese authors | and 81:175 |
| Ramiah and Rao (1953) | 3:1 |
| Choudhari and Sen (1953) | 9:7 |
| Khan (1953) | 3:1 |
| Nair (1958) | 9:7 |
| Aziz and Shafi (1958) | 3:1 |
| Shafi and Khan (1958) | 3:1 |

| | |
|---|---|
| Shafi and Aziz (1959) | 3:1 |
| Butany et al. (1959) | 3:1 |
| Richharia et al. (1960) | 54:10 |
| Unpublished records of CRRI Cuttack quoted by Ghose et al. (1960) | 3:1, 9:7, 27:37, 45:19, 81:175 and 162:94 |
| Kadam and D'Cruz (1960) | 27:37 |
| Ramirez et al. (1960) | 9:7, 27:37 and 81:175 |
| Uzir (1961) | 3:1 |
| Basu and Roy Choudhury (1961) | 3:1 |
| Panda (1962) | 162:94 |
| Dhulappanavar (1962) | 3:1 |
| Sastry and Patnaik (1962) | 3:13, 9:55 |
| Julka (1967) | 12:3:1 |
| Srivastava et al. (1968) | 3:1 |
| Saran and Srivastava (1969) | 162:94 |
| Dhulappanavar and Mensinkai(1970) | 54:10 |
| Ratho and Rao (1972) | 9:7 |
| Dhulappanavar (1973a, 1975) | 9:7 |
| Manjunath (1973) | 54:10 |
| Setty and Misro (1973) and Setty et al. (1973) | 162:94 and 45:19 |
| Satyanarayanaiah and Reddy(1973) | 3:1 |
| Dhulappanavar and Hiremath (1974a, 1974b) | 54:10 |
| Dhulappanavar et al.(1975b and 1975c) | 3:253 |

| | |
|---|---|
| Thimmappaiah (1975) | 255:1 |
| Pavithran (1977) | 9:7 |
| Tripathy and Rao (1979) | 27:37 |
| Kolhe and Bhat (1979, 1982) | 3:1 |
| Hadagal (1980) and Hadagal et al., (1980) | 45:19 |
| Dhulappanavar (1981) | 63:193 |
| Ramesh (1984) | 27:37 |
| Shyla (1985) | 45:19 |
| Rao and Misro (1986) | 9:7 |
| Eruotor (1983, 1986) | 3:1 |
| Prasad et al. (1987) | 3:1, 9:7, 27:37 and 54:10 |
| Shyla and Pavithran (1989) | 54:10 |
| Hadaf (1989) | 3:1 |
| Ahmed and Dar (1990) | 3:1 |
| Sukesh Kumar (1990) | 9:7 |
| Singh et al. (1990) | 162:94 |
| Kashikar and Kulkarni (1990) | 3:1 |

2.1.1.6.2.2 Leaf sheath (internal)

This refers to the pigmentation of the internal side of the leaf sheath that falls above the area of leaf axil. The pigmentation in the leaf sheath (internal) has been reported to differ from the pigmentation of leaf sheath (external) only recently (Kolhe and Bhat, 1979 and 1982). Setty and Misro (1973) have named it as leaf axil extension. Genetic ratios involving one or three genes have been reported by

| | |
|---|---|
| Setty and Misro (1973) | 54:10, 3:1 |
| Kolhe and Bhat (1979, 1982) | 3:1 |
| Nadaf (1989) | 3:1 |

2.1.1.6.3 Prophyllum (Syn. Coleoptyloid)

This is a small, 2-keeled bract enclosed by the leaf sheath with the back against the parent culm and its margins clasping the young tiller. At the maximum tillering stage it is quite distinct being colourless (transparent) to light purple in its pigmentation. Prophyllum as a different part of the plant has received the attention of rice geneticists for studying inheritance of its pigmentation only recently in which it was found to be monogenic dominant (Nadaf, 1989).

2.1.1.6.4 Leaf blade (Syn. lamina), leaf tip and leaf margin

Leaf blade is the linear-lanceolate, flat, sessile and free portion of the leaf and is continuous with the leaf sheath. In leaf blade, pigment may be present on lamina, leaf tip (apex), margins and midrib. The colour of the leaf blade is either full purple or purple wash or normal green. In case of purple wash, the pigmentation slightly spreads down from tip and towards inner side from margins. When the leaf blade is fully purple, the plant is generally called 'purple plant'. Purple leaf character in rice has a special significance in wild rice (Karaga) eradication. Pigment on

leaf tip and margin appears as irregular stripes or as continuous stripes. The pigmentation in the midrib is clearly visible more on the underside of the leaf blade. Inheritance of pigmentation in leaf and the histological distribution of pigment has been thoroughly described and discussed by Nagao and Takahashi (1947). Many workers consider leaf tip and margin as separate characters, although in the past they were taken as single character.

2.1.1.6.4.1 Leaf blade, tip and margin at seedling stage

That the pigmentation due to anthocyanins has been established to be clearly visible during or shortly after anthesis, which then fades as the maturity advances (Nagai, 1958) has not brought the attention of rice geneticists to study the inheritance of pigmentation of these leaf characters during seedling stage. Although foliage colour is reported to be distinguished in the seedling stage (Chang and Bardenas, 1965), there are only a few reports leading to understanding of genetics of these characters. Hsv and Kim (1975) have observed the differential expressions of basal leaf colour in early hybrid generations of rice seedlings.

Genetic ratios involving two to five genes have been reported by Nadaf (1989).

| Character | Authority | Genetic ratio |
|---|---|---|
| i) Leaf blade at seedling stage | Nadaf (1989) | 3:13 |
| ii) Leaf tip at seedling stage | Nadaf (1989) | 45:19 |

| Character | Authority | Genetic ratio |
|---|---|---|
| iii) Leaf margin at seedling stage | Nadaf (1989) | 243:781 |

2.1.1.6.4.2 Leaf blade, tip and margin at flowering stage

Most of the rice geneticists have studied these leaf characters for inheritance of anthocyanin pigmentation at flowering stage only. The reports surveyed have been presented separately hereunder.

1) Leaf blade

| Authority | Genetic ratios |
|---|---|
| Parnell et al. (1917) | 3:1 and 9:7 |
| Takezaki (1923) quoted by Ramiah and Rao(1953) | 27:37 |
| Jones (1930) | 9:7 |
| Kato (quoted by Matsuura (1933)) | 3:1 and 9:7 |
| Sethi (1934) Quoted by Ramiah and Rao(1953) | 9:7 |
| Nakayama (1935) quoted by Ramiah and Rao(1953) | 3:1 |
| Morinaga (1938) | 3:1 |
| Kadam (1936), and Yamaguchi (1937) | 3:13 |
| Hsu and Lu (1943) | 117:139 |
| Dave (1948) and Chakravarty (1948) | 3:13 |
| Dave (1948) | 3:1 |
| Nagao and Takahashi (1951) | 3:13 |
| Ramiah (Unpublished, quoted by Ramiah and Rao, 1953) | 15:1 |

| Authority | Genetic ratios |
|---|---|
| Khan (1953), Shafi and Khan (1958) | 3:13 |
| Aziz and Shafi (1958) | 3:13 |
| Butany et al. (1959) | 9:7 |
| Iyer (1959) | 9:55 and 27:229 |
| Hsieh (1960, 1961) | 9:7 |
| Aziz et al. (1961) | 60 purple: 45 light purple:151 green |
| Panda (1962) | 27:37 |
| Dhulappanavar (1962) | 3:1 |
| Hsieh and Chang (1962) | 3:1 |
| Butany and Bhattacharya (1962) | 3:1, 3:13 |
| Ghose et al. (1963) | 3:13, 9:55, 27:229, 27:37 and 9:7 of purple wash to green |
| | 39 purple wash : 12 dark purple:13 green |
| | 27 purple wash: 9 faint purple:28 green |
| Butany and Ghose (1965) | 1:3, 3:13, 9:55 and 27:229 |
| Saran and Srivastava (1969) | 27:37 |
| Yadav (1971) | 3:13 |
| Setty and Misro (1973) | 9:55 |
| Dhulappanavar (1973 d and 1975) | 15:241 |
| Sastry and Seetharaman (1980) | 3:13 |
| Kinoshita and Maekawa (1986) | 1:3 and 3:13 |

| Authority | Genetic ratios |
|---|---|
| Sahu (1988); Sahu and Sahu (1989) | 13 green:3 purple |
| Kumar and Rangasamy (1989) | 3:1 and 3:13 |
| Nadaf (1989) | 39:25 and 12 purple:<br>27 purple wash :<br>25 green |
| Singh et al. (1990) | 27:37 |

The tetragenic ratio of 60 purple : 45 light purple: 151 green was obtained by Azis et al. (1961) in a cross between full purple and light purple parents. Out of the four genes $Pl_1$, $Pl_2$, I-Pl and Su-Pl involved in the interaction, both $Pl_1$ and $Pl_2$ could produce purple colour in the leaf blade. The gene I-Pl alone was found to be of no effect but in combination with Su-Pl it was found to inhibit purple colour all over the blade apart from the margins and tip. The gene Su-Pl alone reduced the purple colour to light purple.

In crosses involving parents with dark purple and purple wash leaf blade, the $F_2$ segregated into three classes giving a trigenic ratio of 39 purple wash: 12 dark purple: 13 green. Faint purple wash x green cross gave a ratio of 27 purple wash: 9 faint purple wash: 28 green. The faint purple wash was found to be simple recessive to purple wash. Ghose et al. (1963) have explained this inheritance elaborately on several assumptions.

Nadaf (1989) obtained the ratios of 39 non-green (full purple + purple wash) : 25 green or 12 full purple : 27 purple wash : 25 green indicating the operation of trigenic interaction with one basic gene, an inhibitory gene and an anti-inhibitory gene with incomplete penetrance.

(ii) Leaf tip and margin

| Authority | Genetic ratios |
|---|---|
| Dubey (1955) | 9:7, 81:175 and 117:139 |
| Anonymous (1956-57) | 3:1, 9:7, 27:37, 45:19, 54:10 and 162:94 |
| Iyer (1959) | 9:55, 45:19, 27:229 and 81:175 |
| Butany et al. (1959) | 9:7 |
| Kondo (1963a) | Reported 4 genes and also 3 inhibitory genes |
| Dhulappanavar (1973b) | 9:7 |
| Prasad et al. (1987) | 117:139, 9:55 and 27:37 |

(iii) Leaf tip

| | |
|---|---|
| Mitra and Ganguli (1932) | 3:1 |
| Ghose et al. (1960) | 3:1, 9:7, 27:37, 54:10, 45:19, 162:94 |
| Panda (1962) | 27:37, 162:94 |
| Rahman (1964) | 45:19 |
| Saran and Srivastava (1969) | 27:37 |
| Srivastava and Saran (1971) | 81:175 |

| Authority | Genetic ratios |
|---|---|
| Setty and Misro (1973) and Setty et al. (1973) | 702:322, 189:67 |
| Manjunath (1973) | 9:7 |
| Dhulappanavar et al. (1975a) | 1:15 |
| Thimmappaiah (1975) | 255:1 |
| Pavithran (1977) | 9:7 |
| Hadagal (1980) and Hadagal et al.(1980) | 45:19 |
| Rao and Misro (1986) | 45:19 |
| Nadaf (1989) | 3:1 |
| Singh et al. (1990) | 27:37, 162:94 |

An unusual ratio of 1:15 reported by Dhulappanavar et al. (1975a) was explained on the assumption of two inhibitory duplicate genes.

(iv) Leaf margin

| | |
|---|---|
| Mitra and Ganguli (1932) | 3:1 |
| Kuang (1951) including the work of other Chinese workers since 1919 | 3:1, 9:7, 15:1 and 63:1 |
| Panda (1962) | 27:37, 162:94 |
| Saran and Srivastava (1969) | 162:94 |
| Kadam (1970) | 81:175 |
| Setty and Misro (1973) and Setty et al. (1973) | 45:19 and 162:94 |
| Thimmappaiah (1975) | 15:1 |

| Authority | Genetic ratios |
|---|---|
| Pavithran (1977) | 9:7 |
| Hadagal (1980 and Hadagal et al. (1980) | 45:19 |
| Rao and Misro (1986) | 45:19 |
| Nadaf (1989) | 3:1 |
| Singh et al. (1990) | 27:37 and 162:94 |

2.1.1.6.5 Midrib

There are only a few reports available on the inheritance of midrib pigmentation. Genetic ratios involving two to four genes have been reported.

| | |
|---|---|
| Kondo (1963a) | Three genes involved |
| Dhulappanavar (1973b) | 9:7 |
| Dhulappanavar (1981) | 1:3:252 (Purple: Chalky white: White/green) |
| Nadaf (1989) | 27:37 |

The complicated ratio of 1:3:252 (purple:chalky white: white/green) reported by Dhulappanavar (1981), results from the involvement of one complementary gene and three inhibitory duplicate genes in a situation when the gene(s) for purple pigment are not segregating (homozygous) in the parents.

Nagao (1951) established the relationship of the pigmentation genes of leaf blade (Pl), leaf tip (Pla) and

midrib (Plm, now Pmr) in which case the segregation ratio of $F_2$ was always found out to be 12:3:1 showing that the gene Pl was epistatic to both Pla and Plm (now Pmr) and Pla was always epistatic to Plm (now Pmr).

2.1.1.6.6 Leaf axil

This refers to the basal portion of the inside of the leaf sheath. The pigmentation in the axil varies in intensity from light to dark purple either restricted at its lower portion or gets extended in its distribution.

The genetical ratio involving one to five genes have been reported for the inheritance of pigmentation in this part.

| Authority | Genetic ratio |
|---|---|
| Unpublished records quoted by Ramiah and Rao (1953) | 3 dark purple : 1 light purple |
| Unpublished records of CRRI, Cuttack quoted by Ghose et al. (1960) | 3:1, 9:7, 27:37, 45:19, 54:10, 81:175 and 162:94 |
| Nair (1958) | 3:1, and 9:7 |
| Butany et al. (1959) | 3:1 |
| Dhulappanavar (1962) | 9:7 |
| Saran and Srivastava (1969) | 3:1 |
| Dhulappanavar (1973a) | 54:10 |
| Dhulappanavar (1973b) | 162:94 |
| Manjunath (1973) | 54:10 |
| Setty and Misro (1973) | 54:10, 3:1 |

| Authority | Genetic ratio |
|---|---|
| Dhulappanavar and Hiremath (1974a and 1974b) | 54:10 |
| Dhulappanavar (1975; 1976b) | 162:94 |
| Dhulappanavar et al. (1975b) | 567:457 |
| Dhulappanavar (1981) | 27:229 |
| Sadananda (1981) | 39:25 and 117:139 |
| Nadaf (1989) and Sukesh Kumar (1990) | 3:1 |
| Singh et al. (1990) | 27:37 |

The segregation ratio of 27 purple spread : 9 purple base : 28 white axils observed in the study of Dhulappanavar (1973a) was explained on the assumption of three segregating loci controlling pigmentation in the axil. Two of these are basic-complementary for the development of colour at the base of the leaf axil and the third one, acting in conjunction with the two basic genes and under the influence of modifiers extends the pigment in this part to a varying extent.

2.1.1.6.7 Ligule

This is a thin upright membranous structure present on the inside of the juncture at its base where the blade joins the leaf sheath. It is often bilobed, ciliate or glabrous. The pigmentation in ligule varies from few purple specks to full purple and white.

The genetic ratios involving one to four genes have been reported by various authors.

| Authority | Genetic ratio |
|---|---|
| Hector (1922) | 9:7 and 27:37 |
| Chao (1928) | 27:37 |
| Mitra et al. (1928) | 3:1, 9:7 and 27:37 |
| Jones (1930) | 9:7, and 27:37 |
| Mahta and Dave (1931) | 3:1 |
| Mitra and Ganguli (1932) | 3:1 and 9:7 |
| Nakayama (1932) | 3:1 (From $F_3$ data) |
| Kuang (1951) and other Chinese authors since 1919 | 3:1, 9:7, 15:1, 27:37, 63:1 and 81:175 |
| Ramiah unpublished record Quoted by Ramiah and Rao(1953) | 3:1 and 15:1 |
| Dubey (1955) | 117:139 |
| Anonymous (1956-1957) | 9:7 and 117:139 |
| Unpublished records of CRRI, Cuttack, quoted by Ghose et al. (1960) | 3:1, 9:7, 45:19, 54:10 and 162:94 |
| Butany and Bhattacharya (1962) | 27:37 |
| Dhulappanavar (1962) | 3:1 |
| Srivastava et al. (1968) | 3:1 |
| Saran and Srivastava (1969) | 162:94 |
| Dhulappanavar and Menon kai(1970) | 54:10 |
| Dhulappanavar (1973b) | 162:94 |
| Setty and Misro (1973) | 46:19, and 162:94 |

| Authority | Genetic ratio |
|---|---|
| Manjunath (1973) | 189:67 |
| Thimmappaiah (1975) | 15:1 |
| Dhulappanavar (1975, 1976b) | 162:94 |
| Dhulappanavar et al. (1975b) | 27:229 |
| Pavithran and Mohandas (1976 a & b) | 27:37 and 45:19 |
| Pavithran (1977) | 9:7 |
| Dhulappanavar (1979) | 117:139 |
| Dhulappanavar (1980-81) | 243:13 |
| Dhulappanavar (1981) | 9:247 |
| Rao and Misro (1986) | 9:7 |
| Prasad et al. (1987) | 27:37 |
| Nadaf (1989) | 3:1 |
| Singh et al. (1990) | 27:37 and 1:3 |
| Ahmed and Das (1990) | 9:7 |

2.1.1.6.8 Auricles (Syn. sickles)

These are a pair of small, earlike appendages borne at the base of the blade, arise usually at the sides where the ligule and the base of the juncture are joined. The auricles may not persist on the older leaves. They may be colourless or with various shades of purple colour.

The genetic ratios involving one to five genes have been reported by various authors.

| Authority | Genetic ratio |
|---|---|
| Fruwirth (1908) (Quoted by Ramiah and Rao, 1953) | 15:1 |
| Parnell et al. (1917) | 3:1 |
| Hector (1922) | 9:7 |
| Mitra et al. (1928) | 3:1 |
| Mahta and Dave (1931) | 3:1 |
| Mitra and Ganguli (1932) | 9:7 and 1:15 |
| Nakayama (1932) | 3:1 |
| Jones (1930) | 9:7 and 27:37 |
| Dave (1948) | 9:7 |
| Kuang (1951) and other Chinese workers since 1919 | 3:1, 9:7, 15:1 and 63:1 |
| Dubey (1955) | 117:139 |
| Anonymous (1956-57) | 3:1 |
| Butany et al. (1959) | 27:37 |
| Unpublished records of CRRI, Cuttack, quoted by Ghose et al.(1960) | 3:1, 9:7, 54:10 and 162:94 |
| Butany and Bhattacharya (1962) | 9:7 |
| Sastry and Patnaik (1962) | 3:13 |
| Dhulappanavar (1962) | 9:7 |
| Saran and Srivastava (1969) | 162:94 |
| Kadam (1970) | 81:175 |
| Manjunath (1973) | 27:37 |
| Setty and Misro (1973) | 45:19 |
| Dhulappanavar (1973a, 1973c and 1975) | 27:37 |

| Authority | Genetic ratios |
|---|---|
| Dhulappanavar et al. (1973b) | 9:7 |
| Thimmappaiah (1975) | 15:1 |
| Dhulappanavar et al. (1975a and 1975b) | 387:637 |
| Dhulappanavar (1979) | 117:139 |
| Dhulappanavar (1980-81) | 45:19 |
| Shyla (1985) | 54:10 |
| Rao and Misro (1986) | 45:19 |
| Prasad et al. (1987) | 9:55, and 81:175 |
| Shyla and Pavithran (1989) | 54:10 |
| Nadaf (1989) | 27:37 |
| Ahmed and Das (1990) | 9:7 |
| Sukeshkumar (1990) | 9:7 |
| Singh et al. (1990) | 27:37 and 117:139 |

2.1.1.6.9 Junctura (Syn. collar, neck, leaf cushion)

It is the triangular joint portion found at the junction of the leaf sheath and leaf blade. The pigmentation in junctura is independent of that in the leaf sheath or leaf blade. Junctura (Junctura proper) pigmentation shows a wide variation from light purple dots to dark purple.

Genetic ratios involving one to five genes have been reported by various authors.

| Authority | Genetic ratio |
|---|---|
| Parnell et al. (1917) | 9:7, and 27:37 |
| Jones (1930) | 9:7 and 27:37 |
| Kuang (1951) | 3:1 and 3:13 |
| Bhattacharya (1957) | 117:139 |
| Butany et al. (1959); Panda (1962) | 27:37 |
| Iyer (1959) | 27:37, 45:19 and 27:229 |
| Unpublished records of CRRI, Cuttack quoted by Ghose et al. (1960) | 3:1, 9:7, 15:1, 45:19, 54:10, 117:139 and 162:94 |
| Butany and Bhattacharya (1962) | 9:7 |
| Sastry and Patnaik (1962) | 9:7 |
| Dhulappanavar (1962) | 45:19 |
| D'cruz and Dhulappanavar (1963) | 1:3 |
| Rahman and Srivastava (1968) | 81:175 |
| Saran and Srivastava (1969) | 3:1 |
| Dhulappanavar (1973d, 1975) | 1:3 |
| Dhulappanavar et al. (1973b) | 162:94 |
| Setty and Misro (1973) | 27:37 |
| Dhulappanavar et al. (1975b) | 9:7 |
| Thimmappaiah (1975) | 54:10 |
| Dhulappanavar (1977) | 45:19 |
| Dhulappanavar (1979) | 243:781 |
| Hadgal(1980)and Hadagal et al.(1984) | 27:37 |
| Sadananda (1981) | 21:43 and 27:229 |
| Shyla (1985) | 45:19 |

| Authority | Genetic ratio |
|---|---|
| Rao and Misro (1986) | 45:19 |
| Prasad et al. (1987) | 3:61 and 81:175 |
| Nadaf (1989) | 3:1 |
| Sukeshkumar (1990) | 9:7 |
| Ahmed and Das (1990) | 9:7 |
| Singh et al. (1990) | 27:37 and 117:139 |

An inhibitory gene I-Pj has been reported first by D'cruz and Dhulappanavar (1963) as indicated by the ratio 1 purple : 3 green. Dhulappanavar (1979) reported a ratio of 243 pigmented to 781 non-pigmented which indicates the involvement of five complementary genes for pigmentation.

2.1.1.6.10 Juncture back

It is a portion of the midrib present at the junction of leaf blade and leaf sheath behind the juncture. In most of the varieties the pigment in the juncture and that in the juncture back are completely associated but in certain varieties, the two are independently inherited (Ghose et al., 1960). There are only a few reports available on the inheritance of juncture back pigmentation. Genetic ratios involving two to five genes have been reported by the following authors.

| Authority | Genetic ratios |
|---|---|
| Unpublished records of CRRI, Cuttack (Quoted by Ghose et al., 1960) | 9:7, 27:37, 45:19, 54:10, and 162:94 |
| Sastry and Patnaik (1962) | 9:7 |
| Rahman (1964) | 27:37 |
| Saran and Srivastava (1969) | 9:7 |
| Srivastava and Saran (1971) | 81:175 |
| Dhulappanavar (1973d and 1975) | 9:247 |
| Setty and Misro (1973) | 162:94 |
| Dhulappanavar (1980-81) | 129:127 |
| Pavithran and Annie (1980) | 162:94 |
| Hadagal (1980) and Hadagal et al. (1984) | 9:7 |
| Shyla (1985) | 9:7 and 162:94 |
| Annie (1986) | 162:94 |
| Nadaf (1989) | 135:121 |
| Sukeshkumar (1990) | 243:781 |

2.1.1.6.11 Internode

This is the smooth solid (when young) or hollow (when matured) part of the culm, short basally and long apically between the successive nodes. The pigmentation in this part varies from faint purple lines to full purple almost as black. The yellowish colour is also seen some times. The pigmentation is usually observed when the plant is at flowering stage.

The genetic ratio involving one to four genes have been reported by various authors as presented below:

| Authority | Genetic ratio |
|---|---|
| Hector (1922) | 3:1, 9:7 and 27:37 |
| Chao (1928) | 9:7 |
| Mitra et al. (1928) | 3:1 and 9:7 |
| Jones (1930) | 9:7 and 27:37 |
| Ganguli (1942) | 9:7 |
| Kuang (1951) and other Chinese Workers since 1919 | 9:7 |
| Bhattacharya (1957) and Butany et al. (1959) | 81:175 |
| Iyer (1959) | 9:7, 27:37 and 81:175 |
| Unpublished records of CRRI, Cuttack Quoted by Ghose et al. (1960) | 9:7, 27:37, 81:175, 117:139 and 162:94 |
| Hsieh (1960) | 3:1 |
| Uzir (1961) | 3:1 and 9:7 |
| Butany and Bhattacharya (1962) | 27:37 |
| Bhattacharya (1957); Misro (1963) | 3:1 |
| Sastry and Patnaik (1962) | 27:37 |
| Nagai et al. (1962) | 9:7 |
| Kondo (1963a) | 3:1 |
| Rahman and Srivastava (1968) | 45:19 |
| Majumdar et al. (1969) | 9:7 |
| Kadam (1970) | 81:175 |
| Dhulappanavar (1973a, 1975) | 9:7 |

| Authority | Genetic ratio |
|---|---|
| Dhulappanavar et al. (1974) | 9:7 |
| Dhulappanavar et al. (1973b and 1975b) | 27:37 |
| Manjunath (1973) | 54:10 |
| Setty and Misro (1973) | 9:7 |
| Dhulappanavar and Hiremath (1974b) | 54:10 |
| Thimmappaiah (1975) | 57:7 |
| Dhulappanavar (1977 and 1979) | 9:6:1 |
| Kolhe and Bhat (1979) | 27:37 |
| Hadagal (1980) and Hadagal et al. (1980) | 45:19 |
| Sadananda (1981) | 117:139 |
| Dhulappanavar (1981) | 3:13 |
| Mori et al. (1981) | 3:1 |
| Kolhe and Bhat (1982) | 27:37 |
| Nadaf (1989) | 147:109 |
| Ahmed and Das (1990) | 9:7 |
| Singh et al. (1990) | 9:7 and 27:229 |

An unusual tatio of 27 non-pigmented to 37 pigmented for this character was reported by Saran and Srivastava (1969). The $F_1$ was non-pigmented indicating that the pigmentation was recessive and further suggested the involvement of three dominant complementary genes conditioning non-pigmentation in internode.

Dhulappanavar (1977 and 1979) observed three groups of plants exhibiting solid purple, purple lines and yellow pigmentation in $F_2$ in the ratio of 9:6:1. This segregation pattern was explained by the interaction of two complementary duplicate genes ($\underline{Pin}_{a1}$ and $\underline{Pin}_{a2}$) resulting in solid purple colour; whereas purple line pattern was determined by either of them. The recessive condition, however, expressed itself in a green internode at a young stage turning into yellow at maturity.

2.1.1.6.12 Node

This is the solid portion of the culm and is also a point from which a leaf or a tiller or adventitious roots originate. Its pigmentation varies from light purple, violet to dark purple.

The genetic ratio involving one to four genes have been reported by a few authors only.

| Authority | Genetic ratios |
|---|---|
| Lee (1927) quoted by Ramiah and Rao (1953) | 27:37 |
| Jones (1930) | 9:7 |
| Mitra and Ganguli (1932) | 3:1 |
| Silva and Vianna (1950) | 9:7 |
| Nagao (1951) | 3:1 |
| Nagao and Takahashi (1950 and 1951) | 3:1 |

| Authority | Genetic ratios |
|---|---|
| Takahashi (1957) | 3:1 |
| Butany et al. (1959) | 3:1 |
| Butani and Bhattacharya (1962) | 9:7 |
| Misro (1963) | 9:7 |
| Kadam (1970) | 27:37 |
| Dhulappanavar (1973a, 1973d and 1975) | 117:139 |
| Manjunath (1973) | 9:7 |
| Dhulappanavar et al. (1975b and 1975c) | 9:247 |
| Thimmappaiah (1975) | 15:1 |
| Dhulappanavar (1979) | 9:55 |
| Sadananda (1981) | 21:43 and 3:13 |
| Rao and Misro (1986) | 9:55 |
| Singh et al. (1990) | 9:7 and 162:94 |
| Ahmed and Das (1990) | 3:1 |
| Sukeshkumar (1990) | 9:7 |
| Nadaf (1989) | 111:145 |

2.1.1.6.13 Sheath pulvinus (Syn. sheath joint, pulvinus)

This is a small swelling at the base of the leaf sheath just above its point of insertion on the culm. The pulvinus can be colourless (white), green, shades of purple (including red) or purple dots. The reports on the pigmentation of this part are very scanty.

| Authority | Genetic ratios |
|---|---|
| Parnell et al:,(1917) | 3:1 |
| Hector (1922) | 9:7 |
| Mitra et al. (1928) | 9:7 |
| Mitra and Ganguli (1932) | 3:1 |
| Vander stock quoted by Matsuura (1933) | 3:1 and 15:1 |
| Unpublished records of CRRI, Cuttack Quoted by Ghose et al. (1960) | 162:94 |
| Hsieh (1960) | 9:7 |
| Butany and Bhattacharya (1962) | 9:7 |
| Dhulappanavar et al. (1973b) | 81:175 |
| Nadaf (1989) | 21:1003 |

Nadaf (1989) reported an unusual pentagenic ratio of 21:1003 for this character indicating that there was one basic gene conditioning the pigments in this part along with two inhibitory duplicate genes and two inhibitory complementary genes.

2.1.1.6.14 Pulvinus ring

This refers to the portion just above the sheath pulvinus which would appear to be surrounded by either dark green or light/dark purple/violet coloured pigments. As such this is a novel character identified and studied first by Nadaf (1989). The pigmentation around just above the sheath pulvinus confers a typical coloured ring like structure and

hence the name, pulvinus ring. Nadaf (1989) examined this character critically in all the $F_2$, parental and $F_1$ genotypes and suggested that this character being independent of other characters of pigmentation can be used as a marker in the genetical studies. The study indicated the character to be tetragenic, segregating in the ratio of 189 purple : 67 green.

2.1.1.6.15 Glumes (empty) (Syn. glumes, outer glumes, sterile glumes, sterile lemmas)

These are small flowerless bracts present at the base of the spikelets, just above the pedicel. The two glumes may differ in length and shape. The glumes may be colourless (white), straw, gold, brown, red or purple.

Genetic ratios involving one to five genes have been reported for the inheritance of pigmentation in glumes by the following authors.

| Authority | Genetic ratio |
|---|---|
| Hector (1922) | 3:1 and 9:7 |
| Mitra et al. (1928) | 3:1 and 9:7 |
| Jones (1930) | 9:7 |
| Nakayama (1932) | 9:7 |
| Jones (1933) | 3:1 and 9:7 |
| Jodon (1944) | 3:1 |
| Kuang (1951) and other Chinese workers since 1919 | 3:1, 9:7 and 27:37 |

| Authority | Genetic ratio |
|---|---|
| Alim and Sen (1956) | 3:1 |
| Shafi and Aziz (1959) | 45:19, 27:7, 3:1 |
| Iyer (1959) | 3:1 and 9:7 |
| Butany et al. (1959) | 27:37 |
| D'Cruz (1960) | 162:94 |
| Unpublished records of CRRI, Cuttack quoted by Ghose et al. (1960) | 3:1, 9:7, 27:37, 81:175, 117:139 and 243:781 |
| Uzir (1961) | 3:1 |
| Dhulappanavar (1962) | 9:7 |
| D'Cruz and Dhulappanavar (1963) | 3:1 |
| Panda et al. (1967) | 3:13 |
| Rahman and Srivastava (1968) | 9:55 |
| Ratho and Rao (1972) | 3:1 |
| Dhulappanavar (1973a and 1975) | 27:37 |
| Dhulappanavar (1973b) | 9:7 |
| Dhulappanavar et al. (1973a and 1974) | 9:7 |
| Manjunath (1973) | 9:7 |
| Setty and Misro (1973) | 9:7 |
| Dhulappanavar (1976a) and Dhulappanavar et al. (1975a, 1975b) | 117:139 |
| Thimmappaiah (1975) | 15:1 |
| Dhulappanavar (1979) | 9:55 |
| Dhulappanavar (1981) | 9 purple:54 white: and 193 green |
| Sadananda (1981) | 27:37 and 243:781 |

| Authority | Genetic ratio |
|---|---|
| Yadav (1984) | 3:1 and 207:49 |
| Nadaf (1989) | 255 : 769 |
| Ahmed and Das (1990) | 9:7 |

Dhulappanavar (1981) observed a complex nature of inheritance of anthocyanin pigmentation in glume that gave three classes of glumes, purple:white:green (9:54:193). The author presumed the involvement of two sets of four genes, each differing in its action namely, a set of two complementary genes and the other, of two duplicate genes each having ability to complement with the complementary genes. Complementary genes alone complement to produce purple pigment. But with any one of the complementary duplicate genes produce chalky white glumes. The remaining combinations do not produce either purple or white thus resulting in green.

Nadaf (1989) observed a pentagenic ratio of 255:769 for the inheritance of glume pigmentation suggesting the operation of four duplicate genes and one inhibitory gene. Ghose et al. (1960) and Sadananda (1981) have also quoted a ratio of 243:781 suggesting the operation of five complementary genes, to condition the pigment in the glumes.

2.1.1.6.16 Lemma/Palea (Syn.Flowering glume)

The indurate (hardened), 5-nerved bract of the floret partly enclosing the palea is called as the lemma

while the indurate, 3-nerved bract of the floret which fits closely to the lemma is called as the palea. Lemma and palea are the flowering glumes enclosing the essential organs of the spikelet and at later stages, form the husk or hull of the grain. Perhaps there is no other character showing so much colour variation as that of lemma/palea. Colour in young stage is different from the colour in ripening stage and hence, observations are recorded twice, once in the green stage and next in ripening stage (Ramiah and Rao, 1953). The different ratios for this character at ripening stage involves one to five genes as reported by various authors. Nadaf (1989) has recently reported a ratio involving six genes.

| Authority | Genetic ratio |
|---|---|
| Parnell et al. (1917) | 3:1 and 9:7 |
| Hector (1922) | 3:1 and 9:7 |
| Chao (1928) | 9:7 |
| Kato quoted by Matsuura (1933) | 9:3:3:1 |
| Ramiah (1935) | 3:1 |
| Cada (1949) | 9:7 |
| Unpublished records of CRRI, Cuttack Quoted by Ramiah and Rao (1953) | 9:7 |
| Aziz and Shafi (1958) | 9:6:1 (Straw yellow: golden:reddish brown) |
| Unpublished records of CRRI, Cuttack Quoted by Ghose et al. (1960) | 3:1, 9:7, 27:37 and 162:94 |
| Uzir (1961) | 3:1 and 9:7 |

| Authority | Genetic ratio |
|---|---|
| Dhulappanavar (1962) | 9:7 |
| D'Cruz and Dhulappanavar (1963) | 3:1 |
| Setty (1968) | 9:7 |
| Saran and Srivastava (1969) | 162:94 |
| Majumdar et al. (1969) | 1:3 |
| Manjunath (1973), Setty and Misro (1973) | 9:7 |
| Dhulappanavar et al. (1973a and 1974) | 9:7 |
| Dhulappanavar et al. (1975b) | 405:619 |
| Pavithran (1977) | 3:13 |
| Dhulappanavar (1979) | 9:55 |
| Kadam et al. (1980) | 27:37 |
| Dhulappanavar (1981) | 15 chalky white: 49 green |
| Hadagal (1980) and Hadagal et al. (1984) | 3:13 |
| Tripathi and Rao (1985) | 3:13 |
| Nadaf (1989) | 3:4093 |

The segregation ratio of 405 purple:619 green in the study of Dhulappanavar et al. (1975b) indicated the involvement of five genes for the expression of colour in lemma/palea, of which three complementary genes interact with any one or both of the remaining two complementary duplicate genes to produce the phenotype.

Nadaf (1989) has recently reported a hexagenic ratio of 3:4093 for pigmented to non-pigmented in lemma/palea which is the first report suggesting the involvement of one basic gene along with five inhibitory duplicate genes for the expression of colour in lemma/palea.

2.1.1.6.17 Apiculus

Apiculus is the extended tip of the lemma or palea. It also refers to the short excurrent tips of the major fibro-vascular bundles of the lemma-palea (Jones, 1930). The apiculus shows various shades of purple pigmentation which differ during anthesis and ripening. The study of anthocyanin pigmentation in apiculus is of greater importance than other characters because the genes governing expression of colour in apiculus, being basic genes, are known to have the direct effect on pigmentation in other parts of the plant. The role of component genes of the C - A - P complementary genic system has already been discussed.

The genetic ratios involving one to four genes have been reported for pigmentation in apiculus by many authors as mentioned below.

| Authority | Genetic ratio |
|---|---|
| Van der Stok (1908) qhoted by Ramiah and Rao (1953) | 15:1 |
| Hector (1916) | 3:1 and 9:7 |
| Parnell et al. (1917) | 3:1 |

| Authority | Genetic ratio |
|---|---|
| Hector (1922) | 3:1, 9:7, 15:1 and 27:37 |
| Mitra et al. (1928) | 3:1, and 9:7 |
| Chao (1928) | 3:1, 15:1, 9:7, 27:37 and 162:94 |
| Jones (1930) | 3:1 |
| Nakayama (1932) | 3:1 |
| Jones (1933) | 3:1 and 9:7 |
| Jodon (1940) | 3:1 |
| Morinaga et al. (1943) | 3:1 |
| Kuang et al. (1946) | 3:1 |
| Jodon and Chilton (1946) | 3:1, 9:7 and 27:37 |
| Anonymous (1948-49) | 9:7 |
| Silva and Vianna (1950) | 3:1 |
| Kuang (1951) | 3:1 |
| Ramiah and Rao (1953) | 9:7, 15:1, 27:37, 9:3:4, and 27:9:28 |
| Chandraratna (1953) | 3:1 |
| Jodon (1955) (Unpublished record) | 171:85 |
| Dubey (1955) | 3:1, 9:7, 27:37, 54:10 and 162:94 |
| Alim and Sen (1956) | 3:1 |
| Jodon (1957) | 3:1 |
| Butany et al. (1959) | 3:1 |
| Aziz and Shafi (1958) and Shafi and Aziz (1959) | 3:1 and 9:7 |

| Authority | Genetic ratios |
|---|---|
| Unpublished records of CRRI, Cuttack quoted by Ghose et al. (1960) | 45:19, 54:10 and 162:94 |
| Hsieh (1960 & 1961) | 3:1 and 9:7 |
| Kadam and D'Cruz (1960) | 9:7 |
| Ramirez et al. (1960) | 9:7 |
| Uzir (1961) | 3:1 |
| Butany and Bhattacharya (1962) | 45:19 |
| Nagai et al. (1962) | 9:7 and 15:1 |
| Panda (1962) | 162:94 |
| Misro (1963) | 3:1, 9:7 and 162:94 |
| Sastry and Patnaik (1962) | 9:7, 45:19 and 54:10 |
| Hsieh and Chang (1964) | 3:1 and 9:7 |
| Misro and Kulkarni (1965) | 3:1 |
| Panda et al. (1967) | 3:1 |
| Prasada Rao and Misro (1968) | 9:7 |
| Setty (1968); Setty et al.(1973) and Misro (1973) | 3:1, 15:1 and 54:10 |
| Srivastava et al. (1968) | 3:1 |
| Yen and Hsieh (1968) | 3:1 and 9:7 |
| Majumdar et al. (1969) | 9:7 |
| Saran and Srivastava (1969) | 3:1 |
| Dhulappanavar and Mensinkai (1970) | 54:10 |
| Ratho and Rao (1972) | 15:1 |
| Manjunath (1973) | 15:1 |
| Dhulappanavar (1973a) | 15:1 |

| Authority | Genetic ratios |
|---|---|
| Eunus *et al.* (1974) | 3:1 and 15:1 |
| Dhulappanavar *et al.* (1975b) and Dhulappanavar (1976a) | 9 purple: 3 red: 4 White |
| Dhulappanavar (1975 and 1976b) | 162:94 |
| Akbar *et al.* (1975) | 9:7 |
| Pavithran (1977) | 9:7 |
| Nascimento (1977) | 3:1 |
| Dhulappanavar (1977) | 39:25 |
| Tripathi and Rao (1979) | 13 P : 3 Q |
| Hadagal (1980) and Hadagal *et al.* (1980) | 45:19 |
| Pavithran and Annie (1980) | 45:19 |
| Kolhe and Bhat (1979 and 1982) | 45:19 |
| Dhulappanavar (1981) | 135 purple: 90 chalky white: 31 green |
| Sadananda (1981) | 189:67, 135:121 |
| Shyla (1985) | 3:1, 45:19, 54:10 and 162:94 |
| Ramesh (1984) | 27:37 |
| Yadav and Tomar (1984) | 27:37 |
| Tripathi and Rao (1984) | 27:37 |
| Rao and Misro (1986) | 3:1 |
| Paramashivam (1986) | 9:7 |
| Annie (1986) | 3:1, 9:7, 45:19, 54:10 |
| Prasad *et al.* (1987) | 9:7, 27:37 and 54:10 |
| Sano (1988) | 3:1 |

| Authority | Genetic ratios |
|---|---|
| Sahu and Sahu (1989) | 13 purple: 3 green |
| Shyla and Pavithran (1989) | 54:10 |
| Nadaf (1989) | 3:1 |
| Sukeshkumar (1990) | 3:1 |
| Singh et al. (1990) | 162:94 |
| Ahmed and Das (1990) | 3:1 |

A ratio of 171 purple: 85 straw white has been reported by Kadam (cited by Jodon, 1955). The results were explained on the hypothesis that one dominant gene in the presence of basic gene A governs the purple apiculus while the other dominant duplicate gene controls the purple apiculus in the presence of both the basic genes C and A.

Dhulappanavar et al. (1975b) and Dhulappanavar (1976a) reported that the interaction of a supplementary gene with one of the basic genes for pigmentation resulted in the ratio of 9 purple: 3 red: 4 green.

The segregation ratio of 135 purple: 90 chalky white: 31 green reported by Dhulappanavar (1981) was explained on the basis of involvement of two sets of two genes each, one set is of complementary genes and the other, of complementary duplicate genes. Two complementary genes with one or both of complementary duplicate genes produce purple pigmentation and one of the two complementary genes with one

or both complementary duplicate genes produce chalky white apiculus. The remaining combinations do not produce either purple or chalky white thus resulting in green colour.

2.1.1.6.18 Stigma

The short style bears the bifurcate, plumose stigma whose colour varies from colourless white, yellow, light purple to purple, violet and black.

The genetic ratios involving one to four genes have been reported for the stigma pigmentation by many authors as mentioned below:

| Authority | Genetic ratios |
|---|---|
| Van Der Stok (1910) quoted by Ramiah and Rao (1953) | 15:1 |
| Hector (1916) | 3:1 |
| Parnell et al. (1917) | 9:7 |
| Parnell et al. (1922) | 3:1 |
| Hector (1922) | 3:1, 27:37 and 81:175 |
| Chao (1928) | 3:1 and 9:7 |
| Mitra et al. (1928) | 3:1, 9:7 and 15 G:1 P |
| Jones (1930) | 9:7 |
| Mitra and Gangulī (1932) | 3:1 |
| Nakayama (1932) | 9:7 |

| Authority | Genetic ratio |
|---|---|
| Khan (1953) | 3:1 |
| Ramiah and Rao (1953) | 3:1, 9:7, 15 P: 1 G |
| Dubey (1955) | 3:1, 9:7, 27:37, 45:19 81:175 and 162:94 |
| Anonymous (1955-56) | 3:1 |
| Anonymous (1956-57) | 3:1, 9:7, 15:1 and 81:175 |
| Aziz and Shafi (1958) | 3:1 |
| Shafi and Khan (1958) | 3:1 |
| Shafi and Aziz (1959) | 3:1 |
| Butany et al. (1959) | 9:7 |
| D'Cruz (1960) | 162:94 |
| Hsieh (1960 & 1961) | 3:13, and 9:55 |
| Unpublished records of CRRI, Cuttack Quoted by Ghose et al. (1960) | 3:1, 9:7, 27:37, 45:19, 81:175 and 162:94 |
| Kadam and D'Cruz (1960) | 27:37 and 81:175 |
| Ramirez et al. (1960) | 27:37 |
| Butany and Bhattacharya (1962) | 162:94 |
| Nagai et al. (1962) | 9 G:7 P |
| Sastry and Patnaik (1962) | 45:19 |
| D'Cruz and Dhulappanavar (1963) | 3:1 |
| Kondo (1963a) | 3:1 and 1:3 |
| Misro (1963) | 3:1, 9:7 and 27:37 |
| Misro and Kulkarni (1965) | 3:1 |
| Panda et al. (1967) | 3:13 |
| Prasad Rao and Misro (1968) | 9:7 |
| Setty (1968) | 3:1, 54:10 and 162:94 |

| Authority | Genetic ratios |
|---|---|
| Srivastava et al. (1968) | 3:1 |
| Majumdar et al. (1969) | 9:7 |
| Saran and Srivastava (1969) | 162:94 |
| Srivastava and Saran (1971) | 9:7 and 9:55 |
| Ratho and Rao (1972) | 9:7 |
| Kadam and Desale (1972) | 162:94 and 189:67 |
| Manjunath (1973) | 54:10 |
| Setty and Misro (1973) | 54:10 and 162:94 |
| Dhulappanavar (1973a) | 54:10 |
| Dhulappanavar et al.(1973a) and Dhulappanavar (1975, 1976b) | 162:94 |
| Dhulappanavar and Hiremath (1974a and 1974b) | 54:10 |
| Sastry et al. (1975) | 81:175 |
| Pavithran (1977) | 27:37 |
| Hadagal (1980) and Hadagal et al. (1984) | 45:19 |
| Pavithran and Annie (1980) | 9:7 |
| Kolhe and Bhat (1979 and 1982) | 162:94 |
| Dhulappanavar (1981) | 9 purple: 9 chalky White: 46 White |
| Sadananda (1981) | 45:19 and 135:121 |
| Ramesh (1984) | 27:37 |
| Yadav (1984) and Yadav and Tomar (1984) | 3:1, 43:21 and 13:3 |
| Shyla (1985) | 3:1, 45:19, 54:10 and 162:94 |

| Authority | Genetic ratios |
|---|---|
| Annie (1986) | 3:1, 9:7, 54:10 and 162:94 |
| Ghosh and Hossain (1986) | 3:1 |
| Prasad et al. (1987) | 3:1, 9:7, 27:37 and 45:19 |
| Shyla and Pavithran (1989) | 54:10 |
| Nadaf (1989) | 3:1 |
| Sukeshkumar (1990) | 3:1 |
| Ahmed and Das (1990) | 9:7 |
| Singh et al. (1990) | 162:94 |

Dhulappanaver (1981) reported a trigenic interaction giving three classes of pigmentation in stigma in a ratio of 9 purple: 9 chalky white: 46 white, which was explained on the basis of involvement of one basic complementary and two complementary duplicate genes. A basic gene with one complementary duplicate gene will produce purple stigma while the same basic gene with other complementary duplicate gene will produce chalky white stigma. The remaining combinations do not produce either purple or chalky white thus resulting in white.

Oka (1990) made a detailed analysis of genes for stigma colouration. The anthocyanin pigmentation of the apiculus is controlled by three complementary genes, C, A and P which are known to serve as the basic colouration

genes, but the genetic control of stigma colouration is still more complex (Oka, 1990). To throw some light on the gene system, $F_1$ and $F_2$ data for more than 220 crosses were investigated to establish a symbolic logic to explain the whole by assuming a minimum number of genes. The parents involved in the crosses comprised the *indica* and *japonica* types approximately half and half. In ordinary cases, stigma colouration occurs only in plants with C, A and P. The independent genes, $Ps_2$ and $Ps_3$ were involved in this, the former being relatively frequent in *japonicas* and the latter in *indicas*. For $Ps_2$, inhibitory genes were found, that is, $I\text{-}Ps_1^2$, $I\text{-}Ps_2^2$, $I\text{-}Ps_a^2$ and $I\text{-}Ps_b^2$, the last two being complementary. No inhibitory gene was found for $Ps_3$. A Japanese upland variety (Gaisen-mochi) exceptionally showing colourless apiculus and coloured stigma, had $Ps_1$ which expressed stigma colour without P. Its inhibitor, $I\text{-}Ps_1^1$ (tightly linked with $I\text{-}Ps_1^2$), was not effective without P. Four genes, P, $Ps_3$, Ph and $Ps_1$, had their loci in linkage group II in the given order. Their recombination values were estimated (Oka, 1990).

2.1.1.6.19 Panicle base, panicle pulvinus and panicle axis

These are the new characters of the panicle studied first by Nadaf (1989), to understand the inheritance of pigmentation in these parts.

2.1.1.6.19.1 Panicle base

This refers to the nearly solid node between the upper most internode of the culm and the main axis of the panicle. This node gives rise to the first primary branches of the panicle and usually bears no leaf or dominant bud.

2.1.1.6.19.2 Panicle pulvinus

This refers to the swelling in the axils of the primary panicle branches, more noticeable during panicle emergence.

2.1.1.6.19.3 Panicle axis (Syn. rachis)

This refers to the distinctly grooved, main axis of the panicle, extending from the base to the apex. The axis is hollow except at the regions (nodes) where the primary panicle branches are borne.

Pigmentation in these panicle parts is found to be characteristic of genotypes and it is independent of the pigmentation in other plant parts. The pigmentation in these parts vary, ranging from light purple, dark purple to black.

Nadaf (1989) reported a ratio of 171 blackish purple: 853 green showing a pentagenic interaction for each of these traits. Further, pigmentation in panicle base, panicle pulvinus and panicle axis was found to be pleiotropic

and governed by a set of two complementary genes along with one inhibitory basic and two inhibitory complementary duplicate genes.

2.1.1.6.20 Modal septum (Syn. septum)

This is the solid partition in the node which separates two adjoining internodes. The colour of septum is best observed on taking a transverse section of the stem just below the node. The pigment in this part varies from white/light yellow to purple and shades of purple.

The information on septum pigmentation is very scanty. Genetic ratios involving one to four genes have been reported by a few authors.

| Authority | Genetic ratios |
|---|---|
| Ganguli (1942) | 9:7 |
| Dubey (1955) | 3:1, 9:7, 27:37 and 81:175 |
| Anonymous (1956-57) | 3:1 and 9:7 |
| Ghose et al. (1960) | 3:1 |
| Butany et al. (1959) | 3:1 |
| Saran and Srivastava (1969) | 3:1 |
| Srivastava and Saran (1971) | 27:37 |
| Dhulappanavar (1973a and 1975) | 9:7 |
| Manjunath (1973) | 54:10 |

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| Authority | Genetic ratios |
|---|---|
| Setty and Misro (1973) | 3:1 |
| Thimmappaiah (1975) | 15:10 |
| Dhulappanavar and Hiremath(1974b) | 54:10 |
| Dhulappanavar et al. (1975b) | 189 purple: 45 yellow: 22 green/White |
| Dhulappanavar (1981) | 1 purple: 27 yellow: 36 White |
| Sadananda (1981) | 39:25 and 27:37 |
| Rao and Misro (1986) | 9:7 |
| Nadaf (1989) | 81:175 |

Dhulappanavar et al. (1975b) reported a tetragenic ratio comprising of three phenotypic classes in the $F_2$ segregation. The pigmentation in septum showed the segregation ratio of 189 purple:45 yellow:22 white. Here, the complementary gene for purple pigmentation interacts with any one or more of the remaining three complementary duplicate genes to produce purple colour. Of the remaining three genes, one gene in the absence of the complementary gene for purple pigment, acts as a complementary gene for yellow colour which, on interaction with any one or both of the complementary duplicate genes produces yellow colour. The remaining combinations do not produce either purple or yellow colour, thus resulting in white septum. The segregation ratio of 1:27:36 (purple:yellow:white) reported (Dhulappanavar,1981)

was explained on the basis of exclusive role of three segregating complementary duplicate inhibitors in the absence of segregating gene or genes for purple pigment. All the inhibitory duplicate genes in dominant condition complement to produce yellow septum while in their recessive condition purple septum is produced. In rest of the combinations white septum is realized.

2.1.1.6.21 Nodal ring

This is a part of the stem which is just below the node. The pigmentation in this part is usually inseparable from that of node. But in few cases the pigmentation in the nodal ring can be distinguished from that of node. The colour of this part is best observed on taking a transverse section of the stem just below the node. The circular portion that surrounds the central nodal septum is the nodal ring whose pigmentation varies from green, white, yellow to light purple, dark purple, violet and black. There are a very few studies leading to understand the inheritance of nodal ring pigmentation.

| Authority | Genetic ratio |
|---|---|
| Dhulappanavar (1979) | 9:55 |
| Hadagal (1980) and Hadagal et al. (1980) | 45:19 |
| Nadaf (1989) | 63:961 |

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Nadaf (1989) reported a pentagenic ratio of 63 purple: 961 green for this character and showed the involvement of three duplicate genes responsible for colouration along with two inhibitory duplicate genes.

2.1.1.6.22 Awn colour (presence or absence of red awns)

Awn (Syn. arista, beard) is a filiform extension of varying lengths from the Keel (middle nerve) of the lemma. The main colours of the awn are white or straw, pink brown and purple red. The studies and information regarding awn colour inheritance are very few.

| Authority | Genetic ratio |
|---|---|
| Hector (1916) | 3:1 |
| Nagai (1921) | 3:1 |
| Jones (1930) | 3:1 |
| Kato (quoted by Matsuura, 1933) | 9:7, 3:1 and 9:3:4 |
| Okada (quoted by Matsuura, 1933) | 3:1 |
| Sethi et al. (1937) | 3:1 |
| Unpublished records of paddy breeding station, Coimbatore (quoted by Ramiah and Rao, 1953) | 3:1 |
| Dhulappanavar (1981) | 90 purple: 90 white: 76 absent |
| Sadananda (1981) | 39:25 and 117:139 |
| Rao and Misro (1986) | 9:7 |
| Nadaf (1989) | 3:4093 |

The complex ratio of 90 purple awns:90 white awns: 76 absent reported by Dhulappanavar (1981) explains the genetic mechanism of both awning (presence and absence of awns) and awn colour, that involves four interacting genes. Purple awns are determined by the interaction of one basic gene for awning with three complementary duplicate genes, but one of them (independent dominant) interacting with the basic gene also produces the same effect. Two complementary duplicate genes interacting with the basic gene cause white awns which are also produced when a single complementary duplicate gene (independent dominance) interacts with the basic gene.

Nadaf (1989) reported a hexagenic ratio of 3:4093 for pigmented to non-pigmented awn suggesting the involvement of one basic gene along with five inhibitory duplicate genes for the expression of awn colour.

### 2.1.2 Morphological character

#### 2.1.2.1 Awning (presence or absence of awns)

Awning refers to the phenomenon of production of awns, which are filiform extensions of varying lengths from the keel (middle nerve) of the lemma. Awning is considered as an important character as it confers resistance to drought and birds. Awns are prevalent more in the wild varieties than in cultivated varieties. Its development is often

influenced by environmental conditions like spacing, manuring etc. Sethi et al. (1937) found closer spacing to increase the length of the awns, but manuring did not affect in any way. Awn development is also affected by the season, root pruning and level of polyploidy (Sahadevan, 1959).

In the primary tiller, the awns may be absent, but occasionally the secondary tillers are known to develop awns. This is said to be hereditary by Piacco (1917).

Genetic ratios involving one to five genes have been reported for the presence or absence of awn as mentioned below:

| Authority | Genetic ratio |
|---|---|
| Hoshino (1915) and Van der Stok(1915) (quoted by Ramiah and Rao, 1953) | 3:1 |
| Ikeno (1927) | 63:1 and 9 awned: 6 slightly awned: 1 awnless |
| Chao (1928) | 15:1 |
| Alam (1932) | 15:1 |
| Mitra and Ganguli (1932) | 15:1 |
| Ramiah (1935) | 9:7 |
| Sethi et al. (1937) | 63:1 |
| Majid (1939) | 9 awned: 6 slightly awned: 1 awnless |
| Housaye and de La (1942) | 3:13 |

| Authority | Genetic ratios |
|---|---|
| Kuang (1951) | 3:1, 9:7 and 15:1 |
| Ramiah (unpublished; Quoted by Ramiah and Rao, 1953) | 3:1, 15:1, 9:6:1, 12:3:1 and 63:1 |
| Unpublished records of CRRI, Cuttack Quoted by Ghose *et al.* (1960) | 9:7, 27:37, 81:175 and 243:781 |
| Nagai *et al.* (1962) | 9:3:4 |
| Seetharaman (1965) | 3:1 |
| Jodon (1965) | 3:1 and 81:175 |
| Prasad Rao and Misro (1968) | 3:1 |
| Kadam and Pant (1969) | 15:241 |
| Thimmappaiah (1975) | 117:139 |
| Prasad Rao and Misro (1977) | 15:1 |
| Sastry (1977) | 9:7 and 27:37 |
| Dhulappanavar (1981) | 90 purple awns: 90 White awns:76 absent |
| Tripathi and Rao (1985) | 15:1 |
| Rao and Misro (1986) | 45:19 |
| Pavithran (1986) | 3:1, 15:1, 9:7, 27:37, 63:1, 54:10, 81:175, 243:781, and 3:13 |
| Nadaf (1989) | 3:1021 |
| Sahu (1991) | 3:1 |

Inhibitory gene/s for awning have been reported by Housaye (1942), Misro and Misro (1954), Kadam and Pant (1969), Thimmappaiah (1975), Pavithran (1986) and Nadaf (1989). Dhulappanavar (1981) explained both awn colour and awning in

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the segregation ratio of 90 purple awns: 90 white awns: 76 absent on the basis of four interacting genes (Refer awn colour 2.1.1.6.22).

With the wealth of literature surveyed here, it is now evident that the inheritance pattern of anthocyanin colouration has been exhaustively dealt with in conventional genetic studies and the pigmentation genes have been assigned to their linkage groups based on genetic recombination values by almost all the authors mentioned in the above paragraphs. Needless to say, the primary trisomic technique for determining gene-chromosome relationship is an innovative approach which has not been dealt so far; though it has been stressed by many geneticists (Sharma, 1986, Siddiq et al., 1986; Pavithran, 1986; Kinoshita, 1984; 1986a; 1986b and Goud and Nadaf, 1990a).

## 2.2 Primary trisomic analysis

Monosomics and nullisomics of allopolyploids have been extensively used in locating genes to specific chromosomes and in tailoring the genotypes to specific needs through alien addition and substitution of chromosomes (Sears, 1958). However, monosomics and nullisomics were not found useful in crops which are presumably diploids where deficiency for one or both of a pair of chromosomes is deleterious since each chromosome of the complement in such plants is critical for gametophytic and sporophytic

survival. In true diploids or plant species with highly diploidised allopolyploid genomes, aneuploids characterized with extra chromosomes such as trisomics have been found more fruitful.

Trisomics are individuals with one or more (double trisomics, etc.) extra chromosomes in an otherwise disomic chromosome complement. Trisomy is very common in plants. It was first discovered in Jimson weed (Datura stramonium) by Prof. A.F. Blakeslee in 1915. Organisms of this kind are hyperploids. There are five major kinds of trisomy recognized:

i) Primary trisomy: The additional chromosome is completely homologous to one of the chromosome pairs of the complement.

ii) Secondary trisomy: The additional chromosome is a secondary chromosome or an isochromosome.

iii) Tertiary trisomy: The additional chromosome is a translocated or tertiary chromosome consisting of two nonhomologous chromosome segments.

iv) Compensating trisomy: A chromosome is missing and is genetically compensated by two other modified chromosomes.

v) Telosomic trisomy: The additional chromosome is a telocentric chromosome.

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These five types of trisomics can be distinguished cytologically in meiosis. Burnham (1962) and Khush (1973) in their exhaustive reviews provided many examples of such types of trisomics that could be utilized in cytogenetical investigations and plant breeding programmes.

The trisomic series in primary, secondary or tertiary forms are being produced in an increasing number of crop plants (i) to locate genes on specific chromosomes, (ii) to identify chromosomes with their respective linkage groups and (iii) to determine phenotypic effects of individual chromosomes. Employing this technique valuable information on gene-chromosome relationship has been obtained in many crop plants. Tomato (Lesley, 1932; Rick and Barton, 1954; Rick et al., 1964), barley (Ramage, 1965; Tsuchiya, 1958; 1960; 1967; maize (McClintock, 1929; McClintock and Hill, 1931; Rhoades, 1933), sorghum (Schertz, 1966), Nicotiana (Goodspeed and Avery, 1939), emmer wheat (Sears, 1954; Tsunewaki, 1964), spinach (Janick et al., 1959, Tabushi, 1958), rye (Kamanoi and Jenkins, 1962), Capsicum (Pochard, 1968) and pearlmillet (Gill et al., 1970) are some notably classic examples where the trisomic series in primary, secondary or tertiary forms have been developed and where trisomic analyses have been successfully employed. Complete trisomic series have so far been isolated in only 30 flowering plant species (Sareen et al., 1990).

Isolation and extensive study of trisomics have unfolded its usefulness in a variety of ways. While it has proved as an excellent cytogenetic tool in finding solutions to several basic problems that remained topics of great controversy, trisomics have been of help indirectly for crop improvement.

### 2.2.1 Sources of trisomics

The trisomics, like other chromosomal variants such as polyploids, deficiencies, translocations, and inversions, appear spontaneously in natural populations wherein abnormal meiosis leading to the formation of n + 1 gametes occurs; but these variants are so rare that large populations have to be sampled to assemble a reasonable collection of chromosomal deviants.

Of all the sources of trisomics that Khush (1973) has listed, autotriploids and asynaptic plants are considered to be good and most dependable sources for obtaining trisomics. In case of autotriploids, there is a random unequal separation of chromosomes between two poles resulting in the formation of spores with all the chromosome numbers varying from n to 2n; while in asynaptic disomics, a variable number of univalents are present at metaphase I of meiosis due to disturbances in normal pairing. Those univalents segregating at random to the two poles produce n + 1 gametes. Of all the

kinds of trisomics, the primaries are the easiest to obtain (Khush, 1973).

### 2.2.2 Morphology of trisomics

When present in the trisomic condition, the individual chromosome of the complement has a distinct effect on cellular processes and developmental rhythm of the individual. These effects are detected in the altered morphology, anatomy and physiology. The morphological differences between the trisomics and the disomics are of the same nature as the differences between related species and subspecies. Such differences are very helpful in their precise analysis and identification.

Blakeslee (1922, 1930, 1934), Blakeslee and Belling (1924), Avery and Blakeslee (1948) named the trisomics according to the differences in the size and shape of the capsule, size and length of spine, size of the plant and growth habit, shape and form of leaves, flowers and stigmas.

Rick and Barton (1954) reported that in tomato the trisomic for chromosome 3 and 4 produce elongated leaves and leaf segments, longer internode and inflorescences, larger flowers, fruits and even seeds. In triplo 7 and triplo 10, leaves were broader and thicker, the internode inflorescences and flowers were shorter and the fruits were flat. In triplo-1, all plant parts including the fruits were extremely reduced in size.

In addition to gross morphological changes of flower parts, male and female reproductive parts of a trisomic may also be affected. Lin and Ross (1969a, 1969b) described pollen grains with papiliform appendages in the trisomic type-8 of Sorghum vulgare and ovaries with tumerous growth in the trisomic type-9.

Trisomics in general, are less vigorous than normal plants (Mohammed, 1974). Gill et al. (1970) isolated a complete set of seven primary trisomics in pearl millet and found a gradient with regard to vigour which indicated that the vigour of the plant was negatively correlated with the size of the extra chromosome.

Primary trisomics can be distinguished morphologically in Avena sativa (Azael, 1973), Avena strigosa (Rajhathy, 1975) and Pennisetum (Manga, 1976) but morphological differences between trisomics are not large enough to be distinguished in Triticum (Sears, 1954), and Nicotiana (Clausen and Goodspeed, 1924). In maize, only two trisomics, triplo-3 and triplo-5, could be identified morphologically. The rest could not be distinguished from each other nor from the disomics(McClintock, 1929; McClintock and Hill, 1931; Rhoades and McClintock, 1935).

In Sorghum, failure to phenotypically identify trisomic types has been due possibly to the heterozygosity of their source (Price and Ross, 1957). Heterozygous segregation and perhaps heterosis might mark the distinguishable

effect of the extra chromosome on phenotype (Lin and Ross, 1969a).

### 2.2.3 Cytology of trisomics

The cytological behaviour of the extra chromosome in trisomics has an important bearing on the transmission rates of the extra chromosome to the next generation and on the genetic segregation of marker genes.

Since in a primary trisomic one chromosome is represented in three homologous sets, they usually form a trivalent at the pachytene stage of meiosis. Rhoades and McClintock (1935) studied the pairing behaviour of the extra chromosome at pachytene in maize trisomics and reported that of the three chromosomes involved in trivalent formation, any two of them pair at a given point along the length of the chromosome and the third one may pair non-homologously. Rick and Barton (1954) and Rick et al. (1964) observed similar pairing behaviour of extra chromosome in tomato trisomics. The extra chromosomes of seven of the ten trisomics of Sorghum (Poon and Wu, 1967) were identified at the pachytene stage.

Identification of extra chromosome at pachytene is often difficult and less rewarding due to non-homologous pairing of extra chromosome with other chromosomes in the complement as reported in Sorghum (Poon and Wu, 1967;

Venkateswarlu and Reddi, 1968) and Solanum (Vogt and Rowe, 1968). The identification of the trisomics is therefore done at later stages of prophase-I or at the early part of metaphase-I of meiosis. In primary trisomics, pachytene cells with a univalent are extremely rare. Kinset (1943) found only 2 - 4 per cent of pachytene cells with univalents in the primary trisomics of maize. The frequency of cells with univalents increases due to failure of chiasma formation at pachytene and chiasma terminalization at later stages of meiosis (Manga, 1976). At diakinesis and metaphase I, chromosomes are found associated as n-1 bivalents + 1 trivalent or n bivalents + 1 univalent or n-1 bivalents + 3 univalents.

The frequency of trivalents varies for the trisomics of different species and for different trisomics within a species. Kinset (1943) in maize, Rick and Barton (1954) in tomato, Tsuchiya (1960) in barley, Goodspeed and Avery (1939) in Nicotiana, Virmani and Gill (1971) and Minocha et al. (1976) in pearl millet reported that the frequency of trivalents are more in the trisomics for longer chromosomes. In any case primary trisomics are unambiguously identified by the presence of a normal trivalent. Due to the extent of pachytene pairing and the magnitude of chiasma formation, the types of trivalent configurations also vary (Khush, 1973).

At the time of disjunction during anaphase-I, two members of the trivalents go to one pole and the third to

the other pole, but in rare cases all the three might go to the same pole resulting in the production of n-1 and n+2 spores. However, univalent as an extra chromosome is known to behave irregularly ( Minocha et al., 1976).

Due to the irregular behaviour of univalent chromosome, the proportion of n+1 spores will be lower than the expected value of 50 per cent. The deviation from the 1:1 ratio for n and n+1 spores is governed by the degree of irregularity. The trisomics for the longer chromosomes form trivalents more frequently than those for the shorter ones and so there is little chance for the extra chromosome to lag and misdivide. Consequently, n + 1 spores are produced at a frequency which is only slightly lower than expected. In maize, the frequency of production of n + 1 spores for longer chromosomes and shorter chromosomes are 46 and 29 per cent respectively (Einset, 1943). As a result of the irregular behaviour of the univalent, the spored with n + telocentric and n + isochromosome are also produced at a low frequency.

### 2.2.4 Transmission of the extra chromosome in trisomics

Theoretically it is expected that an individual with 2n + 1 chromosomes should produce n and n + 1 gametes in equal frequency on both female and male sides. Similarly, one should be able to obtain 2n + 1 and 2n individuals in equal frequency from (2n + 1) x 2n as well as 2n x (2n + 1) crosses. Furthermore, 2n + 1 individuals when selfed should

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yield 2n 2n + 1, and 2n + 2 progeny in the ratio of 1:2:1. However, these expectations are never realised and the percentage of 2n + 1 individuals in the progeny of a (2n + 1) x 2n cross (transmission of the extra chromosome through the female) is considerably lower than 50 per cent. It is even lower in the 2n x (2n + 1) crosses (transmission of the extra chromosome through the male) and in most cases one fails to recover any 2n + 1 individuals from such a cross. The reduction in the number of 2n + 1 individuals to less than the expected 50 per cent has been attributed to several causes (Khush, 1973).

In maize, a strong correlation exists between chromosome length, univalent frequency at metaphase I, and transmission frequency of the extra chromosome (Einset, 1943); however, this does not hold true for the trisomics of tomato. In the trisomics for the longer chromosomes of the tomato (chromosomes 1, 2 and 3) the univalent frequency is lowest as expected. However, the trisomics of these chromosomes also show the lowest rates of transmission. The trisomes for the shorter chromosomes of the complement (chromosomes 7 to 12) have the higher frequency of univalents, and their transmission rates are also higher (Rick and Barton, 1954). Further, it is interesting to note that in Datura no correlation exists between the length of the extra chromosome and the transmission rates of the trisomics.

The transmission of an extra chromosome through the male, if any, is extremely low compared to that through the female; and hence the tetrasomics rarely appear in the progenies of selfed trisomics. Interestingly, all the trisomics of common wheat are transmitted through the male, although at a lower frequency than through the female. By virtue of this transmission, it has been possible to establish all the 21 tetrasomics in this species.

The female gametophytes are known to tolerate genetic and chromosomal imbalance, as well as various types of environmental stresses, to a greater extent than male gameotyphytes. The reasons for the differential transmission rates of the extra chromosome through the male and fenale should therefore be sought in the differential morphology and development of the male and female gametophytes.

One of the unique and useful features of the breeding behaviour of all kinds of trisomics is that of the appearance of non-parental but related trisomics, and a few unrelated 2n + 1 types in addition to parental trisomics in their progenies. Primary trisomics of Datura and Lotus pedunculatus have been reported to produce some unrelated trisomics in their progenies with a very low frequency (Blakeslee and Avery, 1938; Chen and Grant, 1968). Ashu Bhan et al. (1989) have reported the production of trisomics for a nucleolar chromosome in the progeny of a primary trisomic

of Plantago lagopus for a non-nucleolar chromosome. Why the presence of an extra chromosome increases the rate of spontaneous occurrence of other trisomics is not clear. The increase probably is brought about by the extra chromosome causing interference in the regular mechanics of meiotic division to increase the rate of non-disjunction (Khush, 1973).

### 2.2.5 Genetic segregation in trisomics

Genes situated in trisomic trivalents generally display a mode of inheritance other than that of genes located in normal bivalents. The genetic ratios for the genes which are located on the extra chromosome are modified in the segregating progenies. This modified ratio technique has been used in several plant species. The genotypes Aaa (simplex) and AAa (duplex) segregate with respect to those genes. The expected segregation ratios are calculated on the basis of three different modes of segregation such as, i) random chromosome segregation (Muller, 1914), ii) random chromatid segregation (Haldane, 1930) and iii) maximum equational segregation (Burnham, 1962).

To determine the gene-chromosome relationships the modified ratio technique has been used in locating genes on specific chromosomes (Person, 1956; Hermsen, 1970).

Primary trisomics provide an invaluable tool for testing the independence of linkage groups and for assigning

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linkage groups to particular chromosomes. Since there are three homologous chromosomes in a primary trisomic, the genetic ratio of segregating progenies for genes that are located on this chromosome, are very different from 3:1 ratio obtained in $F_2$ generations of a normal disomic hybrid. Depending upon the genotype of the trisomic, the $F_1$ individual obtained from a cross either with a dominant marker gene or a recessive gene, produces different types of gametes on the basis of chromosomal constitution and allelic constitution. The segregation ratios expected in $F_2$ and backcross are as follows:

| Mode of segregation | | Trisomic genotypes | Trisomic ratios for * entire progeny | |
|---|---|---|---|---|
| | | | 50% transmission | 33.3% transmission |
| $F_2$ ratio | Chromosome segregation | AAa (Duplex) | 17:1 | 12.5 : 1 |
| | | Aaa (Simplex) | 2:1 | 1.7 : 1 |
| | Random chromatid segregation | AAa (Duplex) | 14:1 | 11.27 : 1 |
| | | Aaa (Simplex) | 29:16 | 1. : 1.6 |
| BC ratio | Chromosome segregation | AAa (Duplex) | 5:1 | 3.5 : 1 |
| | | Aaa (Simplex) | 1:1 | 1 : 1.25 |
| | Random chromatid segregation | AAa (Duplex) | 4:1 | 3.1 : 1 |
| | | Aaa (Simplex) | 7:8 | 1 : 0.73 |

* Male transmission of n + 1 gamete is considered as zero.

The above ratios are further modified depending upon the transmission rate of n + 1 gamete in the female and male side. However, the n + 1 gametes are rarely transmitted through male leading to the production of functional male gametes in the proportion of 2:1, in case of duplex (AAa) genotype and 1:2 in case of simplex (Aaa) genotype. Hence, due to the actual transmission frequency of n + 1 gametes through female, there will be deviations in the trisomic ratios obtained as above (Khush, 1973). These ratios are compared with the normal disomic $F_2$ (3:1) and back cross (1:1) ratios to arrive at the location of the genes to specific chromosomes.

With two loci, 24 different trisomic genotypes are possible of which eight are homozygous at both loci, four heterozygous at both loci and 12 heterozygous at one and homozygous at the other locus (Hermsen, 1970).

It is quite evident that the localization of genes for a character becomes more complicated if more than one locus is involved and if the trisomic series is heterozygous at the locus or loci under investigation. In locating major genes it is preferred to use a trisomic series which is homozygous for the gene (s) determining the character (Hermsen, 1970).

If a character is based on many genes with minor effects (quantitative character) the above mentioned clear-cut ratios may no longer be expected. In this case a whole

chromosome has to be taken as a unit and this means that only the effect of an extra chromosome on the character involved can be determined (Hermsen, 1970).

## 2.3 Trisomics of rice

### 2.3.1 Isolation and identification of trisomics

During the first symposium on Rice Genetics and Cytogenetics held at IRRI in 1963, one of the priority research areas identified in rice genetics was the determination of chromosome-linkage group relationships by primary trisomic technique (Kihara, 1964). Though the trisomic method is the most effective way to test the independence of linkage groups, only in past two decades attempts have been made to develop complete set of primary trisomics in rice.

Nayar (1973) has reviewed in detail the work done by several rice cytogeneticists on the trisomics of rice. Primary trisomics of rice have been obtained from the selfed seeds of autotriploids in most of the cases. The progeny of crosses between triploids and diploids have also been employed to isolate trisomic lines.

Little is known about the secondary or tertiary trisomics of rice. Some of the 2n + 1 plants found in the progenies of translocation heterozygotes must have been tertiary trisomics (Khush, 1974).

The efforts done by various rice scientists towards obtaining trisomic progenies have been presented chronologically as follows:

a) Primary trisomics

| Authority | Source of trisomics | Somatic chromosome number | No. of trisomics obtained |
|---|---|---|---|
| 1 | 2 | 3 | 4 |
| Nakamori (1932) | Selfing of a triploid(3n) plant | 25 - 29 | 8 |
| Ichijima (1934) | Artificial induction of mutations | 25 | Few |
| Ramanujam(1937) | Selfed seeds of a triploid | 24 - 30 | 9 |
| Parthasarathy (1938) | X-ray irradiated seeds | 25 | 2 named "stumpy" and "beaked" |
| Jones and Longley (1941) | Progenies of asynaptic and partially sterile plants | 25 - 26 | Few |
| Yunoki and Masuyama(1945) | Progenies of triploids | 25 | 6 |
| Jones (1952) | Progenies of asynaptic and partially sterile plants | 25 - 26 | Few |
| Nagamatsu(1955) and Nishimura(1957) | Nagasaki atomic bomb explosion affected plant progenies | 25 | Few |

| 1 | 2 | 3 | 4 |
|---|---|---|---|
| Karibasappa (1961) | 3n x 2n cross | 26<br>2n + 1 + 1<br>or 2n + 2 | Few |
| Jachuk (1963) | Interplanting stubbles of a triploid plant( T-1242) among diploid plants | 25 - 26 and a tetra-ploid (4n) | Few |
| Katayama(1963) | i. Progenies of two completely asynaptic plants and two partially asynaptic plants | 25 - 26<br>2n + 1<br>and<br>2n + 1 + 1 | Few |
| | ii. Progenies of auto triplo-ids open pollinated as well as 3n x 2n | 25 - 33 | Few |
| Sen (1965) | A natural auto triploid plant of T.1242 x diploids | 25 | 5 |
| Katayama(1966) | Progeny of partially asynaptic plant | 25 | Few |
| Koga et al.(1967) and Koga and Nagamatsu(1967) | 3n x 2n progenies | 25 | Few |
| Hu (1968) | Progeny of a 3n (triploid) plants of a Taiwanese indica variety Kehtze | 25 | A complete set of 12 primary trisomics of indica rice but without cytological confirmation |

| 1 | 2 | 3 | 4 |
|---|---|---|---|
| Watanabe et al. (1969) | Self pollination of triploids as well as 3n x 2n progenies | 25 - 28 | Few |
| Iwata et al. (1970) | Progenies of triploids and tertiary trisomics of four japonica cultivars | 25 | A complete set of 12 primary trisomics of japonica rice |
| Khan and Rutger (1974) and Khan (1974) | Progenies of a triploid of a japonica variety CS-M3 | 25 | A complete set of 12 primary trisomics with cytological confirmation of extra chromosome |
| Watanabe and Koga (1975) | 3n x 2n crosses of a japonica variety "Norin-8" | 25 | A complete set of 12 trisomics designated by the letters A to L. Extra chromosomes were identified with the application of Nishimura's (1961) translocation testers |
| Sur (1975) and Khush et al. (1984) | Triploid progenies of indica varieties IR-841-36-2, IR-36 and IR-3478 | 25 - 29<br>2n+1<br>2n+1+1 and<br>2n+1+1+1<br>2n+1+1+1+1<br>2n+1+1+1+1+1<br>and 2n+2 | A complete set of 12 primary trisomics of indica rice confirmed cytologically at pachytene stage of meiosis. The major distinguishing features of each pachytene chromosome were redescribed |

| 1 | 2 | 3 | 4 |
|---|---|---|---|
| Sen (1977) Jena (1982) Khush and Misra(1983) Misra et al. (1986) | Progeny of a spontaneous autotriploid of an indica cultivar Sona | 25 - 28 (double and triple trisomics) | A complete set of 12 primary trisomics of indica rice. Extra chromosome confirmed cytologically at pachytene |
| Zhang and Zhu (1986) and Zhang et al. (1987) | Progenies of triploides.The triploids were obtained by crossing induced tetraploids of an indica variety Guangluai 4 with diploids | 25 - 35 | A complete set of 12 primary trisomics of indica rice. Only outstanding morphological features have been enumerated |
| Ishiki(1987) | Progenies of autotriploid of African rice O.glaberrima The autotriploid was produced by crossing a colchicine induced autotetraploid with a diploid | 25 | A complete set of primary trisomics of African cultivated rice Oryza glaberrima Steud I |
| Yasui et al. (1989) | A desynaptic mutant MM-15 crossed with a japonica variety Kinmaze | 25 - 26 | A complete set of primary trisomics of japonica rice as judged from morphological features. Cytological examination was obtained in the $F_1$ population |

| 1 | 2 | 3 | 4 |
|---|---|---|---|
| b. Secondary trisomics | | | |
| Khush et al. (1991) | From the progenies of IR-36 primary trisomics | 24 + an extra iso-chromosome | 14 secondary trisomics have been identified representing all the 12 rice chromosomes on the basis of morphological and cytological characteristics |

The first complete set of 12 trisomics in indica rice was obtained by Hu (1968), in the progeny of a triploid plant of a Taiwanese indica variety, Kehtze. In his study, 37 per cent of the progeny survived to maturity and of these, 30 per cent were trisomics and 44 per cent had 2n = 26 chromosomes. Nearly half the progeny of these 2n + 2 plants also turned out to be trisomics. The 12 trisomic plants were identified on the basis of their vigour, habit, stature, rolling of leaves, panicle and spikelet shape and awning. The transmission frequencies of the extra chromosomes upon selfing the 12 trisomics varied from 27 to 48 per cent. The cytological examination of the extra chromosomes was however, not done.

The credit for developing a complete set of 12 primary trisomics for the first time in japonica rice goes

to Iwata et al. (1970). These trisomics have been isolated on the basis of morphological features from four japonica cultivars viz., 'Aikoku', 'Asakaze', 'Benisengoku' and 'Norin-39'. Of the 12 trisomics classified and designated as A to L types (Table 2) 11 trisomics were developed from the progenies of triploids and the twelfth from those of tertiary trisomics. Iwata et al. (1970)have identified the extra chromosome to be different from each other in only five types of trisomics by use of reciprocal translocation testers. Later the chromosomes K1 to K12 were designated in descending order of chromosome length by karyotype analysis of somatic pro-metaphase chromosomes (Kurata and Omura, 1978).

Kurata et al. (1981) further studied 12 trisomic types, A to L developed by Iwata et al. (1970) for cytological evidence. Through karyotype analysis of these trisomics they found that four types, G, I, J and K had the same extra chromosome as already represented by the other types and three types having one of the three longest chromosomes, K1, K2 and K3, as an extra chromosome had not been found at all in the strains. Hence, the three missing trisomic types M, N and O, having the chromosomes, K3, K2 and K1 as extra chromosomes were obtained from progenies of a cross between triploid and diploid Nipponbare (Iwata and Omura, 1984a and 1984b).

Watanabe and Koga (1975) successfully established 12 primary trisomics of *japonica* rice from the progenies of 3n x 2n crosses of *japonica* variety 'Norin-8' and designated them by the letters A to L with the different names based on the most conspicuous characters. The authors further identified the extra chromosome of each trisomic with the application of Nishimura's translocation testers (Nishimura, 1961).

Sur (1975) and Khush *et al.* (1984) have isolated a complete set of 12 primary trisomics of *indica* rice from the progenies of spontaneous triploids. These trisomics were transferred by backcrossing to the genetic background of IR 36, a widely cultivated high yielding rice variety. The 12 trisomics designated by the numbers 1 to 12 were distinct morphologically and were named on the basis of their outstanding features (Table 2). Of these, 11 trisomics can be identified morphologically from one another and from diploids but triplo-11 was difficult to distinguish from diploid sibs. The extra chromosome of each trisomic was identified cytologically at pachytene stage of meiosis, and the chromosomes were numbered in decreasing order of length adopting the chromosome numbering system proposed by Shastry *et al.* (1960). Khush *et al.* (1984) observed that trisomics for the long chromosomes were more distinct from the diploid as well as from each other. They were also slower in growth. This is expected as the longer chromosomes

cause more imbalance than the shorter ones. The authors have redescribed the major distinguishing features of each pachytene chromosome.

At CRRI, Cuttack, India (Sen, 1977; Jena, 1982 and Misra et al., 1986) a complete set of 12 primary trisomics of indica rice has been isolated from the progeny of a spontaneous autotriploid of the cultivar Sona. These trisomics as designated by the numbers 1 to 12 were morphologically different from each other as well as from their disomic parent. Conspicuous morphological differences were observed with regard to growth habit, panicle length, leaf shape and size, ligule length, spikelet size, presence of awn and size of anther and pistil. The trisomics showed pollen and spikelet sterility ranging from 0 to 100 per cent. The extra chromosomes were identified from the pachytene chromosome pairing of all the trisomics. The salient landmarks of each pachytene chromosome helped in their identification. The extra chromosomes were numbered in descending order of length (Shastry et al., 1960), and accordingly these primary trisomics were designated as triplo-1 to triplo-12.

Thus, IRRI in the Philippines and the Central Rice Research Institute (CRRI) in India have independently established all the 12 primary trisomics in the background of two different indica varieties, IR 36 and Sona. A complete correspondence between these two sets of trisomics has been observed (Khush and Misra, 1983).

Primary trisomics of an *indica* variety IR-36 and of a *japonica* variety Nipponbare have been employed for associating linkage groups of rice with respective chromosomes by most of the rice workers. The extra chromosomes of the IR 36 trisomics were identified at pachytene stage of meiosis and that of the Nipponbare trisomics were identified at mitotic prophase. But the results of these two studies, when compared, linkage group-chromosome associations did not agree in all cases (Anonymous, 1990b).

Meanwhile, on the basis of discussions among geneticists in Japan, IRRI and elsewhere, it was decided to hold an International Rice Genetics Symposium, which was held at IRRI in May, 1985. Problems related to gene symbolization and chromosome numbering system in rice were discussed thoroughly during the symposium. It has been recommended that chromosome numbering system based on the length of pachytene stage of meiosis as proposed by Shastry *et al.* (1960) should be followed. Further, a top priority was given to resolve the discrepancies in the chromosome-linkage-group associations determined by Khush *et al.* (1984) and Iwata and Omura (1984a). It was decided that the IR-36 trisomics should be re-examined to see if any error was made in the identification of extra chromosomes of some trisomics (Anonymous, 1990b). On the basis of observations, mutual discussions and taking the re-examination reports by Kurata, (1988) and Wu and Chung (1988) into consideration, it was

decided to change the identification of extra chromosomes of four trisomics. It was agreed that the extra chromosome of triplo 4 is number 3, that of triplo 3 is number 6, that of triplo 6 is number 12 and that of triplo 12 is number 4. The designations of the four trisomics have been consequently changed (Anonymous, 1990b). The old and new designations are presented in Table 2.

Singh and Khush (1978) made an attempt to compare morphological characters of IR-36 trisomic set of Sur (1975) with those established by Hu (1968), Iwata et al. (1970) and Watanabe and Koga (1975). This comparison helped to understand the effect of extra chromosome on morphology of the plants in different rice varieties. Heterogeneity of genetic background has apparently caused considerable differences in the morphological expression of trisomics. When all the trisomics of above authors are critically and closely observed, it appeared that the authors depending upon their observation had given different names (Table 2). The Table 2 presents the latest comparison.

The comparison shown in Table 2 clearly elucidates that, extra chromosome in trisomic condition generally exhibits the same morphological modification regardless of the genetic background. Here, barley could be cited as an example. The trisomics established in wild barley (Hordeum spontaneum) resembled the trisomic in cultivated barley

Table 2. Comparison among different sets of trisomics of rice

| Trisomics | Khush et al.(1984) Misra et al.(1986) indica varieties IR-36 and Sona respectively | Hu (1968) (Taiwanese indica variety Kehtze) | Iwata and Omura (1984 a and b) (Japonica cultivar Nipponbare) | Watanabe and Koga (1975) (japonica variety Norin-8) |
|---|---|---|---|---|
| New designation (Anon.,1990 b) | Old designation | | | |
| Triplo 1 | Triplo 1 (Grassy) | - | O (Grassy) | I (Drooping) |
| Triplo 2 | Triplo 2 (Dwarf) | - | D (Erectoides) | - |
| Triplo 6 | Triplo 3 (AWned) | 5 (Awned) | B (AWned) | E (AWned) |
| Triplo 3 | Triplo 4 (Sterile) | - | M (Sterile) | J (Sterile) |
| Triplo 5 | Triplo 5 (tWisted leaf) | 3 (Adherent panicle) | L (Short panicle) | C (Compact ear) |
| Triplo 12 | Triplo 6 (Bushy) | 2 (Bushy) | A (Pale) | B (Large grain) |
| Triplo 7 | Triplo 7 (Narrow leaf) | 8 (Slender grain) | F (Rolled leaf) | K (Long grain) |
| Triplo 8 | Triplo 8 (Rolled leaf) | 7 (Rolled leaf) | - | D (Erect) |
| Triplo 9 | Triplo 9 (Stout) | 4 (Robust) | H (Large grain) | F (Shattering) |
| Triplo 10 | Triplo 10 (Short grain) | - | C (Small grain) | A (Slender) |
| Triplo 11 | Triplo 11 (Pseudo-normal) | 11(Pseudonormal) | G (Pseudo normal) | H (Pseudonormal) |
| Triplo 4 | Triplo 12 (Tall) | 1 (Spreading)<br>6 (Fine stature)<br>9 (Short stature)<br>10 (Vigorous)<br>12 (Long grain) | E (Spreading)<br>N (Smooth glume) | -<br>G (Dirty glume)<br>L (Subnormal) |

(Tsuchiya, 1960, 1967). All the 12 primary trisomics of African cultivated rice Oryza glaberrima Steud.I., have been isolated by Ishiki (1987). As the article is in Japanese language, the details about these trisomics are not known but it may be conjectured that these trisomics also must have been showing the same morphological modifications as in Oryza sativa L. trisomics.

A complete set of 12 primary trisomics have also been isolated from the colchicine doubled indica variety Guangluai 4, in China (Zhang and Zhu, 1986 and Zhang et al., 1987). The morphology of these trisomics were similar to those of other indica trisomics.

Yasui et al. (1989) have attempted to obtain trisomic plants by using a desynaptic mutant, MM-15, which was induced by a treatment of fertilized egg cells with N-methyl-N-nitroso urea (MNu) in the japonica variety Kinmaze (Kitada and Omura, 1983). Strain MM-15 homozygous for a recessive desynapsis gene and nearly completely self-sterile was crossed with Kinmaze and all the $F_1$ seeds obtained were sown on agar medium. The possible 12 types of trisomic plants obtained in the $F_1$ population were judged from morphological features and confirmed cytologically to have 25 chromosomes. It is interesting to note that these 12 trisomics were in close agreement with those reported by Iwata et al. (1970) and Iwata and Omura (1984a & b) and hence, they were named accordingly. The trisomic series

so far established in *indica* and *japonica* rice varieties have been obtained by screening the progeny of crosses between autotriploid and diploid plants. Thus, this study by Yasui *et al.* (1989) is the first report wherein a new trisomic series has been raised from the progeny of a desynaptic mutant. These mutants are a useful source of trisomic plants (Khush, 1973). The desynapsis gene can be transferred to other varieties by hybridisation.

Shu and Zhang (1986) proposed that the tissue culture method can be used for producing and propagating primary trisomics in rice.

Wang *et al.* (1989) carried out anther culture of the trisomic plants of *japonica* variety Nipponbare in order to obtain x + 1 plants of rice. The regenerated plants were grown in pots in green house. At 5-leaf stage, their morphological features and root-tip chromosomes were examined. A total of 131 plants were examined and they showed chromosome numbers ranging from 2n = 13 to 2n = 73. Plants with 2n = 13, called "disomic haploid" (x + 1) were detected in four trisomics, viz., type A, B, C and H. Plants with 2n = 26 were found in types B, C, D and H, which might have resulted from chromosome doubling in cells with 2n = 13. A few other aneuploids including a monosomic (2n = 23) and 2n = 73 plant were also obtained.

Khush et al. (1991) have isolated secondary trisomics in the background of IR-36 from the progenies of primary trisomics. These trisomics have the normal chromosome complement and an extra isochromosome, they have identified 14 secondary trisomics representing all the 12 rice chromosomes on the basis of morphological and cytological characteristics. The isochromosomes in the trisomics have been identified on the basis of pachytene analysis. These secondary trisomics have been crossed with marker genes. Genetic segregations will help delimit the arm locations of the markers, positions of centromeres and orientations of the linkage groups as proposed by the authors. These trisomics are also being used to locate the centromeres on the RFLP map.

### 2.3.2 Cytology of rice trisomics

The true trisomic nature of an individual is finally established by cytological studies. Since the somatic chromosomes of rice are very small lacking any distinguishing features, the identity of each chromosome is comparatively easier to establish only at pachytene stage of meiosis (Singh and Khush, 1978). A high degree of attenuation of the chromosome, homologous pairing and juxtaposition of the homologous regions render pachytene analysis an ideal tool for the study of not only chromosome morphology but also of chromosome pairing (Shastry, 1964). The chromosomes are fairly long at the pachytene stage of meiosis in the PMCs.

The length of the individual chromosomes or their relative length, their arm ratios, heterochromatic nature of the arms, chromomeric pattern, their role in organizing nucleolus, etc., have been quite helpful in identifying the individual chromosomes without any ambiguity (Sharma, 1986).

Identification of extra chromosomes is one of the most difficult aspects of trisomic analysis in rice (Oka and Wu, 1988) and therefore, cytological study of the extra chromosome in trisomics of rice at pachytene stage has not been done intensively as it has been done in maize, tomato and Datura. Routine analysis of diakinesis and metaphase-I chromosomes have been done mainly to confirm the presence of an extra chromosome.

Parthasarathy (1938) from the cytological study of "stumpy" trisomics reported a chain of five chromosomes in metaphase-I of PMCs and inferred that the extra chromosome was composed of segments derived from the non-homologous chromosomes of the haploid set. But the "beaked" trisomic was of primary type, because the extra chromosome was homologous to one of the haploid set.

Sen (1965) through pachytene chromosome analysis identified the extra chromosome of four out of five trisomics investigated by him and designated them from A to D. The author attempted to associate the diagnostic morphological characters with the cytologically analysed extra

chromosomes in the trisomics. Iwata et al. (1970) identified the extra chromosome of five out of 12 types of trisomics utilizing Nishimura's (1961) translocation testers.

Sen (1977) identified three trisomics precisely through the study of pachytene chromosomes. The author stressed that such studies need further confirmation since the presence of non-homologous pairing of constituent univalents at pachytene in a trivalent association might introduce certain errors.

Jena (1982) identified the extra chromosomes at somatic metaphase from leaf tip and at pachytene stage of meiosis as per the chromosome morphology of the disomic 'Sona' variety. However, the non-homologous pairing of the three homologous chromosomes forming the trivalents, the stretching of the extra chromosome during trivalent formation with its homologs, the absence of chromomeric details on the arms of the univalents not involved in pairing and the stickiness of chromosome ends were found to impose restriction on the identification of extra chromosome with fair degree of certainty (Jena, 1982).

Misra et al. (1986) reported that the salient landmarks of each chromosome helped them in the identification of each trisomic. However, they reported that a disturbing feature of the pachytene chromosome configuration in the trisomics was the formation of a triradial

configuration. Further they reported the difficulty in identifying the extra chromosome remaining as a univalent because of the lack of chromomeric expression. The authors found all the three homologous chromosomes to be paired for a considerable part of their length. In the case of non-homologous pairing of the chromosomes, the extra chromosomes were identified following the chromomeric pattern and centromeric position.

Sur (1975) and Khush et al. (1984) identified all the 12 primary trisomics through pachytene chromosome morphology and their pairing and formation of trivalent association. They reported that the extra, univalent chromosome frequently paired internally and lost identifiable chromomeric patterns. As the trivalents showed non-homologous pairing the identification had to be based upon homologously paired regions. They further reported that the change of pairing partners can occur at any place along the length of the chromosome, but more frequently at the centromere, and hence centromere has to be directly identified from the homologously paired regions of trivalents. Due to the differential condensation of the long and short arms, they frequently observed end to end pairing.

Sometimes in rice trisomics a non-homologous association is observed wherein both the arms are seen paired. Due to this type of pairing, a primary trisomic may be mistaken for a secondary trisomic

(Singh and Khush, 1978). However, close observation of several cells helps to establish the true nature of the trisomic.

The non-homologous association of extra chromosome at pachytene stage has also been observed in maize, tomato, and sorghum with various frequencies (Khush, 1973).

Kurata et al. (1981) were probably the first who tried to identify the extra chromosomes of 12 trisomic strains, by the karyotype analysis of somatic prometaphase chromosomes. Since the morphology of pachytene trivalents were fairly intricate, they used the somatic nuclei for the determination of extra chromosomes. While examining the 12 primary trisomics of japonica rice described by Iwata et al. (1970), four strains, B, C, E and H types were decided to have individual extra chromosomes $K_6$, $K_{12}$, $K_4$ and $K_{10}$ respectively. In well conditioned nuclei, $K_5$, $K_7$, $K_9$ and $K_{11}$ extra chromosomes were found to be carried by the A, D, L and F type trisomics respectively. Extra chromosome $K_8$ was found present in G, I, J and K type trisomics while three kinds of extra chromosomes ($K_1$, $K_2$, $K_3$) were not found in these 12 strains.

Iwata et al. (1984a) crossed four trisomics A, C, D and I with 14 reciprocal translocation lines for the identification of the extra chromosome of each trisomics. Chromosome configuration of a pentavalent and 10 bivalents,

10 II + 1 V, was observed in the $F_1$ hybrids between trisomics A and three reciprocal translocation lines involving chromosome 4, RT 1-4, RT 3-4a and RT 3-4b indicating that the extra chromosome of trisomic A is chromosome 4. Pentavalent formation observed in PMCs of $F_1$ hybrids between trisomic C and five reciprocal translocation lines involving chromosome 7 showed that the extra chromosome of trisomic C was chromosome 7. A similar chromosomal configuration helped to indicate that the extra chromosomes of trisomics D and I were chromosomes 12 and 9 respectively.

Chung and Wu (1987) carried out the karyotype analysis of IR-36 and two trisomic lines of rice, at the pachytene and prometaphase stages. The authors identified 12 pairs of chromosomes in IR-36 and classified them according to their absolute and relative lengths, long arm/short arm ratio and distribution patterns of chromomeres and heterochromatin. The trisomic lines were similar to IR-36 but each had an extra chromosome.

Wu and Chung (1988) carried out pachytene analysis of six IR-36 triplo lines for the identification of extra chromosome, and have designated the extra chromosomes of the six triplo lines as follows: Triplo-3-chromosome 6, Triplo-4-chromosome-2, Triplo-6-chromosome 12, Triplo-8-chromosome-8, Triplo-11-chromosome 11 and Triplo-12-chromosome 4.

Chung and Wu (1988) during the identification work of the IR-36 triplo series have detected structural changes involving extra chromosome in triplos 1 and 12. Subsequently, the occurrence of a pentavalent at diakinesis was recognized in triplos 3, 5, 6 and 7, and pairing of the extra chromosome with "non-homologous" bivalents was also observed in several lines. They have concluded that Triplos 3, 5, 6, 7 and 11 are populations segregating for different chromosome types and a part of the segregants are tertiary trisomics. Probably, the chromosomal imbalance in a trisomic plant some tines provokes structural changes. Primary, secondary and tertiary trisomics are inter-convertible eventually, even though rarely (Khush, 1973). In the IR-36 triplo series, Sur (1975) reported that "both related and unrelated non-parental trisomics were found at a low frequency (0.26%) in the progeny of some primary trisomics". A tertiary trisomic can be derived from a primary trisomic which has a background heterozygous for a reciprocal translocation. In fact, a ring or chain of four indicating the presence of a reciprocal translocation was observed in Triplos 3, 5, 6, 8, 9 and 10. The authors thus have given a warning to the workers who are engaged in the maintenance and use for gene analysis of the triplo series suggesting that they will have to carefully check up segregants with tertiary trisomics and to select the original primary trisomics. However, during the meetings of the coordinating committee of Rice Genetics Cooperative held at IRRI in August 1989 at Tsukuba, Japan, attended by

Drs. Y.Futsuhara, G.S. Khush, T.Kinoshita, N. Kurata, H.I.Oka, T.Takeda and H.I. Wu, the new results and observations shown by Chung and Wu (1988) were reviewed and it was not adopted by the group of geneticists as there was no supporting genetic evidence.

Kurata (1988) identified the trivalent chromosomes of IR-36 triplo plants and his results gave a supporting evidence for the cytological identification that the extra chromosomes of triplos 3, 4 and 12 are chromosomes 6, 3 and 4, respectively. However, the author stated that "when a trivalent chromosome was identified without complete identification of all other bivalents in the nucleus, and when the judgement was not reconfirmed by repeated observations in a number of nuclei, the result could not be considered very reliable. Thus, the author has suggested for re-examination of triplos 5 and 7 and stated that identification as to triplos 6, 8 and 11 in the investigation was tentative.

A trisomic plant having an extra chromosome, when selfed or pollinated by a diploid produces progenies with and without the extra chromosome. Observing the progenies of the trisomic series of cultivar Guangluai 4, Zhang (1988) has found a unique phenomenon that in a few of diploid plants with 2n = 24, a part or whole of the extra chromosome was duplicated and attached longitudinally to its corresponding bivalent. Such plants showed characteristics similar to those of the

trisomic plants. Pachytene analysis revealed that the chromosomes with repeated segments were longer and had more Giemsa bands and chromomeres than normal ones. The author further reported that probably, the segmental repetition is due to the occurrence of breaks at different points in two of three homologous chromosomes, their reciprocal translocation resulting in tandem fusion of partly homologous segments, and their duplication. The author has similarly observed this phenomenon in triplos 5, 8 and 9. Plants with repeated segments had morphological characteristics of corresponding trisomics, namely, the diploid with repeated segments of chromosome 5 had fine hairs on leaf surface similarly as triplo-5, that with chromosome 8 had rolled leaves similarly as triplo-8, and that with chromosome 9 appeared as stout similarly as triplo-9.

Chromosome analysis of trisomics began with a search for mitotic trisomics and meiotic trivalents. Trivalents had complicated morphology in most nuclei and were difficult to identify without the simultaneous detection of all other 11 bivalent chromosomes. In mitosis, chromosomes in the trisomic condition were easily detected comparatively (Kurata, 1986). The condensation pattern analysis by imaging methods (Fukui and Mukai, 1988; Fukui et al., 1988 and Fukui and Iijima, 1991) as well as the application of in situ hybridization methods to chromosome analysis (Kurata, 1986) would be an asset for the identification of an extra chromosome of a trisomic plant.

### 2.3.3 Transmission rate of the extra chromosome

Hu (1968) investigated the transmission rate of extra chromosome of indica trisomics upon self pollination and reported that the transmission of extra chromosome varied between 26.5 and 47.5 per cent depending upon the chromosome involved.

In the primary trisomics of indica series, Sur (1975) reported the range of transmission of extra chromosome between 20.9 and 39.4 per cent with an average rate of 29.8 per cent. The author reported higher transmission of extra chromosome among vigorous seedlings than weak seedlings.

Sen (1977) studied only six trisomics of indica series for examining the transmission rate of extra chromosome. He observed that the transmission rate of the smallest chromosomes were high compared to the longer ones.

Sadananda (1981) studied some of indica as well as japonica trisomics for the relative recovery of trisomics in selfed progenies. In the indica series, triplo-2 and triplo-8 showed the highest (33.0) and lowest (2.03) percentage of recovery respectively. Similarly in japonica series, the recovery was the highest (36.0%) for triplo-4 and for triplo-12 it was the lowest (12.0%).

Jena (1982) and Misra et al. (1986) raised the selfed progenies of different trisomics to study the transmission

rate of extra chromosome based on plant morphology. The transmission frequency ranged from 4.5 per cent in triplo-2 to 50 per cent in triplo-12. There was no seed set in triplo-4 and so it could not be studied. Some non-parental type of trisomics were observed only in the progeny of triplo-11 (0.7%). The authors also noticed that the transmission rate of trisomy for longer chromosomes was lower than that for shorter chromosomes.

Fairly high rate of transmission of the extra chromosome through ovule was found in IR-36 trisomics by Khush et al. (1984). Transmission frequency for triplos 3, 6, 11 and 12 were 37.9, 37.1, 39.7 and 43.9 per cent respectively. Transmission rates were low for triplos 1 and 4 (15.5 and 17.8%). The female transmission rates were calculated from the $F_2$ or back cross progenies of trisomics and marker genes and thus the populations were highly heterozygous. It was believed that heterozygosity might have contributed to the higher transmission frequency of the extra chromosome. As pointed out by Khush (1973), transmission rates are affected by genetic background also.

Sur (1975) also reported that through pollen, the transmission rate for nucleolar chromosome-9 was 15 per cent whereas it was 3.1 per cent for weak nucleolar chromosome-10. Chromosomes 1 and 2 were not transmitted at all and for chromosomes 6 and 12 transmission of extra chromosomes

was very rare. Very low level of transmission of n+1 male gametes was also noticed by Jena (1982) in Sona trisomics. The trisomics for chromosomes 1, 3, 5, 8, 10 and 12 exhibited no transmission of extra chromosomes through male side in contrast to the trisomic for chromosomes 6 and 11 which exhibited 4.0 and 3.1 per cent parental type of trisomics through male (Jena, 1982). However, Khush et al. (1984) reported very high rates of transmission for some of the chromosomes through pollen in IR-36 triplo-8 (14.3%) and triplo-9 (27.3%). In the selfed progenies of triplos-8 and 9 consistent occurrence of few tetrasomic plants was observed earlier by the authors. It was also observed that the longer chromosomes of the complement did not transmit through the pollen as they probably cause greater imbalance. In this respect the behaviour of the trisomics of rice parallel that of tomato and maize trisomics (Khush, 1973).

Jena (1982) also studied the gametic transmission of extra chromosome. The frequency of trivalent associations was observed from the cytological analysis of all triplos at diakinesis and metaphase I. From the total number of trivalents scored, he calculated the expected proportions of trivalents. Triplo-6 exhibited highest percentage of trivalents (88.8%) of the PMCs studied while triplo-5 showed the lowest frequency of trivalents (66.0%). Since the theoretical expectation of transmission of n + 1 gametes and n gametes at the time of meiotic disjunction was 50 per cent, the expected frequency of transmission of extra chromosome for all triplos were calculated on that basis. A 37.4 per cent average rate of transmission

of n + 1 gamete was observed. Triplo-6 exhibited the highest rate of transmission of extra chromosome (n + 1 gamete) which was 44.4 per cent whereas triplo-5 showed the lowest transmission of extra chromosome which was 33 per cent. The author also recorded the number of univalents for all the triplos at diakinesis and metaphase I. Of all the triplos studied, triplo-4 exhibited the lowest percentage of univalents (11.8%) of the total PMCs studied.

Jena (1982) further studied the relation between relative chromosome length, transmission frequency, trivalent frequency and univalent frequency from the correlation coefficients computed between any two of the above variables. He noticed that chromosome length was negatively correlated with trahsmission frequency as well as trivalent frequency though the correlation was not significant. Chromosome length was positively correlated with univalent frequency though the correlation coefficient was non-significant. The frequency of univalents and the frequency of trivalents showed significant negative correlation indicating that higher trivalent formation leads to lower univalent formation which results in lower transmission.

### 2.3.4 Chromosome numbering system in rice

There are three prevalent systems of numbering rice chromosomes.

i) Shastry et al. (1960) numbered the rice chromosomes according to their length at pachytene stage of meiosis. Thus, the longest chromosome was numbered 1 and the shortest as number 12.

ii) Nishimura (1961) assigned arbitrary number (I to XII) to rice chromosomes involved in translocations in the order in which the translocations were discovered. Later, the Roman numerals were changed to Arabic numerals. Nishimura's system of numbering chromosomes is not based on cytological identification and could not be utilised for cytological identification of the extra chromosome of the trisomics (Khush et al., 1984).

iii) Kurata and Omura (1978) numbered the rice chromosomes on the basis of their length at somatic prometaphase. The longest chromosome was numbered as $K_1$ and the shortest as $K_{12}$.

Two primary trisomics series were employed for associating linkage groups of rice with respective chromosomes and the association has been worked out by Khush et al. (1984) using IR-36 indica trisomics as well as by Iwata and Omura (1984 a & b) and Iwata et al. (1984a) using Nipponbare japonica trisomes. However, the numbering systems of chromosomes adopted by the two schools were partly inconsistent and a unified numbering system was required to be established. To settle the discrepancies between the length of chromosomes as reported by different authors and to establish a unified

numbering system, an Interim Committee on chromosome numbering was constituted. As per the report of the Interim Committee on chromosome numbering to the International Rice Genetics Symposium, it was recommended that chromosome numbering system based on the length at pachytene stage of meiosis as proposed by Sastry et al. (1960) should be followed (Anonymous, 1990b).

2.3.5 Genetic analysis through primary trisomics

The 12 possible genetic linkage groups have been proposed for both japonica (Nagao and Takahashi, 1963) and indica (Misro et al., 1966) rices, and a complete trisomic series for both japonica (Iwata and Omura, 1984 a & b) and indica (Khush et al., 1984) rices have also been established.

The trisomic series, upon their isolation, are supposed to be effectively used to assign genetic linkage groups to corresponding chromosomes. Unfortunately, all the primary trisomics in rice have not yet completely and conclusively been identified to both linkage groups and respective extra chromosome. Thus, there is some ambiguity still persisting with regard to clear identification of genetic linkage groups to chromosomes on a one-to-one basis. Thus, rice is lagging behind many crops such as barley, maize and tomato in mapping of genes on chromosomes and on linkage groups.

The literature pertaining to genetic analysis using primary trisomics has been surveyed with the up-to-date information and presented chronologically in tabular form for ready reference and brevity (Table 3). The chromosome numbering systems followed by different authors have been mentioned in the column 7 with abbreviated letters N for Nishimura (1961) and K for Kurata and Omura (1978). The chromosome numbering system in rest of the cases without any abbreviated letter is that of most widely accepted by Shastry et al. (1960). The linkage groups mentioned in column 6 are as designated by Nagao and Takahashi (1963) which have been most widely accepted and the abbreviated letter M (in column 6) is for Misro et. al. (1966).

The literature surveyed on the use of primary trisomics in genetic analysis in rice clearly shows that most of the research on this aspect has been done by testing the genetically identified marker genes for the deviation in disomic segregation and by the confirmation of trisomic segregation.

Further, it is quite clear that the information on the genetic analysis of the most complicated group of characters like that of anthocyanin pigmentation using primary trisomics is very meagre. Anthocyanin pigmentation genes in rice have been traditionally assigned to respective linkage groups based on genetic recombination results. But

the chromosomal location of these genes through primary trisomic technique is equally a systematic approach. Concerted efforts are needed for better understanding of the complex inheritance pattern of these characters to solve the discrepancy of these colour producing genes postulated in japonica and indica rices.

Table 3. Genetic analysis through primary trisomics***

| Authority | Material used | Characters studied | Disomic segregation ratio | Gene symbol | Linkage group | Chromosome |
|---|---|---|---|---|---|---|
| 1 | 2 | 3 | 4 | 5 | 6 | 7 |
| Iwata and Omura (1975) | Primary trisomics (japonica) | Rolled leaf | ( 3:1) | rl | ? | ? |
| | | "Bonsai-to" dwarf | ( 3:1) | dB | | |
| | Type A | Spotted leaf | ( 3:1) | $Spl_1$ | | |
| | | Narrow leaf | (15:1) | $Nal_2$ | | |
| | | Glutinous endosperm | ( 3:1) | Wr | | |
| | | Depressed palea | ( 3:1) | $dp_1$ | | |
| | Type B | White stripe | ( 3:1) | Ws | I | 6 (N) |
| | | Clustered spikelets | ( 3:1) | Cl | | |
| | | Burlush-like panicle | ( 3:1) | Bp | | |
| | Type C | Faded green leaf | ( 3:1) | fl | ? | ? |
| | | Pale green leaf | ( 3:1) | pgl | | |
| | Type D | - | - | - | None | - |
| | Type E | Liguleless | ( 3:1) | lg | II | 11 (N) |
| | | Round kernel | ( 3:1) | rk | | |
| | Type F | Ebisumochi dwarf | ( 3:1) | $d_6$ | IV | 10 (N) |
| | | Long sterile lemma | ( 3:1) | g | | |
| | | Brown pericarp seed coat | ( 3:1) | Rc | | |

Continued

Table 3 continued

| 1 | 2 | 3 | 4 | 5 | 6 | 7 |
|---|---|---|---|---|---|---|
| | | Lazy growth habit | ( 3:1) | la | | |
| | Type G | Short panicle | ( 3:1) | sp | VIII | 9(N) |
| | | Zebra leaf | ( 3:1) | z | | |
| | | Dense panicle | ( 3:1) | Dn | | |
| | Type H | Depressed palea-2 | ( 3:1) | $dp_2$ | VII & V | 1(N) |
| | | Inhibitor for brown furrows | ( 3:1) | I-Bf | | |
| Iwata and Omura (1976) | Primary trisomics (japonica) | "Daikoku"dwarf | ( 3:1) | $d_1$ | | |
| | | Open spikelet sterile | ( 3:1) | ops | VI | 2(N) |
| | Type L | Neck leaf-2 | ( 3:1) | $nl_2$ | | |
| | Type L | Glabrous glume and leaf | ( 3:1) | gl | XII | 2(N) |
| | | Neck leaf-1 | ( 3:1) | $nl_1$ | | |
| | Type L | Verticillate rachies | ( 3:1) | ri | IX | 2(N) |
| Sen (1977) | Primary trisomics (indica) Triplos 2 and 7 | Awn | - | An | - | 2 |
| | | Purple pericarp | - | Prp | - | 7 |
| Sadanada (1981) | Primary trisomics (indica) | Plant habit (erect vs. spreading) | (39:25) | - | - | 3,5,6,9,10, 11 and 12 |
| | Triplos 2,3,5,6, 7,9,10, 11 and 12 | Panicle type (lax vs normal) | (135:121) | lax | - | 2,3,5,6,7, 9,10,11 and 12 |
| | | Grain shattering (shattering vs non-shattering) | (54:10) | - | - | 3,7,11 and 12 |
| | | Resistance to bacterial leaf blight | ( 3:1) | - | - | 3 and 7 |

Continued

Table 3 continued

| 1 | 2 | 3 | 4 | 5 | 6 | 7 |
|---|---|---|---|---|---|---|
| | | Quantitative characters | | | | |
| | | Plant height (cm) | - | - | - | 5,7,9,10,11 and 12 |
| | | Days to flowering | - | - | - | 2,3,5,6,9, 10,11 and 12 |
| | | Panicle length (cm) | - | - | - | 2,5,6,7,11 and 12 |
| | | Tiller number | - | - | - | 3,7, and 9 |
| | | No.of total spikelets per panicle | - | - | - | 5,6,10 and 12 |
| | | Spikelet sterility(%) | - | - | - | 2,5,6 and 10 |
| Sadanada (1981) | Primary trisomics (japonica) | Plant habit (erect vs spreading) | ( 3:1) | - | - | 12 |
| | | Panicle type (lax vs normal) | (54:10) | lax | - | 5 |
| | Triplos 4,5,7,8, 9,10,11 and 12 | Grain shattering(shattering vs non-shattering) | (189 :87) | - | - | 10,11 and 12 |
| | | Glume pubescence (Pubescent vs glabrous) | (183:73) | - | - | 4,5,8,9,10, 11 and 12 |
| | | Leaf pubescence (Pubescent vs.glabrous) | (165:91) | - | - | 4,5,7 and 8 |
| | | Anthocyanin pigmentation | | | | |
| | | Leaf axil | (117:139) | Px | - | 10,11 and 12 |
| | | Internode | (117:139) | Pin | - | 5,10 and 12 |

Continued

Table 3 continued

| 1 | 2 | 3 | 4 | 5 | 6 | 7 |
|---|---|---|---|---|---|---|
| | | Juncture | ( 27:229) | Pj | - | 5,7,8,10, 11 and 12 |
| | | Septum | ( 27: 27) | Pm | - | 10,11 and 12 |
| | | Node | ( 3: 13) | Pn | - | 4,5,8,9, 10,11 and 12 |
| | | Stigma | (135:121) | Ps | - | 4,7 and 10 |
| | | Sterile lemma (glumes) | (243:781) | Pg | - | 4,7,9,10, 11 and 12 |
| | | Apiculus | (135:121) | Pa | - | 7 and 8 |
| | | Awn colour | (117:139) | Ran | - | 7,10 and12 |
| | | <u>Other characters</u> | | | | |
| | | Resistance to bacte-rial blight | ( 3: 1) | - | - | 8 and 10 |
| | | <u>Quantitative traits</u> | | | | |
| | | Plant height (cm) | - | - | - | 4,7,8,9,10 11 and 12 |
| | | Days to flowering | - | - | - | 4,5,7,8,9, 10,11 and 12 |
| | | Panicle length (cm) | - | - | - | 4,5,8,9 and 12 |
| | | Tiller number | - | - | - | 4,5,7,10, 11 and 12 |
| | | No.of total spikelets/ panicle | - | - | - | 4,5,7,8,9, 10,11 and 12 |
| | | Spikelet ste-rility (%) | - | - | - | 4,10 and 12 |

Continued

Table 3 continued

| 1 | 2 | 3 | 4 | 5 | 6 | 7 |
|---|---|---|---|---|---|---|
| Ikeda and Kaneda (1983; 1984) | Primary trisomics (*japonica*) Types A, C, E, G and H | Resistance to brown plant hopper | (3:1) | Bph-1 | II | 11 (N) Type E extra chromosome |
| Sur and Khush (1984) | Primary trisomics (*indica*) 11 of the 12 trisomics used | Resistance to bacterial leaf blight | (3:1 and 1:1) | $Xa_4$ | - | 7 |
| Wu *et al* (1984) | Three trisomic lines (*indica*) of IRRI with extra chromosomes 4,7 and 10 | Brittle culm | (3:1) | bc | - | 7 |
| Khush *et al.* (1984) | Primary trisomics (*indica*) Triplo 1 to Triplo 12 | Extra glume | - | eg | III | 1 |
| | | Lax panicle | - | lax | III | - |
| | | Triangular hull | - | tri | X | 2 |
| | | White stripes | - | ws | I | 3 |
| | | Clustered spikelets | - | Cl | I | 3 |
| | | Purple stigma | - | $PS_1$ | V | 3 |
| | | Chlorina-1 | - | $Ch_1$ | XI | 4 |
| | | Droopy leaf | - | dl | XI | 4 |
| | | Brittle culm | - | $bc_1$ | XI | - |
| | | Gold hull-1 | - | $gh_1$ | VI | 5 |
| | | Glabrous-1 | - | $gl_1$ | XII | 5 |
| | | Neck leaf-1 | - | $nl_1$ | IX | 5 |
| | | Spotted leaf-1 | - | $spl_1$ | - | 6 |
| | | Long empty glume | - | g | IV | 7 |
| | | Virescent-8 | - | $V_8$ | - | 8 |
| | | Depressed palea-2 | - | $dp_2$ | VII | 9 |

Continued

Table 3 continued

| 1 | 2 | 3 | 4 | 5 | 6 | 7 |
|---|---|---|---|---|---|---|
| | | Inhibitor of brown furrow | - | I-Bf | V | 9 |
| | | Faded leaf | - | fl | - | 10 |
| | | Pale green leaf | - | pgl | - | 10 |
| | | Lazy | - | la | VIII | 11 |
| | | Zebra striped leaves -2 | - | $Z_2$ | VIII | 11 |
| | | Liguleless | - | lg | XI | 12 |
| Iwata and Omura (1984 a & b) | Primary trisomics (*japonica*) Type M | Drooping leaf | (3:1) | dl | XI | 5 (N) ($K_3$) |
| | | Chlorina-2 | (3:1) | Ch-2 | | |
| | | Virescent-2 | (3:1) | V-2 | | |
| | Type N | Gold hull and internode-2 | (3:1) | gh-2 | X | 8 (N) |
| | | Virescent-1 | (3:1) | V-1 | | ($K_2$) |
| | Type O | Lax panicle | (3:1) | lax | III | 3 (N) |
| | | Dwarf-18 | (3:1) | d-18 | | ($K_1$) |
| Iwata *et al* (1984a) | Primary trisomics (*japonica*) | Sugary endosperm | (3:1) | su | ? | 12 (N) |
| | | Virescent-8 | (3:1) | V-8(t) | | |
| Iwata *et al* (1984b) | Primary trisomics (*japonica*) | Neck leaf-2 | (3:1) | nl-2 | VI | 2 (N) |
| | Type L | Verticillate rachies | (3:1) | ri | IX | 2 (N) |
| | | Bright green leaf | (3:1) | bgl | XII | 2 (N) |

Continued

Table 3 continued

| 1 | 2 | 3 | 4 | 5 | 6 | 7 |
|---|---|---|---|---|---|---|
| | | **Anthocyanin pigmentation** | | | | |
| Siddiq et al. (1986) | Primary trisomics (*indica*) Triplos 2,3,5,6, 7,9,10, 11 and 12 | Leaf axil | ( 39: 25) | Pk | - | 5,6,7,10, 11 and 12 |
| | | Internode | (117:139) | Pin | - | 5 and 10 |
| | | Juncture | ( 21: 43) | pj | - | 2,3,6,7, 9,10 and 12 |
| | | Septum | ( 39: 25) | pm | - | 5,7,9,11 and 12 |
| | | Node | ( 21: 43) | Pn | - | 3,10,11 and 12 |
| | | Stigma | ( 45: 19) | Ps | - | 2,3,5,7, 9,11 and 12 |
| | | Sterile lemma | ( 27: 37) | Pg | - | 3,6 and 9 |
| | | Apiculus | (189: 67) | Pa | - | 2,3,5,6, 7,11 and 12 |
| | | Awn colour | ( 39: 25) | Ran | - | 5,6,7,10 11 and 12 |
| | | **Other characters** | | | | |
| | | Resistance to bacterial leaf blight | ( 54: 10) | $Xa_4$ | - | 3 and 9 |
| | | **Basmati quality attributes** | | | | |
| | | Aroma | ( 54: 10) | - | - | 5 and 9 |
| | | Kernel length (mm) | - | - | - | 2,3,5,6, 7 and 10 |
| | | Kernel breadth (mm) | - | - | - | 2,3,5,6, 7,9,10 and 12 |
| | | Kernel L/B ratio | - | - | - | 3,5,6,7, 9,10 and 12 |
| | | Kernel elongation(mm) | - | - | - | 3,5,6,7, 9 and 12 |

Continued

Table 3 continued

| 1 | 2 | 3 | 4 | 5 | 6 | 7 |
|---|---|---|---|---|---|---|
| | | Water absorption (mg) | - | - | - | 2,3,5,6,7, 9,10 and 12 |
| | | Volume expansion (ml) | - | - | - | 2,3,5,6,7, 9,10 and 12 |
| | | Gelatinization temperature (Alkali score) | - | - | - | 2,3,5,6,7, 9,10 and 12 |
| | | Amylose content % | - | - | - | 2,3,5,6,7, 9,10 and 12 |
| | | Gel consistency (mm) | - | - | - | 3,6,7,9, 10, 11 and 12 |
| Misra & Jena (1986) | Primary trisomics (*indica*) | Clustered spikelets | (45:19) | Cl | (M) I, VI | 7,8 and 12 |
| | | Gold hull | (55: 9) | gh | (M) VI | 2,5 and 12 |
| Jena & Misra (1983) | Triplos 1 to 12 except Triplo 4 | Long glume | ( 3: 1) | g | (M) IV | 9 |
| | All triplos except triplos 4 and 5 | Brittle culm | ( 3: 1) | bc | (M) XI | 6 |
| Librojo and Khush (1986) | Primary trisomics (*indica*) Triplos 1 to 12 | Hairy glume | - | Hg | - | 4 |
| | | Multiple pistil-1 | - | mp-1 | - | 1 |
| | | Multiple pistil-2 | - | mp-2 | - | 3 |
| | | Chlorina | - | ch | - | 3 |
| | | Yellow green leaves | - | ygl | - | 10 |
| | | Elongated upper most internode | - | eui | - | 5 |

Continued

Table 3 continued

| 1 | 2 | 3 | 4 | 5 | 6 | 7 |
|---|---|---|---|---|---|---|
| | | Auricleless | - | aul | - | 12 |
| | | Brittle culm | - | bc-4 | - | 3 |
| Ishikawa *et al* (1986) | Primary trisomics (*japonica*) Types A, C, D, E, F, G and H | Amino-peptidase-2 | - | Amp-2 | Su | 12 (N) D type |
| | | Phospho-gluconate dehydroge-nase-1 | - | Pgd-1 | VIII | 9 (N) G type |
| | | Shikimate dehydro-genase-1 | - | Sdh-1 | d-33 | 4 (N) A type |
| Ranjhan *et al* (1986, 1988) | Primary trisomics (*indica*) Triplos 1 to 12 | Phospho-glucose isomerase | - | Pgi-1 | - | 4 |
| | | Shikimate dehydro-genase | - | Sdh-1 | - | 6 |
| | | Esterase | - | Est-9 (Est-8) | - | 7 |
| | | Alcohol de-hydrogenase | - | Adh-1 | - | 11 |
| Matsuo *et al* (1986) | Primary trisomics (*japonica*) Type D | Shrunken endosperm | (3:1) | Shr-2 | - | 12 (N) |
| Ishikawa *et al* (1987) | Primary trisomics (*japonica*) Types A, B, C, D, E, F, G & H | Catalase-1 | - | Cat-1 | I | 6(B type extra chromosome) |
| | | Acid phos-Phatase-1 | - | Acp-1 | d-33 | 4(A type) |
| | | Peroxidase-2 | - | Pox-2 | d-33 | 4(A type extra chromosome) |

Continued

Table 3 continued

| 1 | 2 | 3 | 4 | 5 | 6 | 7 |
|---|---|---|---|---|---|---|
| Khush and Cruz (1988) | Primary trisomics (*indica*) | Chlorina-8 | - | Chl-8 | Sug | 8 |
| | Triplo-1 to 12 | Chlorina-9 | - | Chl-9 | Sug | 8 |
| Wu *et al* (1988) | Primary trisomics (*indica*) Triplos 1 to 12 and (*japonica*) few triplos) | Peroxidase-5 | - | Pox-5 | I | 3 |
| | | Catalase-1 | - | Cat-1 | I | Type B |
| | | Esterase-5 | - | Est-5 | III | 1 |
| | | Aspartate amino transferase-1 | - | Got-1 | III | 1 |
| | | Isocitrate dehydrogenase-1 | - | Icd-1 | III | 1 |
| | | Phosphoglyconate dehydrogenase-1 | - | Pgd-1 | VIII | 11 |
| | | Aminopeptidase-1 | - | Amp-1 | X | 2 |
| | | Acid phosphates 1 and 2 | - | Acp-1 Acp-2 | d-33 | 6 |
| | | Aminopeptidase-2 | - | Amp-2 | Sug | 8 |
| | | Aminopeptidase-4 | - | Amp-4 | Sug | 8 |
| Yano *et al.* (1988) | Primary trisomics (*japonica*) | Low amylose (dull) | - | $du_1$ | - | 7 (N) |
| | | Low amylose (dull) | - | $du_4$ | - | 4 (N) |
| Ishikawa *et al* (1989) | Primary trisomics (*japonica*) | Phosphogluconate dehydrogenase-1 | - | Pgd-1 | VIII | 11 |
| | | Alcohol dehydrogenase | - | Adh-1 | VIII | 11 |

Continued

Table 3 continued

| 1 | 2 | 3 | 4 | 5 | 6 | 7 |
|---|---|---|---|---|---|---|
| Kumamaru *et al* (1989) | Primary trisomics (*japonica*) Types A, C, D, E, G and L | Rice storage protein | (3:1) | esp-3 | - | 9 (N) G type extra chromosome |
| Ishikawa *et al* (1990) | Primary trisomics (*japonica*) | Glutamate dehydrogenase | - | Gdh-1 | - | 4 |
| Brar *et al* (1990) | Primary trisomics (*indica*) | Asperatate aminotransferase-3 | - | Got-3 | - | 2 |
| | | Endopeptidase-1 | - | Enp-1 | - | 3 |
| | | Esterase-1 | - | Est-1 | - | 7 |
| | | Fructose-1, 6-diphosphatase | - | Fdp-1 | - | 11 |

*** Relationships among various systems of numbering linkage groups, chromosomes and trisomics (Kinoshita, 1986a) has been presented in Table 4 for ready reference and comparison.

Table 4. Relationships among various systems of numbering linkage groups, chromosomes and trisomics (Kinoshita, 1986a)

| Linkage groups | | Chromosomes | | | Trisomics | |
|---|---|---|---|---|---|---|
| Kinoshita (1984) | Misro * (1981) | Nishimura (1961) | Shastry et al. (1960) | Kurata et al. (1981) | Khush et al. (1984) | Iwata et al. (1984a) |
| I | I | 6 | 3 | K6 | Triplo-3 | B |
| II | II | 11 | 12 | K4 | Triplo-12 | E |
| III | III | 3 | 1 | K1 | Triplo-1 | O |
| IV | IV | 10 | 7 | K11 | Triplo-7 | F |
| VI + IX | VI, XII | 2 | 5 | K9 | Triplo-5 | L |
| V | IX | 1 | - | - | - | H |
| VII | - | 1 | 9 | K10 | Triplo-9 | H |
| VIII | VIII | 9 | 11 | K8 | Triplo-11 | G |
| X | - | 8 | 2 | K2 | Triplo-2 | N |
| XI | XI | 5 | 4 | K3 | Triplo-4 | M |
| XII | - | - | - | - | - | - |
| fgl | - | 7 | 10 | K12 | Triplo-10 | C |
| d-33 | - | 4 | 6 | K5 | Triplo-6 | A |
| Su | - | 12 | 8 | K7 | Triplo-8 | D |

*Groups V, VII and X were not correlated with Kinoshita's groups.

## CHAPTER - III

# MATERIAL AND METHODS

## III. MATERIAL AND METHODS

The present investigations were initiated in kharif 1987 and 1988 at the University of Agricultural Sciences (UAS), Dharwad Campus, with the stubble progeny of 'Sona' trisomics brought from CRRI, Cuttack, Orissa State. The unfavourable climatic conditions namely, dry weather and very low morning temperatures in winter season rendered the maintenance of this genetically abnormal material, extremely difficult. Using four different local cultivars as donor parent, the hybridization programme remained almost unsuccessful. In both the seasons a soil-borne root-rot disease complex, namely, Rhizoctonia solani etc., further weakened the trisomics and on vegetative multiplication, most of them could not survive. Hence, the present investigations were continued during the years 1989-90 (wet and dry seasons) and 1990-91 (wet and dry seasons) at the Central Rice Research Institute (CRRI), Indian Council of Agricultural Research (ICAR), Cuttack, Orissa State.

The hot and humid weather at CRRI, Cuttack was found congenial for trisomics to flourish. With intensive and concerted efforts, the trisomics were successfully maintained and the hybridization programme was attempted. All the subsequent studies were also carried out at the CRRI Institute Cuttack.

The material used and the methods followed for carrying out these investigations are presented in the following sections.

3.1 Parental material and

3.2 methods, for the sake of convenience .

## 3.1 Parental material

The material for crossing programme consisted of a green disomic indica cultivar, Sona, a complete set of primary trisomics (Triplos 1 to 12) in its background and an indica cultivar 'Crossa' with purple leaf blade having pigmentation in most of its other plant parts also. All the parental material was obtained through the courtesy of Dr. R.N. MISRA, Principal Scientist, Cytogenetics Laboratory, CRRI, Cuttack. The morphological description of the parents are given in Tables 5 and 6 and their pedigree and origin are presented in Table 7. The photographs displaying the phenotypes of all the 12 primary trisomics, along with the disomic control Sona, the triploid (3N) Sona along with the disomic control Sona, and the completely purple marker Crossa, are depicted in plates 1 to 14.

The purple marker 'Crossa' was utilized in cross combinations with all the 12 primary trisomics as well as with the disomic control 'Sona' for locating the genes on specific chromosomes. All the 12 primary trisomics as well

Table 5. Morphological description of parental material

| Female parents | Morphological details |
|---|---|
| 1 | 2 |
| Sona (disomic) | Medium duration, photoinsensitive, high yielding variety having long panicles with awnless, slender spikelets and superfine grains. This widely adapted, hard to thresh variety has an average growth period of 130 days and was released earlier in Punjab. In 1973 Central Variety Release Committee (CVRC) released it. It is designated as RP-5-3. It has no anthocyanin pigmentation in any of the plant parts [Plate 1 cultivar Sona (2n) along with Sona (3n)]. |
| Triplo-1 (grassy) | Plants are slightly shorter in height than the control and show slender culm, profuse tillering, and pale, narrow, drooping leaves. The presence of thin and droopy leaves gives the characteristic grassy appearance. Spikelets are short and slender. It flowers very late - almost always in December - January after the Winter. Spikelet fertility is very low (Plate 2). |
| Triplo-2 (dwarf) | This is the shortest among the trisomics and has a semi spreading appearance due to nodal bending after flowering. Leaves are short and slightly twisted at the base. The panicle is small, with poor exsertion, spikelets are short and broad with a slight tip awn. It is self-incompatible but cross-compatible (Plate 3). |
| Triplo-3 (awned) | Due to the presence of long awns in the spikelets, this can be easily distinguished from other trisomics as well as from the disomic. It has short height with short, erect, dark green leaves. Panicles are short with moderate spikelet fertility (Plate 4). |




Plate 1: Cultivar Sona (2n) and Sona (3N)




Plate 2: Sona disomic and triplo-1




Plate 3: Sona disomic and triplo-2




Plate 4: Sona disomic and triplo-3

Table 5 continued

| 1 | 2 |
|---|---|
| Triplo-4 (sterile) | This plant is sterile and low tillering and has short, thick, dark green leaves and short, compact panicles. The spikelets are shorter, hairy and the palea is smaller than the lemma. The stamens are small, rudimentary, and vary from 5 to 6. The stigma is mostly tri to quadrifid. The ovary is comparatively the shortest. It is completely pollen sterile, and only 0.2% of the seeds set on open pollination (Plate 5). |
| Triplo-5 (twisted leaf) | This is characterized by the presence of pale green, narrow, twisted, hairy leaves. Plants are short with compact, short panicles with poor exsertion. Spikelets are partially fertile. The lemma and palea often remain open after fertilization (Plate 6). |
| Triplo-6 (bushy) | Short height, light green leaves, long flag leaves, and a profuse tillering habit are characteristic features of this trisomic. Panicles are long with moderate spikelet fertility. Spikelets have short or little long tip awns. Degenerated spikelets at the tips of the panicles are a characteristic feature of this trisomic (Plate 7). |
| Triplo-7 (narrow leaf) | Plants are short and have low tillering with characteristic long, narrow leaves that sometimes appear slightly rolled. Spikelets are slender with low fertility (Plate 8). |
| Triplo-8 (rolled leaf) | Short height, low tillering, rolled, erect, acicular leaves with short bold spikelets, and poor panicle exsertion are the distinguishing features of this trisomic. Pollen and spikelet fertility are low (Plate 9). |




Plate 5: Sona disomic and triplo-4




Plate 6: Sona disomic and triplo-5




Plate 7: Sona disomic and triplo-6




Plate 8: Sona disomic and triplo-7

Table 5 continued

| 1 | 2 |
|---|---|
| Triplo-9 (stout) | This trisomic has a stout habit with thick, dark green leaves and large spikelets. The stout culm is due to a larger culm diameter. Panicle is long with low pollen and spikelet fertility. The spikelets have short tip awns. Degenerated spikelets are more in the incompletely exserted panicles (Plate 10). |
| Triplo-10 (short grain) | This trisomic is slightly taller than the disomic with an erect habit. Its long leaves have the margin curved somewhat inwards. The panicles are long and the rachis is wavy with small, round spikelets. It also flowers very late like that of trisomic-1. Dehusking is difficult due to tightly enveloped hull. It has the longest ovary among the trisomics. The spikelets are awned. (Plate 11) |
| Triplo-11 (pseudo-normal) | Morphologically this trisomic is not distinguishable from the disomic except by the presence of a lax type panicle and the longest ligule. It has high fertility (Plate 12). |
| Triplo-12 (tall) | This is the tallest among the trisomics and the disomic. It has a robust habit in all morphological traits; long broad leaves; long panicles with bold spikelets; and long anthers. It tillers profusely and has a moderate fertility (Plate 13). |
| Male parent | |
| Crossa | A completely purple marker having anthocyanin pigmentation in almost all the plant parts except leaf sheath (internal), juncture, node, internode, nodal septum, nodal ring, lemma and palea, sheath pulvinus, panicle base and panicle pulvinus. The leaf blade is full purple (Plate 14) |

Note: Plate 12 has a freshly planted triplo-11 stubble and plates 7 and 8 have 3 to 4 stubbles of triplo-6 and triplo-7 respectively.




Plate 9: Sona disomic and triplo-8




Plate 10: Sona disomic and triplo-9




Plate 11: Sona disomic and triplo-10




Plate 12: Sona disomic and triplo-11




Plate 13: Sona disomic and triplo-12

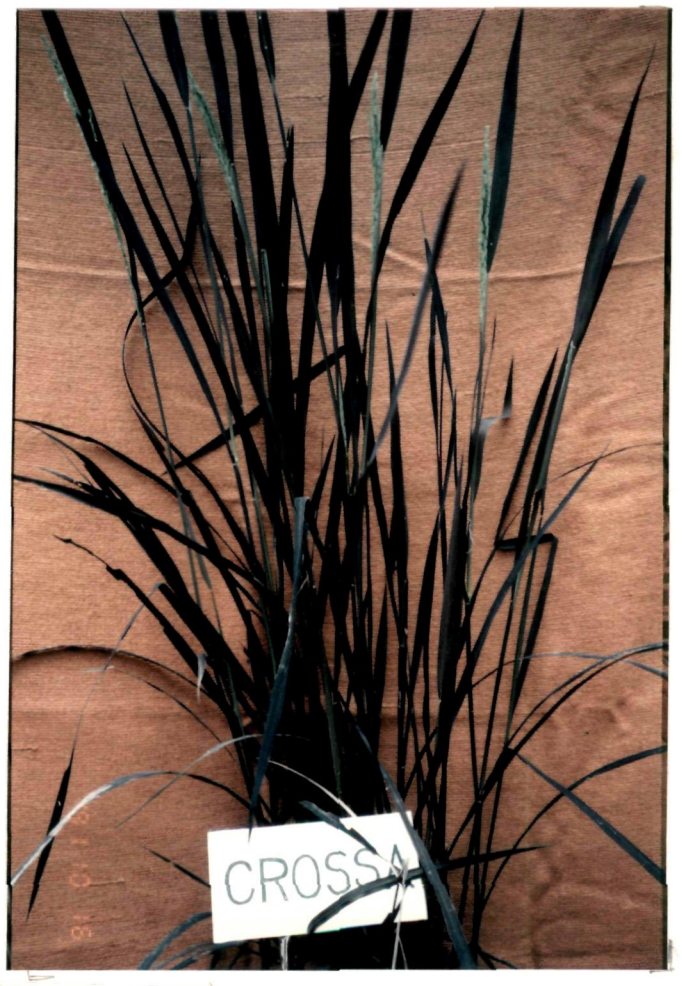



Plate 14: Cultivar Crossa

Table 6. Morphological and reproductive features of the parental material

| Parents | Plant height (cm) | Leaf length (cm) | Flag leaf length (cm) | Ligule length (cm) | Panicle length (cm) | No.of panicles | No.of spikelets/ panicle | Pollen fertility (%) | Spikelet fertility (%) |
|---|---|---|---|---|---|---|---|---|---|
| **Female parents** | | | | | | | | | |
| Sona (2n) | 82.5 | 42.8 | 32.9 | 2.4 | 28.5 | 18 | 145 | 94.3 | 97.7 |
| *Triplo-1 | 78.5 | 44.4 | 37.5 | 1.6 | 24.0 | 22 | 184 | 4.0 | 7.6 |
| *Triplo-2 | 42.0 | 18.6 | 12.0 | 1.0 | 12.0 | 11 | 40 | - | 0.0 |
| *Triplo-3 | 61.1 | 39.8 | 31.0 | 2.4 | 23.2 | 17 | 63 | 75.3 | 27.7 |
| *Triplo-4 | 46.8 | 22.0 | 16.5 | 0.5 | 15.5 | 20 | 79 | 1.0 | 0.0 |
| *Triplo-5 | 53.5 | 35.9 | 26.8 | 0.7 | 18.8 | 18 | 104 | 55.5 | 25.3 |
| *Triplo-6 | 66.0 | 38.2 | 21.2 | 1.8 | 24.5 | 43 | 98 | 62.7 | 54.9 |
| *Triplo-7 | 70.2 | 37.7 | 28.0 | 1.4 | 25.0 | 20 | 100 | 43.9 | 17.0 |
| *Triplo-8 | 57.5 | 33.4 | 19.2 | 0.8 | 21.2 | 20 | 103 | 14.9 | 9.0 |
| *Triplo-9 | 71.3 | 46.4 | 32.5 | 1.7 | 23.5 | 09 | 70 | 26.4 | 2.0 |
| *Triplo-10 | 94.4 | 52.6 | 22.5 | 1.5 | 27.8 | 22 | 122 | - | 8.0 |
| *Triplo-11 | 60.0 | 40.3 | 20.2 | 3.2 | 28.4 | 20 | 110 | 77.7 | 50.0 |
| *Triplo-12 | 117.2 | 57.2 | 40.4 | 1.8 | 32.1 | 26 | 160 | 72.2 | 20.0 |
| **Male parent** | | | | | | | | | |
| Crossa | 115.5 | 58.0 | 38.0 | 2.5 | 28.0 | 20 | 150 | 96.1 | 90.9 |

(-) = Not studied

* The data from stubble progeny of trisomics

Table 7. Pedigree and origin of the parental material

| Parents (*indica*) | Pedigree and origin |
|---|---|
| Sona (disomic) control (Female parent) | GEB-24 x T (N) - 1 CVRC released high yielding variety. Year 1973 |
| Trisomics 1 to 12 (Female parents) | A spontaneous autotriploid of the cultivar Sona obtained at CRRI, Cuttack. [Plate 1 Sona (2n) and Sona (3n)] * |
| Crossa (male parent) | A derivative of Nungi x Nagkesar also called as Cross-1. Nagkesar is a purple local land race with purple colour foliage, grown to eradicate weedy wild rices in Madhya Pradesh (Plate 14) |

*Note: The Sona (3n) in plate-1 being a stubble progeny is not an exact representative of triploid.

as the disomic control (Sona) were devoid of pigmentation in any plant part and thus were completely green (acyanic). The $F_1$ hybrids were produced using the purple marker as male parent with each of the trisomics keeping always as female parent. The different cross combinations of Crossa marker with control and trisomics, the number of $F_1$s and actual trisomic $F_1$s obtained are given in Table 8.

## 3.2 Methods

### 3.2.1 Growing of the trisomics and the marker parent

The complete set of 12 primary trisomics and the control disomic 'Sona' were grown in uniform size earthen pots from the stubbles, carefully selecting young tillers of equal size and age. Before planting in puddled pots, the stubbles were treated with insecticidal solution for 20 to 30 minutes followed by proper washing in running water. The fertilizer requirements as basal dose as well as top dressing were properly taken care of as per the package of practices.

The seeds of the marker male parent were sown in shallow pans at two to three different timings and 21 to 28 days seedlings were transplanted in small experimental plots following a staggered planting. As per the recommended schedule, proper plant protection care and fertilizer application were followed.

Table 8. Different cross combinations with control and trisomics

| Sl. no. | Cross combinations | Total No.of $F_1$s/ $BC_1F_1$s obtained | (2n+1) $F_1$s/ $BC_1F_1$ | Remarks |
|---|---|---|---|---|
| 1 | Disomic x Crossa | 3 | - | - |
| 2 | Triplo-1 x Crossa | 6 | 2 | - |
| 3 | Triplo-2 x Crossa | 5 | 1 | - |
| 4 | Triplo-3 x Crossa | 7 | 2 | - |
| 5a | Triplo-4 x Crossa | 2 | 1* | $F_1$ caryopses rescued on 1/4 M.S. media |
| 5b | (Triplo-4 x Crossa) $F_1$ x Sona | 2 | 1 | - |
| 6 | Triplo-5 x Crossa | 6 | 1 | - |
| 7 | Triplo-6 x Crossa | 6 | 1 | - |
| 8 | Triplo-7 x Crossa | 6 | 1 | - |
| 9 | Triplo-8 x Crossa | 6 | 1 | - |
| 10 | Triplo-9 x Crossa | 5 | 2 | - |
| 11 | Triplo-10 x Crossa | 9 | 2* | Degenerated caryopsis of $F_1$ was inoculated on 1/4 M.S media, but was lost due to contamination |
| 12 | Triplo-11 x Crossa | 14 | 1 | - |
| 13 | Triplo-12 x Crossa | 15 | 2 | - |

*The Triplo-4 and one of the Triplo-10 $F_1$s being highly sterile; backcrosses were made and (2n + 1) $BC_1F_1$ plant was recovered in case of Triplo-4.

### 3.2.2 Technique of hybridization

The 12 primary trisomics and the disomic were used as the female parents and the genetic marker parent was utilized as the male parent in the hybridization programme. The crosses in all combinations were effected in the dry and wet seasons of 1989. The trisomics and the marker male parent were grown in such a way that they coincided in flowering. In the morning at 7.30 a.m. healthy spikelets were hand emasculated with a fine forceps prior to anthesis and were covered with butter paper bags to protect the emasculated spikelets from foreign-pollen contamination. For pollination, during 8.00 a.m. to 11.00 a.m. healthy anthers ready for dusting were collected from healthy spikelets of the marker plants and were carefully pollinated by inserting a piece or a whole of well dehisced anther into the emasculated spikelets of the trisomics and the disomic. The crossed spikelets were protected under butter paper bags until they were ready for harvest. Nearly, 10,000 pollinations were attempted to obtain a complete set of cross combinations (Table 8).

### 3.2.3 Raising the $F_1$ plants

The crossed seeds collected separately for each cross were stored in the desiccator after proper drying. For raising the $F_1$ plants crossed seeds were treated with 0.1 per cent $HgCl_2$ solution for 10 minutes, thoroughly washed in

water and then they were kept in sterilized petridishes for germination. The petridishes were kept inside properly maintained germinator box for quick germination. On seventh day, germinated seeds were transplanted to shallow pans with pulverized soil and after 21 days, the seedlings were transplanted individually to puddled soil in earthen pots of suitable size.

Surprisingly and unprecedentally two underdeveloped $F_1$ caryopses were recovered from so far called highly sterile trisomic-4. Needless to say that utmost care was taken to rescue them. The $F_1$ caryopses were soaked in distilled water for 10 minutes, dehusked gently and disinfected in sodium hypochloride for five minutes. After washing thoroughly with three to four changes of sterile water the surface sterilized caryopses were transferred to sterilized distilled water under the Laminar flow hood, and were inoculated on 1/4 M.S. media (Murashige and Skoog, 1962) in the culture tubes aseptically. The juvenile seedlings on reaching the plug head of the test tube were transferred and retained in distilled water for nearly 48 hours and then were carefully transplanted to the loose pulverized autoclave sterilized soil in a autoclave sterilized micro pot.

### 3.2.4 Morphological observations on $F_1$ plants

The morphology of all the $F_1$ plants were recorded from seedling to adult stage and were compared with the morphology

of the concerned trisomic and the genetic marker parent. True hybrids were identified by the dominance of pigmented characters in the seedlings. A detailed study of qualitative traits that is, presence or absence of pigmentation in various plant parts was made in all the $F_1$ plants as well as the parents at appropriate stages of plant growth. The qualitative traits studied have been presented in later paragraphs (3.2.11 and Table 10). In addition to this, a few quantitative traits (ancillary morphological traits) namely, (i) plant height (cm), (ii) leaf length (cm), (iii) flag leaf length (cm) (iv) ligule length (cm) (v) panicle length (cm), (vi) No. of panicles and (vii) No. of spikelets per panicle were recorded at maturity in all the $F_1$ plants and the parents (Tables 6 and 9). The general appearance of the $F_1$ plants at maximum tillering stage was also recorded to distinguish the trisomic (2n + 1) $F_1$ plants from the disomic (2n) $F_1$s. The photographs depicting the phenotypes of few of the representative trisomic $F_1$s and a $BC_1F_1$ of trisomic-4 have been displayed in plates 15 TP-1 x Crossa; 16 TP-4 x Crossa ($F_1$ and $BC_1F_1$ together), 17 TP-4 x Crossa ($F_1$), 18 TP-4 x Crossa ($BC_1F_1$) 19 TP-7 x Crossa and 20 TP-12 x Crossa.

### 3.2.5 Reproductive features of $F_1$ plants

The percentage of pollen fertility and spikelet fertility of the parents as well as the trisomic and control $F_1$s was computed as depicted in Tables 6 and 9. The study




Plate 15: $F_1$ Triplo-1 x Crossa




Plate 16: $F_1$ and $BC_1F_1$ Triplo-4 x Crossa




Plate 17: $F_1$ Triplo-4 x Crossa



Plate 18: $BC_1F_1$ (Triplo-4 x Crossa) x Sona disomic

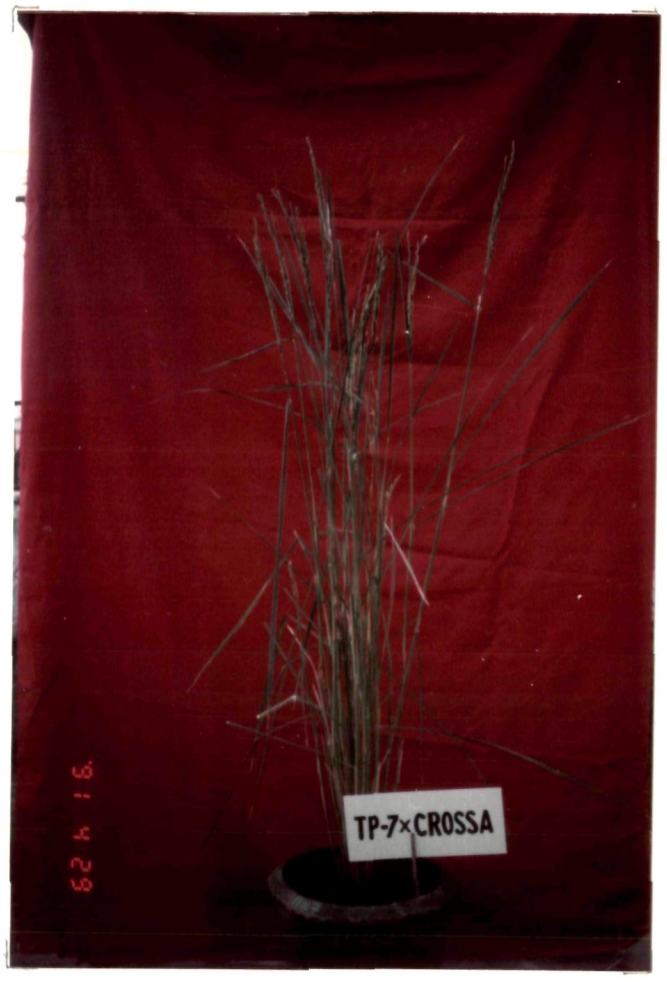



Plate 19: $F_1$ Triplo-7 x Crossa




Plate 20: $F_1$ Triplo-12 x Crossa

Table 9. Morphological and reproductive features of the disomic and trisomic $F_1/BC_1F_1$ plants

| $F_1/BC_1F_1$ *plants | Plant height (cm) | Leaf length (cm) | Flag leaf length (cm) | Ligule length (cm) | Panicle length (cm) | No. of panicles | No. of spikelets/ panicle | Pollen fertility % | Spikelet fertility % |
|---|---|---|---|---|---|---|---|---|---|
| Triplo-1 x Crossa | 87.4 | 48.5 | 33.6 | 1.8 | 21.3 | 19 | 136 | - | 6.4 |
| Triplo-2 x Crossa | 69.0 | 32.0 | 25.4 | 1.5 | 16.4 | 17 | 70 | - | 3.5 |
| Triplo-3 x Crossa | 94.0 | 49.2 | 33.9 | 2.0 | 18.5 | 19 | 58 | 68.8 | 42.0 |
| Triplo-4 x Crossa (TP-4 x Crossa) | 75.0 | 40.0 | 36.0 | 0.5 | 17.5 | 12 | 100 | 0.0 | 0.09 |
| Sona (2n) | 112.5 | 55.0 | 43.0 | 2.0 | 27.5 | 17 | - | - | - |
| Triplo-5 x Crossa | 80.0 | 49.0 | 30.5 | 1.7 | 20.2 | 26 | 111 | 40.0 | 27.0 |
| Triplo-6 x Crossa | 96.5 | 70.2 | 51.0 | 2.7 | 25.0 | 20 | 96 | 72.6 | 53.0 |
| Triplo-7 x Crossa | 118.0 | 55.0 | 37.0 | 2.2 | 23.0 | 21 | 104 | - | 44.6 |
| Triplo-8 x Crossa | 132.0 | 58.8 | 30.2 | 2.4 | 26.2 | 22 | 128 | 45.5 | 69.9 |
| Triplo-9 x Crossa | 83.0 | 48.5 | 31.0 | 1.5 | 22.0 | 11 | 70 | - | 32.8 |
| Triplo-10 x Crossa | 134.0 | 59.0 | 36.5 | 1.8 | 22.6 | 15 | 90 | 80.0 | 11.2 |
| Triplo-11 x Crossa | 106.5 | 72.0 | 46.3 | 2.8 | 27.3 | 18 | 132 | 61.5 | 78.0 |
| Triplo-12 x Crossa | 100.3 | 54.0 | 33.3 | 1.7 | 23.5 | 27 | 98 | 50.5 | 25.6 |
| (Sona control) x Crossa | 105.5 | 59.5 | 30.7 | 1.5 | 20.5 | 14 | 102 | 95.5 | 92.8 |

(-) = Not studied

however, was a tentative study. Further the data in few of the cases were not recorded and hence the study requires further confirmation.

3.2.5.1 Pollen sterility

The healthy spikelets of all the parents and $F_1$s under study were collected at the time of anthesis and were kept in 70 per cent alcohol. One per cent KI solution (1 part of iodine with 3 parts of potassium iodide dissolved in 100 cc glycerine) was used for study of pollen sterility. Two to three anthers of each trisomics were kept on the slide with a drop of KI solution and covered with a cover glass and was pressed. The stained and unstained pollens were observed in the microscope and a number of cells were counted. Grains which were full and deeply stained were taken as fertile while the lightly stained ones with shrivelled margins were classified as sterile. The percentage of pollen sterility was recorded from the observation of mean number of cells.

3.2.6 Cytological preparations and microphotography

Young healthy panicles at suitable stages were fixed between 7 a.m. to 8 a.m. in aceto-alcohol solution (1:3 by volume) with traces of ferric chloride and were kept at room temperature for nearly 48 hours. Fixed panicles were stored in a refrigerator until they were cytologically observed. The selected spikelets were squashed in 1 per cent aceto carmine solution (prepared by boiling 1 g of carmine dye powder

in 100 cc of 45 per cent acetic acid); when the anthers were stained, a clean cover slip was placed on it and the anthers were gently tapped with a needle. Uniform gentle pressure with the thumb was applied in order to spread the chromosomes. The PMCs at suitable stages were pressed gently from time to time with alternate warming over the flame of spirit lamp. After proper spreading of the PMCs the slides were sealed with paraffin wax and the cytological observations were made from temporary slides.

The chromosome numbers of all the trisomic parents and $F_1$ plants under study were counted at metaphase-I or metaanaphase stages of meiosis, as 12 II + 1 I or 11 II + 3 I. Sometimes, diakinesis and anaphase-I stages were also considered for the presence of the trivalent and a univalent (laggard) respectively. Some of the PMCs with well spread, countable chromosomes were photographed with NP-15 (ORWO) slow film of ASA 25/15 DIN in Olympus camera attached with GTR-11A and Meiji microscopes thoroughly limiting the source of light and time of exposure.

The microslides were made permanent by allowing the cover glass to be separated in a solution of 1:1 acetic acid (45%) and n-butanol and then after passing them through 1:1 butanol and xylol, and pure n-butanol. Finally, Canada balsam was used as a mounting medium.

The microphotographs of a few of the representative PMCs have been displayed in plates 21 to 35.

### 3.2.7 Raising the $F_2$ population

The seeds collected from $F_1$ trisomic plants (critical $F_1$s) from each of the cross as well as the control disomic $F_1$ were properly dried and were sown in powdered loose soil of shallow pans under green house condition. Further, the disomic populations obtained from $F_1$ plants of trisomics 1 to 12 (non-critical $F_1$s) were also grown in a similar fashion in the separate shallow pans.

To obtain sufficient number of seeds, the trisomic $F_1$ plants were multiplied vegetatively through stubbles and the seeds were harvested after maturity. Inspite of this, there were only a few $F_2$ seeds in case of trisomics 2, 4, 8, 9, 10 and 12 and hence the $F_2$ seeds of these trisomics were dehusked, disinfected and kept on sterile petriplates for germination. On seventh day, the juvenile germinated seeds were transplanted to pulverized soil in shallow pans inside the net house. The 28 to 30 days old seedlings were very carefully uprooted and transplanted in a properly prepared 20 cent plot of cytogenetics unit at 'B' block. The $F_2$ seedlings were transplanted in a random manner with 30 cm x 20 cm spacing to facilitate recording of observations at later stages. All cultural practices like weeding, stirring, irrigation and application of fertilizer as well as plant protection measures were undertaken as and when necessary.

Plate 21: Metaanaphase showing 12 III in 3 N 'Sona'

Plate 22: Metaphase I showing 12 II in disomic "Sona"

Plate 23: Metaanaphase showing 12 II in disomic "Sona"

Plate 24: Telophase I showing a persistent laggard univalent in the $F_1$ of triplo-4

Plate 21

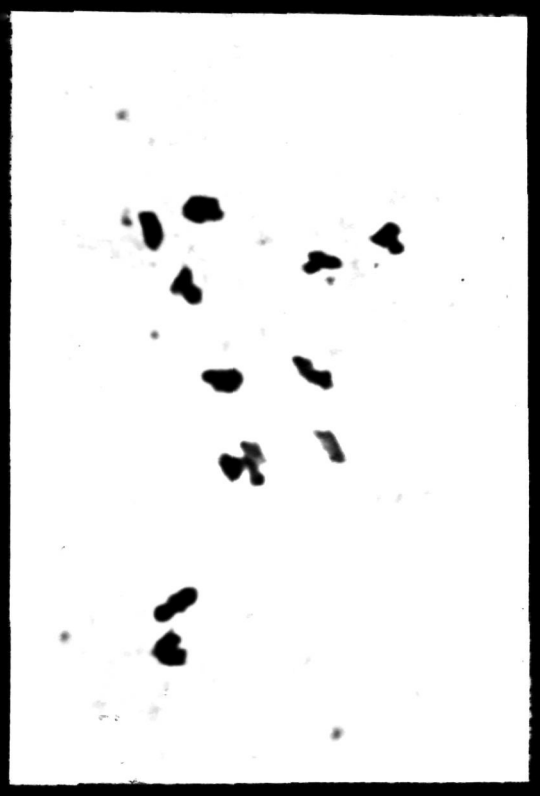


Plate 22





Plate 23

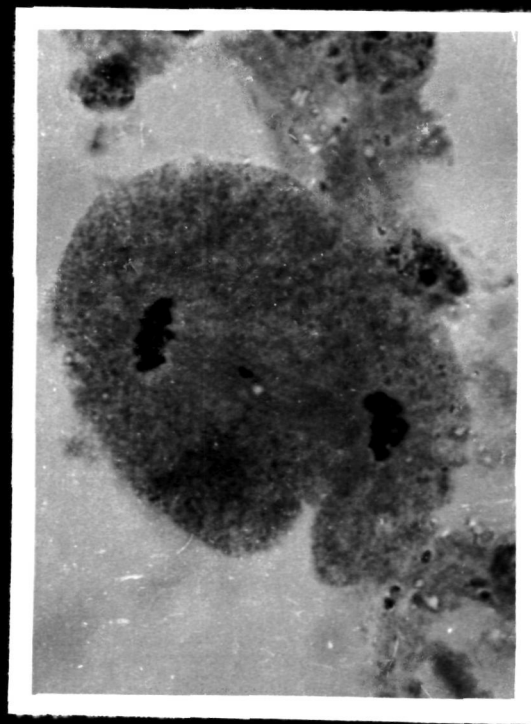


Plate 24

Plate 25: Anaphase I showing a bridge in the $BC_1F_1$ of triplo-4

Plate 26: Metaanaphase showing 12 II + 1 I in the $BC_1F_1$ of triplo-4

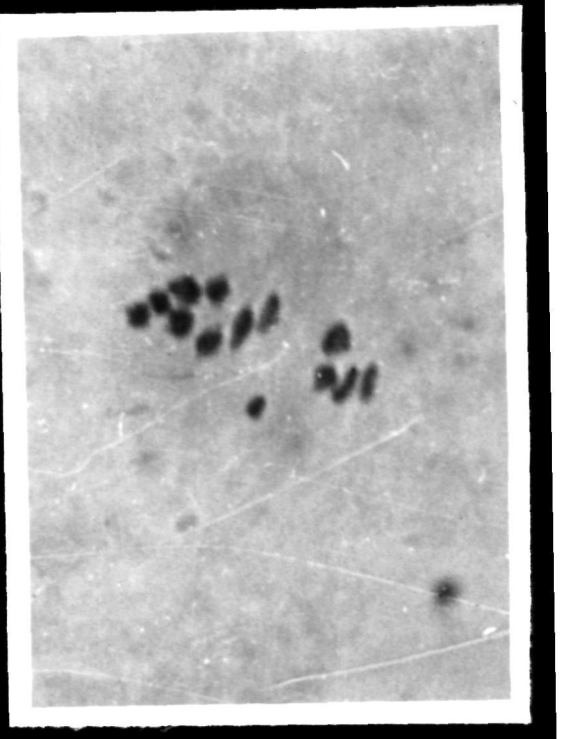
Plate 27: Metaanaphase I showing 12 II and a lagging univalent in the $F_1$ of triplo-5

Plate 28: Metaanaphase I showing 12 II and a lagging univalent in the $F_1$ of triplo-5

Plate 25



Plate 26



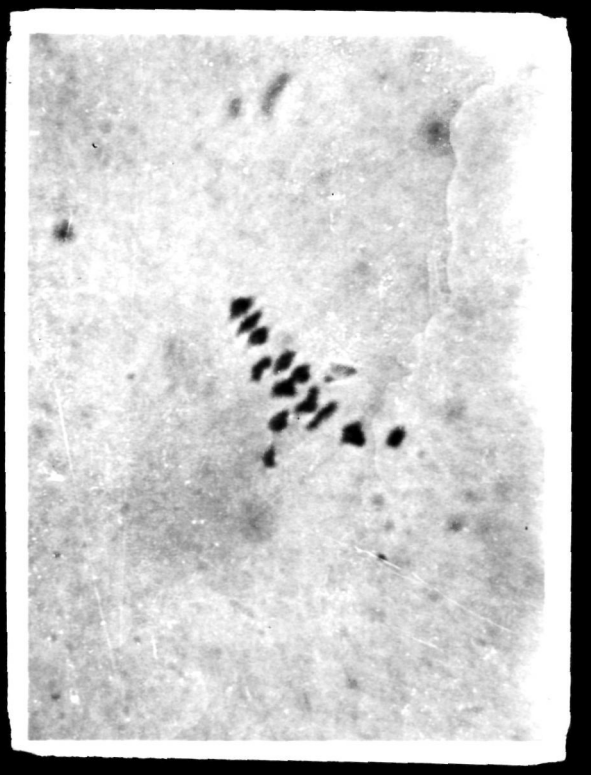




Plate 27

Plate 28

Plate 29: Metaanaphase showing 12 II + 1 I in the $F_1$ of triplo-6

Plate 30: Anaphase I showing uneQual distribution (12,13) in the $F_1$ of triplo-7

Plate 31: Metaanaphase depicting 12 II + 1 I in the $F_1$ of triplo-8

Plate 32: Metaanaphase depicting 12 II + 1 I in the $F_1$ of triplo-9

Plate 29

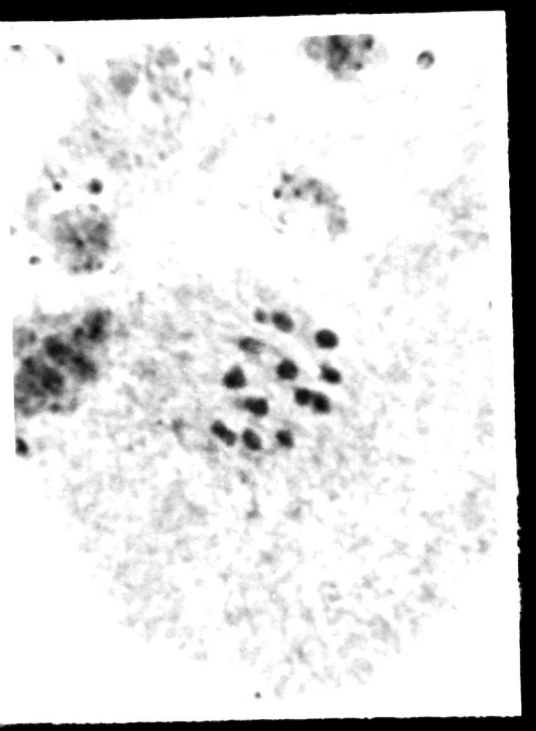


Plate 30





Plate 31

Plate 32

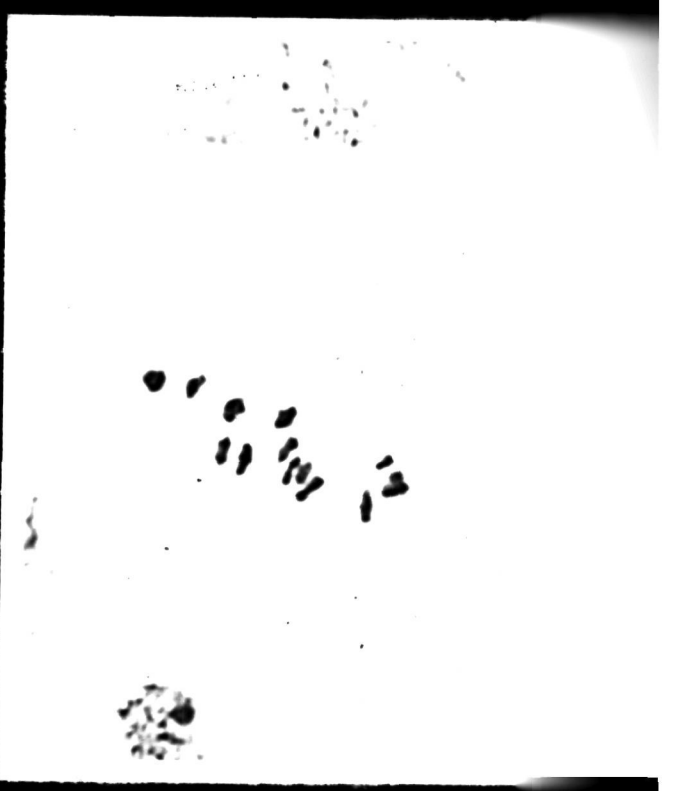


Plate 33: Polar view of metaanaphase showing 12 II + 1 I in the $F_1$ of triplo-10

Plate 34: Telophase I showing a dividing laggard in the $F_1$ of triplo-11

Plate 35: Diakinesis showing 12 II + 1 I in the $F_1$ of triplo-12

Plate 33

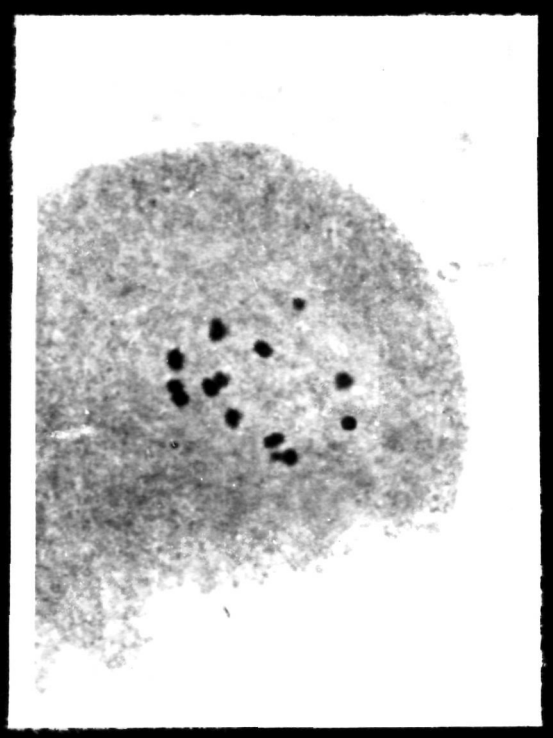


Plate 34



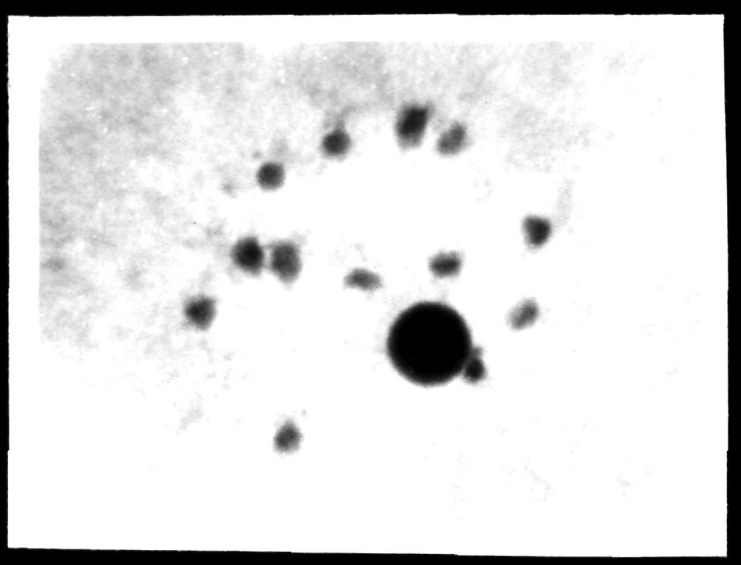


Plate 35

as per the recommended package of practices.

The observations regarding awning and presence or absence of pigmentation in various parts of the plant were recorded carefully at suitable stages.

3.2.8 Backcrossing with the disomic control Sona

Of the parental trisomics, triplo-2 and triplo-4 were self-incompatible and highly sterile respectively. They were backcrossed by dusting the pollen grains of the disomic control - Sona on the stigma of respective trisomics. Only two seeds were recovered in case of triplo-2 while none could be obtained in triplo-4.

Of the trisomic $F_1$s obtained, the $F_1$ of Triplo-4 x Crossa and one of the two $F_1$s of Triplo-10 x Crossa were highly sterile. The stubbles of these $F_1$ plants were vegetatively multiplied so as to obtain a few $F_2$ seeds. Inspite of this, only eight $F_2$ seeds of triplo-4 and two $F_2$ seeds of triplo-10 were obtained. Out of these only six $F_2$ plants of triplo-4 survived after utmost care, but none could survive in triplo-10. Under such circumstances, backcrossing was resorted to. The stigma of the $F_1$s of triplo-4 x Crossa and triplo-10 x Crossa were dusted with the pollen grains of disomic control Sona. Out of 500 back-pollinations four $BC_1F_1$ seeds were obtained in case of trisomic-4; whereas in case of trisomic-10, eight $BC_1F_1$ seeds were obtained after 400 back-pollinations.

Inspite of utmost care only two $BC_1F_1$ plants could survive in each of trisomic-4 as well as trisomic-10. The cytological examination of these plants were made to search for a trisomic $BC_1F_1$ plant. Both the $BC_1F_1$ plants of trisomic-10 and one of $BC_1F_1$ plants of trisomic-4 turned out to be normal disomics showing 12 II only, while, fortunately, one of the $BC_1F_1$ plants of trisomic-4 showed trisomic configuration of 12 II + 1 I as depicted in plate 26 (see also coloured plates 16 and 18).

### 3.2.8.1 Raising the $BC_1F_2$ population

The cytologically confirmed (2n + 1) $BC_1F_1$ plant of triplo-4 on selfing yielded 78 $BC_1F_2$ seeds. The properly dried $BC_1F_2$ seeds were gently dehusked, disinfected and were germinated in sterile petriplates. The germinated seeds were transferred in pulverized soil of shallow pots and the 30 days old seedlings were transplanted to properly puddled soil in earthen pots. All the 72 surviving $BC_1F_2$ plants were critically studied for the presence or absence of pigmentation in various plant parts at suitable stages of plant growth as well as for awning.

### 3.2.9 Raising the $F_3$ population

Out of the total 900 disomic $F_2$ plants, 29 $F_2$ plants were selected at random and corresponding 29 $F_3$ families were raised in the dry season of 1991. All the recommended

agronomic practices were followed in the nursery as well as in the field. The number of plants per family varied from 100 to 140 in the $F_3$ generation. The breeding behaviour of the $F_3$ families was critically studied by recording the presence or absence of pigmentation in different plant parts at suitable stages, as well as the presence or absence of awns.

3.2.10 Transmission rate studies

The transmission rate of the extra chromosome was studied critically from the selfed progenies of parental triplos as well as from the $F_2$ or backcross progenies of trisomics and marker crosses.

After the emergence of the panicle from the boot leaf, the panicles of each trisomics were covered with butter paper bags before the occurrence of anthesis. The fertile spikelets were collected after 30 days of proper maturation and kept inside desiccator. To obtain seeds, backcrosses were made in triplos-2 and 4 as discussed earlier (Refer 3.2.8).

The selfed seeds of all the trisomics except triplo-4 were gently dehusked, disinfected and were kept on sterile petriplates for germination. The germinated seeds were transferred to the pulverized soil in the shallow pots and the 30 to 35 days old surviving seedlings were transplanted in the properly prepared microplot. The recommended agronomic practices were properly taken care of.

The phenotypes of the individual plants raised from selfed seeds of all Sona trisomics were critically observed at different growth stages and were classified into disomics (2n) like, trisomics (2n + 1) like and non-disomic like plants. The percentage of 2n + 1 (trisomic) plants was computed. The "non-disomic like" plants were unusual, as phenotypically they were neither disomic-like nor like parental trisomics.

The experiment was conducted two times that is, in dry seasons of 1990 and 1991. Similar observations were made on $F_2$ or $BC_1F_2$ populations to estimate transmission frequencies of the extra chromosome.

### 3.2.11 Study of individual characters

The description of different parts of rice plant as standardized by Chang and Bardenas (1965) as well as others (Anonymous, 1980) and the system of gene symbols as reported by the Committee on gene symbolization, nomenclature and linkage groups (Anonymous, 1986) have been adopted in the present investigation. These rules for gene symbolization in principle, follow the international rules adopted earlier by International Rice Commission (Anonymous, 1959 and 1963).

A total of 31 characters comprising of 30 anthocyanin pigmentation characters and one morphological character that is, awning were chosen for this investigation. The characters considered and the phenotypic alternatives

studied are listed below:

| Characters | | Contrasting expression |
|---|---|---|
| I. | Anthocyanin pigmentation characters | |
| 1 | Culm base* | Green vs purple |
| 2 | Leaf sheath (external) | Green vs purple |
| 3 | Leaf sheath (internal) | Green vs purple |
| 4 | Prophyllum | Green vs purple |
| 5a | i) Leaf blade (seedling stage) | Green vs purple |
| | ii) Leaf tip (seedling stage) | Green vs purple |
| | iii) Leaf margin (seedling stage) | Green vs purple |
| 5b | i) Leaf blade (flowering stage) | Green vs purple |
| | ii) Leaf tip (flowering stage) | Green vs purple |
| | iii) Leaf margin (flowering stage) | Green vs purple |
| 6 | Leaf midrib | Green vs purple |
| 7 | Leaf axil | Green vs purple |
| 8 | Ligule | Green vs purple |
| 9 | Auricle | Green vs purple |
| 10 | Juncture | Green vs purple |
| 11 | Juncture back | Green vs purple |
| 12 | Internode | Green vs purple |
| 13 | Node | Green vs purple |
| 14 | Sheath pulvinus | Green vs purple |
| 15 | Pulvinus ring | Green vs purple |
| 16 | Glumes (empty) | Green vs purple |
| 17 | Lemma/palea | Green vs purple |
| 18 | Apiculus | Green vs purple |
| 19 | Stigma | Green vs purple |
| 20 | i) Panicle base | Green vs purple |
| | ii) Panicle pulvinus | Green vs purple |
| | iii) Panicle axis | Green vs purple |
| 21 | Nodal septum | Green vs purple |
| 22 | Nodal ring | Green vs purple |
| 23 | Awn colour (red awn) | Absence vs presence |
| II. | Morphological character | |
| 24 | Awning | Absence vs presence |

*Culm base pigmentation has been studied only in disomic $F_2$ population and not in trisomic $F_2$ and $F_3$ populations

The anthocyanin pigmentation characters in various plant parts were recorded at suitable stages. Critical observations were made wherever differences between pigmented and non-pigmented conditions were not distinct using a 10 X lens. Pigmentation in the nodal septum and the nodal ring was observed by taking a transverse section of the culm just below the basal node. The plants were finally classified as pigmented (purple) or non-pigmented (green) with reference to each plant part. Similarly, plants were classified as either awned or awnless.

### 3.2.12 Genetical analysis

A total of 900 disomic $F_2$ plants of cultivar Sona (control) x Crossa were critically observed for the presence or absence of pigmentation in all the different parts of the rice plant as well as for awning. The 12 trisomic $F_2$ populations were also observed similarly for all the characters under study. The disomic segregation ratio for individual character was computed for $F_2$ as well as for $BC_1F_2$ populations. For computation of $BC_1F_2$ ratio of individual character, the genotypes of the parents, $F_1$ and $BC_1F_1$ were worked out (Table 19).

If a gene is located on the extra chromosome of a trisomic, then the trisomic $F_2$ populations are likely to deviate from the disomic segregation ratios. This is known as a critical combination. On the other hand if the gene concerned is not located on the extra chromosome then the trisomic $F_2$ population may exhibit a fit with the disomic

segregation ratio which is often termed as a non-critical combination.

The trisomic families showing significant deviation at the 5 and 1 per cent levels in heterogeneity tests were marked as critical.

3.2.13 Statistical techniques

The expected values corresponding to the observed values for each character were calculated based on the genetic ratio hypothesized. The chi-square test was applied to test the significance of deviation using the formula -

$$\chi^2 = \sum \frac{(O - E)^2}{E} \quad \text{with } (n-1) \text{ df}$$

Where, $\sum$ = Summation over all classes

O = Observed frequencies

E = Expected frequencies and

n = Number of classes

The genetic hypothesis was confirmed fit when the calculated chi-square value was less than the table value (3.841) at 5 per cent level for one degree of freedom. However, the most appropriate genetic ratio for the character concerned was taken only after confirming the $F_3$ breeding behaviour.

In case of $F_3$, the data on segregating families were statistically analysed by applying the chi-square test. The families breeding true for particular characters were noted.

The whole data relating to the $F_3$ population were used to confirm the $F_2$ ratios.

3.2.13.1 Heterogeneity test

The chi-square test of heterogeneity was carried out to find out whether the observed segregation in trisomic $F_2$ populations was in conformity with that of disomic $F_2$ population. To test this hypothesis the following formula[95] in Panse and Sukhatme (1961) was employed.

$$\chi^2 = \frac{(n_1 + n_2)^2}{n_1 n_2} \left\{ \sum \frac{a^2}{a + a'} - \frac{n_1^2}{n_1 + n_2} \right\}$$

Where $n_1$ and $n_2$ are the corresponding column totals in the two fold classification and $a$ and $a'$ are the cell frequencies in a row; and $\sum$ corresponds to summation over all classes. When the calculated chi-square value was more than the table value at (n-1) df, it was inferred that there is distorted segregation thereby revealing that the gene(s) in question is/ are located on the critical chromosome.

3.2.14 Description of certain terms used

"Pigmented" invariably means purple anthocyanin pigment and "non-pigmented" is referred to green or white as applicable. Hence, these terms are synonymously used. In some instances, 'leaf sheath' has been mentioned as "sheath", and "nodal septum" as "septum".

The $F_1$ arising out of a cross Sona (2n)control x Crossa is termed as a '<u>disomic $F_1$</u>'. The'<u>disomic $F_1$s</u>' obtained out of the crosses, trisomics 1 to 12 x Crossaare termed as <u>non-critical $F_1$s</u> whereas the trisomic $F_1$s are termed as '<u>Critical $F_1$s</u>'. In a similar fashion the terms '<u>disomic $F_2$</u>', '<u>non-critical $F_2$s</u>' and '<u>critical $F_2$s</u>' have been used in the present investigations. The "non-disomic like" plants are those that appeared phenotypically different from the designated trisomics as well as the disomic. Such unusual plants were found in the selfed progenies as well as the $F_2$ progenies of trisomics.

3.2.15 <u>Abbreviations used</u>

BTD - Breeding true for dominant character,

BTR - Breeding true for recessive character,

O - Observed number of individuals,

E - Expected number of individuals

3.2.16 <u>Designations for trisomics</u>

In the present investigations old designations of the trisomics prevalent till December, 1990 have been used everywhere. However, wherever necessary, the new designations of triplos (Anonymous, 1990b) have been given along with the old designations.

CHAPTER - IV

# EXPERIMENTAL RESULTS

# IV. EXPERIMENTAL RESULTS

As detailed in the preceding chapters, the present investigation comprised three aspects of study on primary trisomic series of Oryza sativa L., sub sp. indica.

1. The inheritance pattern of the anthocyanin pigmentation characters and awning

2. Chromosomal location of the genes for pigmentation and awning, establishing gene-chromosome relationships and

3. Study on the transmission of extra chromosome through selfed progenies of parental triplos as well as through the $F_2$ or backcross populations of trisomics and marker crosses.

The results obtained from the investigation are presented aspectwise in the following pages.

## 4.1 Studies on inheritance pattern

The characters of the 13 female parents disomic Sona-control and its triplos-1 to 12, one male parent (Crossa), their 13 $F_1$s and one $BC_1F_1$ of triplo-4 along with autotriploid Sona (3N) were compared and the observations are presented in Table 10.

In disomic $F_1$, with regard to the expression of pigmentation in the characters namely, culm base, leaf sheath (external), prophyllum, leaf tip at seedling and flowering

Table 10. The qualitative characters of the parents along with the disomic and trisomic $F_1$s/$BC_1F_1$

| Parents $F_1$s and $BC_1F_1$ | | Culm base | Leaf sheath (Ext) | Leaf sheath (Int) | Prophyllum | Leaf blade (Seedling stage) | Leaf tip (S.S) | Leaf margin(S.S) | Leaf blade (Flowering stage) | Leaf tip (F.S) | Leaf margin (F.S) | Leaf midrib | Leaf axil | Ligule | Auricle | Junctura | Junctura back | Internode | Node | Sheath pulvinus | Pulvinus ring | Glumes(empty) | Lemma and palea | Apiculus | Stigma | Panicle base | Panicle pulvinus | Panicle axis | Nodal septum | Nodal ring | Red awn | Awning |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Sona (3N) | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | Absent | Present |
| $P_1$ | Sona disomic control & TP-1, 4, 5, 7, 8, 11 & 12 | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | Absent | Absent |
| $P_1$ | TP-2, 3, 6, 9 & 10 | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | G | Absent | Present |
| $P_2$ | Crossa | P | P | G | P | P | P | P | P | P | P | P | P | P | P | G | P | G | G | G | P | P | G | P | P | G | G | P | G | G | Absent | Absent |
| $F_1$ | Sona control(2n) x crossa | P | P | G | P | G | P | G | G | P | G | G | P | G | G | G | G | G | G | G | P | G | G | P | P | G | G | G | G | G | Absent | Absent |
| $F_1$ | TP-1 x Crossa | P | P | G | P | G | P | G | G | P | G | G | P | G | G | G | G | G | G | G | P | G | G | P | P | G | G | G | P | P | Absent | Absent |
| $F_1$ | TP-2 x Crossa | P | P | G | P | G | P | G | G | P | G | G | P | G | P | G | G | G | G | G | P | G | G | P | P | G | G | G | G | P | Present | Present |
| $F_1$ | TP-3 x Crossa | P | P | G | P | G | P | G | G | P | G | G | P | G | P | G | G | G | G | G | P | G | G | P | P | G | G | G | P | P | Present | Present |
| $F_1$ | TP-4 x Crossa | P | P | G | P | G | P | G | G | P | G | G | P | G | G | G | G | G | G | G | P | G | G | P | P | G | G | G | G | P | Absent | Absent |
| $BC_1F_1$ | (TP-4 x Crossa) x Sona control | P | P | G | P | G | P | G | G | P | G | G | P | G | G | G | G | G | G | G | P | G | G | P | P | G | G | G | P | G | Absent | Absent |
| $F_1$ | TP-5 x Crossa | P | P | G | P | G | P | G | G | P | G | G | P | G | G | G | G | G | G | G | P | G | G | P | P | G | G | G | G | G | Absent | Absent |
| $F_1$ | TP-6 x Crossa | P | P | G | P | G | P | G | G | P | G | G | P | G | P | P | G | G | G | G | P | G | G | P | P | G | G | G | G | P | Present | Present |
| $F_1$ | TP-7 x Crossa | P | P | G | P | G | P | G | G | P | G | G | P | G | G | G | G | G | G | G | P | G | G | P | P | G | G | G | G | P | Absent | Absent |
| $F_1$ | TP-8 x Crossa | P | P | G | P | G | P | P | G | P | P | G | P | G | P | P | G | G | G | G | P | G | G | P | P | G | G | G | G | G | Absent | Absent |
| $F_1$ | TP-9 x Crossa | P | P | G | P | G | P | G | G | P | G | G | P | G | G | G | G | G | G | G | P | G | G | P | P | G | G | G | G | G | Absent | Absent |
| $F_1$ | TP-10 x Crossa | P | P | G | P | G | P | G | G | P | G | G | P | G | G | G | G | G | G | G | P | G | G | P | P | G | G | G | G | G | Present | Present |
| $F_1$ | TP-11 x Crossa | P | P | G | P | G | P | G | G | P | G | G | P | G | G | G | G | G | G | G | P | G | G | P | P | G | G | G | G | G | Absent | Absent |
| $F_1$ | TP-12 x Crossa | P | P | G | P | G | P | G | G | P | G | G | P | G | G | G | G | G | G | G | P | G | G | P | P | G | G | G | G | P | Absent | Absent |

P = purple

G = green

stage, leaf axil, pulvinus ring, apiculus and stigma, the presence of pigmentation was dominant over its absence. All the remaining characters studied, showed a recessive nature in the disomic $F_1$'s due to the involvement of inhibitory genes.

The details of the observed frequencies of phenotypic segregations in disomic $F_2$ for each of the character, the expected frequencies of phenotypic segregations based on the assumed phenotypic ratios, the calculated chi-square with the levels of probability indicating the goodness of fit of observed frequencies to the assumed ratios are presented in Table 11. The different phenotypic ratios obtained in $F_2$ were confirmed by the $F_3$ breeding behaviour.

The segregation ratios for different characters obtained and their genetic explanations are as follows.

### 4.1.1 Pigmentation in leaf margin at seedling and flowering stage and awning

Phenotypic segregation in both of the above pigmentation characters and a morphological character showed a good fit for 3:13 ratio of pigmented to non-pigmented leaf margin at seedling and flowering stage and presence vs absence of awning indicating digenic interaction of a basic gene and an inhibitory gene.

The 3:13 ratio obtained in $F_2$ was confirmed by the $F_3$ breeding behaviour. The summary of $F_3$ breeding behaviour

Table 11. Phenotypic segregation of various characters in the $F_2$

| Sl. no. | Character | Contrasting expressions | Expected ratio | Phenotypic segregation in $F_2$ | | | $X^2$ | P |
|---|---|---|---|---|---|---|---|---|
| 1 | 2 | 3 | 4 | 5 | | | 6 | 7 |
| I. | Pigmentation characters | (+) (-) | | | (+) | (-) | | |
| 1. | Culm base* | Purple:Green | 45:19 | O | 707.00 | 268.00 | 2.2614 | 0.20-0.10 |
| | | | | E | 685.55 | 289.45 | | |
| 2. | Leaf sheath(External) | Purple:Green | 45 :19 | O | 651.00 | 249.00 | 1.7607 | 0.20-0.10 |
| | | | | E | 632.81 | 267.19 | | |
| 3. | Leaf sheath(Internal) | Purple :Green | 27:229 | O | 77.00 | 823.00 | 3.7827 | 0.10-0.05 |
| | | | | E | 94.92 | 805.08 | | |
| 4. | Prophyllum | Purple:Green | 45:19 | O | 631.00 | 269.00 | 0.0175 | 0.90-0.80 |
| | | | | E | 632.81 | 267.19 | | |
| 5. | Leaf blade (Seedling stage) | Purple:Green | 9:55 | O | 116.00 | 784.00 | 1.0258 | 0.30-0.20 |
| | | | | E | 126.56 | 773.44 | | |
| 6. | Leaf tip (Seedling stage) | Purple:Green | 45:19 | O | 655.00 | 245.00 | 2.6204 | 0.20-0.10 |
| | | | | E | 632.81 | 267.19 | | |
| 7. | Leaf margin (Seedling stage) | Purple:Green | 3:13 | O | 181.00 | 719.00 | 1.0945 | 0.30-0.20 |
| | | | | E | 168.75 | 731.25 | | |
| 8. | Leaf blade (Flowering stage) | Purple:Green | 9:55 | O | 116.00 | 784.00 | 1.0258 | 0.30-0.20 |
| | | | | E | 126.56 | 773.44 | | |
| 9. | Leaf tip (Flowering stage) | Purple:Green | 45:19 | O | 640.00 | 260.00 | 0.2750 | 0.70-0.50 |
| | | | | E | 632.81 | 267.19 | | |
| 10. | Leaf margin (Flowering stage) | Purple:Green | 3:13 | O | 179.00 | 721.00 | 0.7663 | 0.50-0.30 |
| | | | | E | 168.75 | 731.25 | | |
| 11. | Leaf midrib | Purple:Green | 9:55 | O | 116.00 | 784.00 | 1.0258 | 0.30-0.20 |
| | | | | E | 126.56 | 773.44 | | |
| 12. | Leaf axil | Purple:Green | 45:19 | O | 614.00 | 286.00 | 1.8838 | 0.20-0.10 |
| | | | | E | 632.81 | 267.19 | | |
| 13. | Ligule | Purple:Green | 9:55 | O | 147.00 | 753.00 | 3.8403 | 0.10-0.05 |
| | | | | E | 126.56 | 773.44 | | |
| 14. | Auricle | Purple:Green | 9:55 | O | 137.00 | 763.00 | 1.0016 | 0.50-0.30 |
| | | | | E | 126.56 | 773.44 | | |
| 15. | Junctura | Purple:Green | 3:253 | O | 12.00 | 888.00 | 0.2026 | 0.70-0.50 |
| | | | | E | 10.55 | 889.45 | | |
| 16. | Junctura back | Purple:Green | 9:55 | O | 147.00 | 753.00 | 3.8403 | 0.10-0.05 |
| | | | | E | 126.56 | 773.44 | | |
| 17. | Internode | Purple:Green | 9:247 | O | 33.00 | 867.00 | 0.0605 | 0.90-0.80 |
| | | | | E | 31.64 | 868.36 | | |

Continued..

Table 11 continued

| 1 | 2 | 3 | 4 | 5 | | | 6 | 7 |
|---|---|---|---|---|---|---|---|---|
| | | (+) (-) | | | (+) | (-) | | |
| 18. | Node | Purple:Green | 9:247 | O | 30.00 | 870.00 | 0.0882 | 0.80-0.70 |
| | | | | E | 31.64 | 868.36 | | |
| 19. | Sheath pulvinus | Purple:Green | 3:253 | O | 10.00 | 890.00 | 0.0287 | 0.90-0.80 |
| | | | | E | 10.55 | 889.45 | | |
| 20. | Pulvinus ring | Purple:Green | 45:19 | O | 651.00 | 249.00 | 1.7607 | 0.20-0.10 |
| | | | | E | 632.81 | 267.19 | | |
| 21. | Glumes(empty) | Purple:Green | 9:55 | O | 115.00 | 785.00 | 1.2292 | 0.30-0.20 |
| | | | | E | 126.56 | 773.44 | | |
| 22. | Lemma/Palea | Purple:Green | 9:247 | O | 21.00 | 879.00 | 3.7088 | 0.10-0.05 |
| | | | | E | 31.64 | 868.36 | | |
| 23. | Apiculus | Purple:Green | 45:19 | O | 655.00 | 245.00 | 2.6204 | 0.20-0.10 |
| | | | | E | 632.81 | 267.19 | | |
| 24. | Stigma | Purple:Green | 45:19 | O | 651.00 | 249.00 | 1.7607 | 0.20-0.10 |
| | | | | E | 632.81 | 267.19 | | |
| 25. | Panicle base | Purple:Green | 9:247 | O | 31.00 | 869.00 | 0.0134 | 0.95-0.90 |
| | | | | E | 31.64 | 868.36 | | |
| 26. | Panicle pulvinus | Purple:Green | 9:247 | O | 26.00 | 874.00 | 1.0422 | 0.50-0.30 |
| | | | | E | 31.64 | 868.36 | | |
| 27. | Panicle axis | Purple:Green | 27:229 | O | 101.00 | 799.00 | 0.4351 | 0.70-0.50 |
| | | | | E | 94.92 | 805.08 | | |
| 28. | Nodal septum | Purple:Green | 21:43 | O | 274.00 | 626.00 | 2.2894 | 0.20-0.10 |
| | | | | E | 295.13 | 604.69 | | |
| 29. | Nodal ring | Purple:Green | 27:229 | O | 84.00 | 816.00 | 1.4049 | 0.30-0.20 |
| | | | | E | 94.92 | 805.08 | | |
| 30. | Awn colour (Red awn) | Presence:Absence | 9:55 | O | 111.00 | 789.00 | 2.2267 | 0.20-0.10 |
| | | | | E | 126.56 | 773.44 | | |
| II. | Morphological character | | | | | | | |
| 31. | Awning | Presence:Absence | 3:13 | O | 167.00 | 733.00 | 0.0223 | 0.90-0.80 |
| | | | | E | 168.75 | 731.25 | | |

* Culm base pigmentation has not been studied in trisomic $F_2$ and $F_3$ populations; Only while uprooting the disomic $F_2$ seedlings culm base pigmentation was recorded.

in respect of these characters is presented in Table 12.

4.1.2 Pigmentation in culm base, leaf sheath (external), prophyllum, leaf tip at seedling and flowering stage, leaf axil, pulvinus ring, apiculus and stigma

Phenotypic segregations for all the nine pigmentation characters showed a segregation ratio of 45 pigmented to 19 non-pigmented indicating the involvement of three interacting genes namely, one basic complementary and two complementary duplicate genes.

The 45:19 ratio obtained in the $F_2$ was confirmed by the $F_3$ breeding behaviour in all the above characters except in case of culm base pigmentation. The summary of $F_3$ breeding behaviour in respect of rest of the eight characters is presented in Table 13.

4.1.3 Pigmentation in leaf blade at seedling and flowering stage, leaf midrib, ligule, auricle, juncture back, glumes and presence vs absence of red awn

All the above eight pigmentation characters showed a good fit to a 9:55 ratio of pigmented to non-pigmented, meaning thereby that pigmentation in these parts was due to the interaction of three genes namely, two complementary and one inhibitory genes.

The 9:55 ratio observed in the $F_2$ was further confirmed by the breeding behaviour of $F_3$ in all these

Table 12. Breeding behaviour of the characters showing 3:13 $F_2$ ratio in $F_3$ generation

| Sl. no. | Expected frequencies based on the $F_2$ ratio of 3:13(1:2:2:4:7) | | Number of families | | | | | $x^2$ | 'p' |
|---|---|---|---|---|---|---|---|---|---|
| | | | BTD | 1:3 | 3:1 | 3:13 | BTR | | |
| | | E | 1.81 | 3.63 | 3.63 | 7.25 | 12.68 | | |
| | Characters | | | | | | | | |
| 1 | Leaf margin (seedling stage) | O | 1.00 | 3.00 | 2.00 | 7.00 | 16.00 | 2.0816 | 0.80 - 0.70 |
| 2 | Leaf margin (flowering stage) | O | 1.00 | 3.00 | 2.00 | 7.00 | 16.00 | 2.0816 | 0.80 - 0.70 |
| 3 | Awning | O | 2.00 | 2.00 | 4.00 | 7.00 | 14.00 | 0.9356 | 0.95 - 0.90 |

O = Observed number of families

E = Expected number of families

Table 13. $F_3$ breeding behaviour of the characters showing 45:19 $F_2$ ratio

| Sl. no. | Expected frequencies based on the $F_2$ ratio of 45:19 (7:18:8:4:8:19) | | Number of families | | | | | | $X^2$ | 'p' |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | BTD | 3:1 | 9:7 | 15:1 | 45:19 | BTR | | |
| | | E | 3.17 | 8.16 | 3.63 | 1.81 | 3.63 | 8.60 | | |
| | Characters | | | | | | | | | |
| 1 | Leaf sheath (external) | O | 5.00 | 8.00 | 5.00 | 0.00 | 4.00 | 7.00 | 3.7220 | 0.70 - 0.50 |
| 2 | Prophyllum | O | 5.00 | 7.00 | 5.00 | 1.00 | 4.00 | 7.00 | 2.4363 | 0.80 - 0.70 |
| 3 | Leaf tip (seedling stage) | O | 5.00 | 7.00 | 5.00 | 1.00 | 4.00 | 7.00 | 2.4363 | 0.80 - 0.70 |
| 4 | Leaf tip (flowering stage) | O | 5.00 | 7.00 | 5.00 | 1.00 | 4.00 | 7.00 | 2.4363 | 0.80 - 0.70 |
| 5 | Leaf axil | O | 5.00 | 7.00 | 5.00 | 1.00 | 4.00 | 7.00 | 2.4363 | 0.80 - 0.70 |
| 6 | Pulvinus ring | O | 5.00 | 7.00 | 5.00 | 1.00 | 4.00 | 7.00 | 2.4363 | 0.80 - 0.70 |
| 7 | Apiculus | O | 5.00 | 7.00 | 5.00 | 1.00 | 4.00 | 7.00 | 2.4363 | 0.80 - 0.70 |
| 8 | Stigma | O | 5.00 | 7.00 | 5.00 | 1.00 | 4.00 | 7.00 | 2.4363 | 0.80 - 0.70 |

O = Observed number of families

E = Expected number of families

characters. The summary of $F_3$ breeding behaviour in respect of these characters is presented in Table 14.

4.1.4 Pigmentation in nodal septum

Pigmented and non-pigmented nodal septum segregated in the ratio of 21:43 which suggested the operation of three interacting genes, of which, one was a basic gene and two were complementary inhibitory genes to condition pigmentation in the nodal septum.

The 21:43 ratio observed in the $F_2$ was further confirmed by the $F_3$ breeding behaviour. The summary of $F_3$ breeding behaviour for this character is presented in Table 15.

4.1.5 Pigmentation in internode, node, lemma/palea, panicle base and panicle pulvinus

All the above five pigmentation characters showed a phenotypic segregation of 9:247 for pigmented to non-pigmented implying thereby the role of four interacting genes. Thus, two complementary genes and two inhibitory duplicate genes were involved for the expression of these characters.

The tetragenic ratio of 9:247 observed in the $F_2$ was also confirmed by the $F_3$ breeding behaviour; the summary of which is presented in Table 16.

Table 14. $F_3$ breeding behaviour of the characters showing 9:55 $F_2$ ratio

| Sl. no. | Expected frequencies based on the $F_2$ ratio of 9:55(1:2:4:8:4:8:37 | | Number of families | | | | | | | $\chi^2$ | 'P' |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | BTD | 1:3 | 3:1 | 3:13 | 9:7 | 9:55 | BTR | | |
| | | E | 0.45 | 0.91 | 1.81 | 3.63 | 1.81 | 3.63 | 16.76 | | |
| | **Characters** | | | | | | | | | | |
| 1 | Leaf blade (seedling stage) | O | 1.00 | 1.00 | 2.00 | 3.00 | 0.00 | 6.00 | 16.00 | 4.2022 | 0.70 - 0.50 |
| 2 | Leaf blade (flowering stage) | O | 1.00 | 1.00 | 2.00 | 3.00 | 0.00 | 6.00 | 16.00 | 4.2022 | 0.70 - 0.50 |
| 3 | Leaf midrib | O | 1.00 | 1.00 | 2.00 | 3.00 | 0.00 | 6.00 | 16.00 | 4.2022 | 0.70 - 0.50 |
| 4 | Ligule | O | 1.00 | 1.00 | 2.00 | 2.00 | 1.00 | 6.00 | 16.00 | 3.3773 | 0.80 - 0.70 |
| 5 | Auricle | O | 1.00 | 1.00 | 2.00 | 3.00 | 0.00 | 6.00 | 16.00 | 4.2022 | 0.70 - 0.50 |
| 6 | Junctura back | O | 1.00 | 1.00 | 2.00 | 3.00 | 1.00 | 5.00 | 16.00 | 1.7244 | 0.95 - 0.90 |
| 7 | Glumes | O | 1.00 | 1.00 | 2.00 | 3.00 | 0.00 | 6.00 | 16.00 | 4.2022 | 0.70 - 0.50 |
| 8 | Red awn (Awn colour) | O | 1.00 | 2.00 | 1.00 | 2.00 | 1.00 | 2.00 | 20.00 | 4.7930 | 0.70 - 0.50 |

O = Observed number of families

E = Expected number of families

Table 15. $F_3$ breeding behaviour of the character showing 21:43 $F_2$ ratio

| Expected frequencies based on the $F_2$ ratio of 21:43 (7:4:14:8:4:8:19) | | Number of families | | | | | | | $x^2$ | 'p' |
|---|---|---|---|---|---|---|---|---|---|---|
| | | BTD | 1:3 | 3:1 | 3:13 | 7:9 | 21:43 | BTR | | |
| | E | 3.17 | 1.81 | 6.34 | 3.63 | 1.81 | 3.63 | 8.61 | | |
| Character | | | | | | | | | | |
| Modal septum | O | 4.00 | 3.00 | 4.00 | 1.00 | 3.00 | 2.00 | 12.00 | 6.6179 | 0.50-0.30 |

O = Observed number of families
E = Expected number of families

Table 16. $F_3$ breeding behaviour of the characters showing 9:247 $F_2$ ratio

| Sl. No. | Expected frequencies based on the $F_2$ ratio of 9:247 (1:4:4:4:16:4:16:16:16:175) | | Number of families | | | | | | | | | BTR | $x^2$ | 'P' |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | BTD | 1:3 | 3:1 | 1:15 | 3:13 | 9:7 | 1:63 | 9:55 | 9:247 | | | |
| | | E | 0.13 | 0.45 | 0.45 | 0.45 | 1.81 | 0.45 | 1.81 | 1.81 | 1.81 | 19.83 | | |
| | Character | | | | | | | | | | | | | |
| 1 | Internode | 0 | 0.00 | 0.00 | 1.00 | 1.00 | 0.00 | 0.00 | 0.00 | 1.00 | 2.00 | 24.00 | 7.2538 | 0.70 - 0.50 |
| 2 | Node | 0 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 1.00 | 28.00 | 11.0885 | 0.30 - 0.20 |
| 3 | Lemma/ Palea | 0 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 29.00 | 13.4105 | 0.20 - 0.10 |
| 4 | Panicle pulvinus | 0 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 1.00 | 2.00 | 1.00 | 25.00 | 5.8128 | 0.80 - 0.70 |
| 5 | Panicle base | 0 | 0.00 | 0.00 | 0.00 | 1.00 | 0.00 | 0.00 | 0.00 | 2.00 | 1.00 | 25.00 | 7.5026 | 0.70 - 0.50 |

O = Observed number of families

E = Expected number of families

### 4.1.6 Pigmentation in leaf sheath (internal), panicle axis and nodal ring

Phenotypic segregations for all these three pigmentation characters showed a tetragenic ratio of 27 pigmented to 229 non-pigmented indicating that there existed three complementary genes and one inhibitory gene.

The tetragenic ratio of 27:229 obtained in the $F_2$ was confirmed by the $F_3$ breeding behaviour in all the characters. The summary of breeding behaviour of $F_3$ is presented in Table 17.

### 4.1.7 Pigmentation in juncture and sheath pulvinus

A tetragenic interaction involving one basic gene and three duplicate inhibitory genes was observed for the expression of pigmentation in these two parts.

The phenotypic segregation of 3 pigmented to 253 non-pigmented observed in $F_2$ was further confirmed by the $F_3$ breeding behaviour. The summary of the $F_3$ breeding behaviour is presented in Table 18.

From the study of the inheritance of individual characters in $F_2$ and $F_3$ generations in the cross Sona (disomic) control x Crossa, it was possible to assign the genotypic constitution for each character as observed in the two parents, and these are presented in Table 19. The genotypic constitution assigned to the parents for each of the

Table 17. $F_3$ breeding behaviour of the characters showing 27:229 $F_2$ ratio

| Sl. No. | Expected frequencies based on the $F_2$ ratio of 27:229 (1:2:6:12:12:24:8:16:175) | | Number of families | | | | | | | | | $\chi^2$ | 'p' |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | BTD | 1:3 | 3:1 | 3:13 | 9:7 | 9:55 | 27:37 | 27:229 | BTR | | |
| | | E | 0.11 | 0.23 | 0.68 | 1.36 | 1.36 | 2.72 | 0.91 | 1.81 | 19.82 | | |
| | Characters | | | | | | | | | | | | |
| 1 | Leaf sheath (internal) | O | 0.00 | 1.00 | 2.00 | 0.00 | 0.00 | 2.00 | 1.00 | 0.00 | 23.00 | 10.4809 | 0.30 - 0.20 |
| 2 | Panicle axis | O | 0.00 | 1.00 | 1.00 | 2.00 | 1.00 | 3.00 | 0.00 | 2.00 | 19.00 | 4.2276 | 0.90 - 0.80 |
| 3 | Nodal ring | O | 0.00 | 0.00 | 0.00 | 1.00 | 0.00 | 2.00 | 0.00 | 2.00 | 24.00 | 4.4774 | 0.90 - 0.80 |

O = Observed number of families

E = Expected number of families

Table 18. $F_3$ breeding behaviour of the characters showing 3:253 $F_2$ ratio

| Sl. no. | Expected frequencies based on the $F_2$ ratio of 3:253 (1:6:2:12:12:8:24:16:175) | | Number of families | | | | | | | | | $x^2$ | 'p' |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | BTD | 1:3 | 3:1 | 1:15 | 3:13 | 1:63 | 3:61 | 3:253 | BTR | | |
| | | E | 0.11 | 0.68 | 0.23 | 1.36 | 1.36 | 0.91 | 2.72 | 1.81 | 19.82 | | |
| | Character | | | | | | | | | | | | |
| 1 | Juncture | 0 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 1.00 | 28.00 | 11.1085 | 0.20 - 0.10 |
| 2 | Sheath pulvinus | 0 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 29.00 | 13.4319 | 0.10 - 0.05 |

O = Observed number of families

E = Expected number of families

Table 19 . Phenotype and genotype of characters of the parents, Sona and Crossa

| Sl. No. | Characters | Sona Phenotype | Sona Genotype | Crossa Phenotype | Crossa Genotype |
|---|---|---|---|---|---|
| I. | Anthocyanin Pigmentation characters | | | | |
| 1. | Culm base | Green | $pcb_a\ pcb_a\ pcb_{b_1}\ pcb_{b_1}\ pcb_{b_2}\ pcb_{b_2}$ | Purple | $Pcb_aPcb_a\ Pcb_{b_1}Pcb_{b_1}\ Pcb_{b_2}\ Pcb_{b_2}$ |
| 2. | Leaf sheath (external) | Green | $psh_a\ psh_a\ psh_{b_1}\ psh_{b_1}\ psh_{b_2}\ psh_{b_2}$ | Purple | $Psh_aPsh_a\ Psh_{b_1}Psh_{b_1}\ Psh_{b_2}\ Psh_{b_2}$ |
| 3. | Leaf sheath (internal) | Green | $psh(int)_a\ psh(int)_a psh(int)_b\ psh_{(int)_b}$ $psh(int)_c\ psh(int)_c i psh(int) i psh_{(int)}$ | Green | $Psh(int)_aPsh(int)_aPsh(int)_bPsh(int$ $Psh(int)_cPsh(int)_cI\text{-}Psh(int)$ $I\text{-}Psh(int)$ |
| 4. | Prophyllum | Green | $ppr_appr_a\ ppr_{b_1}\ ppr_{b_1}ppr_{b_2}\ ppr_{b_2}$ | Purple | $ppr_a\ ppr_a\ ppr_{b_1}\ ppr_{b_1}\ ppr_{b_2}\ ppr_{b_2}$ |
| 5a. | Leaf blade i. (Seedling stage) | Green | $pl(s)_a\ pl(s)_a\ pl(s)_b\ pl(s)_b$ I-Pl I-Pl | Purple | $Pl(s)_aPl(s)_aPl(s)_bPl(s)_b$ i-Pl i-Pl |
| 5a. | Leaf tip ii(Seedling stage) | Green | $pla(s)_apla(s)_a\ pla(s)_{b_1}\ pla(s)_{b_1}$ $pla(s)_{b_2}\ pla(s)_{b_2}$ | Purple | $Pla(S)_aPla(S)_aPla(S)b_1Pla(S)_{b_1}$ $Pla(S)_{b_2}\ Pla(S)_{b_2}$ |
| 5a. | Leaf margin iii.(Seedling stage) | Green | plm(s) plm(s) I-Plm(s) I-Plm(s) | Purple | Plm(s)Plm(s) iPlm(s) iPlm(s) |
| 5b. | Leaf blade i. (Flowering stage) | Green | plb pla plb plb I-Pl I-Pl | Purple | $Pl_aPl_aPl_bPl_b$ i-Pl i-Pl |
| 6b. | Leaf tip ii.(Flowering stage) | Green | $pla_a\ pla_a\ pla_{b_1}\ pla_{b_1}\ pla_{b_2}\ pla_{b_2}$ | Purple | $Pla_aPla_aPla_{b_1}\ Pla_{b_1}Pla_{b_2}\ Pla_{b_2}$ |
| 5b. | Leaf margin iii.(flowering stage) | Green | plm plm I-Plm I-Plm | Purple | Plm Plm i-Plm i-Plm |
| 6. | Leaf midrib | Green | $pmr_a\ pmr_a\ pmr_b\ pmr_b$ I-Pmr I-Pmr | Purple | $Pmr_aPmr_aPmr_bPmr_b$ i-Pmr i-Pmr |
| 7. | Leaf axil | Green | $pxa\ pxa\ pxb_1\ pxb_1\ pxb_2\ pxb_2$ | Purple | $Px_aPx_aPx_{b_1}Px_{b_1}Px_{b_2}\ Px_{b_2}$ |
| 8. | Ligule | Green | $plg_a\ plg_a\ plg_b\ plg_b$ I-Plg I-Plg | Purple | $Plg_aPlg_aPlg_bPlg_b$ i-Plg i-Plg |
| 9. | Auricle | Green | $pau_a\ pau_a\ pau_b\ pau_b$ I-Pau I-Pau | Purple | $Pau_aPau_aPau_bPau_b$ i-Pau i-Pau |
| 10. | Juncture | Green | pjpj i-$Pj_1$ i$Pj_1$ i$Pj_2$ i$Pj_2$ i$Pj_3$ i$Pj_3$ | Green | $P_jP_jI\text{-}P_{j_1}I\text{-}P_{j_1}I\text{-}P_{j_2}IP_{j_2}\ I\text{-}P_{j_3}\ I\text{-}P_{j_3}$ |
| 11. | Juncture back | Green | $pjb_a\ pjb_a\ pjb_bpjb_b$ I-Pjb I-Pjb | Purple | $P_{jb_a}P_{jb_a}P_{jb_b}P_{jb_b}$ i-Pjb i-Pjb |
| 12. | Internode | Green | $pin_apin_a\ pin_b\ pin_b$ I-$Pin_1$ I-$Pin_1$ I-$Pin_2$ I-$Pin_2$ | Green | $Pin_aPin_aPin_bPin_b$ I-$Pin_1$ I-$Pin_1$ I-$Pin_2$ I-$Pin_2$ |
| 13. | Node | Green | $pn_apn_a\ pn_b\ pn_b$ i-$Pn_1$ i-$Pn_1$ i-$Pn_2$ i-$Pn_2$ | Green | $Pn_aPn_aPn_bPn_b$ I-$Pn_1$I-$Pn_1$I-$Pn_2$ I-$Pn_2$ |
| 14. | Sheath pulvinus | Green | pupu i-$Pu_1$i$Pu_1$ i-$Pu_2$ i-$Pu_2$ i-$Pu_3$ i-$Pu_3$ | Green | PuPu I-$Pu_1$ I-$Pu_1$ I-$Pu_2$ I-$Pu_2$ I-$Pu_3$ I-$Pu_3$ |
| 15. | Pulvinus ring | Green | $pur_a\ pur_a\ pur_{b_1}\ pur_{b_1}\ pur_{b_2}\ pur_{b_2}$ | Purple | $Pur_aPur_aPur_{b_1}\ Pur_{b_1}\ Pur_{b_2}Pur_{b_2}$ |
| 16. | Glumes(empty) | Green | $pg_apg_a\ pg_bpg_b$ I-Pg I-Pg | Purple | $Pg_aPg_a\ Pg_bPg_b$ i-Pg i-Pg |
| 17. | Lemma/palea | Green | $pr_apr_a\ pr_b\ pr_b$ i-$Pr_1$ i-$Pr_1$ i-$Pr_2$ i-$Pr_2$ | Green | $Pr_aPr_aPr_bPr_b$ I-$Pr_1$ I-$Pr_1$ I-$Pr_2$ I-$Pr_2$ |
| 18. | Apiculus | Green | $pa_apa_a\ pa_{b_1}\ pa_{b_1}\ pa_{b_2}\ pa_{b_2}$ | Purple | $Pa_a\ Pa_aPa_{b_1}\ Pa_{b_1}\ Pa_{b_2}\ Pa_{b_2}$ |
| 19. | Stigma | Green | $ps_a\ ps_a\ ps_{b_1}\ ps_{b_1}\ ps_{b_2}\ ps_{b_2}$ | Purple | $Ps_aPs_aPs_{b_1}\ Ps_{b_1}\ Ps_{b_2}Ps_{b_2}$ |

continued ..

Table 19 contd.

| 1 | 2 | 3 | 4 | 5 | 6 |
|---|---|---|---|---|---|
| 20.i. | Panicle base | Green | $ppb_a$ $ppb_a$ $ppb_b$ $ppb_b$ i-$Ppb_1$ i-$Ppb_1$ i-$Ppb_2$ i-$Ppb_2$ | Green | $P_{pb_a}$ $P_{pb_a}$ $P_{pb_b}$ $P_{pb_b}$ I-$P_{pb_1}$ I-$P_{pb_1}$ |
| 20.ii. | Panicle pulvines | Green | $ppu_a$ $ppu_a$ $ppu_b$ $ppu_b$ i-$Ppu_1$ i-$Ppu_1$ i-$Ppu_2$ i-$Ppu_2$ | Purple | $P_{pu_a}$ $P_{pu_a}$ $P_{pu_b}$ $P_{pu_b}$ I-$P_{pu_1}$ I-$P_{pu_1}$ I-$P_{pu_2}$ I-$P_{pu_2}$ |
| 20.iii. | Panicle axis | Green | $ppa_a$ $ppa_a$ $ppa_b$ $ppa_b$ $ppa_b$ $ppa_c$ I-Ppa I-Ppa | Purple | $P_{pa_a}$ $P_{pa_a}$ $P_{pa_b}$ $P_{pa_b}$ $P_{pa_c}$ $P_{pa_c}$ i-Ppa i-Ppa |
| 21. | Nodal septum | Green | pm pm i-$Pm_a$ i-$Pm_a$ i-$Pm_b$ i-$Pm_b$ | Green | Pm Pm I-$Pm_a$ I-$Pm_a$ I-$Pm_b$ I-$Pm_b$ |
| 22. | Nodal ring | Green | $pnr_a$ $pnr_a$ $pnr_b$ $pnr_b$ $pnr_c$ $pnr_c$ i-Pnr i-Pnr | Green | $Pnr_a$ $Pnr_a$ $Pnr_b$ $Pnr_b$ $Pnr_c$ $Pnr_c$ I-Pnr I-Pnr |
| 23. | Awn colour (Red awn) | Absent | $Ran_a$ $Ran_a$ $ran_b$ $ran_b$ i-Ran i-Ran | Absent | $ran_a$ $ran_a$ $Ran_b$ $Ran_b$ I-Ran I-Ran |
| II. | Morphological character: | | | | |
| 24. | Awning | (absent) Awnless | An An I-An I-An | (Absent) Awnless | an an i-An i-An |

* Both the parents are homozygous for 'C', 'A' and 'P' loci of C-A-P Complementary genic system.

character disregards the phenomenon of linkage and pleiotropy and hence is tentative.

## 4.2 Chromosomal location of genes for pigmentation and awning

Since the beginning of genetical studies in rice, the anthocyanin pigmentation genes have been studied extensively by various workers. Based on the cross-over values, these genes have been positioned to the corresponding linkage groups routinely by almost all the workers, hitherto. In the present study a novel attempt has been made for the chromosomal location of the pigmentation genes using the primary trisomic technique.

A total of 29 pigmentation characters and a morphological character were chosen for study. The marker parent (Crossa) having these genes was crossed with all the 12 primary trisomics as well as with the disomic 'Sona' using the marker as donor parent. The $F_1$s having 2n + 1 chromosomes were selfed and the $F_2$ populations were grown for critical observations for the presence of the marker characters.

Of the parental trisomics, triplo-4 is highly sterile; and hence, it is extremely difficult to obtain any seed out of it. Still, however, after attempting several pollinations, two underdeveloped $F_1$ caryopses were obtained. The seedlings were raised aseptically by inoculating the $F_1$ caryopses on 1/2 M.S. media. The 2n + 1 $F_1$ obtained was

also highly sterile and so the $F_1$ plant was multiplied vegetatively to obtain adequate $F_2$ population. Only six $F_2$ plants were recovered out of 10 $F_2$ seeds obtained. As discussed in preceding chapter, 72 $BC_1F_2$ plants were also obtained in case of this trisomic. Both the $F_2$ as well as the $BC_1F_2$ populations were grown and critical observations were made for the presence of the marker character. However, in case of $F_2$ involving trisomic-4 the population of only six plants is too small to arrive at any conclusion and hence the data of $BC_1F_2$ plants have been relied upon.

The number and nature of genes controlling the characters under study were determined by subjecting the observed frequencies in the $F_2$ of the cross involving the diploid parent Sona (control) and the donor parent Crossa, to chi-square test so as to find out the goodness of fit of different genetic ratios. The ratio that was statistically and genetically found valid was selected (Table 11). The $F_2$ segregations were tested for expected normal segregation ratios for each of the trisomic crosses.

The critical trisomic (s) was identified for each character based on the extent of deviation of the observed frequencies in the $F_2$ population of crosses of each of the trisomics with the donor parent from that of the observed frequencies in the $F_2$ population of the cross of disomic control with donor parent. Wherever the deviation was significant as evidenced from heterogeneity test at 5 per cent and 1 per

cent levels (0.05 and 0.01 levels of probability), the trisomic was marked as critical and as carrying gene(s) governing the character.

### 4. 2.1 Anthocyanin pigmentation characters

#### 4.2.1.1 Leaf sheath (external) pigmentation

The pigmentation of leaf sheath (external) in the parents as well as in the disomic and trisomic $F_1$s is presented in Table 10. The presence of pigmentation was dominant over its absence in case of leaf sheath (external) and hence, as expected, the disomic and all the trisomic $F_1$s showed purple pigmentation in this part.

The $F_2$ segregation of leaf sheath (external pigmentation in trisomics and marker crosses is presented in Table 20. The data revealed that pigmentation in leaf sheath (external) was controlled by three interacting genes as presented earlier (refer 4.1.2). The $F_2$ population of trisomics 1, 2, 10 and 12 and the $BC_1F_2$ population of trisomic-4 showed significant deviation from the expected 45:19 and 19.02 : 44.98 segregation ratios respectively at 0.01 level of probability. The $BC_1F_2$ population involving trisomic-4 showed a higher proportion of plants with non-pigmented leaf sheath (external) and with a calculated chi-square value of 20.1266. The $F_2$ populations of trisomics 1, 2 and 10 also had more than expected proportion of plants with non-pigmented leaf sheath

Table 20. $F_2$ segregation of leaf sheath (external) pigmentation in trisomics and their crosses

| Cross combination | | $F_2$ phenotypic segregation | | Total | Confirmed genetic ratio | $\chi^2$ | 'P' |
|---|---|---|---|---|---|---|---|
| | | Purple(+) | Green(-) | | | | |
| Disomic x Crossa (Sonia control) | O | 651 | 249 | 900 | 45:19 | 1.7607 | 0.20 - 0.10 |
| | E | 632.81 | 267.19 | | | | |
| Triplo-1 x Crossa | O | 134 | 86 | 220 | 45:19 | 9.3194** | < 0.01 |
| | E | 154.69 | 65.31 | | | | |
| Triplo-2 x Crossa | O | 44 | 53 | 097 | 45:19 | 28.9311** | < 0.01 |
| | E | 68.20 | 28.80 | | | | |
| Triplo-3 x Crossa | O | 248 | 127 | 375 | 45:19 | 3.1376 | 0.10 - 0.05 |
| | E | 263.67 | 111.33 | | | | |
| Triplo-4 x Crossa ($F_2$) | O | 04 | 02 | 006 | 45:19 | 0.0382 | 0.90 - 0.80 |
| | E | 4.22 | 1.78 | | | | |
| Triplo-4 x Crossa ($BC_1F_2$) | O | 04 | 68 | 072 | 19.02:44.98 | 20.1366** | < 0.01 |
| | E | 21.40 | 50.60 | | | | |
| Triplo-5 x Crossa | O | 133 | 42 | 175 | 45:19 | 2.7119 | 0.10 - 0.05 |
| | E | 123.05 | 51.95 | | | | |
| Triplo-6 x Crossa | O | 172 | 68 | 240 | 45:19 | 0.2108 | 0.70 - 0.50 |
| | E | 168.75 | 71.25 | | | | |
| Triplo-7 x Crossa | O | 216 | 79 | 295 | 45:19 | 1.1950 | 0.30 - 0.20 |
| | E | 207.42 | 87.58 | | | | |
| Triplo-8 x Crossa | O | 72 | 27 | 099 | 45:19 | 0.2766 | 0.70 - 0.50 |
| | E | 69.61 | 29.39 | | | | |
| Triplo-9 x Crossa | O | 65 | 17 | 082 | 45:19 | 3.1508 | 0.10 - 0.05 |
| | E | 57.66 | 24.34 | | | | |
| Triplo-10 x Crossa | O | 23 | 73 | 096 | 45:19 | 98.8195** | < 0.01 |
| | E | 67.50 | 28.50 | | | | |
| Triplo-11 x Crossa | O | 148 | 77 | 225 | 45:19 | 2.2166 | 0.20 - 0.10 |
| | E | 158.20 | 66.80 | | | | |
| Triplo-12 x Crossa | O | 73 | 08 | 081 | 45:19 | 15.2296** | < 0.01 |
| | E | 56.95 | 24.05 | | | | |

** = Significant deviation at 0.01 probability level

(external). The calculated chi-square values of these trisomics were 9.3194, 28.9311 and 98.8195 respectively. With respect to the $F_2$ population of trisomic 12, a significant skewed segregation towards plants with pigmented leaf sheath (external) was realized; with a calculated chi-square value of 15.2296.

Thus, trisomics 1, 2, 4, 10 and 12 seem to be the critical trisomics, contrary to the expectation for a trigenic segregation ratio.

4.2.1.2 Leaf sheath (internal)pigmentation

As has been depicted in Table 10, all the female parents, the donor parent, the disomic and trisomic $F_1$s and the trisomic $BC_1F_1$ were devoid of any pigmentation in leaf sheath (internal) due to the action of an inhibitory gene.

The $F_2$ segregation of leaf sheath (internal) pigmentation in trisomics and marker crosses is presented in Table 21. The data revealed that pigmentation in leaf sheath (internal) was governed by four interacting genes as depicted earlier (refer 4.1.6). The $F_2$ population of trisomics 3, 4, 5, 7, 8 and 11 as well as the $BC_1F_2$ population of trisomic-4 showed a significant departure from the expected 27:229 and 12.03:243.97 segregation ratios, respectively at 0.01 level of probability. The $F_2$ and $BC_1F_2$ populations involving trisomic-4 showed a skewed segregation in the occurrence of purple plants; with the calculated chi-square values of higher

Table 21. $F_2$ Segregation of leaf sheath (internal) pigmentation in trisomics and marker crosses

| Cross combination | | $F_2$ phenotypic segregation | | Total | Confirmed genetic ratio | $x^2$ | 'P' |
|---|---|---|---|---|---|---|---|
| | | Purple(+) | Green(-) | | | | |
| Disomic x Crossa (Sona control) | O | 77 | 823 | 900 | 27:229 | 3.7827 | 0.10 - 0.05 |
| | E | 94.92 | 805.08 | | | | |
| Triplo-1 x Crossa | O | 17 | 203 | 220 | 27:229 | 1.8539 | 0.20 - 0.10 |
| | E | 23.20 | 196.80 | | | | |
| Triplo-2 x Crossa | O | 9 | 88 | 097 | 27:229 | 0.1654 | 0.70 - 0.50 |
| | E | 10.23 | 86.77 | | | | |
| Triplo-3 x Crossa | O | 9 | 366 | 375 | 27:229 | 26.3812** | < 0.01 |
| | E | 39.55 | 335.45 | | | | |
| Triplo-4 x Crossa ($F_2$) | O | 6 | 0 | 006 | 27:229 | 50.8889** | < 0.01 |
| | E | 0.63 | 5.37 | | | | |
| Triplo-4 x Crossa ($BC_1F_2$) | O | 49 | 23 | 072 | 12.03:243.97 | 645.3428** | < 0.01 |
| | E | 3.38 | 68.62 | | | | |
| Triplo-5 x Crossa | O | 4 | 171 | 175 | 27:229 | 12.6590** | < 0.01 |
| | E | 18.46 | 156.54 | | | | |
| Triplo-6 x Crossa | O | 16 | 224 | 240 | 27:229 | 3.8300 | 0.10 - 0.05 |
| | E | 25.31 | 214.69 | | | | |
| Triplo-7 x Crossa | O | 10 | 285 | 295 | 27:229 | 16.0166** | < 0.01 |
| | E | 31.11 | 263.89 | | | | |
| Triplo-8 x Crossa | O | 0 | 99 | 099 | 27:229 | 11.6725** | < 0.01 |
| | E | 10.44 | 88.56 | | | | |
| Triplo-9 x Crossa | O | 10 | 72 | 082 | 27:229 | 0.2361 | 0.70 - 0.50 |
| | E | 8.65 | 73.35 | | | | |
| Triplo-10 x Crossa | O | 05 | 91 | 096 | 27:229 | 2.9000 | 0.10 - 0.05 |
| | E | 10.13 | 85.87 | | | | |
| Triplo-11 x Crossa | O | 03 | 222 | 225 | 27:229 | 20.2449** | < 0.01 |
| | E | 23.73 | 201.27 | | | | |
| Triplo-12 x Crossa | O | 04 | 77 | 081 | 27:229 | 2.7007 | 0.20 - 0.10 |
| | E | 8.54 | 72.46 | | | | |

** = Significant deviation at 0.01 probability level

magnitude that is, 50.8889 and 645.3428 respectively. With regard to the $F_2$ populations of trisomics 3, 5, 7, 8 and 11, the skewness of segregation was towards the green plants, predominating the populations. The calculated chi-square values of these trisomics, 3, 5, 7, 8 and 11 were of the magnitude 26.3812, 12.6590, 16.0166, 11.6725 and 20.2449 respectively.

Thus, trisomics 3, 4, 5, 7, 8 and 11 seem to be the critical trisomics, contrary to the expectation for a tetragenic segregation ratio.

4.2.1.3 Prophyllum pigmentation

The pigmentation in prophyllum in the parents as well as in the disomic (2n) and trisomic (2n + 1) $F_1$s is displayed in Table 10. The presence of pigmentation in prophyllum was dominant over its absence and so, as expected, the disomic and all the trisomic $F_1$s showed purple pigmentation in the prophyllum.

The $F_2$ segregation for prophyllum pigmentation in trisomics and marker crosses is presented in Table 22. The data revealed that pigmentation in prophyllum was governed by three interacting genes as depicted earlier (refer 4.1.2). The $BC_1F_2$ population involving trisomic-4 and the $F_2$ populations involving trisomics 2 and 12 showed significant deviation from the expected 19.02:44.98 and 45:19 segregation

Table 22. $F_2$ segregation of prophyllum pigmentation in trisomics and marker crosses

| Cross combination | $F_2$ phenotypic segregation | | Total | Confirmed genetic ratio | $x^2$ | 'P' |
|---|---|---|---|---|---|---|
| | Purple(+) | Green(-) | | | | |
| Disomic x crossa (Sona control) | O 631 | 269 | 900 | 45:19 | 0.0175 | 0.90-0.80 |
| | E 632.81 | 267.19 | | | | |
| Triplo-1 x Crossa | O 150 | 70 | 220 | 45:19 | 0.4785 | 0.50-0.30 |
| | E 154.69 | 65.31 | | | | |
| Triplo-2 x Crossa | O 51 | 46 | 97 | 45:19 | 14.6163** | < 0.01 |
| | E 68.20 | 28.80 | | | | |
| Triplo-3 x Crossa | O 250 | 125 | 375 | 45:19 | 2.3879 | 0.20-0.10 |
| | E 263.67 | 111.33 | | | | |
| Triplo-4 x Crossa ($F_2$) | O 6 | 0 | 06 | 45:19 | 2.5333 | 0.20-0.10 |
| | E 4.22 | 1.78 | | | | |
| Triplo-4 x Crossa ($BC_1F_2$) | O 49 | 23 | 72 | 19.02:44.98 | 50.6634** | < 0.01 |
| | E 21.40 | 50.60 | | | | |
| Triplo-5 x Crossa | O 134 | 41 | 175 | 45:19 | 3.2842 | 0.10-0.05 |
| | E 123.05 | 51.95 | | | | |
| Triplo-6 x Crossa | O 172 | 68 | 240 | 45:19 | 0.2108 | 0.70-0.50 |
| | E 168.75 | 71.25 | | | | |
| Triplo-7 x Crossa | O 216 | 79 | 295 | 45:19 | 1.1950 | 0.30-0.20 |
| | E 207.42 | 87.58 | | | | |
| Triplo-8 x Crossa | O 76 | 23 | 99 | 45:19 | 1.9763 | 0.20-0.10 |
| | E 69.61 | 29.39 | | | | |
| Triplo-9 x crossa | O 65 | 17 | 82 | 45:19 | 3.1508 | 0.10-0.05 |
| | E 57.66 | 24.34 | | | | |
| Triplo-10 x Crossa | O 60 | 36 | 96 | 45:19 | 2.8070 | 0.10-0.05 |
| | E 67.50 | 28.50 | | | | |
| Triplo-11 x Crossa | O 148 | 77 | 225 | 45:19 | 2.2166 | 0.20-0.10 |
| | E 158.20 | 66.80 | | | | |
| Triplo-12 x Crossa | O 73 | 08 | 81 | 45:19 | 15.2296** | < 0.01 |
| | E 56.95 | 24.05 | | | | |

** = Significant deviation at 0.01 probability level

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ratios, respectively at 0.01 level of probability.

The $BC_1F_2$ population of trisomic-4 and the $F_2$ population of trisomic-12 showed a skewed segregation in the occurrence of purple plants predominating the populations with the calculated chi-square values of 50.6634 and 15.2296 respectively. While the $F_2$ population of trisomic-2 showed a different behaviour with the occurrence of more than expected green plants and with a calculated chi-square value of lower magnitude that is, 14.6163.

Thus, trisomics 2, 4, and 12 seem to be the critical trisomics.

4.2.1.4 Leaf blade pigmentation at seedling and flowering stage and leaf midrib pigmentation

The pigmentation of these two plant parts in the parents as well as in the disomic and trisomic $F_1$s is depicted in Table 10. Although the donor parent showed purple pigmentation in these two plant parts, all the trisomic and the disomic $F_1$s remained devoid of pigmentation owing to the action of inhibitory gene.

The $F_2$ segregation for pigmentation in these two plant parts in trisomics and marker crosses is depicted in Table 23. The data revealed that pigmentation in leaf blade at seedling stage and at flowering stage as well as in leaf midrib was under the control of three interacting genes as

Table 23. $F_2$ segregation of leaf blade (seedling and flowering stage) and leaf midrib pigmentation in trisomics and marker crosses

| Cross combination | | $F_2$ Phenotypic segregation | | Total | Confirmed genetic ratio | $X^2$ | 'P' |
|---|---|---|---|---|---|---|---|
| | | Purple(+) | Green(-) | | | | |
| Disomic x crossa (Sona control) | O | 116 | 784 | 900 | 9:55 | 1.0258 | 0.50-0.30 |
| | E | 126.56 | 773.44 | | | | |
| Triplo-1 x Crossa | O | 35 | 185 | 220 | 9:55 | 0.6208 | 0.50-0.30 |
| | E | 30.94 | 189.06 | | | | |
| Triplo-2 x Crossa | O | 8 | 89 | 97 | 9:55 | 2.7142 | 0.10-0.05 |
| | E | 13.64 | 83.36 | | | | |
| Triplo-3 x Crossa | O | 48 | 327 | 375 | 9:55 | 0.4946 | 0.50-0.30 |
| | E | 52.73 | 322.27 | | | | |
| Triplo-4 x Crossa ($F_2$) | O | 0 | 6 | 06 | 9:55 | 0.9818 | 0.50-0.30 |
| | E | 0.84 | 5.16 | | | | |
| Triplo-4 x Crossa ($BC_1F_2$) | O | 0 | 72 | 72 | 0.64:63.36 | 0.7273 | 0.50-0.30 |
| | E | 0.72 | 71.28 | | | | |
| Triplo-5 x Crossa | O | 22 | 153 | 175 | 9:55 | 0.3220 | 0.70-0.50 |
| | E | 24.61 | 150.39 | | | | |
| Triplo-6 x Crossa | O | 21 | 219 | 240 | 9:55 | 5.6048* | 0.02-0.01 |
| | E | 33.75 | 206.25 | | | | |
| Triplo-7 x Crossa | O | 39 | 256 | 295 | 9:55 | 0.1731 | 0.70-0.50 |
| | E | 41.48 | 253.52 | | | | |
| Triplo-8 x Crossa | O | 6 | 93 | 99 | 9:55 | 5.2454* | 0.05-0.02 |
| | E | 13.92 | 85.08 | | | | |
| Triplo-9 x Crossa | O | 12 | 70 | 82 | 9:55 | 0.0222 | 0.90-0.80 |
| | E | 11.53 | 70.47 | | | | |
| Triplo-10 x Crossa | O | 10 | 86 | 96 | 9:55 | 1.0559 | 0.50-0.30 |
| | E | 13.50 | 82.50 | | | | |
| Triplo-11 x Crossa | O | 30 | 195 | 225 | 9:55 | 0.0990 | 0.80-0.70 |
| | E | 31.64 | 193.36 | | | | |
| Triplo-12 x Crossa | O | 25 | 56 | 81 | 9:55 | 18.9211** | < 0.01 |
| | E | 11.39 | 69.61 | | | | |

** = Significant deviation at 0.01 probability level

* = Significant deviation at 0.05 probability level

has been elaborated previously ( refer 4.1.3). The $F_2$ populations of trisomics 6 and 8 showed significant deviation from the expected 9:55 segregation ratio at 0.05 level of probability, whereas the $F_2$ population of trisomic-12 deviated significantly at 0.01 level of probability.

The data further revealed that all the three $F_2$ populations showed a skewed segregation. The $F_2$ populations of trisomics 6 and 8 had more than expected proportion of green plants and they showed calculated chi-square values of 5.6048 and 5.2454 respectively. With respect to the $F_2$ population of trisomic 12 the calculated value of chi-square was 18.9211 and the skewness of segregation was towards the purple plants predominating the population.

Thus, trisomics 6, 8 and 12 seem to be the critical trisomics.

4.2.1.5 Leaf tip pigmentation at seedling and flowering stages

The leaf tip pigmentation at seedling and flowering stages in the parents as well as in the disomic and trisomic $F_1$s is displayed in Table 10. The presence of pigmentation in this part of the plant at both the stages of growth was dominant over its absence; and hence, as per expectation the disomic and all the trisomic $F_1$s showed purple pigmentation.

The $F_2$ segregation for leaf tip pigmentation at

seedling stage and at flowering stage in the trisomics and marker crosses is presented in Tables 24 and 26, respectively. The data revealed that pigmentation in leaf tip at seedling and flowering stages was under the control of three interacting genes as shown earlier ( refer 4.1.2). The $F_2$ populations of trisomics-2 and 12 and the $BC_1F_2$ population of trisomic-4 exhibited a significant deviation from the expected 45:19 and 19.02:44.98 segregation ratios respectively at 0.01 level of probability for pigmentation of leaf tip at both the seedling and flowering stages.

The data further revealed that the calculated value of chi-square for the $BC_1F_2$ population of trisomic-4 was with a higher magnitude of 50.6634 at both the stages of growth. This population constituted predominantly of plants with purple leaf tip. With respect to the $F_2$ population of trisomic-12 the skewness of segregation was also towards purple plants predominating the population; with a calculated chi-square value of 15.2296.

As regards the $F_2$ population of trisomic-2, the calculated value of chi-square for leaf tip pigmentation at seedling stage was 24.3473, while the value was 14.6163 for leaf tip pigmentation at flowering stage. In both these cases the skewness of segregation was due to more than expected proportion of green plants.

Table 24. $F_2$ segregation of leaf tip (seedling stage) pigmentation in trisomics and marker crosses

| Cross combination | | $F_2$ phenotypic segregation | | Total | Confirmed genetic ratio | $X^2$ | 'P' |
|---|---|---|---|---|---|---|---|
| | | Purple(+) | Green(-) | | | | |
| Disomic x Crossa (Sona control) | O | 655 | 245 | 900 | 45:19 | 2.8224 | 0.20-0.10 |
| | E | 632.81 | 267.19 | | | | |
| Triplo-1 x Crossa | O | 148 | 72 | 220 | 45:19 | 0.9739 | 0.50-0.30 |
| | E | 154.69 | 65.31 | | | | |
| Triplo-2 x Crossa | O | 46 | 51 | 97 | 45:19 | 24.3473** | < 0.01 |
| | E | 68.20 | 28.80 | | | | |
| Triplo-3 x Crossa | O | 250 | 125 | 375 | 45:19 | 2.3879 | 0.20-0.10 |
| | E | 263.67 | 111.33 | | | | |
| Triplo-4 x Crossa ($F_2$) | O | 6 | 0 | 06 | 45:19 | 2.5333 | 0.20-0.10 |
| | E | 4.22 | 1.78 | | | | |
| Triplo-4 x Crossa ($BC_1F_2$) | O | 49 | 23 | 72 | 19.02:44.98 | 50.5634** | < 0.01 |
| | E | 21.40 | 50.60 | | | | |
| Triplo-5 x Crossa | O | 129 | 46 | 175 | 45:19 | 0.9702 | 0.50-0.30 |
| | E | 123.05 | 51.95 | | | | |
| Triplo-6 x Crossa | O | 172 | 68 | 240 | 45:19 | 0.2108 | 0.70-0.50 |
| | E | 168.75 | 71.25 | | | | |
| Triplo-7 x Crossa | O | 215 | 80 | 295 | 45:19 | 0.9326 | 0.50-0.30 |
| | E | 207.42 | 87.58 | | | | |
| Triplo-8 x Crossa | O | 78 | 21 | 99 | 45:19 | 3.4068 | 0.10-0.05 |
| | E | 69.61 | 29.39 | | | | |
| Triplo-9 x Crossa | O | 65 | 17 | 82 | 45:19 | 3.1508 | 0.10-0.05 |
| | E | 57.66 | 24.34 | | | | |
| Triplo-10 x Crossa | O | 59 | 37 | 96 | 45:19 | 3.6055 | 0.10-0.05 |
| | E | 67.50 | 28.50 | | | | |
| Triplo-11 x Crossa | O | 148 | 77 | 225 | 45:19 | 2.2166 | 0.20-0.10 |
| | E | 158.20 | 66.80 | | | | |
| Triplo-12 x Crossa | O | 73 | 08 | 81 | 45:19 | 15.2296** | < 0.01 |
| | E | 56.95 | 24.05 | | | | |

** = Significant deviation at 0.01 probability level.

Table 26. $F_2$ segregation of leaf tip (flowering stage) pigmentation in trisomics and marker crosses

| Cross combination | | $F_2$ phenotypic segregation | | Total | Confirmed genetic ratio | $\chi^2$ | 'P' |
|---|---|---|---|---|---|---|---|
| | | Purple(+) | Green(-) | | | | |
| Disomic x Crossa (Sona control) | O | 640 | 260 | 900 | 45:19 | 0.2750 | 0.70 - 0.50 |
| | E | 632.81 | 267.19 | | | | |
| Triplo-1 x Crossa | O | 149 | 71 | 220 | 45:19 | 0.7044 | 0.50 - 0.30 |
| | E | 154.69 | 65.31 | | | | |
| Triplo-2 x Crossa | O | 51 | 46 | 97 | 45:19 | 14.6163** | < 0.01 |
| | E | 68.20 | 28.80 | | | | |
| Triplo-3 x Crossa | O | 250 | 125 | 375 | 45:19 | 2.3879 | 0.20 - 0.10 |
| | E | 263.67 | 111.33 | | | | |
| Triplo-4 x Crossa ($F_2$) | O | 6 | 0 | 06 | 45:19 | 2.5333 | 0.20 - 0.10 |
| | E | 4.22 | 1.78 | | | | |
| Triplo-4 x Crossa ($BC_1F_2$) | O | 49 | 23 | 72 | 19.02:44.98 | 50.6634** | < 0.01 |
| | E | 21.40 | 50.60 | | | | |
| Triplo-5 x Crossa | O | 134 | 41 | 175 | 45:19 | 3.2642 | 0.10 - 0.05 |
| | E | 123.05 | 51.95 | | | | |
| Triplo-6 x Crossa | O | 172 | 68 | 240 | 45:19 | 0.2108 | 0.70 - 0.50 |
| | E | 168.75 | 71.25 | | | | |
| Triplo-7 x Crossa | O | 215 | 80 | 295 | 45:19 | 0.9326 | 0.50 - 0.30 |
| | E | 207.42 | 87.58 | | | | |
| Triplo-8 x Crossa | O | 78 | 21 | | | | |
| | E | 69.61 | 29.39 | 99 | 45:19 | 3.4068 | 0.10 - 0.05 |
| Triplo-9 x Crossa | O | 65 | 17 | 82 | 45:19 | 3.1508 | 0.10 - 0.05 |
| | E | 57.66 | 24.34 | | | | |
| Triplo-10 x Crossa | O | 59 | 37 | 96 | 45:19 | 3.6055 | 0.10 - 0.05 |
| | E | 67.50 | 28.50 | | | | |
| Triplo-11 x Crossa | O | 148 | 77 | 225 | 45:19 | 2.2166 | 0.20 - 0.10 |
| | E | 158.20 | 66.80 | | | | |
| Triplo-12 x Crossa | O | 73 | 8 | 81 | 45:19 | 15.2296** | < 0.01 |
| | E | 56.95 | 24.05 | | | | |

** = Significant deviation at 0.01 probability level

Thus, trisomics-2, 4 and 12 appear to be the critical trisomics.

4.2.1.6 Leaf margin pigmentation at seedling and flowering stage

The leaf margin pigmentation at seedling and flowering stages in the parents as well as in the disomic and trisomic $F_1$s is presented in Table 10. Although the donor parent showed presence of pigmentation in leaf margin at seedling and flowering stages, the disomic and all the trisomic $F_1$s - except the $F_1$ involving trisomic-8 remained, devoid of any purple pigmentation due to the involvement of an inhibitory gene. Interestingly, the $F_1$ of trisomic-8 showed pigmented leaf margin at both the seedling and flowering stages of growth.

The $F_2$ segregation for leaf margin pigmentation at seedling stage and at flowering stage in the trisomics and marker crosses is presented in Tables 25 and 27 respectively. The data revealed that pigmentation in these parts was under digenic control as indicated earlier (refer 4.1.1). The $F_2$ populations of trisomics-6, 8 and 12 exhibited a significant deviation from the expected 3:13 digenic ratio (at 0.01 level of probability) for leaf margin pigmentation at seedling and flowering stages.

The data revealed further that the calculated chi-square values for the $F_2$ populations of trisomics-6 and 12 was 9.8735 and 17.7806 respectively. The skewness of

Table 25. $F_2$ segregation of leaf margin (seedling stage) pigmentation in trisomics and marker crosses

| Cross combination | | $F_2$ phenotypic segregation | | Total | Confirmed genetic ratio | $\chi^2$ | 'P' |
|---|---|---|---|---|---|---|---|
| | | Purple(+) | Green(-) | | | | |
| Disomic x Crossa (Sona control) | O | 181 | 719 | 900 | 3:13 | 1.0945 | 0.30-0.20 |
| | E | 168.75 | 731.25 | | | | |
| Triplo-1 x crossa | O | 36 | 184 | 220 | 3:13 | 0.8224 | 0.50-0.30 |
| | E | 41.25 | 178.75 | | | | |
| Triplo-2 x crossa | O | 21 | 76 | 97 | 3:13 | 0.5353 | 0.50-0.30 |
| | E | 18.19 | 78.81 | | | | |
| Triplo-3 x Crossa | O | 64 | 311 | 375 | 3:13 | 0.6975 | 0.50-0.30 |
| | E | 70.31 | 304.69 | | | | |
| Triplo-4 x Crossa ($F_2$) | O | 0 | 6 | 06 | 3:13 | 1.3846 | 0.30-0.20 |
| | E | 1.13 | 4.88 | | | | |
| Triplo-4 x Crossa ($BC_1F_2$) | O | 0 | 72 | 72 | 0.44:15.56 | 2.0360 | 0.20-0.10 |
| | E | 1.98 | 70.02 | | | | |
| Triplo-5 x Crossa | O | 29 | 146 | 175 | 3:13 | 0.5452 | 0.50-0.30 |
| | E | 32.81 | 142.19 | | | | |
| Triplo-6 x Crossa | O | 26 | 214 | 240 | 3:13 | 9.8735** | <0.01 |
| | E | 45.00 | 195.00 | | | | |
| Triplo-7 x Crossa | O | 44 | 251 | 295 | 3:13 | 2.8475 | 0.10-0.05 |
| | E | 55.31 | 239.69 | | | | |
| Triplo-8 x Crossa | O | 41 | 58 | 99 | 3:13 | 33.3802** | <0.01 |
| | E | 18.56 | 80.44 | | | | |
| Triplo-9 x Crossa | O | 22 | 60 | 82 | 3:13 | 3.5134 | 0.10-0.05 |
| | E | 15.38 | 66.63 | | | | |
| Triplo-10 x Crossa | O | 22 | 74 | 96 | 3:13 | 1.0940 | 0.30-0.20 |
| | E | 18.00 | 78.00 | | | | |
| Triplo-11 x Crossa | O | 32 | 193 | 225 | 3:13 | 3.0278 | 0.10-0.05 |
| | E | 42.19 | 182.81 | | | | |
| Triplo-12 x Crossa | O | 30 | 51 | 81 | 3:13 | 17.7806** | <0.01 |
| | E | 15.19 | 65.81 | | | | |

** = Significant deviation at 0.01 probability level

Table 27. $F_2$ segregation of leaf margin (flowering stage pigmentation in trisomics and marker crosses

| Cross combination | | $F_2$ phenotypic segregation | | Total | Confirmed genetic ratio | $X^2$ | 'P' |
|---|---|---|---|---|---|---|---|
| | | Purple(+) | Green(-) | | | | |
| Disomic x Crossa (Sona control) | O | 179 | 721 | 900 | 3:13 | 0.7663 | 0.50 - 0.30 |
| | E | 168.75 | 731.25 | | | | |
| Triplo-1 x Crossa | O | 37 | 183 | 220 | 3:13 | 0.5389 | 0.50 - 0.30 |
| | E | 41.25 | 178.75 | | | | |
| Triplo-2 x Crossa | O | 22 | 75 | 97 | 3:13 | 0.9836 | 0.50 - 0.30 |
| | E | 18.19 | 78.81 | | | | |
| Triplo-3 x Crossa | O | 66 | 309 | 375 | 3:13 | 0.3255 | 0.70 - 0.50 |
| | E | 70.31 | 304.69 | | | | |
| Triplo-4 x Crossa ($F_2$) | O | 0 | 6 | 06 | 3:13 | 1.3846 | 0.30 - 0.20 |
| | E | 1.13 | 4.87 | | | | |
| Triplo-4 x Crossa ($BC_1F_2$) | O | 0 | 72 | 72 | 0.44:15.56 | 2.0360 | 0.20-0.10 |
| | E | 1.98 | 70.02 | | | | |
| Triplo-5 x Crossa | O | 26 | 149 | 175 | 3:13 | 1.7408 | 0.20 - 0.10 |
| | E | 32.81 | 142.19 | | | | |
| Triplo-6 x Crossa | O | 26 | 214 | 240 | 3:13 | 9.8735** | < 0.01 |
| | E | 45.00 | 195.00 | | | | |
| Triplo-7 x Crossa | O | 43 | 252 | 295 | 3:13 | 3.3732 | 0.10 - 0.05 |
| | E | 55.31 | 239.69 | | | | |
| Triplo-8 x Crossa | O | 9 | 90 | 99 | 3:13 | 6.0629* | 0.02 - 0.01 |
| | E | 18.56 | 80.44 | | | | |
| Triplo-9 x Crossa | O | 22 | 60 | 82 | 3:13 | 3.5134 | 0.10 - 0.05 |
| | E | 15.38 | 66.63 | | | | |
| Triplo-10 x Crossa | O | 23 | 73 | 96 | 3:13 | 1.7094 | 0.20 - 0.10 |
| | E | 18.00 | 78.00 | | | | |
| Triplo-11 x Crossa | O | 32 | 193 | 225 | 3:13 | 3.0278 | 0.10 - 0.05 |
| | E | 42.19 | 182.81 | | | | |
| Triplo-12 x Crossa | O | 30 | 51 | 81 | 3:13 | 17.7806** | < 0.01 |
| | E | 15.19 | 65.81 | | | | |

** = Significant deviation at 0.01 probability level

* = Significant deviation at 0.05 probability level

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segregation in case of the $F_2$ population of trisomic-6 was due to the occurrence of more than expected proportion of green plants, whereas that in the case of trisomic-12, $F_2$ it was due to more than expected proportion of purple plants.

The $F_2$ population of trisomic-8 showed a strange behaviour interestingly. In this case, the leaf margin pigmentation at seedling stage exhibited a skewed segregation with the occurrence of more than expected proportion of purple plants and showed a high calculated chi-square value of 33.3802. whereas, in case of leaf margin pigmentation at flowering stage, the skewness of segregation was due to the occurrence of green plants predominating the population, thereby showing a calculated chi-square value of 6.0629 at 0.05 level of probability.

Thus, it appears that trisomics-6, 8 and 12 are the critical trisomics.

4.2.1.7 Leaf axil pigmentation

Table 10 details the description of leaf axil pigmentation in the parents as well as in the disomic (2n) and trisomic (2n + 1) $F_1$s. The presence of pigmentation in leaf axil was dominant over its absence and hence, as expected the disomic and all the trisomic $F_1$s showed pigmented leaf axil.

Table 28 details the $F_2$ segregation of leaf axil pigmentation in trisomics and marker crosses. The data revealed that pigmentation in leaf axil was under a trigenic control as indicated elsewhere (refer 4.1.2). The $F_2$ populations of trisomics-2 and 12 and the $BC_1F_2$ population of trisomic-4 showed significant deviation from the expected 45:19 and 19.02:44.98 segregation ratios respectively at 0.01 probability level.

The data further revealed that the $BC_1F_2$ population involving trisomic-4 and the $F_2$ population of trisomic-12 showed a skewness of segregation in the occurrence of purple plants predominating the populations, thereby showing the calculated chi-square values of 50.6634 and 15.2296 respectively. With respect to the $F_2$ population involving trisomic-2 a calculated chi-square value of 28.9311 was realized. The deviation of the value from that of the disomic was due to the occurrence of green plants predominating the population.

Thus, it appears that the trisomics-2, 4 and 12 are the critical trisomics.

4.2.1.8 Ligule pigmentation

The ligule pigmentation in the parents as well as in the disomic (2n) and trisomic (2n + 1) $F_1$s is detailed in Table 10. Although the donor parent showed purple pigmentation in ligule, all the trisomic and the disomic $F_1$s remained devoid of any pigmentation due to the action of an inhibitory gene.

Table 28. $F_2$ Segregation of leaf axil pigmentation in triploxdiploid and marker crosses

| Cross combination | | $F_2$ Phenotypic segregation | | Total | Confirmed genetic ratio | $x^2$ | 'P' |
|---|---|---|---|---|---|---|---|
| | | Purple(+) | Green(-) | | | | |
| Disomic x Crossa (Sona control) | O | 614 | 286 | 900 | 45:19 | 1.8838 | 0.20 - 0.10 |
| | E | 632.81 | 267.19 | | | | |
| Triplo-1 x Crossa | O | 150 | 70 | 220 | 45:19 | 0.4785 | 0.50 - 0.30 |
| | E | 154.69 | 65.31 | | | | |
| Triplo-2 x Crossa | O | 44 | 53 | 97 | 45:19 | 28.9311** | < 0.01 |
| | E | 68.20 | 28.80 | | | | |
| Triplo-3 x Crossa | O | 249 | 126 | 375 | 45:19 | 2.7500 | 0.10 - 0.05 |
| | E | 263.67 | 111.33 | | | | |
| Triplo-4 x Crossa ($F_2$) | O | 6 | 0 | 06 | 45:19 | 2.5333 | 0.20 - 0.10 |
| | E | 4.22 | 1.78 | | | | |
| Triplo-4 x Crossa ($BC_1F_2$) | O | 49 | 23 | 72 | 19.02:44.98 | 50.6634** | < 0.01 |
| | E | 21.40 | 50.60 | | | | |
| Triplo-5 x Crossa | O | 132 | 43 | 175 | 45:19 | 2.1943 | 0.20 - 0.10 |
| | E | 123.05 | 51.95 | | | | |
| Triplo-6 x Crossa | O | 172 | 68 | 240 | 45:19 | 0.2108 | 0.70 - 0.50 |
| | E | 168.75 | 71.25 | | | | |
| Triplo-7 x Crossa | O | 216 | 79 | 295 | 45:19 | 1.1950 | 0.30 - 0.20 |
| | E | 207.42 | 87.58 | | | | |
| Triplo-8 x Crossa | O | 77 | 22 | 99 | 45:19 | 2.6431 | 0.20 - 0.10 |
| | E | 69.61 | 29.39 | | | | |
| Triplo-9 x Crossa | O | 65 | 17 | 82 | 45:19 | 3.1508 | 0.10 - 0.05 |
| | E | 57.66 | 24.34 | | | | |
| Triplo-10 x Crossa | O | 59 | 37 | 96 | 45:19 | 3.6055 | 0.10 - 0.05 |
| | E | 67.50 | 28.50 | | | | |
| Triplo-11 x Crossa | O | 148 | 77 | 225 | 45:19 | 2.2166 | 0.20 - 0.10 |
| | E | 158.20 | 66.80 | | | | |
| Triplo-12 x Crossa | O | 73 | 08 | 81 | 45:19 | 15.2296** | < 0.01 |
| | E | 56.95 | 24.05 | | | | |

** = Significant deviation at 0.01 probability level

The $F_2$ segregation for ligule pigmentation in trisomics and marker crosses is presented in Table 29. The data revealed that ligule pigmentation was governed by three interacting genes as indicated elsewhere (refer 4.1.3). The $F_2$ populations of trisomics-9 and 12 showed a significant deviation from the expected 9:55 segregation ratio at 0.01 level of probability, whereas the deviation in case of the $F_2$ population of trisomic-11 was at 0.05 level of probability.

As displayed in the table, the deviation in the case of all the three $F_2$ populations was due to higher proportion of plants with purple ligule. The $F_2$ populations of trisomics-9 and 12 showed the calculated chi-square values of higher magnitude that is, 46.5109 and 31.6780 respectively; whereas the $F_2$ population involving trisomic-11 had the calculated chi-square value of 6.5636 only.

Thus, trisomics-9, 11 and 12 seem to be the critical trisomics.

4.2.1.9 Auricle pigmentation

The auricle pigmentation in the parents as well as in the disomic (2n) and trisomic (2n + 1) $F_1$s is detailed in Table 10. Although the donor parent showed pigmented auricle, the 2n and all the 2n + 1 $F_1$s except the $F_1$s involving trisomics-2, 3, 6 and 8 remained devoid of any pigmentation in auricle due to the action of an inhibitory gene. Interestingly,

Table 29. $F_2$ segregation of ligule pigmentation in trisomics and marker crosses

| Cross combination | | $F_2$ phenotypic segregation | | Total | Confirmed genetic ratio | $x^2$ | 'P' |
|---|---|---|---|---|---|---|---|
| | | Purple(+) | Green(-) | | | | |
| Disomic x Crossa (Sona control) | O | 147 | 753 | 900 | 9:55 | 3.8403 | 0.10 - 0.05 |
| | E | 126.56 | 773.44 | | | | |
| Triplo-1 x Crossa | O | 39 | 181 | 220 | 9:55 | 2.4450 | 0.20 - 0.10 |
| | E | 30.94 | 189.06 | | | | |
| Triplo-2 x Crossa | O | 13 | 84 | 97 | 9:55 | 0.0350 | 0.90 - 0.80 |
| | E | 13.64 | 83.36 | | | | |
| Triplo-3 x Crossa | O | 54 | 321 | 375 | 9:55 | 0.0353 | 0.90 - 0.80 |
| | E | 52.73 | 322.27 | | | | |
| Triplo-4 x Crossa (F2) | O | 0 | 6 | 06 | 9:55 | 0.9818 | 0.50 - 0.30 |
| | E | 0.84 | 5.16 | | | | |
| Triplo-4 x Crossa ($BC_1F_2$) | O | 0 | 72 | 72 | 0.64:63.36 | 0.7273 | 0.50 - 0.30 |
| | E | 0.72 | 71.28 | | | | |
| Triplo-5 x Crossa | O | 30 | 145 | 175 | 9:55 | 1.3740 | 0.30 - 0.20 |
| | E | 24.61 | 150.39 | | | | |
| Triplo-6 x Crossa | O | 43 | 197 | 240 | 9:55 | 2.9500 | 0.10 - 0.05 |
| | E | 33.75 | 206.25 | | | | |
| Triplo-7 x Crossa | O | 40 | 255 | 295 | 9:55 | 0.0618 | 0.90 - 0.80 |
| | E | 41.48 | 253.52 | | | | |
| Triplo-8 x Crossa | O | 20 | 79 | 99 | 9:55 | 3.0879 | 0.10 - 0.05 |
| | E | 13.92 | 85.08 | | | | |
| Triplo-9 x Crossa | O | 33 | 49 | 82 | 9:55 | 46.5109** | < 0.01 |
| | E | 11.53 | 70.47 | | | | |
| Triplo-10 x Crossa | O | 11 | 85 | 96 | 9:55 | 0.5387 | 0.50 - 0.30 |
| | E | 13.50 | 82.50 | | | | |
| Triplo-11 x Crossa | O | 45 | 180 | 225 | 9:55 | 6.5636* | 0.02 - 0.01 |
| | E | 31.64 | 193.36 | | | | |
| Triplo-12 x Crossa | O | 29 | 52 | 81 | 9:55 | 31.6780** | < 0.01 |
| | E | 11.39 | 69.61 | | | | |

** = Significant deviation at 0.01 probability level

* = Significant deviation at 0.05 probability level

the $F_1$s of trisomics-2, 3, 6 and 8 showed pigmented auricles. Of these trisomics, triplo-6 consistently exhibited dark purple auricles till maturity without any dilution or fading.

The $F_2$ segregation for auricle pigmentation in trisomics and marker crosses is detailed in Table 30. The data revealed that the pigmentation in auricle was under a trigenic control as elaborated earlier (refer 4.1.3). The $F_2$ populations involving trisomics-6 and 12 showed a significant departure from the expected 9:55 segregation ratio at 0.01 level of probability. The data further revealed that, the calculated chisquare value was of a larger magnitude that is, 195.2345 for the $F_2$ population of trisomic-6, whereas in case of the $F_2$ population of trisomic-12 the calculated chi-square value was 31.6780. Both the $F_2$ populations showed a skewed distribution with a higher proportion of plants with purple auricles.

Although the trisomics-2, 3 and 8 showed pigmented auricles in the $F_1$ (Table 10), non-significant values of calculated chi-square were realised in the $F_2$ populations of these trisomics.

Thus, the trisomics-6 and 12 appear to be the critical trisomics.

4.2.1.10 Junctura (collar) pigmentation

The description of junctura pigmentation in the

Table 30 $F_2$ Segregation of auricle pigmentation in trisomics and marker crosses

| Cross combination | | $F_2$ phenotypic segregation | | Total | Confirmed Genetic ratio | $X^2$ | 'P' |
|---|---|---|---|---|---|---|---|
| | | purple (+) | Green(-) | | | | |
| Disomic x Crossa (Sona control) | O | 137 | 763 | 900 | 9:55 | 1.0016 | 0.50 - 0.30 |
| | E | 126.56 | 773.44 | | | | |
| Triplo-1 x Crossa | O | 40 | 180 | 220 | 9:55 | 3.0691 | 0.10 - 0.05 |
| | E | 30.94 | 189.06 | | | | |
| Triplo-2 x Crossa | O | 19 | 78 | 97 | 9:55 | 2.4503 | 0.20 - 0.10 |
| | E | 13.64 | 83.35 | | | | |
| Triplo-3 x Crossa | O | 54 | 321 | 375 | 9:55 | 0.0353 | 0.90 - 0.80 |
| | E | 52.73 | 322.27 | | | | |
| Triplo-4 x Crossa ($F_2$) | O | 2 | 4 | 06 | 9:55 | 1.8438 | 0.20 - 0.10 |
| | E | 0.84 | 5.16 | | | | |
| Triplo-4 x Crossa ($BC_1F_2$) | O | 2 | 70 | 72 | 0.64:63.36 | 2.2985 | 0.20 - 0.10 |
| | E | 0.72 | 71.28 | | | | |
| Triplo-5 x Crossa | O | 28 | 147 | 175 | 9:55 | 0.5436 | 0.50 - 0.30 |
| | E | 24.51 | 150.39 | | | | |
| Triplo-6 x Crossa | O | 109 | 131 | 240 | 9:55 | 195.2345** | < 0.01 |
| | E | 33.75 | 206.25 | | | | |
| Triplo-7 x Crossa | O | 40 | 255 | 295 | 9:55 | 0.0618 | 0.90 - 0.80 |
| | E | 41.48 | 253.52 | | | | |
| Triplo-8 x Crossa | O | 20 | 79 | 99 | 9:55 | 3.0879 | 0.10 - 0.05 |
| | F | 13.92 | 85.08 | | | | |
| Triplo-9 x Crossa | O | 17 | 65 | 82 | 9:55 | 3.0180 | 0.10 - 0.05 |
| | E | 11.53 | 70.47 | | | | |
| Triplo-10 x Crossa | O | 11 | 85 | 96 | 9:55 | 0.5387 | 0.50 - 0.30 |
| | E | 13.50 | 82.50 | | | | |
| Triplo-11 x Crossa | O | 40 | 185 | 225 | 9:55 | 2.5699 | 0.20 - 0.10 |
| | E | 31.64 | 193.36 | | | | |
| Triplo-12 x Crossa | O | 29 | 52 | 81 | 9:55 | 31.6780** | <0.01 |
| | E | 11.39 | 69.61 | | | | |

** = Significant deviation at 0.01 probability level

parents as well as in the disomic and trisomic $F_1$s is detailed in Table 10. As may be seen from the table, all the female parents, the donor parent, the disomic $F_1$ and all the trisomic $F_1$s except the $F_1$s involving chromosomes 6 and 8 remained devoid of any pigmentation in juncture region. It was interesting to observe pigmentation in juncture in the $F_1$s of trisomics-6 and 8. Of these two trisomics, juncture pigmentation in trisomic-6 was remarkably consistent, as a dark purple juncture was noticed without any dilution till maturity.

The $F_2$ segregation of juncture pigmentation in trisomics and marker crosses is detailed in Table 31. The data revealed that juncture pigmentation was under the control of four interacting genes as has been indicated earlier (refer 4.1.7). The $F_2$ populations involving trisomics-2, 4, 6 and 11 displayed a significant deviation from the expected 3:253 segregation ratio at 0.01 level of probability. With respect to the $F_2$ population of trisomic-4, the population comprised of only six plants. A highly significant value of calculated chi-square of 12.4383 in this case is likely to be due to the sampling error. The $F_2$ populations of trisomics-2, 6 and 11 showed the calculated chi-square values of 55.0394, 2313.3442 and 7.3060 respectively. All the three $F_2$ populations showed a skewed segregation in the occurrence of higher proportion of plants with purple juncture.

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Table 31 $F_2$ segregation of juncture (collar) pigmentation in trisomics and marker crosses

| Cross combination | | $F_2$ phenotypic segregation | | Total | Confirmed genetic ratio | $x^2$ | 'P' |
|---|---|---|---|---|---|---|---|
| | | Purple(+) | Green(-) | | | | |
| Disomic x Crossa (Sona control) | O | 12 | 888 | 900 | 3:253 | 0.2026 | 0.70 - 0.50 |
| | E | 10.55 | 889.45 | | | | |
| Triplo-1 x Crossa | O | 0 | 220 | 220 | 3:253 | 2.6087 | 0.20 - 0.10 |
| | E | 2.58 | 217.42 | | | | |
| Triplo-2 x Crossa | O | 9 | 88 | 97 | 3:253 | 55.0394** | < 0.01 |
| | E | 1.14 | 95.86 | | | | |
| Triplo-3 x Crossa | O | 2 | 373 | 375 | 3:253 | 1.3202 | 0.30 - 0.20 |
| | E | 4.39 | 370.61 | | | | |
| Triplo-4 x Crossa ($F_2$) | O | 1 | 5 | 06 | 3:253 | 12.4383** | < 0.01 |
| | E | 0.07 | 5.93 | | | | |
| Triplo-4 x Crossa ($BC_1F_2$) | O | 2 | 70 | 72 | 19.93:236.07 | 2.5147 | 0.20 - 0.10 |
| | E | 5.61 | 66.39 | | | | |
| Triplo-5 x Crossa | O | 0 | 175 | 175 | 3:253 | 2.0751 | 0.20 - 0.10 |
| | E | 2.05 | 172.95 | | | | |
| Triplo-6 x Crossa | O | 83 | 157 | 240 | 3:253 | 2313.3442** | < 0.01 |
| | E | 2.81 | 237.19 | | | | |
| Triplo-7 x Crossa | O | 1 | 294 | 295 | 3:253 | 1.7670 | 0.20 - 0.10 |
| | E | 3.46 | 291.54 | | | | |
| Triplo-8 x Crossa | O | 0 | 99 | 99 | 3:253 | 1.1739 | 0.30 - 0.20 |
| | E | 1.16 | 97.84 | | | | |
| Triplo-9 x Crossa | O | 0 | 82 | 82 | 3:253 | 0.9723 | 0.50 - 0.30 |
| | E | 0.96 | 81.04 | | | | |
| Triplo-10 x Crossa | O | 0 | 96 | 96 | 3:253 | 1.1383 | 0.30 - 0.20 |
| | E | 1.13 | 94.87 | | | | |
| Triplo-11 x Crossa | O | 7 | 218 | 225 | 3:253 | 7.3060** | < 0.01 |
| | E | 2.64 | 222.36 | | | | |
| Triplo-12 x Crossa | O | 0 | 81 | 81 | 3:253 | 0.9605 | 0.50 - 0.30 |
| | E | 0.95 | 80.05 | | | | |

** = Significant deviation at 0.01 probability level

A very high magnitude of calculated chi-square in case of trisomic-6 was due to a very high proportion of plants with purple juncture in the $F_2$ population.

Although the $F_1$ of trisomic-8 showed pigmented juncture (Table 10), a non-significant value of calculated chisquare was realized in the $F_2$ segregation unlike the $F_1$ and $F_2$ of trisomic-6.

As inferred from the modified ratios in the trisomic $F_2$s of triplos-2, 6 and 11, the respective chromosomes-2, 6 and 11 seem to be critical.

4.2.1.11 Juncture back pigmentation

The juncture back pigmentation in the parents as well as in the disomic and trisomic $F_1$s is detailed in Table 10. Although the donor parent exhibited a purple juncture back, all the trisomic and disomic $F_1$s remained devoid of any pigmentation in juncture back due to the action of an inhibitory gene.

The $F_2$ segregation for juncture back pigmentation in trisomics and marker crosses is presented in Table 32. The data revealed that juncture back pigmentation was governed by an interaction of three genes as indicated earlier (refer 4.1.3). The $F_2$ populations of trisomics-1, 5, 9 and 12 exhibited a significant deviation from the expected 9:55 segregation ratio at 0.01 level of probability. These

Table 32 $F_2$ segregation of junctura back pigmentation in trisomics and marker crosses

| Cross combination | | $F_2$ phenotypic segregation | | Total | Confirmed genetic ratio | $X^2$ | 'p' |
|---|---|---|---|---|---|---|---|
| | | Purple(+) | Green(-) | | | | |
| Disomic x Crossa (Sona control) | O | 147 | 753 | 900 | 9:55 | 3.8403 | 0.10 - 0.05 |
| | E | 126.56 | 773.44 | | | | |
| Triplo-1 x Crossa | O | 56 | 164 | 220 | 9:55 | 23.5255** | < 0.01 |
| | E | 30.94 | 189.06 | | | | |
| Triplo-2 x Crossa | O | 14 | 83 | 97 | 9:55 | 0.0110 | 0.95- 0.90 |
| | E | 13.64 | 83.36 | | | | |
| Triplo-3 x crossa | O | 53 | 322 | 375 | 9:55 | 0.0016 | > 0.99 |
| | E | 52.73 | 322.27 | | | | |
| Triplo-4 x Crossa ($F_2$) | O | 2 | 4 | 06 | 9:55 | 1.8438 | 0.20 - 0.10 |
| | E | 0.84 | 5.16 | | | | |
| Triplo-4 x Crossa ($BC_1F_2$) | O | 0 | 72 | 72 | 0.64:63.36 | 0.7273 | 0.50 - 0.30 |
| | E | 0.72 | 71.28 | | | | |
| Triplo-5 x Crossa | O | 48 | 127 | 175 | 9:55 | 25.8702** | < 0.01 |
| | E | 24.61 | 150.39 | | | | |
| Triplo-6 x Crossa | O | 28 | 212 | 240 | 9:55 | 1.1399 | 0.30 - 0.20 |
| | E | 33.75 | 206.25 | | | | |
| Triplo-7 x Crossa | O | 40 | 255 | 295 | 9:55 | 0.0618 | 0.90 - 0.80 |
| | E | 41.48 | 253.52 | | | | |
| Triplo-8 x Crossa | O | 20 | 79 | 99 | 9:55 | 3.0879 | 0.10 - 0.05 |
| | E | 13.92 | 85.08 | | | | |
| Triplo-9 x Crossa | O | 33 | 49 | 82 | 9:55 | 46.5109** | < 0.01 |
| | E | 11.53 | 70.47 | | | | |
| Triplo-10 x Crossa | O | 12 | 84 | 96 | 9:55 | 0.1939 | 0.70 - 0.50 |
| | E | 13.50 | 82.50 | | | | |
| Triplo-11 x Crossa | O | 31 | 194 | 225 | 9:55 | 0.0151 | 0.95 - 0.90 |
| | E | 31.64 | 193.36 | | | | |
| Triplo-12 x Crossa | O | 29 | 52 | 81 | 9:55 | 31.6780** | < 0.01 |
| | E | 11.39 | 69.61 | | | | |

** = Significant deviation at 0.01 probability level

trisomic $F_2$ populations showed a skewed segregation in the occurrence of purple plants predominating the populations, which was reflected in higher values of calculated chi-square. The calculated chi-square values for trisomics- 1, 5, 9 and 12 were 23.6255, 25.8702, 46.5109 and 31.6780 respectively.

It is evident that contrary to the expectation for a tri hybrid interaction, trisomics-1, 5, 9 and 12 seem to be the critical trisomics.

4.2.1.12 Internode pigmentation

As depicted in Table 10, all of the female parents, the donor parent, the disomic and trisomic $F_1$s and the trisomic $BC_1F_1$ were devoid of any pigmentation in the internode owing to the action of an inhibitory gene.

The $F_2$ segregation of internode pigmentation in trisomics and marker crosses is detailed in Table 33. A perusal of the table revealed that four interacting genes were involved for realization of internode pigmentation as detailed earlier (refer 4.1.5). The $F_2$ populations of trisomics-6, 7 and 11 showed a significant deviation from the expected 9:247 segregation ratio at 0.01 probability level, whereas the deviation was at 0.05 level of probability in case of $BC_1F_2$ population of trisomic-1, wherein a ratio of 15.61:240.39 was realized.

Table 33 $F_2$ segregation of internode pigmentation in trisomics and marker crosses

| Cross combination | | $F_2$ phenotypic segregation | | Total | Confirmed genetic ratio | | 'P' |
|---|---|---|---|---|---|---|---|
| | | Purple (+) | Green (-) | | | | |
| Disomic x Crossa (Sona control) | O | 33 | 867 | 900 | 9:247 | 0.0605 | 0.90 - 0.80 |
| | E | 31.64 | 868.36 | | | | |
| Triplo-1 x Crossa | O | 12 | 208 | 220 | 9:247 | 2.4383 | 0.20 - 0.10 |
| | E | 7.73 | 212.27 | | | | |
| Triplo-2 x Crossa | O | 2 | 95 | 97 | 9:247 | 0.5044 | 0.50 - 0.30 |
| | E | 3.41 | 93.59 | | | | |
| Triplo-3 x Crossa | O | 17 | 358 | 375 | 9:247 | 1.1450 | 0.30 - 0.20 |
| | E | 13.18 | 361.82 | | | | |
| Triplo-4 x Crossa ($F_2$) | O | 0 | 6 | 06 | 9:247 | 0.2186 | 0.70--0.50 |
| | E | 0.21 | 5.79 | | | | |
| Triplo-4 x Crossa ($BC_1F_2$) | O | 0 | 72 | 72 | 15.61:240.39 | 4.5754* | 0.05 - 0.02 |
| | E | 4.39 | 67.61 | | | | |
| Triplo-5 x Crossa | O | 7 | 168 | 175 | 9:247 | 0.1210 | 0.80 - 0.70 |
| | E | 6.15 | 168.85 | | | | |
| Triplo-6 x Crossa | O | 83 | 157 | 240 | 9:247 | 682.9205** | < 0.01 |
| | E | 8.44 | 231.56 | | | | |
| Triplo-7 x Crossa | O | 0 | 295 | 295 | 9:247 | 10.7490** | < 0.01 |
| | E | 10.37 | 284.63 | | | | |
| Triplo-8 x Crossa | O | 5 | 94 | 99 | 9:247 | 0.6876 | 0.50 - 0.30 |
| | E | 3.48 | 95.52 | | | | |
| Triplo-9 x Crossa | O | 0 | 82 | 82 | 9:247 | 2.9879 | 0.10 - 0.05 |
| | E | 2.88 | 79.12 | | | | |
| Triplo-10 x Crossa | O | 2 | 94 | 96 | 9:247 | 0.5806 | 0.50 - 0.30 |
| | E | 3.38 | 92.63 | | | | |
| Triplo-11 x Crossa | O | 0 | 225 | 225 | 9:247 | 8.1984** | < 0.01 |
| | E | 7.91 | 217.09 | | | | |
| Triplo-12 x Crossa | O | 0 | 81 | 81 | 9:247 | 2.9514 | 0.10 - 0.05 |
| | E | 2.85 | 78.15 | | | | |

** = Significant deviation at 0.01 probability level

* = Significant deviation at0.05 probability level

The calculated chi-square values for the $BC_1F_2$ of trisomic-4 was 4.6754, whereas the values of 10.7490 and 8.1984 were realized for the $F_2$ populations involving trisomics-7 and 11, respectively. All these three populations exhibited, a skewed segregation in the occurrence of more than expected proportion of green plants. Interesting was the case of the $F_2$ population involving trisomic-6. A significantly higher value of calculated chi-square, that is, 682.9205, was realized in this case wherein the skewness of $F_2$ segregation was due to the higher proportion of plants with purple internode.

It appears that trisomics-4, 6, 7 and 11 are the critical trisomics.

4.2.1.13 Nodal pigmentation

Table 10 details the description of node pigmentation in the parents as well as in the disomic and trisomic $F_1$s. As may be seen from the table, all the parents as well as the disomic and trisomic $F_1$s remained devoid of nodal pigmentation as in the case of internode.

The $F_2$ segregation for node pigmentation in trisomics and marker crosses is presented in Table 34. A perusal of the table revealed that pigmentation in nodal region was due to the operation of four interacting genes as mentioned earlier (refer 4.1.5).

Table 34. $F_2$ segregation of node pigmentation in trisomics and marker crosses

| Cross combination | | $F_2$ phenotypic segregation | | Total | Confirmed genetic ratio | $x^2$ | 'P' |
|---|---|---|---|---|---|---|---|
| | | Purple(+) | Green(-) | | | | |
| Disomic x Crossa (Sona control) | O | 30 | 870 | 900 | 9:247 | 0.0882 | 0.80 - 0.70 |
| | E | 31.64 | 868.36 | | | | |
| Triplo-1 x Crossa | O | 4 | 216 | 220 | 9:247 | 1.8688 | 0.20 - 0.10 |
| | E | 7.73 | 212.27 | | | | |
| Triplo-2 x Crossa | O | 2 | 95 | 97 | 9:247 | 0.6044 | 0.50 - 0.30 |
| | E | 3.41 | 93.59 | | | | |
| Triplo-3 x Crossa | O | 13 | 362 | 375 | 9:247 | 0.0026 | > 0.99 |
| | E | 13.18 | 361.82 | | | | |
| Triplo-4 x Crossa ($F_2$) | O | 0 | 6 | 06 | 9:247 | 0.2186 | 0.70 - 0.50 |
| | E | 0.21 | 5.79 | | | | |
| Triplo-4 x Crossa ($BC_1F_2$) | O | 1 | 71 | 72 | 15.61:240.39 | 2.7881 | 0.10 -0.05 |
| | E | 4.39 | 67.61 | | | | |
| Triplo-5 x Crossa | O | 0 | 175 | 175 | 9:247 | 6.3765* | 0.02 - 0.01 |
| | E | 6.15 | 168.85 | | | | |
| Triplo-6 x Crossa | O | 16 | 224 | 240 | 9:247 | 7.0252** | < 0.01 |
| | E | 8.44 | 231.56 | | | | |
| Triplo-7 x Crossa | O | 10 | 285 | 295 | 9:247 | 0.0138 | 0.95- 0.90 |
| | E | 10.37 | 284.63 | | | | |
| Triplo-8 x Crossa | O | 0 | 99 | 99 | 9:247 | 3.6073 | 0.10 - 0.05 |
| | E | 3.48 | 95.52 | | | | |
| Triplo-9 x Crossa | O | 1 | 81 | 82 | 9:247 | 1.2745 | 0.30 - 0.20 |
| | E | 2.88 | 79.12 | | | | |
| Triplo-10 x Crossa | O | 0 | 96 | 96 | 9:247 | 3.4980 | 0.10 - 0.05 |
| | E | 3.38 | 92.62 | | | | |
| Triplo-11 x Crossa | O | 28 | 197 | 225 | 9:247 | 52.8824** | < 0.01 |
| | E | 7.91 | 217.09 | | | | |
| Triplo-12 x Crossa | O | 10 | 71 | 81 | 9:247 | 18.6188** | < 0.01 |
| | E | 2.85 | 78.15 | | | | |

** = Significant deviation at 0.01 probability level

* = Significant deviation at 0.05 probability level

The $F_2$ populations of trisomics-6, 11 and 12 exhibited a significant departure from the expected 9:247 segregation ratio at 0.01 level of probability, whereas, the $F_2$ population of trisomic-5 showed deviation at 0.05 level of probability. The calculated chi-square values of 7.0252, 52.8824 and 18.6188 were realized for the $F_2$ populations of trisomics-6, 11 and 12 respectively. The deviation of the chi-square in case of these trisomics was due to the skewness of segregation towards more than expected plants with purple node. On the contrary, trisomic-5 revealed the calculated chi-square value of 6.3765, wherein the skewness of segregation was due to a higher proportion of green plants.

As indicated by the modified ratios in the trisomics-5, 6, 11 and 12, the respective chromosomes 5, 6, 11 and 12 seem to be critical.

4.2.1.14 Sheath pulvinus pigmentation

The sheath pulvinus pigmentation in the parents as well as in the disomic and trisomic $F_1$s is detailed in Table 10, similar to node and internode pigmentation, the sheath pulvinus was also devoid of any pigmentation in all the parents and the $F_1$s.

The $F_2$ segregation of sheath pulvinus pigmentation in trisomics and marker crosses is presented in Table 35. The data revealed that pigmentation in sheath pulvinus was due to the operation of four genes, as mentioned earlier (refer 4.1.7).

Table 35 $F_2$ segregation of sheath pulvinus pigmentation in trisomics and marker crosses

| Cross combination | | $F_2$ phenotypic segregation | | Total | Confirmed genetic ratio | $x^2$ | 'P' |
|---|---|---|---|---|---|---|---|
| | | Purple(+) | Green(-) | | | | |
| Disomic x Crossa (Sona control) | O | 10 | 890 | 900 | 3:253 | 0.0287 | 0.90 - 0.80 |
| | E | 10.55 | 889.45 | | | | |
| Triplo-1 x Crossa | O | 0 | 220 | 220 | 3:253 | 2.6087 | 0.20 - 0.10 |
| | E | 2.58 | 217.42 | | | | |
| Triplo-2 x Crossa | O | 0 | 97 | 97 | 3:253 | 1.1502 | 0.30 - 0.20 |
| | E | 1.14 | 95.86 | | | | |
| Triplo-3 x Crossa | O | 0 | 375 | 375 | 3:253 | 4.4466* | 0.05 - 0.02 |
| | E | 4.39 | 370.61 | | | | |
| Triplo-4 x Crossa ($F_2$) | O | 0 | 6 | 06 | 3:253 | 0.0711 | 0.80 - 0.70 |
| | E | 0.07 | 5.93 | | | | |
| Triplo-4 x Crossa ($BC_1F_2$) | O | 0 | 72 | 72 | 19.93:236.07 | 6.0785* | 0.02 - 0.01 |
| | E | 5.61 | 66.39 | | | | |
| Triplo-5 x Crossa | O | 0 | 175 | 175 | 3:253 | 2.0751 | 0.20 - 0.10 |
| | E | 2.05 | 172.95 | | | | |
| Triplo-6 x Crossa | O | 0 | 240 | 240 | 3:253 | 2.8458 | 0.10 - 0.05 |
| | E | 2.81 | 237.19 | | | | |
| Triplo-7 x Crossa | O | 0 | 295 | 295 | 3:253 | 3.4980 | 0.10 - 0.05 |
| | E | 3.46 | 291.54 | | | | |
| Triplo-8 x Crossa | O | 0 | 99 | 99 | 3:253 | 1.1739 | 0.30 - 0.20 |
| | E | 1.16 | 97.84 | | | | |
| Triplo-9 x Crossa | O | 0 | 82 | 82 | 3:253 | 0.9723 | 0.50 - 0.30 |
| | E | 0.96 | 81.04 | | | | |
| Triplo-10 x Crossa | O | 0 | 96 | 96 | 3:253 | 1.1383 | 0.30 - 0.20 |
| | E | 1.12 | 94.88 | | | | |
| Triplo-11 x Crossa | O | 0 | 225 | 225 | 3:253 | 2.6680 | 0.20 - 0.10 |
| | E | 2.64 | 222.36 | | | | |
| Triplo-12 x Crossa | O | 0 | 81 | 81 | 3:253 | 0.9605 | 0.50 - 0.30 |
| | E | 0.95 | 80.05 | | | | |

* = Significant deviation at 0.05 probability level

It is interesting to note that all the trisomic $F_2$ populations have shown a skewed segregation in the occurrence of only green plants predominating the population. Further, the populations of all the trisomic $F_2$ was of a small size and this might be the reason for not recovering a single purple plant in the trisomic $F_2$ populations.

However, based on the extent of deviation from the expected 3:253 and 19.93:236.07 segregation ratios for trisomic-3 and 4 respectively, it is evident that these are probably the critical trisomics. The value of calculated chi-square in case of the $F_2$ population of trisomic-3 was 4.4466 at 0.05 level of probability, whereas at the same level of probability, the $BC_1F_2$ population of trisomic-4 showed the calculated chi-square value of 6.0785.

4.2.1.15 Pulvinus ring pigmentation

Table 10 details the description of pulvinus ring pigmentation in the parents as well as in the disomic and trisomic $F_1$s. The presence of pigmentation in pulvinus ring was dominant over its absence and hence, as per expectation, the disomic and the trisomic $F_1$s showed purple pigmentation in the pulvinus ring region.

The $F_2$ segregation for pulvinus ring pigmentation in trisomics and marker crosses is detailed in Table 36. The data revealed that pigmentation in pulvinus ring was due to the interaction of four genes as depicted earlier( refer 4.1.2).

Table 36. $F_2$ Segregation of pulvinus ring pigmentation in trisomics and marker crosses

| Cross combination | | $F_2$ phenotypic segregation | | Total | Confirmed genetic ratio | $x^2$ | 'P' |
|---|---|---|---|---|---|---|---|
| | | Purple (+) | Green(-) | | | | |
| Disomic x Crossa (Sona control) | O | 651 | 249 | 900 | 45:19 | 1.7607 | 0.20 - 0.10 |
| | E | 632.81 | 267.19 | | | | |
| Triplo-1 x Crossa | O | 145 | 75 | 220 | 45:19 | 2.0436 | 0.20 - 0.10 |
| | E | 154.69 | 65.31 | | | | |
| Triplo-2 x Crossa | O | 51 | 46 | 97 | 45:19 | 14.6163** | < 0.01 |
| | E | 68.20 | 28.80 | | | | |
| Triplo-3 x Crossa | O | 250 | 125 | 375 | 45:19 | 2.3879 | 0.20 - 0.10 |
| | E | 263.67 | 111.33 | | | | |
| Triplo-4 x Crossa ($F_2$) | O | 6 | 0 | 06 | 45:19 | 2.5333 | 0.20 - 0.10 |
| | E | 4.22 | 1.78 | | | | |
| Triplo-4 x Crossa ($BC_1F_2$) | O | 49 | 23 | 72 | 19.02:44.98 | 50.6634** | < 0.01 |
| | E | 21.40 | 50.60 | | | | |
| Triplo-5 x Crossa | O | 132 | 43 | 175 | 45:19 | 2.1943 | 0.20 - 0.10 |
| | E | 123.05 | 51.95 | | | | |
| Triplo-6 x Crossa | O | 172 | 68 | 240 | 45:19 | 0.2108 | 0.70 - 0.50 |
| | E | 168.75 | 71.25 | | | | |
| Triplo-7 x Crossa | O | 215 | 80 | 295 | 45:19 | 0.9326 | 0.50 - 0.30 |
| | E | 207.42 | 87.58 | | | | |
| Triplo-8 x Crossa | O | 73 | 26 | 99 | 45:19 | 0.5563 | 0.50 - 0.30 |
| | E | 69.61 | 29.39 | | | | |
| Triplo-9 x Crossa | O | 65 | 17 | 82 | 45:19 | 3.1508 | 0.10 - 0.05 |
| | E | 57.66 | 24.34 | | | | |
| Triplo-10 x Crossa | O | 45 | 51 | 96 | 45:19 | 25.2632** | < 0.01 |
| | E | 67.50 | 28.50 | | | | |
| Triplo-11 x Crossa | O | 148 | 77 | 225 | 45:19 | 2.2166 | 0.20 - 0.10 |
| | E | 158.20 | 66.80 | | | | |
| Triplo-12 x Crossa | O | 73 | 08 | 81 | 45:19 | 15.2296** | < 0.01 |
| | E | 56.95 | 24.05 | | | | |

** = Significant deviation at 0.01 probability level

As may be seen from the table, the $F_2$ populations of trisomics-2, 10 and 12 and the $BC_1F_2$ population involving trisomic-4 showed a significant deviation from the expected trigenic ratios of 45:19 and 19.02:44.98 respectively at 0.01 level of probability.

The $BC_1F_2$ population of trisomic-4 and the $F_2$ populations of trisomics-2, 10 and 12 showed calculated chi-square values of 50.6634, 14.6163, 25.2632 and 15.2296 respectively. Such deviated values of chi-square were due to a skewed segregation. With regard to the $BC_1F_2$ population of trisomic-4 and the $F_2$ population of trisomic 12 the skewness of segregation was towards the purple plants predominating the population whereas on the contrary, the $F_2$ populations of trisomics-2 and 10 constituted more than expected proportion of green plants.

It is needless to say that the trisomics-2, 4, 10 and 12 seem to be the critical trisomics.

4.2.1.16 Glumes pigmentation

The pigmentation of glumes in the parents as well as in the disomic and trisomic $F_1$s is detailed in Table 10. Although the donor parent showed pigmented glumes, all the trisomic and the disomic $F_1$s remained devoid of any pigmentation due to the action of an inhibitory gene.

The $F_2$ segregation for pigmentation of glumes in trisomics and marker crosses is presented in Table 37. A perusal of the table revealed that pigmentation in glumes was under a trigenic control as elaborated earlier ( refer 4.1.3). The $F_2$ populations of trisomics-6 and 8 showed a significant deviation from the expected 9:55 segregation ratio at 0.05 level of probability, whereas the $F_2$ of trisomic 12 deviated significantly at 0.01 level of probability. The calculated chi-square values of these three trisomic $F_2$s that is, trisomics-6, 8 and 12 were 5.6048, 5.2454 and 18.9211 respectively. The deviation of the chi-square values from that of disomic control was due to a skewed segregation. The $F_2$ populations of trisomics-6 and 8 showed a skewness of segregation due to the occurrence of more than expected proportion of green plants, while on the contrary the $F_2$ of trisomic 12 had purple plants in abundance.

As inferred from the modified ratios in the trisomic $F_2$s of triplos-6, 8 and 12, the respective chromosomes-6, 8 and 12 seem to be the critical trisomics.

4.2.1.17 Lemma and palea pigmentation

As detailed in Table 10, all the female parents, the donor parent, the disomic and trisomic $F_1$s and the trisomic $BC_1F_1$ were devoid of any pigmentation in lemma and palea.

The $F_2$ segregation of lemma and palea pigmentation in trisomics and marker crosses is presented in Table 38. The

Table 37. $F_2$ segregation of glumes pigmentation in trisomics and marker crosses

| Cross combination | | $F_2$ phenotypic segregation | | Total | Confirmed genetic ratio | $\chi^2$ | 'P' |
|---|---|---|---|---|---|---|---|
| | | Purple(+) | Green(-) | | | | |
| Disomic x Crossa (Sona control) | O | 115 | 785 | 900 | 9:55 | 1.2292 | 0.30 - 0.20 |
| | E | 126.56 | 773.44 | | | | |
| Triplo-1 x Crossa | O | 34 | 186 | 220 | 9:55 | 0.3528 | 0.70 - 0.50 |
| | E | 30.94 | 189.06 | | | | |
| Triplo-2 x Crossa | O | 8 | 89 | 97 | 9:55 | 2.7142 | 0.10 - 0.05 |
| | E | 13.64 | 83.36 | | | | |
| Triplo-3 x Crossa | O | 46 | 329 | 375 | 9:55 | 1.0007 | 0.50 - 0.30 |
| | E | 52.73 | 322.27 | | | | |
| Triplo-4 x Crossa ($F_2$) | O | 0 | 6 | 06 | 9:55 | 0.9818 | 0.50 - 0.30 |
| | E | 0.84 | 5.16 | | | | |
| Triplo-4 x Crossa ($BC_1F_2$) | O | 0 | 72 | 72 | 0.64:63.36 | 0.7273 | 0.50 - 0.30 |
| | E | 0.72 | 71.28 | | | | |
| Triplo-5 x Crossa | O | 22 | 153 | 175 | 9:55 | 0.3220 | 0.70 - 0.50 |
| | E | 24.61 | 150.39 | | | | |
| Triplo-6 x Crossa | O | 21 | 219 | 240 | 9:55 | 5.6048* | 0.02 - 0.01 |
| | E | 33.75 | 206.25 | | | | |
| Triplo-7 x Crossa | O | 39 | 256 | 295 | 9:55 | 0.1731 | 0.70 - 0.50 |
| | E | 41.48 | 253.52 | | | | |
| Triplo-8 x Crossa | O | 6 | 93 | 99 | 9:55 | 5.2454* | 0.05 - 0.02 |
| | E | 13.92 | 85.08 | | | | |
| Triplo-9 x Crossa | O | 12 | 70 | 82 | 9:55 | 0.0222 | 0.90 - 0.80 |
| | E | 11.53 | 70.47 | | | | |
| Triplo-10 x Crossa | O | 10 | 86 | 96 | 9:55 | 1.0559 | 0.50 - 0.30 |
| | E | 13.50 | 82.50 | | | | |
| Triplo-11 x Crossa | O | 28 | 197 | 225 | 9:55 | 0.4974 | 0.50 - 0.30 |
| | E | 31.64 | 193.36 | | | | |
| Triplo-12 x Crossa | O | 25 | 56 | 81 | 9:55 | 18.9211** | < 0.01 |
| | E | 11.39 | 69.61 | | | | |

** = Significant deviation at 0.01 probability level
* = Significant deviation at 0.05 probability level

Table 38. $F_2$ segregation of lemma and palea pigmentation in trisomics and marker crosses

| Cross combination | | $F_2$ Phenotypic segregation | | Total | Confirmed genetic ratio | $x^2$ | 'P' |
|---|---|---|---|---|---|---|---|
| | | Purple(+) | Green(-) | | | | |
| Disomic x Crossa (Sona control) | O | 21 | 879 | 900 | 9:247 | 3.7088 | 0.10 - 0.05 |
| | E | 31.64 | 868.36 | | | | |
| Triplo-1 x Crossa | O | 1 | 219 | 220 | 9:247 | 6.0773* | 0.02 - 0.01 |
| | E | 7.73 | 212.27 | | | | |
| Triplo-2 x Crossa | O | 1 | 96 | 97 | 9:247 | 1.7655 | 0.20 - 0.10 |
| | E | 3.41 | 93.59 | | | | |
| Triplo-3 x Crossa | O | 2 | 373 | 375 | 9:247 | 9.8327** | < 0.01 |
| | E | 13.18 | 361.82 | | | | |
| Triplo-4 x Crossa ($F_2$) | O | 0 | 6 | 06 | 9:247 | 0.2186 | 0.70 - 0.50 |
| | E | 0.21 | 5.79 | | | | |
| Triplo-4 x Crossa ($BC_1F_2$) | O | 1 | 71 | 72 | 15.61:240.39 | 2.7881 | 0.10 - 0.05 |
| | E | 4.39 | 67.61 | | | | |
| Triplo-5 x Crossa | O | 3 | 172 | 175 | 9:247 | 1.6741 | 0.20 - 0.10 |
| | E | 6.15 | 168.85 | | | | |
| Triplo-6 x Crossa | O | 0 | 240 | 240 | 9:247 | 8.7449** | < 0.01 |
| | E | 8.44 | 231.56 | | | | |
| Triplo-7 x Crossa | O | 0 | 295 | 295 | 9:247 | 10.7490** | < 0.01 |
| | E | 10.37 | 284.63 | | | | |
| Triplo-8 x Crossa | O | 0 | 99 | 99 | 9:247 | 3.6073 | 0.10 - 0.05 |
| | E | 3.48 | 95.52 | | | | |
| Triplo-9 x Crossa | O | 0 | 82 | 82 | 9:247 | 2.9879 | 0.10 - 0.05 |
| | E | 2.88 | 79.12 | | | | |
| Triplo-10 x Crossa | O | 0 | 96 | 96 | 9:247 | 3.4980 | 0.10 - 0.05 |
| | E | 3.38 | 92.63 | | | | |
| Triplo-11 x Crossa | O | 0 | 225 | 225 | 9:247 | 8.1984** | < 0.01 |
| | E | 7.91 | 217.09 | | | | |
| Triplo-12 x Crossa | O | 0 | 81 | 81 | 9:247 | 2.9514 | 0.10 - 0.05 |
| | E | 2.85 | 78.15 | | | | |

** = Significant deviation at 0.01 probability level

* = Significant deviation at 0.05 probability level

data revealed that pigmentation in lemma and palea was under tetragenic control as detailed earlier (refer 4.1.5). As may be seen from the table, the $F_2$ populations involving trisomics-3, 6, 7 and 11 exhibited a significant deviation from the expected 9:247 segregation ratio at 0.01 level of probability, whereas in case of the $F_2$ of trisomic-1, significant deviation was realized at 0.05 level of probability. The $F_2$ populations of these trisomics-1, 3, 6, 7 and 11 showed a skewed segregation with the occurrence of a higher proportion of green plants predominating in the populations, which was reflected in the higher values of calculated chi-square. The calculated chi-square values for trisomics-1, 3, 6, 7 and 11 were 6.0773, 9.8327, 8.7449, 10.7490 and 8.1984 respectively.

Thus, it is evident that the trisomics-1, 3, 6, 7 and 11 seem to be the critical trisomics, contrary to the expectation for a tetragenic segregation ratio.

4.2.1.18 Apiculus pigmentation

The Table 10 details the description of apiculus pigmentation in the parents as well as in the disomic and trisomic $F_1$s. The presence of pigmentation in apiculus was dominant over its absence and hence, as per expectation, the disomic and the trisomic $F_1$s showed pigmented apiculus.

The $F_2$ segregation of apiculus pigmentation in trisomics and marker crosses is detailed in Table 39. The data

Table 39. $F_2$ segregation of apiculus pigmentation in trisomics and marker crosses

| Cross combination | | $F_2$ phenotypic segregation | | Total | Confirmed genetic ratio | $x^2$ | 'P' |
|---|---|---|---|---|---|---|---|
| | | Purple(+) | Green(-) | | | | |
| Disomic x Crossa (Sona control) | O | 665 | 245 | 900 | 45:19 | 2.6204 | 0.20 - 0.10 |
| | E | 632.91 | 267.19 | | | | |
| Triplo-1 x crossa | O | 150 | 70 | 220 | 45:19 | 0.4785 | 0.50 - 0.30 |
| | E | 154.69 | 65.31 | | | | |
| Triplo-2x Crossa | O | 51 | 46 | 97 | 45:19 | 14.6163** | < 0.01 |
| | E | 68.20 | 28.80 | | | | |
| Triplo-3 x Crossa | O | 250 | 125 | 375 | 45:19 | 2.3879 | 0.20 - 0.10 |
| | E | 263.67 | 111.33 | | | | |
| Triplo-4 x Crossa ($F_2$) | O | 6 | 0 | 06 | 45:19 | 2.5333 | 0.20 - 0.10 |
| | E | 4.22 | 1.78 | | | | |
| Triplo-4 x Crossa ($BC_1F_2$) | O | 49 | 23 | 72 | 19.02:44.98 | 50.6634** | < 0.01 |
| | E | 21.40 | 50.60 | | | | |
| Triplo-5 x Crossa | O | 134 | 41 | 175 | 45:19 | 3.2842 | 0.10 - 0.05 |
| | E | 123.05 | 51.95 | | | | |
| Triplo-6 x Crossa | O | 170 | 70 | 240 | 45:19 | 0.0312 | 0.90 - 0.80 |
| | E | 168.75 | 71.25 | | | | |
| Triplo-7 x Crossa | O | 213 | 82 | 295 | 45:19 | 0.5053 | 0.50 - 0.30 |
| | E | 207.42 | 87.58 | | | | |
| Triplo-8 x Crossa | O | 77 | 22 | 99 | 45:19 | 2.5431 | 0.20 - 0.10 |
| | E | 69.61 | 29.39 | | | | |
| Triplo-9 x Crossa | O | 65 | 17 | 82 | 45:19 | 3.1508 | 0.10 - 0.05 |
| | E | 57.66 | 24.34 | | | | |
| Triplo-10 x Crossa | O | 59 | 37 | 96 | 45:19 | 3.6055 | 0.10 - 0.05 |
| | E | 67.50 | 28.50 | | | | |
| Triplo-11 x Crossa | O | 145 | 80 | 225 | 45:19 | 3.7116 | 0.10 - 0.05 |
| | E | 158.20 | 66.80 | | | | |
| Triplo-12 x Crossa | O | 73 | 08 | 81 | 45:19 | 15.2296** | < 0.01 |
| | E | 56.95 | 24.05 | | | | |

** = Significant deviation at 0.01 probability level.

clearly revealed that three interacting genes were responsible for governing pigmentation in apiculus, as elaborated elsewhere ( refer 4.1.2). As may be seen from the table, the $F_2$ populations of trisomic-2 and 12 and the $BC_1F_2$ population of trisomic-4 revealed a significant deviation from the expected 45:19 and 19.02:44.98 trigenic segregation ratios respectively at 0.01 level of probability. The $F_2$ of trisomic-2 showed a skewed segregation in the occurrence of more than expected proportion of green plants, thereby showing a calculated chi-square value of 14.6163. On the contrary, the $BC_1F_2$ of trisomic-4 and the $F_2$ of trisomic-12 constituted predominantly of purple plants, thereby showing the calculated chi-square values of 50.6634 and 15.2296 respectively.

It appears that the trisomics-2, 4 and 12 are the critical trisomics.

4.2.1.19 Stigma pigmentation

As depicted in Table 10 the donor parent as well as the disomic and trisomic $F_1$s showed pigmented stigma indicating that the pigmentation in stigma was a dominant trait.

The $F_2$ segregation for stigma pigmentation in trisomics and marker crosses is presented in Table 40. It revealed that stigma pigmentation was also under a trigenic control like that of apiculus ( refer 4.1.2). As detailed in the table, the $F_2$ populations of trisomics 4, 2 and 12 and the $BC_1F_2$ of trisomic-4 showed a significant deviation from the

Table 40 $F_2$ segregation of stigma pigmentation in trisomics and marker crosses

| Cross combination | | $F_2$ phenotypic segregation | | Total | Confirmed genetic ratio | $\chi^2$ | 'P' |
|---|---|---|---|---|---|---|---|
| | | Purple(+) | Green(-) | | | | |
| Disomic x Crossa (Gona control) | O | 651 | 249 | 900 | 45:19 | 1.7607 | 0.20 - 0.10 |
| | E | 632.81 | 267.19 | | | | |
| Triplo-1 x Crossa | O | 121 | 99 | 220 | 45:19 | 24.7120** | < 0.01 |
| | E | 154.69 | 65.31 | | | | |
| Triplo-2 x Crossa | O | 51 | 46 | 97 | 45:19 | 14.6163** | < 0.01 |
| | E | 68.20 | 28.80 | | | | |
| Triplo-3 x Crossa | O | 250 | 125 | 375 | 45:19 | 2.3879 | 0.20 - 0.10 |
| | E | 263.67 | 111.33 | | | | |
| Triplo-4 x Crossa ($F_2$) | O | 6 | 0 | 06 | 45:19 | 2.5333 | 0.20 - 0.10 |
| | E | 4.22 | 1.78 | | | | |
| Triplo-4 x Crossa ($BC_1F_2$) | O | 49 | 23 | 72 | 19.02:44.98 | 50.6634** | < 0.01 |
| | E | 21.40 | 50.60 | | | | |
| Triplo-5 x Crossa | O | 134 | 41 | 175 | 45:19 | 3.2842 | 0.10 - 0.05 |
| | E | 123.05 | 51.95 | | | | |
| Triplo-6 x Crossa | O | 172 | 68 | 240 | 45:19 | 0.2108 | 0.70 - 0.50 |
| | E | 168.75 | 71.25 | | | | |
| Triplo-7 x Crossa | O | 216 | 79 | 295 | 45:19 | 1.1950 | 0.30 - 0.20 |
| | E | 207.42 | 87.58 | | | | |
| Triplo-8 x Crossa | O | 78 | 21 | 99 | 45:19 | 3.4068 | 0.10 - 0.05 |
| | E | 69.61 | 29.39 | | | | |
| Triplo-9 x Crossa | O | 65 | 17 | 82 | 45:19 | 3.1508 | 0.10 - 0.05 |
| | E | 57.66 | 24.34 | | | | |
| Triplo-10 x Crossa | O | 59 | 37 | 96 | 45:19 | 3.6055 | 0.10 - 0.05 |
| | E | 67.50 | 28.50 | | | | |
| Triplo-11 x Crossa | O | 148 | 77 | 225 | 45:19 | 2.2166 | 0.20 - 0.10 |
| | E | 158.20 | 66.80 | | | | |
| Triplo-12 x Crossa | O | 73 | 08 | 81 | 45:19 | 15.2296** | < 0.01 |
| | E | 56.95 | 24.05 | | | | |

** = Significant deviation at 0.01 probability level

expected trigenic 45:19 and 19.02:44.98 segregation ratios respectively at 0.01 probability level. The $F_2$ populations of trisomics-1 and 2 had the calculated chi-square values of 24.7120 and 14.6163 respectively. Such a deviation in calculated chi-squares from that of the value of disomic chi-square was due to the skewness of segregation. In case of both the populations, plants with colourless stigma were in more than expected proportion. With regard to the $BC_1F_2$ population of trisomic-4 and the $F_2$ populations of trisomics-4 and 12, the skewness of segregation was towards higher proportion of plants with purple stigma; thereby showing the calculated chi-square values of 50.6634 and 15.2296 respectively.

As evidenced from the data, the trisomics-1, 2, 4 and 12 seem to be the critical trisomics, contrary to the expectation for a trihybrid ratio.

4.2.1.20 Panicle base pigmentation

As detailed in Table 10, all the parents as well as the $F_1$s were devoid of any pigmentation in panicle base.

The $F_2$ segregation of panicle base pigmentation in trisomics and marker crosses is presented in Table 41. It revealed that panicle base pigmentation was due to the operation of four interacting genes, as detailed earlier (refer 4.1.5). As may be seen from the table the $F_2$ populations

Table 41. $F_2$ segregation of panicle base pigmentation in trisomics and marker crosses

| Cross combination | $F_2$ phenotypic segregation | | | Total | Confirmed genetic ratio | $\chi^2$ | 'P' |
|---|---|---|---|---|---|---|---|
| | | Purple(+) | Green(-) | | | | |
| Disomic x Crossa (Sona control) | O | 31 | 869 | 900 | 9:247 | 0.0134 | 0.95 - 0.90 |
| | E | 31.64 | 868.36 | | | | |
| Triplo-1 x Crossa | O | 3 | 217 | 220 | 9:247 | 3.0036 | 0.10 - 0.05 |
| | E | 7.33 | 212.27 | | | | |
| Triplo-2 x Crossa | O | 2 | 95 | 97 | 9:247 | 0.6044 | 0.50 - 0.30 |
| | E | 3.41 | 93.59 | | | | |
| Triplo-3 x Crossa | O | 14 | 361 | 375 | 9:247 | 0.0524 | 0.90 - 0.80 |
| | E | 13.18 | 361.82 | | | | |
| Triplo-4 x Crossa ($F_2$) | O | 0 | 6 | 06 | 9:247 | 0.2186 | 0.70 - 0.50 |
| | E | 0.21 | 5.79 | | | | |
| Triplo-4 x Crossa ($BC_1F_2$) | O | 0 | 72 | 72 | 15.61:240.39 | 4.6754* | 0.05- 0.02 |
| | E | 4.39 | 67.61 | | | | |
| Triplo-5 x Crossa | O | 2 | 173 | 175 | 9:247 | 2.9046 | 0.10 - 0.05 |
| | E | 6.15 | 168.85 | | | | |
| Triplo-6 x Crossa | O | 2 | 238 | 240 | 9:247 | 5.0905* | 0.05 - 0.02 |
| | E | 8.44 | 231.56 | | | | |
| Triplo-7 x Crossa | O | 0 | 295 | 295 | 9:247 | 10.7490** | < 0.01 |
| | E | 10.37 | 284.63 | | | | |
| Triplo-8 x Crossa | O | 0 | 99 | 99 | 9:247 | 3.6073 | 0.10 - 0.05 |
| | E | 3.48 | 95.52 | | | | |
| Triplo-9 x Crossa | O | 0 | 82 | 82 | 9:247 | 2.9879 | 0.10 - 0.05 |
| | E | 2.68 | 79.12 | | | | |
| Triplo-10 x Crossa | O | 1 | 95 | 96 | 9:247 | 1.7322 | 0.20 - 0.10 |
| | E | 3.38 | 92.63 | | | | |
| Triplo-11 x Crossa | O | 1 | 225 | 225 | 9:247 | 8.1984** | < 0.01 |
| | E | 7.91 | 217.09 | | | | |
| Triplo-12 x Crossa | O | 0 | 81 | 81 | 9:247 | 2.9514 | 0.10 - 0.05 |
| | E | 2.85 | 78.15 | | | | |

** = Significant deviation at 0.01 probability level

* = Significant deviation at 0.05 probability level

of trisomics 6, 7 and 11 and the $BC_1F_2$ population of trisomic-4 showed a significant departure from the expected tetragenic 9:247 and 15.61:240.39 segregation ratios respectively. The deviation was significant at 0.05 level of probability for the $BC_1F_2$ population of trisomic-4 and the $F_2$ of trisomic-6; whereas, with respect to the $F_2$ populations of trisomics-7 and 11 the deviation was significant at 0.01 level of probability. The data revealed further, that all these populations also showed a skewed segregation in the occurrence of more than expected proportion of green plants predominating the populations. As a result the $BC_1F_2$ of trisomic-4 showed a calculated chi-square value of 4.6754 and the $F_2$ populations of trisomics-6, 7 and 11 showed the values of 5.0905, 10.7490 and 8.1984 respectively.

Thus, the trisomics-4, 6, 7 and 11 seem to be the critical trisomics.

4.2.1.21 Panicle pulvinus pigmentation

As detailed in Table 10, all the female parents, the donor parent, the disomic and trisomic $F_1$s and the trisomic $BC_1F_1$ remained devoid of any pigmentation in panicle pulvinus, due to the action of an inhibittory gene.

The $F_2$ segregation of panicle pulvinus pigmentation in trisomics and marker crosses is presented in Table 42. It revealed that like panicle base pigmentation, the pigmentation

Table 42. $F_2$ [illegible] gation of panicle colour pigmentation in trisomics and marker crosses

| Cross combination | | $F_2$ phenotypic segregation Purple(+) | . (-) | Total | Confirmed genetic ratio | $\chi^2$ | 'P' |
|---|---|---|---|---|---|---|---|
| Disomic x Crossa (Sona control) | O | 26 | 874 | 900 | 9:247 | 1.0422 | 0.50 - 0.30 |
| | E | 31.64 | 868.36 | | | | |
| Triplo-1 x Crossa | O | 3 | 217 | 220 | 9:247 | 3.0036 | 0.10 - 0.05 |
| | E | 7.73 | 212.27 | | | | |
| Triplo-2 x Crossa | O | 2 | 95 | 97 | 9:247 | 0.6044 | 0.50 - 0.30 |
| | E | 3.41 | 93.59 | | | | |
| Triplo-3 x Crossa | O | 4 | 371 | 375 | 9:247 | 6.6303* | 0.02 - 0.01 |
| | E | 13.18 | 361.82 | | | | |
| Triplo-4 x Crossa ($F_2$) | O | 0 | 6 | 06 | 9:247 | 0.2196 | 0.70 - 0.50 |
| | E | 0.21 | 5.79 | | | | |
| Triplo-4 x Crossa ($BC_1F_2$) | O | 0 | 72 | 72 | 15.6_:240.39 | 4.6754* | 0.05 - 0.02 |
| | E | 4.39 | 67.61 | | | | |
| Triplo-5 x Crossa | O | 2 | 173 | 175 | 9:247 | 2.9046 | 0.10 - 0.05 |
| | E | 6.15 | 168.85 | | | | |
| Triplo-6 x Crossa | O | 2 | 238 | 240 | 9:247 | 5.0905* | 0.05 - 0.02 |
| | E | 8.44 | 231.56 | | | | |
| Triplo-7 x Crossa | O | 0 | 295 | 295 | 9:247 | 10.7490** | < 0.01 |
| | E | 10.37 | 284.63 | | | | |
| Triplo-8 x Crossa | O | 0 | 99 | 99 | 9:247 | 3.6073 | 0.10 - 0.05 |
| | E | 3.48 | 95.52 | | | | |
| Triplo-9 x Crossa | O | 0 | 82 | 82 | 9:247 | 2.9879 | 0.10 - 0.05 |
| | E | 2.88 | 79.12 | | | | |
| Triplo-10 x Crossa | O | 1 | 95 | 96 | 9:247 | 1.7322 | 0.20 - 0.10 |
| | E | 3.38 | 92.63 | | | | |
| Triplo-11 x Crossa | O | 0 | 225 | 225 | 9:247 | 8.1984** | < 0.01 |
| | E | 7.91 | 217.09 | | | | |
| Triplo-12 x Crossa | O | 0 | 81 | 81 | 9:247 | 2.9514 | 0.10 - 0.05 |
| | E | 2.85 | 78.15 | | | | |

** = Significant deviation at 0.01 probability level

* = Significant deviation at 0.05 probability level

in panicle pulvinus was also under the control of four genes (refer 4.1.5). The $F_2$ populations involving trisomics-7 and 11 showed a significant deviation from the expected tetragenic 9:247 segregation ratio at 0.01 level of probability. In case of the $F_2$ populations involving trisomics-3 and 6, the $BC_1F_2$ of trisomic-4, the deviation realized was significant at 0.05 level of probability.

The data further revealed that all the critical populations showed a skewed segregation in that the number of plants with green pulvinus was more than expected. The calculated chi-square value for the $F_2$ of trisomic-3 was 6.6303, for the $BC_1F_2$ of trisomic-4 it was 4.6754 and for the $F_2$s of trisomics-6, 7 and 11 it was 5.0905, 10.7490 and 8.1984 respectively.

Thus, trisomics-3, 4, 6, 7 and 11 appear to be the critical trisomics, contrary to the expectation for a tetra hybrid ratio.

4.2.1.22 Panicle axis pigmentation

The panicle axis pigmentation in the parents as well as in the disomic and trisomic $F_1$s is displayed in Table 10. Although the donor parent showed pigmented panicle axis, all the trisomic and the disomic $F_1$s remained devoid of any pigmentation due to the inhibitory action of a gene.

The $F_2$ segregation for pigmentation in panicle axis in trisomics and marker crosses is presented in Table 43. It revealed that four interacting genes were responsible for conditioning pigmentation in the panicle axis, as detailed earlier (refer 4.1.6). The $F_2$ populations of trisomics-2, 10 and 12 showed a significant deviation from that of the expected tetragenic, 27:229 segregation ratio. The deviation in case of the $F_2$s of trisomics 2 and 12 was significant at 0.01 level of probability, whereas in case of the $F_2$ of trisomic-10 it was significant at 0.05 probability level. The calculated chi-square values realized for the $F_2$ of trisomics-2 and 10 were 7.4022 and 5.6050 respectively. Such a deviation of the chi-square values was due to a skewed segregation constituting more than expected proportion of plants with green panicle axis. Contrary to this, the $F_2$ population of trisomic-12 had higher proportion of plants having pigmented panicle axis and showed a calculated chi-square value of 23.6970.

The trisomic lines 2, 10 and 12 appear to be the critical lines..

4.2.1.23 Nodal septum pigmentation

The nodal septum pigmentation in the parents as well as in the disomic and trisomic $F_1$s is shown in Table 10. As may be seen from the table, all the parents, the disomic $F_1$

Table 43. $F_2$ segregation of panicle axis pigmentation in triploids and marker crosses

| Cross combination | | $F_2$ phenotypic segregation | | Total | Confirmed genetic ratio | $\chi^2$ | 'P' |
|---|---|---|---|---|---|---|---|
| | | Purple (+) | Green(-) | | | | |
| Diploid x Crossa (Sona control) | O | 101 | 799 | 900 | 27:229 | 0.4351 | 0.70 - 0.50 |
| | E | 94.92 | 805.08 | | | | |
| Triplo-1 x Crossa | O | 26 | 194 | 220 | 27:229 | 0.3769 | 0.70 - 0.50 |
| | E | 23.20 | 196.80 | | | | |
| Triplo-2 x Crossa | O | 2 | 95 | 97 | 27:229 | 7.4022** | < 0.01 |
| | E | 10.23 | 86.77 | | | | |
| Triplo-3 x Crossa | O | 38 | 337 | 375 | 27:229 | 0.0680 | 0.80 - 0.70 |
| | E | 39.55 | 335.45 | | | | |
| Triplo-4 x Crossa ($F_2$) | O | 0 | 6 | 06 | 27:229 | 0.7074 | 0.50 - 0.30 |
| | E | 0.63 | 5.37 | | | | |
| Triplo-4 x Crossa ($BC_1F_2$) | O | 0 | 72 | 72 | 1.31:254.69 | 0.3703 | 0.70 - 0.50 |
| | E | 0.37 | 71.63 | | | | |
| Triplo-5 x Crossa | O | 20 | 155 | 175 | 27:229 | 0.1442 | 0.80 - 0.70 |
| | E | 18.46 | 156.54 | | | | |
| Triplo-6 x Crossa | O | 20 | 220 | 240 | 27:229 | 1.2464 | 0.30 - 0.20 |
| | E | 25.31 | 214.69 | | | | |
| Triplo-7 x Crossa | O | 35 | 260 | 295 | 27:229 | 0.5428 | 0.50 - 0.30 |
| | E | 31.11 | 263.89 | | | | |
| Triplo-8 x Crossa | O | 7 | 92 | 99 | 27:229 | 1.2680 | 0.30 - 0.20 |
| | E | 10.44 | 88.56 | | | | |
| Triplo-9 x Crossa | O | 12 | 70 | 82 | 27:229 | 1.4520 | 0.30 - 0.20 |
| | E | 8.65 | 73.35 | | | | |
| Triplo-10 x Crossa | O | 3 | 93 | 96 | 27:229 | 5.6050* | 0.02 - 0.01 |
| | E | 10.13 | 85.87 | | | | |
| Triplo-11 x Crossa | O | 19 | 206 | 225 | 27:229 | 1.0542 | 0.50 - 0.30 |
| | E | 23.73 | 201.27 | | | | |
| Triplo-12 x Crossa | O | 22 | 59 | 81 | 27:229 | 23.6970** | < 0.01 |
| | E | 8.54 | 72.46 | | | | |

** = Significant deviation at 0.01 probability level

* = Significant deviation at 0.05 probability level

and all the trisomic $F_1$s except the $F_1$s of triplos-1 and 3 and the $BC_1F_1$ of triplo-4, remained devoid of any pigmentation in the nodal septum. It was interesting to notice that the $F_1$s of triplos-1 and 3 and the $BC_1F_1$ of triplo-4 had pigmented nodal septum.

The $F_2$ segregation of nodal septum pigmentation in trisomics and marker crosses is presented in Table 44. It revealed that three genes were responsible for conditioning pigmentation in nodal septum, as elaborated elsewhere (refer 4.1.5). The $F_2$ populations of trisomics-1, 2, 3, 4, 6, 10 and 11 and the $BC_1F_2$ of trisomic-4 showed a significant deviation from the expected trigenic, 21:43 and 22.64:41.36 segregation ratios respectively at 0.01 level of probability.

It was also interesting to note that all the $F_2$ populations involving trisomics-1, 2, 3, 6, 10 and 11 showed a skewed segregation characterized with more than expected proportion of plants with colourless nodal septum, which was reflected in their respective calculated chi-square values. The calculated chi-square values of these critical lines were 69.8086, 11.7155, 36.6527, 111.3314, 14.4703 and 20.4226 for trisomics-1, 2, 3, 6, 10 and 11 respectively.

With respect to the $F_2$ and $BC_1F_2$ populations involving trisomic-4, a different behaviour was noticed, as, these populations were characterized with more than expected proportion of plants having purple nodal septum. The calculated

Table 44. $F_2$ segregation of nodal septum pigmentation in trisomics and marker crosses

| Cross combination | $F_2$ phenotypic segregation | | | Total | Confirmed genetic ratio | $X^2$ | 'P' |
|---|---|---|---|---|---|---|---|
| | | Purple(+) | Green(-) | | | | |
| Disomic x Crossa (Sona control) | O | 274 | 626 | 900 | 21:43 | 2.2893 | 0.20 - 0.10 |
| | E | 295.31 | 604.69 | | | | |
| Triplo-1 x Crossa | O | 14 | 206 | 220 | 21:43 | 69.8086** | < 0.01 - |
| | E | 72.19 | 147.81 | | | | |
| Triplo-2 x Crossa | O | 16 | 81 | 97 | 21:43 | 11.7155** | < 0.01 |
| | E | 31.83 | 65.17 | | | | |
| Triplo-3 x Crossa | O | 68 | 307 | 375 | 21:43 | 36.6527** | < 0.01 |
| | E | 123.05 | 251.95 | | | | |
| Triplo-4 x Crossa ($F_2$) | O | 6 | 0 | 06 | 21:43 | 12.2857** | < 0.01 |
| | E | 1.97 | 4.03 | | | | |
| Triplo-4 x Crossa ($BC_1F_2$) | O | 49 | 23 | 72 | 22.64:41.36 | 33.6368** | < 0.01 |
| | E | 25.47 | 46.53 | | | | |
| Triplo-5 x Crossa | O | 60 | 115 | 175 | 21:43 | 0.1723 | 0.70 - 0.50 |
| | E | 57.42 | 117.58 | | | | |
| Triplo-6 x Crossa | O | 2 | 238 | 240 | 21:43 | 111.3314** | < 0.01 |
| | E | 78.75 | 161.25 | | | | |
| Triplo-7 x Crossa | O | 97 | 198 | 295 | 21:43 | 0.0006 | > 0.99 |
| | E | 96.80 | 198.20 | | | | |
| Triplo-8 x Crossa | O | 25 | 74 | 99 | 21:43 | 2.5665 | 0.20 - 0.10 |
| | E | 32.48 | 66.52 | | | | |
| Triplo-9 x Crossa | O | 34 | 48 | 82 | 21:43 | 2.7836 | 0.10 - 0.05 |
| | E | 26.91 | 55.09 | | | | |
| Triplo-10 x Crossa | O | 14 | 82 | 96 | 21:43 | 14.4703** | < 0.01 |
| | E | 31.50 | 64.50 | | | | |
| Triplo-11 x Crossa | O | 42 | 183 | 225 | 21:43 | 20.4226** | < 0.01 |
| | E | 73.83 | 151.17 | | | | |
| Triplo-12 x Crossa | O | 29 | 52 | 81 | 21:43 | 0.3285 | 0.70 - 0.50 |
| | E | 26.58 | 54.42 | | | | |

** = Significant deviation at 0.01 probability level

chi-square values of these critical lines were 12.2857 and 33.6368 respectively.

Contrary to the expectation for a character determined by three operating genes, interestingly, trisomics-1, 2, 3, 4, 6, 10 and 11 seem to be the critical trisomics.

4.2.1.24 Nodal ring pigmentation

The nodal ring pigmentation in the parents as well as in the disomic and trisomic $F_1$s is presented in Table 10. As may be seen from the table, all the parents, the disomic $F_1$ and all the trisomic $F_1$s except the $F_1$s of triplos-1, 2, 3, 4, 6, 7 and 12, remained devoid of any pigmentation in the nodal ring. It was really surprising to notice that seven $F_1$s that is of trisomics-1, 2, 3, 4, 6, 7 and 12 showed a purple nodal ring.

The $F_2$ segregation of nodal ring pigmentation in trisomics and marker crosses is detailed in Table 45. As shown in the table, nodal ring pigmentation was under tetragenic control (refer 4.1.6). The $F_2$ populations involving trisomics-1, 3, 6, 7 and 11 showed a significant departure from the expected tetragenic 27:229 segregation ratio at 0.01 level of probability. All these critical lines were characterized with relatively higher proportion of plants bearing colourless nodal ring thus exhibiting the skewness of segregation towards green plants predominating the populations. This was reflected, as expected in deviated values of

Table 45. $F_2$ segregation of nodal ring pigmentation in trisomics and marker crosses

| Cross combination | | $F_2$ phenotypic segregation | | Total | Confirmed genetic ratio | $X^2$ | 'P' |
|---|---|---|---|---|---|---|---|
| | | Purple(+) | Green(-) | | | | |
| Disomic x Crossa (Sona control) | O | 84 | 816 | 900 | 27:229 | 1.4049 | 0.30 - 0.20 |
| | E | 94.92 | 805.08 | | | | |
| Triplo-1 x Crossa | O | 10 | 210 | 220 | 27:229 | 8.3987** | < 0.01 |
| | E | 23.20 | 196.80 | | | | |
| Triplo-2 x Crossa | O | 10 | 87 | 97 | 27:229 | 0.0058 | 0.95 - 0.90 |
| | E | 10.23 | 86.77 | | | | |
| Triplo-3 x Crossa | O | 7 | 368 | 375 | 27:229 | 29.9483** | < 0.01 |
| | E | 39.55 | 335.45 | | | | |
| Triplo-4 x Crossa ($F_2$) | O | 0 | 6 | 06 | 27:229 | 0.7074 | 0.50 - 0.30 |
| | E | 0.63 | 5.37 | | | | |
| Triplo-4 x Crossa ($BC_1F_2$) | O | 0 | 72 | 72 | 12.03:243.97 | 3.5503 | 0.10 - 0.05 |
| | E | 3.38 | 68.62 | | | | |
| Triplo-5 x Crossa | O | 19 | 156 | 175 | 27:229 | 0.0179 | 0.90 - 0.80 |
| | E | 18.46 | 156.54 | | | | |
| Triplo-6 x Crossa | O | 8 | 232 | 240 | 27:229 | 13.2370** | < 0.01 |
| | E | 25.31 | 214.69 | | | | |
| Triplo-7 x Crossa | O | 0 | 295 | 295 | 27:229 | 34.7817** | < 0.01 |
| | E | 31.11 | 263.89 | | | | |
| Triplo-8 x Crossa | O | 5 | 94 | 99 | 27:229 | 3.1701 | 0.10 - 0.05 |
| | E | 10.44 | 88.56 | | | | |
| Triplo-9 x Crossa | O | 4 | 78 | 82 | 27:229 | 2.7931 | 0.10 - 0.05 |
| | E | 8.65 | 73.35 | | | | |
| Triplo-10 x Crossa | O | 5 | 91 | 96 | 27:229 | 2.9000 | 0.10 - 0.05 |
| | E | 10.12 | 85.88 | | | | |
| Triplo-11 x Crossa | O | 0 | 225 | 225 | 27:229 | 26.5284** | < 0.01 |
| | E | 23.73 | 201.27 | | | | |
| Triplo-12 x Crossa | O | 4 | 77 | 81 | 27:229 | 2.7007 | 0.20 - 0.10 |
| | E | 8.54 | 72.46 | | | | |

** = Significant deviation at 0.01 probability level

calculated chi-square. The calculated chi-square values for the $F_2$ of triplos 1, 3, 6, 7 and 11 were 8.3987, 29.9483, 13.2370, 34.7817 and 26.5284 respectively.

As may be inferred from the modified ratio technique, the triplos-1, 3, 6, 7 and 11 seem to be the critical trisomics. Interestingly, the $F_1$s of trisomics-2, 4 and 12 that had shown pigmented nodal ring (Table 10) exhibited non-significant values of chi-square in the $F_2$ segregation.

4.2.1.25 Awn colour (red awns)

The presence or absence of red awns in the parents as well as in the disomic and trisomic $F_1$s is presented in Table 10. As may be seen from the table, the $F_1$s of trisomics-2, 3, 6 and 10 showed the presence of red awns.

The $F_2$ segregation for presence or absence of red awns in trisomics and marker crosses is presented in Table 46. It revealed that the presence of red awns was under the control of three interacting genes as detailed earlier (refer 4.1.3). Further, the $F_2$ populations involving trisomics-3, 5, 7, 8 and 10 showed significant deviation from the expected trigenic 9:55 segregation ratio at 0.01 level of probability. The $F_2$ populations of trisomics-3, 7 and 10 showed a skewed segregation in the occurrence of more than expected proportion of plants with presence of red awns, which was reflected in the magnitude of calculated chi-square. These populations showed the calculated chi-square values of 32.3103, 11.8060 and

Table 4b. $F_2$ segregation for presence/absence of red anthocyanin trisomics and marker crossers

| Cross combination | | $F_2$ trisomic segregation | | Total | Confirmed Genetic Ratio | $X^2$ | 'P' |
|---|---|---|---|---|---|---|---|
| | | Present(+) | Absent(-) | | | | |
| Disomic x Disomic (Diploid control) | O | 111 | 789 | 900 | 9:55 | 2.2267 | 0.20 - 0.10 |
| | E | 126.56 | 773.44 | | | | |
| Triplo-1 x Crossa | O | 26 | 194 | 220 | 9:55 | 0.9170 | 0.50 - 0.30 |
| | E | 30.94 | 189.06 | | | | |
| Triplo-2 x Crossa | O | 13 | 84 | 97 | 9:55 | 0.0350 | 0.90 - 0.80 |
| | E | 13.64 | 83.36 | | | | |
| Triplo-3 x Crossa | O | 91 | 284 | 375 | 9:55 | 32.3103** | < 0.01 |
| | E | 52.73 | 322.27 | | | | |
| Triplo-4 x Crossa ($F_2$) | O | 2 | 4 | 06 | 9:55 | 1.8438 | 0.20 - 0.10 |
| | E | 0.84 | 5.16 | | | | |
| Triplo-4 x Crossa ($BC_1F_2$) | O | 11 | 61 | 72 | 14.77:49.23 | 2.4678 | 0.20 - 0.10 |
| | E | 16.62 | 55.38 | | | | |
| Triplo-5 x Crossa | O | 8 | 167 | 175 | 9:55 | 13.0444** | < 0.01 |
| | E | 24.61 | 150.39 | | | | |
| Triplo-6 x Crossa | O | 44 | 196 | 240 | 9:55 | 3.6224 | 0.10 - 0.05 |
| | E | 33.75 | 206.25 | | | | |
| Triplo-7 x Crossa | O | 62 | 233 | 295 | 9:55 | 11.8060** | < 0.01 |
| | E | 41.48 | 253.52 | | | | |
| Triplo-8 x Crossa | O | 3 | 96 | 99 | 9:55 | 9.9704** | < 0.01 |
| | E | 13.92 | 85.08 | | | | |
| Triplo-9 x Crossa | O | 16 | 66 | 82 | 9:55 | 2.0152 | 0.20 - 0.10 |
| | E | 11.53 | 70.74 | | | | |
| Triplo-10 x Crossa | O | 36 | 60 | 96 | 9:55 | 43.6364** | < 0.01 |
| | E | 13.50 | 82.50 | | | | |
| Triplo-11 x Crossa | O | 22 | 203 | 225 | 9:55 | 3.4181 | 0.10 - 0.05 |
| | E | 31.64 | 193.36 | | | | |
| Triplo-12 x Crossa | O | 7 | 74 | 81 | 9:55 | 1.9693 | 0.20 - 0.10 |
| | E | 11.39 | 69.61 | | | | |

** = Significant deviation at 0.01 probability level

43.6364 respectively. On the contrary, the $F_2$s of trisomics-5 and 8 had more than expected proportion of plants wherein red awns were absent. The calculated chi-square values for these trisomics were 13.0444 and 9.9704 respectively.

Although the $F_1$s of triplos-2 and 6 had long red awns (Table 10) their $F_2$ populations showed non-significant values of calculated chi-square. It was interesting to note that, contrary to the expectation for a character under a trigenic control, the $F_2$ populations, trisomic for chromosomes-3, 5, 7, 8 and 10 behaved as the critical populations.

### 4.2.2 Morphological character

#### 4.2.2.1 Awning

The presence or absence of awns in the parents as well as in the disomic and trisomic $F_1$s is displayed in Table 10. As may be seen from the table, triplos-2, 3, 6, 9 and 10 and their corresponding trisomic $F_1$s except the $F_1$ of triplo-9 showed the presence of awns. The disomic parent, the donor parent and their disomic $F_1$ remained devoid of awns due to the action of an inhibitory gene.

The $F_2$ segregation for presence or absence of awns in trisomics and marker crosses is presented in Table 47. As revealed in the table, awning was under a digenic control as detailed earlier (refer 4.1.1). A perusal of the table, revealed further, that the $F_2$ populations of trisomics-2, 3,

Table 47. $F_2$ segregation for presence or absence of awning in trisomics and marker crosses

| Cross combination | | $F_2$ phenotypic segregation | | Total | Confirmed genetic ratio | $x^2$ | 'P' |
|---|---|---|---|---|---|---|---|
| | | Present(+) | Absent(-) | | | | |
| Disomic x Crossa (Sona control) | O | 167 | 733 | 900 | 3:13 | 0.0223 | 0.90 - 0.80 |
| | E | 168.75 | 731.25 | | | | |
| Triplo-1 x Crossa | O | 42 | 178 | 220 | 3:13 | 0.0168 | 0.90 - 0.80 |
| | E | 41.25 | 178.75 | | | | |
| Triplo-2 x Crossa | O | 31 | 66 | 97 | 3:13 | 11.1089** | < 0.01 |
| | E | 18.19 | 78.81 | | | | |
| Triplo-3 x Crossa | O | 131 | 244 | 375 | 3:13 | 64.4678** | < 0.01 |
| | E | 70.31 | 304.69 | | | | |
| Triplo-4 x Crossa ($F_2$) | O | 2 | 4 | 06 | 3:13 | 0.8376 | 0.50 - 0.30 |
| | E | 1.12 | 4.88 | | | | |
| Triplo-4 x Crossa ($BC_1F_2$) | O | 15 | 57 | 72 | 0.94:15.06 | 29.1331** | < 0.01 |
| | E | 4.23 | 67.77 | | | | |
| Triplo-5 x Crossa | O | 8 | 167 | 175 | 3:13 | 23.0929** | < 0.01 |
| | E | 32.81 | 142.19 | | | | |
| Triplo-6 x Crossa | O | 74 | 166 | 240 | 3:13 | 23.0017** | < 0.01 |
| | E | 45.00 | 195.00 | | | | |
| Triplo-7 x Crossa | O | 98 | 197 | 295 | 3:13 | 40.5466** | < 0.01 |
| | E | 55.31 | 239.69 | | | | |
| Triplo-8 x Crossa | O | 12 | 87 | 99 | 3:13 | 2.8555 | 0.10 - 0.05 |
| | E | 18.56 | 80.44 | | | | |
| Triplo-9 x Crossa | O | 31 | 51 | 82 | 3:13 | 19.5435** | < 0.01 |
| | E | 15.38 | 66.62 | | | | |
| Triplo-10 x Crossa | O | 68 | 28 | 96 | 3:13 | 170.9402** | < 0.01 |
| | E | 18.00 | 78.00 | | | | |
| Triplo-11 x Crossa | O | 44 | 181 | 225 | 3:13 | 0.0958 | 0.90 - 0.70 |
| | E | 42.19 | 182.81 | | | | |
| Triplo-12 x Crossa | O | 9 | 72 | 81 | 3:13 | 3.1026 | 0.10 - 0.05 |
| | E | 15.19 | 65.81 | | | | |

** = Significant deviation at 0.01 probability level

5, 6, 7, 9 and 10 and the $BC_1F_2$ of trisomic-4 showed a significant deviation from the expected digenic, 3:13 and 0.94:15.06 segregation ratios respectively at 0.01 level of probability. The $F_2$ populations of trisomics-2, 3, 6, 7, 9 and 10 and $BC_1F_2$ of triplo-4 showed a skewed segregation in the occurrence of higher proportion of plants with the presence of awns, which was reflected in their respective values of calculated chi-square. The $F_2$ populations of all these trisomics showed calculated chi-square values of 11.1089 for triplo-2, 64.4678 for triplo-3, 23.0017 for triplo-6, 40.5466 for triplo-7, 19.5435 for triplo-9 and 170.9402 for triplo-10, whereas the $BC_1F_2$ of triplo-4 had a value of 29.1331.

Contrary to this, the $F_2$ population of triplo-5 interestingly showed the skewness of segregation towards higher proportion of awnless plants thereby showing a calculated chi-square value of 23.0929. Interesting was the case of triplo-9, as the parental trisomic-9 showed the presence of awns, its corresponding $F_1$ was awnless, but the $F_2$ involving triplo-9 showed a highly significant value of calculated chi-square, following a skewed segregation in the occurrence of awned plants.

Thus, contrary to the expectation for a character showing a digenic control, the trisomics-2, 3, 4, 5, 6, 7, 9 and 10 behaved as critical trisomics.

## 4.3 Transmission of the extra chromosome

In the present investigation, the transmission of the extra chromosome of the primary trisomics was critically studied from:

1. The selfed progenies of parental triplos as well as
2. the $F_2$ and back cross progenies of trisomics and marker crosses.

### 4.3.1 Selfed progenies of parental trisomics

The selfed progenies of all triplos except triplo-4, were raised. They were classified as disomic like, trisomic like (appearing like the designated triplos) and non-disomic like plants based on their phenotypic appearance as described in Material and Methods section (refer 3.2.10 and 3.2.14). The transmission of the extra chromosome in primary trisomics upon selfing is presented in Table 48.

A perusal of the table reveals that the transmission frequency of the extra chromosome varied from 17.58 per cent in triplo-5 to 45.52 per cent in triplo-12. Triplo-1 showed 19.44 per cent transmission frequency through female. In triplo-2 after several pollinations with disomic Sona, only two seeds were obtained and hence the percentage transmission has not been worked out. The non-disomic like plants appeared in the selfed progenies of triplos-3, 5, 6, 7, 8 and 12, with a frequency ranging between 0.97 per cent in

Table 48. The transmission frequency of extra chromosome in primary trisomics upon selfing

| New designations of trisomics | Trisomic | Total No.of plants | Progeny (No. of plants) | | | | |
|---|---|---|---|---|---|---|---|
| | | | Disomics 2n | Disomic like | | Trisomics 2n+1 | Trisomics 2n+1 (%) |
| | | | | No. | % | | |
| Triplo- 1 | Triplo- 1 | 36 | 29 | 0 | 0 | 7 | 19.44 |
| Triplo- 2 | Triplo- 2 | 02 | 2 | 0 | 0 | 0 | - |
| Triplo- 6 | Triplo- 3 | 103 | 71 | 1 | 0.97 | 31 | 30.09 |
| Triplo- 3 | Triplo- 4 | - | - | - | - | - | - |
| Triplo- 5 | Triplo- 5 | 91 | 73 | 2 | 2.20 | 16 | 17.58 |
| Triplo-12 | Triplo- 6 | 99 | 64 | 2 | 2.02 | 33 | 33.33 |
| Triplo- 7 | Triplo- 7 | 95 | 65 | 2 | 2.11 | 28 | 29.47 |
| Triplo- 8 | Triplo- 8 | 101 | 70 | 4 | 3.96 | 27 | 26.73 |
| Triplo- 9 | Triplo- 9 | 22 | 18 | 0 | 0 | 04 | 18.18 |
| Triplo-10 | Triplo-10 | 95 | 70 | 0 | 0 | 25 | 26.32 |
| Triplo-11 | Triplo-11 | 104 | 81 | 0 | 0 | 23 | 22.12 |
| Triplo- 4 | Triplo-12 | 134 | 71 | 2 | 1.49 | 61 | 45.52 |

- Not studied

triplo-3 to 3.96 per cent in triplo-8. The trisomics 5, 6, and 7 showed on an average, 2 per cent of such unusual plants, whereas in case of trisomic-12, the frequency was only 1.49 per cent.

### 4.3.2 $F_2$ or backcross progenies

The transmission frequency of the extra chromosome was also studied in a similar way from the $F_2$ and/or backcross progenies of trisomics and marker crosses. The results are presented in Table 49. A perusal of the table reveals that transmission frequency of the extra chromosome varied from 5.56 per cent in triplo-4 to 38.27 per cent in triplo-12. Triplo-1 showed 12.27 per cent transmission. In case of triplo-4, the $F_1$ being highly sterile, vegetative multiplication of the same was resorted to. Only six $F_2$ plants could be obtained in this case and hence percentage transmission has not been worked out. The non-disomic like plants also appeared in the progenies of triplos-6 and 7 with a frequency of 1.25 and 2.03 per cent respectively. However, the occurrence of such unusual plants was only in two trisomics as against six trisomics in the previous case.

## 4.4 Characteristic recovery of trisomic like plants from the non-critical disomic $F_2$ population

It is obvious that a trisomic plant having an extra chromosome, when selfed or pollinated by a diploid, produces progenies with or without the extra chromosome.

Table 49. The transmission rate of extra chromosome in primary trisomics calculated from the $F_2$ or backcross progenies of trisomics and marker crosses

| New designations of triplos | Trisomic x marker | Total No.of plants* | Progeny (No. of plants) | | | | |
|---|---|---|---|---|---|---|---|
| | | | Disomic 2n | Non-disomic like | | Trisomics 2n+1 | Trisomics (2n+1) |
| | | | | No. | % | | |
| Triplo- 1 | Triplo- 1 | 220 | 193 | - | - | 27 | 12.27 |
| Triplo- 2 | Triplo- 2 | 97 | 82 | - | - | 15 | 15.46 |
| Triplo- 6 | Triplo- 3 | 375 | 283 | - | - | 92 | 24.53 |
| Triplo- 3 | Triplo- 4 ($F_2$) | 06 | 06 | - | - | 0 | - |
| Triplo- 3 | Triplo- 4 ($BC_1F_2$) | 72 | 68 | - | - | 4 | 5.56 |
| Triplo- 5 | Triplo- 5 | 175 | 146 | - | - | 29 | 16.57 |
| Triplo-12 | Triplo- 6 | 240 | 158 | 3 | 1.25 | 79 | 32.92 |
| Triplo- 7 | Triplo- 7 | 295 | 215 | 6 | 2.03 | 74 | 25.08 |
| Triplo- 8 | Triplo- 8 | 99 | 65 | - | - | 34 | 34.34 |
| Triplo- 9 | Triplo- 9 | 82 | 71 | - | - | 11 | 13.41 |
| Triplo-10 | Triplo-10 | 96 | 69 | - | - | 27 | 28.13 |
| Triplo-11 | Triplo-11 | 225 | 186 | - | - | 39 | 17.33 |
| Triplo- 4 | Triplo-12 | 81 | 50 | - | - | 31 | 38.27 |

In the present investigation (as has been mentioned in sections 3.2.7 and 3.2.14) the non-critical disomic $F_2$ populations obtained from $F_1$ plants of trisomics-1 to 12 x Crossa were also raised to study their behaviour. It is expected that the non-critical disomic $F_2$ plants obtained from the crosses of Sona trisomics-1 to 12 x Crossa would be quite similar to that of the normal disomic $F_2$ plants of Sona disomic (control) x Crossa. But it did not hold true in a few cases during the course of present investigations.

In the non-critical disomic $F_2$ populations involving trisomics-1, 4, 8, 10 and 12 many trisomic like plants with a phenotype of their trisomic parent/s appeared, as identified on the basis of phenotypic expressions of marker characters. The $F_1$'s of these populations showed 12 II only, yet these $F_2$ populations were remarkably different, in general, from that of the normal disomic $F_2$ population. The phenotypic segregation revealed trisomic like plants as if there was an impact of the concerned parental trisomic. Such plants of characteristic trisomic nature, were observed in populations comprising 238, 244, 636, 635 and 1746 $F_2$ plants respectively of crosses involving TP-1, TP-4, TP-8, TP-10 and TP-12 with marker parent Crossa. Thus, a total of 3519 plants were critically observed for all the characters studied in the present investigation to recover individuals of trisomic nature. However, it was not possible to study the cytological details of such plants.

## 4.5 Isolation of possible aneuploid derivatives and rare segregants of morphological variations during investigation

### 4.5.1 Possible aneuploid derivatives

It is known that the extra chromosome in trisomic condition generally exhibits the same morphological modifications regardless of the genetic background. Thus, the trisomics in the background of IR-36 and Sona cultivars have been designated according to their distinctive morphological appearance as grassy (triplo-1), dwarf (triplo-2), Awned (triplo-3), sterile (triplo-4), twisted leaf (triplo-5), bushy (triplo-6), narrow leaf (triplo-7), rolled leaf (triplo-8), stout (triplo-9), short grain (triplo-10), pseudonormal (triplo-11) and tall (triplo-12).

A very interesting feature of the present investigation was the isolation of completely purple trisomic like derivatives from the $F_2$ generation plants of trisomics and marker crosses. Thus, a grassy like derivative from the $F_2$ of triplo-1, dwarf like from triplo-2, awned from triplo-3, twisted leaf from triplo-5, bushy from triplo-6, narrow leaf from triplo-7, rolled leaf from triplo-8, stout from triplo-9, short grain from triplo-10, pseudonormal from triplo-11 and tall from triplo-12 were isolated. These derivatives had pigmentation in almost all the plant parts as in the donor parent crossa. However, from the $F_2$ and $BC_1F_2$ populations of triplo-4, such a derivative could not be isolated.

Although, it was not possible to investigate the cytological details of such possible aneuploid derivatives, the suitable progeny has been maintained in the microplots of CRRI, Cuttack, Orissa State.

4.5.2 Rare segregants of morphological variations

The following rare segregants of morphological variations were obtained from the non-critical disomic $F_2$ population of trisomics 1 to 12 and marker crosses.

1. An open spikelet segregant and clustered-compact spikelet segregants from the populations involving triplo-1.

2. A bent node segregant from the population of triplo-2.

3. A triplo-4 like highly sterile but completely purple segregant from population involving trisomic-4.

4. Twisted leaf derivatives from population involving triplo-5.

5. Small flag leaf derivatives from populations involving triplo-6.

6. Needle like narrow leaf and rolled leaf derivatives from populations involving triplos-7 and 8.

7. A red pericarp kernel derivative from a triplo-8 population.

8. A round kernel derivative and a plant with lemmal and paleal awns from triplo-10 population.

9. A derivative having 4 cm long ligule from a population involving triplo-11.

10. A very long flag leaf segregant, a big bold grain segregant and a few derivatives with lazy-spreading growth habit from populations involving triplo-12.

11. Superfine grain derivatives from populations involving triplos-1, 7, 8 and 11.

12. Segregants with very poor exserted panicles to fully exserted panicles from populations involving triplos-1 to 12 in general.

In addition to the above derivatives, an autotriploid (3 N) like plant, a profusely tillering derivative and a plant with a long panicle (35 cm) were isolated from the $F_3$ families.

A wealth of interesting derivatives observed during the course of investigations have been isolated, the seed material has been preserved and the stubble progeny is being maintained at CRRI, Cuttack, Orissa state.

# CHAPTER - V

# DISCUSSION

# V. DISCUSSION

Rice is the principal food, that feeds more than 54 per cent of the mankind to-day. In comparison to its importance, rice genetics lags far behind that of other genetically well-known crops such as wheat, maize, barley and tomato (Khush, 1974; Khush et al., 1984). Although the genetic investigations were started from the beginning of the present century, the linkage maps are far from complete.

Linkage studies of rice are of course, plentiful but so are the disagreements regarding the similarities of the linkage maps of indica and japonica varietal groups. There prevail diverse opinions regarding the similarities of the indica and japonica linkage groups.

Several Indian workers, especially Misro et al.(1966); Misro (1981); Misra et al. (1986).(Misro School, CRRI, Cuttack), Pavithran and co-workers (unpublished), Pavithran (1986), Sukeshkumar (1990) (Pavithran School, University of Calicut, Kerala) and Dhulappanavar et al. (1975a), Dhulappanavar (1977), Goud and Nadaf (1985, 1990)and Nadaf (1989) (Dhulappanavar School, UAS, Dharwad) believed that linkage groups of indica rice differ from those of japonica rice, and thus following linkage analysis, they have contributed to the linkage maps of indica rice as proposed by Misro et al. (1966), and Misro (1981). However, on the other hand, several Japanese workers, especially Nagao and Takahashi (1963), Kinoshita and

Takahashi (1991) (Japanese School) and Singh and Khush (1978); Khush et al.(1984); Khush and Singh (1986) (IRRI School, Philippines), felt that the linkage maps for the two ecotypes cannot be so much different and Khush et al. (1984) have found complete correspondence between the linkage groups of japonica and indica rices. Thus, following linkage analysis, they have contributed to the linkage maps of japonica rice as proposed by Nagao and Takahashi (1963).

These differences could be resolved by establishing the identity of chromosomes, developing translocation stocks and primary trisomics in japonica as well as in indica rices and locating the genes on the chromosomes with the help of these stocks (Sharma, 1986).

Kinoshita and Takahashi (1991) have presented the updated current linkage maps (Anonymous, 1990a) admitting that it is difficult to consolidate the Indian linkage groups published by Misro (1981) in the maps. Because of the differences in the genetic scheme for anthocyanin colouration and scarcity of identical genes involved in the two series of linkage groups at that time, it has been difficult to establish 12 groups common to indica and japonica types. Kinoshita (1984, 1986a, 1986b) has reviewed the discrepancy of the colour producing genes postulated in japonica and indica rices stressing the need for further efforts for the identification of genic system used in colour characters.

The inheritance pattern of the anthocyanin pigmentation characters has been extensively dealt with by Indian workers in *indica* rice and a wealth of data has been accumulated. Out of a total of 133 genes located to different linkage groups of *indica* rice, 87 genes are associated with pigmentation alone in different parts of the rice plant. These 87 pigmentation genes are distributed in first ten linkage groups with three genes in I group, 26 in II, 23 in III, 16 in IV, 3 in V, 1 in VI, 3 in VII, 1 in VIII, 3 in IX and 8 in X group. The distribution of the genes, which have been already located, clearly demonstrates that the pigmentation genes are mostly concentrated in II, III, IV and X groups in order of their magnitude (Goud and Nadaf, 1990a).

The three basic genes C, A and P, responsible for the anthocyanin pigmentation, seem to have several multiple alleles (refer Figure 1). These three basic genes or their multiple alleles are expected to be located in not more than three linkage groups; but in fact, genes for anthocyanin pigmentation are found in eight linkage groups namely, I, II, III, IV, V, VII, IX and X. Further, it is evident that the localization genes governing purple pigmentation of the same plant parts are located in more than one linkage group (linkage map). In view of such distribution of genes responsible for same character in different groups, association of gene chromosome relationship appears to be a difficult task.

A detailed cytogenetic analysis using trisomics and translocation stocks would certainly throw light on such a fascinating aspect (Goud and Sadaf, 1990a).

Trisomics are useful in linkage studies. Striking morphological changes that usually accompany the addition of each extra chromosome make it very easy to follow the trisomics through any generation. Trisomics also show aberrant genetic ratios which are expressed whenever the cross is made to the critical trisomic.

The primary trisomic series, upon its establishment should have been effectively studied to probe into the complex inheritance mode of pigmentation characters. Since the initiation of genetical research on rice in India, anthocyanin pigmentation genes have been traditionally assigned to their respective linkage groups based on cross over values. The chromosomal location of these genes through primary trisomic technique, although being an innovative area, has remained almost untouched and unprobed.

Keeping the foregoing lacuna in view, an attempt has been made in the present investigations to locate the genes governing anthocyanin pigmentation characters to specific chromosomes utilizing a complete set of primary trisomics of *indica* rice so as to establish gene-chromosome relationship.

In accordance with the objectives of the present investigations, it would be appropriate to discuss the results

under three major headings:

5.1 Inheritance pattern of anthocyanin pigmentation in different plant parts and a morphological character namely, awning ,

5.2 Chromosomal location of genes governing pigmentation and awning, and

5.3 Studies on the transmission frequency of the extra chromosome.

## 5.1 Studies on inheritance pattern

### 5.1.1 Anthocyanin pigmentation characters

Colouration, due to the presence of anthocyanin and its related pigments, occurs quite commonly in several parts of the rice plant. The anthocyanin colour shows a wide range of variation from pink to purplish black; and its related colour expression, the so called 'tawny', ranges from light to dark brown (Takahashi, 1957). Inheritance of pigmentation mostly of the presence and the absence of the pigment in various parts of the plant has been studied by many and only a few have considered the intensity of the pigment. A wealth of literature has been accumulated on this aspect, which has been periodically reviewed by many authors (Yamaguchi, 1927; Ikeno, 1927; Matsuura, 1933; Sakai, 1935; Richharia, 1945; Jodon, 1948, 1955, 1956 and 1957; Nagao, 1951; Kuang, 1951; Ramiah and

Rao, 1953; Nagai, 1958; Ghose et al., 1960 and Chang, 1964). Many of these authors have highlighted the importance of the basic nature of the genes governing apiculus colour in developing pigment in other plant parts.

The production of pigment in apiculus or in any other plant part requires the presence of two complementary genes C and A; C produces a precursor (chromogen), while A produces an enzyme which converts the precursor into anthocyanin (Parnell et al., 1917; Yamaguchi, 1932 and Nakayama, 1935). Apart from these two basic genes, the localisation gene of apiculus P is also found to be basic for the pigmentation in other plant parts (Nagao, 1951; Nagao and Takahashi, 1956; Takahashi, 1957; Manjunath, 1973; Setty and Misra, 1973 and Hadagal et al., 1980). Contrary to the above, some authors have reported the development of pigment in other plant parts even in the absence of pigment in apiculus (Jones, 1929; Ramiah, 1945; Misra et al., 1960 and Dhulappanavar and Mensinkai, 1965). This acts as an example to show the complexity that is involved in the anthocyanin pigmentation. In addition to this, such unusual ratios as 45:19, 117:139, 162:94, 81:175, 243:781 etc., have been reported for one and the same character in different crosses, thus showing altogether different gene combinations and types of interaction in different varieties. Several localisation genes in other plant parts have also been recognized apart from the basic C-A-P genes and they are found to be necessary for the

development of pigment in those plant parts. While each gene is specific for pigmentation of a particular organ (Table 1), instances are not uncommon that show more than one localization gene responsible for pigmentation in one and the same plant part. Two to seven genes are supposed to be involved in the production of different types of pigmentation in the various parts of rice plant (Chang, 1964). Also, there are instances that a localization gene reported for pigmentation in one part is also found to be responsible for localizing pigment in other parts, thus indicating pleiotropy. For example, the gene Pl governs pigmentation on leaf blade, leaf sheath, pulvinus, auricle, ligule, internode, node and rachis, while the gene Pn does the same for leaf apex, leaf margin, node, pulvinus, auricle and ligule (Takahashi, 1957 and 1964 and Nagao et al., 1962). Such cases of pleiotropy have also been reported by many in the past for various pigmentation characters (Ramiah and Rao, 1953; Takahashi, 1957; Richharia et al., 1960; Dhulappanavar, 1973a,b,c; Manjunath, 1973; Setty and Misro, 1973; Thimmappaiah, 1975; Dhulappanavar, 1980-81; Pavithran, 1985; Pavithran and Annie, 1985; Nadaf, 1989; Nadaf et al., 1990 and Goud and Nadaf, 1990b). Along with basic and localizing genes, a battery of regulatory genes such as inhibitory, anti-inhibitory, intensifying and diluting genes have also been found to play an important role in pigmentation of plant parts (Ramiah and Rao, 1953). The present investigation in this context is yet another attempt

to probe further into the genetics of pigmentation in different plant parts.

The results of the present investigation, in the light of the enormous variation observed in the pattern of inheritance of anthocyanin pigmentation are discussed in the following paragraphs.

5.1.1.1 Inheritance of culm base pigmentation

A perusal of Table 11 reveals that the pigmentation in culm base segregated into 45 purple:19 green indicating the existence of three interacting localisation genes for pigment formation, namely, one basic complementary gene designated as $\underline{Pcb}_a$ and two complementary duplicate genes designated as $\underline{Pcb}_{b1}$ and $\underline{Pcb}_{b2}$. The literature pertaining to inheritance of culm base pigmentation is very scanty as only two reports, that of Majumdar (1985) and Nadaf (1989), are available. Both the authors have reported a monogenic inheritance of culm base pigmentation conditioned by a localization gene Pcb. Thus, the nature of inheritance pattern observed in the present investigation could be considered as a new report.

5.1.1.2 Inheritance of leaf sheath (external) pigmentation

As may be seen from Table 11, the pigmentation in leaf sheath (external) also segregated into 45 purple: 19 green indicating the involvement of three interacting genes for pigment formation, namely, one basic complementary gene -

$Psh_a$, and two complementary duplicate genes $Psh_{b1}$ and $Psh_{b2}$. This finding is in consonance with the several similar observations made earlier (Unpublished record of CRRI, Cuttack quoted by Ghose et al., 1960; Setty and Misro, 1973; Setty et al., 1973; Hadagal, 1980; Hadagal et al., 1984 and Shyla, 1985).

A survey of the available literature revealed further, that the operation of complementary duplicate genes conditioning leaf sheath pigmentation is not uncommon. As evidenced by a segregation ratio of 54:10, many authors have earlier reported the involvement of any two of the three complementary duplicate genes (Richharia et al., 1960; Dhulappanavar and Mensinkai, 1970; Manjunath, 1973; Dhulappanavar and Hiremath, 1974a and 1974b; Prasad et al., 1987 and Shyla and Pavithran, 1989). Further, Ghose et al. (1960) from unpublished records of CRRI; Panda (1962), Saran and Srivastava (1969); Setty and Misro (1973); Setty et al. (1973) and Singh et al. (1990) have reported a tetragenic ratio of 162:94 indicating the involvement of one basic gene and any two of the three complementary duplicate genes.

Thus, the nature of inheritance pattern observed in the present study could be considered as confirmatory to earlier findings.

5.1.1.3 Inheritance of leaf sheath (internal) pigmentation

The pigmentation in leaf sheath (internal) segregated into 27 purple:229 green (Table 11) indicating the operation of four interacting genes namely, three complementary genes designated as Psh (int)$_a$ Psh (int)$_b$ and Psh (int)$_c$ and one inhibitory gene designated as I-Psh (int) for the formation of pigment. The literature pertaining to inheritance of leaf sheath (internal) is very scanty. Setty and Misro (1973), Kolhe and Bhat (1979, 1982) and Nadaf (1989) have reported a monogenic inheritance conditioned by a localization gene Psh (int) for its pigmentation. A trigenic interaction involving any two of the three complementary duplicate genes, was also realized with a segregation ratio of 54 purple : 10 green (Setty and Misro, 1973). Thus, a tetragenic interaction as realized in the present investigation could be considered as a new report.

5.1.1.4 Inheritance of prophyllum pigmentation

As may be seen from Table 11, the prophyllum pigmentation segregated into 45 purple: 19 green ratio suggesting the existence of three interacting genes namely, one basic complementary gene Ppr$_a$ and two complementary duplicate genes Ppr$_{b1}$ and Ppr$_{b2}$. So far, only one report, that of Nadaf (1989), is available for inheritance of prophyllum pigmentation, wherein it was found to be monogenically inherited as conditioned by a localization gene Ppr. Thus, a trihybrid

ratio obtained in the present investigation could be considered as a new report.

5.1.1.5 Inheritance of pigmentation in leaf characters

Anthocyanin pigmentation characters are generally clearly visible at the time of anthesis/flowering stage (Nagai, 1958). However, foliage colour could be distinguished in the seedling stage also (Chang and Bardenas, 1965). This fact was later confirmed by Hev and Kim (1975) as the authors observed the differential expression of basal leaf colour in early hybrid generations of rice seedlings. Nadaf (1989) was the first to study the leaf characters elaborately at seedling and flowering stages and the author has reported different segregation ratios at different stages. In view of this possible differential expression of pigmentation in leaf parts, pigmentation in leaf blade, leaf tip and leaf margin, was studied in the present investigation at both seedling and flowering stages to comprehend their inheritance pattern.

5.1.1.5.1 Leaf blade pigmentation

A perusal of Table 11 reveals that at both the seedling and flowering stages, pigmentation in entire leaf blade segregated into 9 purple: 55 green plants indicating the involvement of two complementary genes ($\underline{Pl_a}$, $\underline{Pl_b}$) and one inhibitory gene ($\underline{I\text{-}Pl}$). Nadaf (1989) reported a digenic interaction as realized by a 3:13 genetic ratio for pigmentation of leaf blade at seedling stage whereas a trigenic

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ratio of 39 purple:25 green and/or 12 purple:27 purple wash:25 green was observed at flowering stage. Thus, the involvement of a basic gene and an inhibitory gene for leaf blade pigmentation at seedling stage and the involvement of a basic gene, an inhibitory gene and an anti-inhibitory gene at flowering stage was hypothesized. Further, the author postulated a phenomenon of genetic silence with respect to the production of anthocyanin at seedling stage due to a latent anti-inhibitory gene with incomplete penetrance. In the present investigation, however, such a phenomenon of genetic silence was not observed.

With regard to the observations made on the leaf blade pigmentation by earlier authors, Iyer (1959), Ghose et al. (1963); Butany and Ghose (1965) and Setty and Misro (1973) have also reported a segregation ratio of 9 purple: 55 green. The operation of complementary genes along with an inhibitory gene to condition leaf blade pigmentation was also reported earlier with the segregation ratios of 117:139 (Hsu and Lu, 1943) and 27:229 (Ghose et al., 1963; Iyer, 1959; and Butany and Ghosh, 1965). Thus, the nature of inheritance pattern observed in the present study could be considered as confirmatory to these earlier findings.

5.1.1.5.2 Leaf tip pigmentation

As may be seen from Table 11, the leaf tip pigmentation at the seedling and flowering stages, segregated into 45 purple:19 green plants indicating the operation of three interacting genes namely, one basic complementary gene $\underline{Pla}_a$ and two complementary duplicate genes $\underline{Pla}_{b1}$ and $\underline{Pla}_{b2}$. Nadaf (1989) also reported such a ratio for leaf tip pigmentation at seedling stage but pigmentation in leaf tip at flowering stage was shown to be inherited monogenically.

Earlier authors have also reported a segregation ratio of 45 purple:19 green (Anonymous, 1956-57; Iyear, 1959; Rahman, 1964; Ghose et al., 1960 - from unpublished records of CRRI, Cuttack; Hadagal et al., 1980; Hadagal, 1980 and Rao and Misro, 1986).

A survey of the available literature further revealed that the operation of complementary duplicate genes for this character was also realized with the segregation ratios of 54:10 (Anonymous, 1956-57; and Ghose et al.(1960 - from unpublished records of CRRI, Cuttack) and 162:94 (Anonymous, 1956-57; Panda, 1962; Ghose et al., 1960 - from unpublished records of CRRI, Cuttack and Singh et al.,1990).

Thus, the nature of inheritance pattern observed in the present investigation could be considered as a confirmatory evidence of the earlier findings.

Although the leaf tip pigmentation at seedling and flowering stages have exhibited a similar gene action, the frequency of $F_2$s with pigmentation in this part at different stages was dissimilar. A perusal of Table 11 revealed that numerically 15 more $F_2$ plants had leaf tip pigmentation at seedling stage. In respect of the action of these genes in sequential stages of development, a clear picture would certainly emerge as a confirmatory evidence to the finding of Nadaf (1989) by studying larger sized populations.

5.1.1.5.3 Leaf margin pigmentation

A perusal of Table 11 displays that at both seedling and flowering stages, pigmentation in leaf margin segregated into 3 purple:13 green plants suggesting the involvement of a basic gene Plm and one inhibitory gene I-Plm. Nadaf(1989) reported a pentagenic ratio of 243 purple:781 green for the pigmentation of leaf margin at seedling stage indicating the involvement of five complementary genes. However, the author reported that pigmentation in leaf margin at flowering stage was inherited monogenically.

The literature surveyed in the present study clearly showed that a dihybrid ratio of 3:13 involving an inhibitory gene in case of leaf margin pigmentation has not been reported, so far, and thus the nature of gene action observed in the present study could be considered as a new report.

In the present investigation, the pigmentation in leaf tip and leaf margin has been studied separately. However, there are many reports wherein leaf tip and margin pigmentation have been studied together. Of these, Dubey (1955) and Prasad et al. (1987) have reported a segregation ratio of 117:139 indicating the involvement of four interacting genes namely, two complementary genes, one inhibitory gene and one anti-inhibitory gene for realizing leaf tip and margin pigmentation.

5.1.1.6 Leaf midrib pigmentation

As may be seen from Table 11, the midrib pigmentation segregated into a 9 purple:55 green plants suggesting the operation of two complementary genes ($\underline{Pmr}_a$, $\underline{Pmr}_b$) and an inhibitory gene ($\underline{I}$-Pmr). The literature pertaining to inheritance of midrib pigmentation is meagre. Existence of two complementary genes for pigmentation in this part was reported earlier by Dhulappanavar (1977b) although Kondo (1963a) presumed the availability of three genes earlier to that. Nadaf (1989) reported a genetic ratio of 27:37 indicating the involvement of three complementary genes for midrib pigmentation. The prediction of involvement of two genes of complementary nature along with an inhibitory gene was realized in the present investigation and hence this seems to be the first report. However, Dhulappanavar (1981) reported an usual ratio of 1:3:252 (Purple:chalky white: white/green) indicating the involvement of one complementary

gene and three inhibitory duplicate genes.

5.1.1.7 Inheritance of leaf axil pigmentation

This character was governed by three interacting genes namely, one basic complementary gene, $\underline{Pk}_a$ and two complementary duplicate genes $\underline{Pk}_{b1}$ and $\underline{Pk}_{b2}$, as indicated by a segregation ratio of 45 purple: 19 green (Table 11). This finding is in confirmation with the similar observation reported earlier (unpublished record of CRRI, Cuttack quoted by Ghose et al., 1960).

The involvement of complementary duplicate genes for conditioning pigmentation in leaf axil was also reported with the segregation ratios of 54:10 (unpublished records of CRRI, Cuttack quoted by Ghose et al., 1960; Dhulappanavar, 1973a; Manjunath, 1973; Setty and Misro, 1973 and Dhulappanavar and Hiremath 1974a, and 1974b); 162:94 (Unpublished records of CRRI, Cuttack quoted by Ghose et al., 1960; Dhulappanavar, 1973b; 1975 and 1976b) and 567:457 (Dhulappanavar et al., 1975b). Thus, these reports further substantiate the present finding.

5.1.1.8 Inheritance of ligule pigmentation

A perusal of Table 11 reveals that pigmentation in ligule was trigenic, conditioned by two complementary genes ($\underline{Plg}_a$, $\underline{Plg}_b$) and an inhibitory gene ($\underline{I\text{-}Plg}$). The survey of literature has revealed that such a trigenic interaction ratio of 9 purple:55 green has not been reported, so far. Thus,

this seems to be a new report. Further, it revealed that the involvement of inhibitory genes for conditioning ligule pigmentation is not uncommon. Dubey (1955), Anonymous (1956-1957) and Dhulappanavar (1979) have reported a segregation ratio of 117:139, wherein one of the four genes was an inhibitory gene. Similarly, the segregation ratios of 1:3 (Singh et al., 1990), 9:247 (Dhulappanavar, 1981) and 27:229 (Dhulappanavar et al., 1975b) revealed the operation of inhibitory genes.

5.1.1.9 Inheritance of auricle pigmentation

The pigmentation in auricle was also observed to be governed by three interacting genes namely, two complementary genes ($Pau_a$, $Pau_b$) and one inhibitory gene (I-Pau) as evidenced by a trihybrid ratio of 9 purple:55 green (Table 11). Prasad et al. (1987) also reported a similar ratio of 9:55 for auricle pigmentation. Thus, the nature of inheritance pattern observed in the present study could be considered as confirmatory to this earlier finding. Inhibitory genes were also reported to be involved for auricle pigmentation in the ratios of 3:13 (Shastry and Patnaik, 1962), 117:139 (Dubey, 1955; Dhulappanavar, 1979 and Singh et al., 1990) and 387:637 (Dhulappanavar et al., 1975a and 1975b) which further substantiate the present finding.

5.1.1.10 Inheritance of juncture pigmentation

The pigmentation in juncture segregated in the ratio of 3 purple:253 green (Table 11) which indicated a tetragenic

interaction involving one basic gene Pj and three duplicate inhibitory genes I-$Pj_1$, I-$Pj_2$ and I-$Pj_3$. Although tetragenic nature of inheritance with operation of inhibitors has been reported earlier by several authors (Bhattacharya, 1957; Iyer, 1959; Ghose et al., 1960 - from unpublished records of CRRI, Cuttack; Sadananda, 1981 and Singh et al., 1990), the segregation ratio of 3 purple:253 green has not been reported, so far, and hence the present finding could be considered as a new report.

The involvement of inhibitory genes has also been reported earlier by other workers with the segregation ratios of 1:3 (D'Cruz and Dhulappanavar, 1963; Dhulappanavar, 1973d and 1975), 3:13 (Kuang, 1951) and 3:61 (Prasad et al., 1987).

5.1.1.11 Inheritance of juncture back pigmentation

A perusal of Table 11 reveals that pigmentation in juncture back was conditioned by three interacting genes, namely, two complementary genes ($Pjb_a$, $Pjb_b$) and one inhibitory gene (I-Pjb) as evidenced by a 9 purple:55 green segregation ratio. This ratio (9:55) has not been reported so far, although complementary genes were reported to be involved in the ratios of 9:7 (Ghose et al., 1960; from unpublished records of CRRI, Cuttack; Sastry and Srivastava, 1969; Hadagal, 19[illegible]984 and Shyla, 1985), 27:37 (Ghose et [illegible]ished records of CRRI Cuttack and Rahman, 1964), 81:175(Srivastava

and Saran, 1971) and 243:781 (Sukeshkumar, 1990). Thus, the ratio obtained in this investigation is the first report wherein a trigenic interaction is revealed with the involvement of an inhibitory gene. However, a tetragenic interaction with the involvement of inhibitory genes was reported earlier with the segregation ratios of 9:247 (Dhulappanavar, 1973d and 1975) and 129:127 (Dhulappanavar, 1980-81). These reports of involvement of inhibitory genes to condition pigmentation in juncture back corroborate the present finding.

5.1.1.12 Inheritance of internode pigmentation

A segregation ratio of 9 purple:247 green obtained for internode pigmentation (Table 11) indicated a tetragenic interaction involving two complementary genes ($\underline{Pin_a}$, $\underline{Pin_b}$) and two inhibitory duplicate genes ($\underline{I\text{-}Pin_1}$, $\underline{I\text{-}Pin_2}$). This ratio of 9:247 for internode pigmentation has not been reported earlier by any worker and hence, the present ratio is a new report. In this investigation, the Genes $\underline{Pin_a}$ and $\underline{Pin_b}$ are of complementary nature to condition pigmentation in internode, with two independent inhibitory genes, giving a ratio of 9:247. It is evident, that in the absence of the inhibitory genes it would be a case of digenic inheritance with complementary nature of genes. A digenic inheritance with a 9:7 genetical ratio for internode pigmentation was earlier reported by many workers (Hector, 1922; Chao, 1928; Mitra et al., 1928; Jones, 1930; Ganguli, 1942; Iyer, 1959; Uzir, 1961; Nagai et al., 1962; Majumdar et al., 1969;

Dhulappanavar, 1973a, 1975; Dhulappanavar et al., 1974; Setty and Misro, 1973; Dhulappanavar, 1977 and 1979; Ahmed and Das, 1990 and Singh et al., 1990). However, the operation of inhibitors were also noticed in the studies of Ghose et al. (1960 - quoted from unpublished records of CRRI, Cuttack; Sadananda (1981); Dhulappanavar (1981), Nadaf (1989) and Singh et al. (1990). So far as the involvement of inhibitory gene is concerned, the present finding is in agreement with the previous reports.

5.1.1.13 Inheritance of nodal pigmentation

As may be seen from Table 11, the nodal pigmentation also segregated into 9 purple:247 green plants indicating the operation of four genes, namely, two complementary genes designated as $\underline{Pn_a}$, $\underline{Pn_b}$ and two inhibitory duplicate genes designated as $\underline{I\text{-}Pn_1}$, $\underline{I\text{-}Pn_2}$. This finding is in consonance with earlier reports of Dhulappanavar et al. (1975b and 1975c). The involvement of inhibitory genes for nodal pigmentation has been reported by several earlier workers and the genetic ratios of 3:13 (Sadananda, 1981), 117:139 (Dhulappanavar, 1973a, 1973d and 1975), 9:55 (Dhulappanavar, 1979 and Rao and Misro, 1986) and 111:145 (Nadaf, 1989) have been obtained. In this investigation the genes $\underline{Pn_a}$ and $\underline{Pn_b}$ are of complementary nature to condition nodal pigmentation along with two inhibitory duplicate genes. In the absence of inhibitors it would be a case of digenic inheritance with complementary nature of genes. A 9:7 genetic ratio for

nodal pigmentation was reported in the past by several authors (Jones, 1930; Silva and Vianna, 1950; Butany and Bhattacharya, 1962; Misro, 1963; Manjunath, 1973; Singh et al., 1990 and Sukeshkumar, 1990), indicating the involvement of two complementary genes.

5.1.1.14 Inheritance of sheath pulvinus pigmentation

A perusal of Table 11 indicates that sheath pulvinus pigmentation was governed by four interacting genes as evidenced by 3 purple:253 green segregation ratio. The interacting genes involved a basic gene Pu along with three duplicate inhibitory neighbours I-$Pu_1$, I-$Pu_2$ and I-$Pu_3$. This inhibitory complex for pigmentation in sheath pulvinus was detected to be present in the purple parent (donor) of this investigation (Table 19). Inhibitory genes of varied action for sheath pulvinus pigmentation were shown to exist for the first time by Nadaf (1989). The author obtained an unusual pentagenic ratio of 21 purple:1003 green indicating the involvement of a basic gene conditioning the pigmentation along with two inhibitory duplicate genes and two inhibitory complementary genes. The tetragenic ratio of 3:253, obtained in the present investigation has not been reported earlier and hence it could be considered as a new report.

5.1.1.15 Inheritance of pulvinus ring pigmentation

As may be seen from Table 11, the pulvinus ring pigmentation segregated into a 45 purple:19 green ratio,

suggesting the existence of three interacting genes, namely, a basic complementary gene ($\underline{Pur_a}$) and two complementary duplicate genes ($\underline{Pur_{b1}}$ and $\underline{Pur_{b2}}$). The pulvinus ring pigmentation was first observed and investigated by Nadaf (1989). The author reported this character to be tetragenic, segregating into a ratio of 189 purple:67 green, suggesting the operation of one basic complementary gene along with three complementary duplicate genes. The nature of inheritance pattern observed for this character in the present investigation could be considered as a first report as nobody has, so far, studied and reported such a segregation ratio of 45:19. With regard to the involvement of complementary duplicate genes along with a basic gene, the present finding is in close agreement with that of Nadaf (1989).

5.1.1.16 Inheritance of pigmentation in glumes (empty)

A segregation ratio of 9 purple:55 green realized for pigmentation in glumes (Table 11) indicated a trigenic interaction involving two complementary genes $\underline{Pg_a}$ and $\underline{Pg_b}$ and an inhibitory gene $\underline{I\text{-}Pg}$. A similar ratio of 9:55 for this character was earlier observed by Rahman and Srivastava (1968) and Dhulappanavar (1979). Thus, the nature of inheritance observed in the present investigation could be considered as a confirmatory evidence of the earlier findings. A survey of the literature showed that inhibitory genes were reported earlier for the pigmentation in glumes in the ratios

of 117:139 (Ghose et al., 1960 from unpublished records of CRRI, Cuttack, Dhulappanavar, 1976a, Dhulappanavar et al., 1975a and 1975b), 3:13 (Panda et al., 1967), 9:54:193 (Dhulappanavar, 1981) and 255:769 (Nadaf, 1989). A pentagenic ratio of 255 purple:769 green involving the operation of four duplicate genes and one inhibitory gene was reported for the first time for this character by Nadaf (1989). Many of the earlier workers have reported a digenic complementary ratio of 9:7 for glume pigmentation (Hector, 1922; Mitra et al., 1928; Jones, 1930, 1933; Nakayama, 1932; Iyer, 1959; Ghose et al., 1960 - from unpublished records of CRRI, Cuttack; Dhulappanavar, 1962, 1973b; Dhulappanavar et al., 1973a, 1974, Manjunath, 1973; Setty and Misro, 1973 and Ahmed and Das, 1990), whereas a trigenic complementary ratio of 27:37 was also reported earlier (Shafi and Aziz, 1959; Butany et al., 1959; Ghose et al., 1960 - from unpublished records of CRRI, Cuttack; Dhulappanavar, 1973a and 1975 and Sadanand, 1981).

5.1.1.17 Inheritance of lemma/palea pigmentation

A perusal of Table 11 reveals that pigmentation in lemma/palea was controlled by four interacting genes, as evidenced by 9 purple:247 green segregation ratio. The interacting genes involved two complementary genes, designated as $\underline{Pr_a}$ and $\underline{Pr_b}$ and two inhibitory duplicate genes designated as $\underline{I\text{-}Pr_1}$ and $\underline{I\text{-}Pr_2}$. Such a segregation ratio of tetragenic nature has not been reported earlier by any worker, and hence,

the present ratio is a new report. The operation of inhibitory genes for lemma-palea pigmentation has been reported earlier in the segregation ratios of 1:3 (Majumdar et al., 1969), 3:13 (Pavithran, 1977; Hadagal, 1980; Hadagal et al., 1984 and Tripathi and Rao, 1985); 15:49 (Dhulappanavar, 1981); 9:55 (Dhulappanavar, 1979); and 3:4093 (Nadaf, 1989). Of these genetic ratios, the hexagenic ratio of 3 pigmented:4093 non-pigmented realised for this character seems to be the first report (Nadaf, 1989), which suggested the operation of six interacting genes namely, one basic gene and five inhibitory duplicate genes.

In the present investigation the genes $\underline{Pr_a}$ and $\underline{Pr_b}$ are of complementary nature for pigmentation in lemma/palea along with two independent inhibitory genes. It is quite evident that, in the absence of the inhibitory genes, it would be a case of digenic inheritance with complementary gene action. A complementary nature of genes as evidenced by 9 purple:7 green segregation ratio has been reported earlier by many authors (Parnell et al., 1917; Hector, 1922; Chao, 1928; Cada, 1949 - unpublished records quoted by Ramiah and Rao, 1953; Aziz and Shafi, 1958; Ghose et al., 1960 - from unpublished records of CRRI; Uzir, 1961; Dhulappanavar, 1962; Setty, 1968; Manjunath, 1973; Setty and Misro, 1973 and Dhulappanavar et al., 1973a and 1974).

5.1.1.18 Inheritance of apiculus pigmentation

As may be seen from Table 11, the apiculus pigmentation segregated into 45 purple:19 green plants indicating the involvement of three interacting genes namely, one basic complementary gene, $\underline{Pa}_a$, and two complementary duplicate genes, $\underline{Pa}_{b1}$ and $\underline{Pa}_{b2}$. The present finding is in consonance with several reports namely Ghose et al. (1960) from unpublished records of CRRI, Butany and Bhattacharya (1962), Shastry and Patnaik (1962), Hadagal (1980), Hadagal et al. (1980), Pavithran and Annie (1980), Kolhe and Bhat (1979 and 1981), Shyla (1985) and Annie (1986). Thus, in apiculus, the basic complementary gene with either of the two complementary duplicate genes produced the colour.

As detailed earlier, the pigmentation in apiculus or in any plant part is determined by three basic complementary genes (C, A and P) in association with the concerned localisation gene and a set of regulatory genes with inhibitory and/or anti-inhibitory effects. The C-A-P complementary genic system suggested first for the purple apiculus in japonica rice was subsequently established in indica rice as well (Kondo, 1963a; Ghose et al., 1963; Setty and Misro, 1973; Hadagal, 1980 and Pavithran, 1986).

A systematic investigation regarding the genetics of anthocyanin pigmentation of indica rice was made by Setty and Misro (1973). The authors obtained segregation ratios of

15:1 and 54:10 for apiculus pigmentation. Accordingly, they proposed that in addition to the two basic genes C and A, there were three more duplicate complementary genes $P_a$, $P_b$ and $P_c$ involved in the pigmentation of apiculus, A modified C-A-P complementary genic system was proposed by them. In accordance with their postulation, the complementary genic scheme for *indica* group of rice was C-A-$P_a$, $P_b$ or C-A-$P_a$, $P_c$ or C-A-$P_b$, $P_c$.

The results of the present investigation support the findings of Setty and Misro (1973). A segregation ratio of 45:19 was observed for pigmentation in apiculus. The genes C and $P_c$ must be in homozygous condition in both the parents while the genes A, $P_a$ and $P_b$ must be segregating. The gene A must be the basic complementary gene causing pigmentation in collaboration with either $P_a$ or $P_b$ since the other gene $P_c$ is in homozygous condition. Accordingly, the genetic constitution of the two parents used in the present study, with reference to anthocyanin pigmentation in the apiculus would be as follows:

Sona - CC, aa, $P_a$ $P_a$, $P_b$ $P_b$, $P_c$ $P_c$

Crossa - CC, AA, $P_a$ $P_a$, $P_b$ $P_b$, $P_c$ $P_c$

As per the revised international nomenclature of genes in rice (Anonymous, 1986), the genotypes of both the parents with reference to pigmentation in apiculus would be as follows:

Sona - $\underline{CC}\ \underline{Pa}_a\ \underline{Pa}_a,\ \underline{Pa}_{b1}\ \underline{Pa}_{b1},\ \underline{Pa}_{b2}\ \underline{Pa}_{b2},\ \underline{Pa}_{c3}\ \underline{Pa}_{c3}$

Crossa - $\underline{CC}\ \underline{Pa}_a\ \underline{Pa}_a\ \underline{Pa}_{b1}\ \underline{Pa}_{b1}\ \underline{Pa}_{b2}\ \underline{Pa}_{b2}\ \underline{Pa}_{c3}\ \underline{Pa}_{c3}$

The phenotypes and genotypes of the parents used in the present study with reference to pigmentation in different plant parts have been presented in Table 19.

The genes involved in apiculus pigmentation appeared to coexist with additional loci of C-A-P genic system in the two parents chosen for genetic studies in this investigation. Establishment of trigenic inheritance of apiculus coupled with critical examination of the results pertaining to the inheritance pattern of each of the other anthocyanin pigmentation characters revealed that both the parents namely, Sona, and Crossa were homozygous for C, A and P loci of C-A-P complementary genic system.

### 5.1.1.19 Inheritance of stigma pigmentation

A perusal of Table 11 reveals that pigmentation in stigma was also governed by three interacting genes as realised by a 45 purple:19 green segregation ratio. The interacting genes involved a basic complementary gene $\underline{Ps}_a$ along with two complementary duplicate genes $\underline{Ps}_{b1}$ and $\underline{Ps}_{b2}$. A 45:19 segregation ratio was also reported earlier by Dubey (1955), unpublished records of CRRI, Cuttack quoted by Ghose et al. (1960), Sastry and Patnaik (1962); Hadagal (1980),

Hadagal et al. (1984); Sadananda (1981); Shyla (1985) and Prasad et al. (1987). Thus, the nature of inheritance pattern of stigma observed in the present investigation could be considered as a confirmatory evidence of the earlier findings. The basic complementary gene with either of the two complementary duplicate genes produced a pigmented stigma.

Dhulappanavar (1981) reported an unusual segregation ratio of 9 purple:9 chalky white:46 white with a similar explanation of trigenic interaction. The segregation in these three classes was explained on the basis of involvement of a basic complementary gene and two complementary duplicate genes. A basic gene with one complementary duplicate gene will produce purple stigma while the same basic gene with the other complementary duplicate gene will produce chalky white stigma. The remaining combinations do not produce either purple or chalky white, thus resulting in white. The nature of interacting genes for development of pigmentation in stigma realized in the present study is further substantiated with the findings of Dhulappanavar (1981).

5.1.1.20 Inheritance of panicle-base, pulvinus and axis pigmentation

Nadaf (1989) for the first time identified and investigated this new group of characters to be suitable for genetic studies. The author reported that these three parts of the panicle namely, panicle base, panicle pulvinus and panicle axis were governed by only one set of five interacting genes as indicated by a segregation ratio of 171 purple/

black:853 green. The interacting genes involved were two complementary genes along with one inhibitory-basic and two inhibitory complementary duplicate genes.

As may be seen from Table 11, the panicle base and panicle pulvinus pigmentation segregated into 9 purple:247 green plants indicating the involvement of two complementary genes $\underline{Ppb_a}$, $\underline{Ppb_b}$ for panicle base and $\underline{Ppu_a}$, $\underline{Ppu_b}$ for panicle pulvinus along with two inhibitory duplicate genes designated as $\underline{I\text{-}Ppb_1}$, $\underline{I\text{-}Ppb_2}$ for panicle base and $\underline{I\text{-}Ppu_1}$, $\underline{I\text{-}Ppu_2}$ for panicle pulvinus. The present investigation is only the second report to observe these novel characters. Thus the nature of inheritance pattern observed in the present study could be considered as a new report.

As regards panicle axis, a segregation ratio of 27 purple:229 green (Table 11) realized for pigmentation indicated the involvement of four interacting genes, namely, three complementary genes ($\underline{Ppa_a}$, $\underline{Ppa_b}$ and $\underline{Ppa_c}$) and one inhibitory gene ($\underline{I\text{-}Ppa}$). A tetrahybrid ratio of 27:229 observed in the present investigation is a new finding.

The present investigation could, however, be considered as in conformity with the singular previous report of Nadaf (1989) as evidenced by the operation of complementary and inhibitory genes for controlling pigmentation in panicle base, panicle pulvinus and panicle axis.

5.1.1.2 1 Inheritance of nodal septum pigmentation

A perusal of Table 11, reveals that pigmentation in nodal septum is controlled by three interacting genes as evidenced by a 21 purple:43 green segregation ratio. The interacting genes involved a basic gene Pm and two complementary inhibitory genes I-$Pm_a$ and I-$Pm_b$. A survey of the available literature showed that such a segregation ratio of trigenic nature involving a basic gene along with two complementary inhibitory genes has not been reported earlier. Thus the present finding could be considered as a new report.

The involvement of inhibitory gene for governing nodal septum pigmentation was earlier reported by Sadananda (1981). The author obtained a trihybrid ratio of 39 purple: 25 green indicating the involvement of a basic gene, an inhibitory gene and an anti-inhibitory gene. Most of the earlier authors have reported the involvement of genes with complementary nature in the segregation ratios of 9:7 (Ganguli, 1942; Dubey, 1955; Anonymous, 1956-57; Dhulappanavar, 1973a and 1975; and Rao and Misro, 1986); 27:37 (Dubey, 1955; Srivastava and Saran, 1971; and Sadananda, 1981); 54:10 (Manjunath, 1973 and Dhulappanavar and Hiremath, 1974b) and 81:175 (Dubey, 1955 and Nadaf, 1989).

A complicated segregation ratio of 1:27:36 (Purple: yellow:white) reported by Dhulappanavar (1981) was explained on the basis of exclusive role of three segregating

complementary duplicate inhibitors in the absence of segregating gene or genes for purple pigment.

5.1.1.22 Inheritance of nodal ring pigmentation

With respect to nodal ring pigmentation, a segregation ratio of 27 purple:229 green was realized (Table 11) indicating the role of four interacting genes namely, three complementary genes $\underline{Pnr_a}$, $\underline{Pnr_b}$ and $\underline{Pnr_c}$ along with an inhibitory gene $\underline{I\text{-}Pnr}$. The literature pertaining to pigmentation in nodal ring is meagre. However, a survey of available literature revealed that the involvement of inhibitory genes to condition pigmentation in nodal ring is not uncommon. Among the only three reports available in respect of its inheritance, Dhulappanavar (1979) observed the ratio of 9 purple:55 green which was explained on the basis of two complementary genes and one inhibitory gene, while Nadaf (1989) reported a segregation ratio of 63 purple:961 green indicating the involvement of five interacting genes, namely, three duplicate genes and two inhibitory duplicate genes.

Hadagal (1980) and Hadagal et al. (1980) have reported a trigenic ratio of 45:19 suggesting the operation of a basic complementary gene along with two complementary duplicate genes.

A tetragenic ratio of 27:229 realized in the present investigation has not been reported earlier and hence, this could be considered as a new report.

5.1.1.23 Inheritance of red awn

A perusal of Table 11 displays that the presence of red awn was controlled by three interacting genes as evidenced by a 9 present:55 absent ratio, explained on the basis of two complementary genes and an inhibitory gene. A survey of the literature revealed that such a trigenic ratio involving complementary genes $\underline{R\text{Awn}}_a$ and $\underline{R\text{Awn}}_b$ along with an inhibitory gene $\underline{I\text{-RAwn}}$ has not been reported earlier and hence the present finding could be called as a new report. Although the literature pertaining to the inheritance of red awn is meagre, it appears that the operation of inhibitory gene action for the presence of red awn is not uncommon. Sadananda (1981) reported trigenic and tetragenic ratios of 39:25, and 117:139, respectively indicating the involvement of one inhibitory gene. A hexagenic ratio of 3 present:4093 absent was realised by Nadaf (1989) which was explained on the basis of one basic gene along with five inhibitory duplicate genes. As far as the involvement of inhibitory gene/s is concerned, the present finding is in agreement with the reports of Sadananda (1981) and Nadaf (1989).

5.1.2 Morphological character

The rice plant shows tremendous variability with regard to the morphological characters also which include presence or absence of certain structures. The only one character that is, awning studied for its inheritance in the

present investigation has been discussed hereunder.

5.1.2.1 Inheritance of awning

As may be seen from Table 11, the presence of awns was under a digenic control as realised by a segregation ratio of 3 present:13 absent involving a basic gene An along with its inhibitor I-An.

A survey of the literature revealed that the present finding is in close corroboration with similar findings reported earlier by Housaye (1942) and Pavithran (1986). Further, inhibitory genes for awning seems to be common as many earlier authors have reported the involvement of such genes (Misro and Misro, 1954; Kadam and Pant, 1969; Thimmappiah, 1975; and Nadaf, 1989) as evidenced by tetragenic and pentagenic segregation ratios like, 15:241, 117:139 and 3:1021.

It is interesting to note that most of the authors have reported the involvement of duplicate genes for governing the presence of awn. A segregation ratio of 15:1 indicating the operation of two duplicate genes has been reported (Ikeno, 1927; Chao, 1928; Alam, 1932; Mitra and Ganguli, 1932; Majid, 1939; Kuang, 1951; Ramiah - unpublished, quoted by Ramiah and Rao, 1953, Prasad Rao and Misro, 1977, Tripathi and Rao, 1985, and Pavithran, 1986); whereas the operation of three duplicate genes as realized by a segregation ratio of 63:1 was also reported (Ikano, 1927; Sethi et al., 1937; Ramiah - unpublished, quoted by Ramiah and Rao, 1953 and Pavithran, 1986).

Dhulappanavar (1981) explained both awning and its colour in the segregation ratio of 90 purple awns:90 white awns: 76 absent on the basis of four interaction genes.

5.1.3 Inheritance of pigmentation characters and awning - an overview

In the present investigation, a critical study of the inheritance pattern of 30 anthocyanin pigmentation characters along with a morphological character was undertaken.

Although presence of pigmentation has been reported to be dominant over its absence, in general, by various workers including in the present study, exceptions have been noticed in respect of eight plant parts, namely, ligule, auricle, juncture back, glumes, panicle axis, midrib, leaf blade and leaf margin during the course of the present investigation. These plant parts remained green in $F_1$ probably due to the involvement of inhibitory gene/s for the expression of pigments (Table 10).

With regard to the mode of inheritance of pigmentation in various plant parts, the present findings corroborate with the previous reports in certain cases and also furnish altogether new information in many cases. A summarized overview of the present findings has been presented in Table 50. As may be seen from the table in as many as 19 characters pertaining to anthocyanin pigmentation the present findings

Table 30. Inheritance of pigmentation characters and awning - A Summary of the present study

| Sl. no. | Genetic ratio | Genetic explanation for the ratio | Characters studied in the present investigations | |
|---|---|---|---|---|
| | | | As a confirmatory evidence | As a new report |
| Digenic | | | | |
| 1. | 3:13 | One base gene and one inhibitory gene | Awning | 1. Leaf margin (seedling stage)<br>2. Leaf margin (flowering stage |
| Trigenic | | | | |
| 2. | 45:19 | One basic gene and two complementary duplicate genes | 1) Leaf sheath (external)<br>2) Leaf tip (seedling stage)<br>3) Leaf tip (flowering stage)<br>4) Leaf axil<br>5) Apiculus<br>6) Stigma | 1. Culm base<br>2. Prophyllum<br>3. Pulvinus ring |
| 3. | 9:55 | Two complementary genes and one inhibitory gene | 1) Leaf blade (seedling stage)<br>2) Leaf blade (flowering stage)<br>3) Auricles<br>4) Glumes | 1. Leaf midrib<br>2. Ligule<br>3. Juncture back<br>4. Red awn |
| 4. | 21:43 | One basic gene and two complementary inhibitory genes | - | 1. Nodal septum |
| Tetragenic | | | | |
| 5. | 9:247 | Two complementary genes and two inhibitory duplicate genes | 1) Node | 1. Internode<br>2. Lemma/palea<br>3. Panicle base<br>4. Panicle pulvinus |
| 6. | 27:229 | Three complementary genes and one inhibitory gene | - | 1. Leaf sheath (internal)<br>2. Panicle axis<br>3. Nodal ring |
| 7. | 3:253 | One basic gene and three duplicate inhibitory genes | - | 1. Juncture<br>2. Sheath pulvinus |

provide new information with previously unresorted segregation ratios.

Thus, the survey of literature reveals $F_2$ segregation ratios that are different from the ratios reported here for the same set of characters. This is as per expectations due to different cross combinations involving parents with different gene constellations studied by different workers. It is obvious that the $F_2$ segregation ratios depend upon the number of genes for which the two parents involved in the cross differ, the rest of the genes responsible for the same character expression remaining homozygous in both the parents.

Inheritance pattern of all the characters under investigation, except for the culm base pigmentation, was confirmed by their respective breeding behaviour in $F_3$ families. The non-significant chi-square values obtained in the tests of $F_3$ breeding behaviour showed agreement to the hypotheses of breeding behaviour on the basis of inheritance pattern in $F_2$ for each of the character (Tables 12 to 18). It was however, not possible to study the culm base pigmentation in $F_3$ families.

## 5.2 Chromosomal location of genes for pigmentation and awning

### 5.2.1 Chromosomal location of genes through primary trisomic technique

Both trisomics and monosomics have been used to provide information on the chromosomal location of genes of

significance in varietal synthesis. Such information is of importance in enabling breeders to determine the likelihood of synthesising genotypes with desired combinations of beneficial alleles (Riley, 1979). In addition to the assignment of genes to particular chromosomes, primary trisomics provide an excellent cytogenetic tool for testing the independence of linkage groups.

The primary trisomics technique is most widely used to determine chromosome-linkage group relationships since it is based on chromosomal segregation without recombinations (Burnham, 1962).

Assignment of genes to chromosomes using trisomics is done in the following two ways. The first procedure involves making all primary trisomics heterozygous for the gene to be located by crossing trisomic series with the contrasting parent. In the trisomic $F_2$ populations, the segregation pattern is recorded. In cases where trisomics are distinguishable morphologically from disomics, segregation pattern of both the genotypes are recorded separately. The trisomic family showing a segregation pattern that significantly deviates from that of normal disomic $F_2$ family is assumed to carry the gene(s) governing the character under investigation. If the segregation pattern is recorded separately on disomic and trisomic plants of a trisomic family, the family in which trisomic ratio deviates significantly from

the normal disomic ratio is assumed to carry the gene concerned. In this type of analysis no definite trisomic segregation ratio can be used as criteria for assigning genes because of variation in the rate of transmission of extra chromosome through both female and male gametes and the occurrence of either chromosome or chromatid segregation. Once a gene is located on a chromosome by aneuploid analysis of this kind, using that very gene as a marker, location of other genes on the same chromosome can be made precisely through conventional linkage analysis.

The second procedure of assigning genes to chromosomes using trisomics can be practiced only in those cases where established linkage maps are already available. Here, each of the primary trisomics as in the previous procedure is made heterozygous for one of the representative marker genes from each linkage group by crossing primary trisomics with all marker genotypes. Association between linkage group and chromosome can be made by studying $F_2$ or backcross progeny. When the linkage groups are independent, one of the trisomic families, which carries the linkage group in question, tends to show significantly deviated ratio for the marker loci and all others show normal disomic ratio. If the linkage groups are not independent, that is, when more than one established linkage group is situated on the same chromosome, some of the trisomic families give deviated ratios

for more than one marker gene. Once the linkage groups are assigned to chromosomes, for any new gene to be assigned, it will be sufficient to ascertain the linkage group to which it belongs, through conventional linkage analysis. The result can be confirmed by the first procedure, described previously.

Using these techniques many genes have been located on chromosomes in a wide range of crop plants which include maize (McClintock, 1929; McClintock and Hill, 1931 and Rhodes, 1933), tomato (Lesley, 1926; 1928; 1937; Rick and Barton, 1954; Rick et al., 1964; Hagemann, 1969 and Khush, 1973), barley (Tsuchiya, 1956; 1958; 1959; 1969, 1971, 1979; Kedak et al., 1972 and Shahta and Tsuchiya, 1980); potato (Hermsen et al., 1973, 1978), soybean (Hedges and Palmer, 1991), Spinach (Janick et al., 1959), Sorghum vulgare (Hanna and Schertz, 1970, durum wheat ( Sridevi and Goud, 1988) and rice (Iwata and Omura, 1975, 1976; Sadananda, 1981; Wu et al., 1984; Khush et al., 1984; Siddiq et al., 1986; Librojo and Khush, 1986; Ishikawa et al., 1986, 1987, 1989; Ranjhan et al., 1986, 1988; Misra and Jena, 1986; Wu et al., 1988; Kumamaru et al., 1989 and Brar et al., 1990). Further, fairly complete linkage maps have been consolidated in tomato (Khush, 1973) and Barley (Tsuchiya, 1979) by combining trisomic analysis with conventional techniques.

Trisomics are used in genetic studies not only for locating genes and linkage groups but also to determine the validity of linkage groups already established by conventional analysis. There are several instances to show how useful the trisomic analysis has been in delineating the physical linkage groups. Two of the linkage groups in maize which were considered to be independent, for instance, were consolidated into one chromosome by trisomic analysis (Rhoades and McClintock, 1935) and four linkage groups in tomato were assigned to two chromosomes (Khush, 1973). Similarly, in japonica rice, three linkage groups were assigned to one chromosome (Iwata and Omura, 1976) thereby reducing the number of already established linkage groups.

In addition to their use in genetic studies, the trisomics have been employed in several other basic studies also. Effect of extra chromosome on anatomy, physiology and morphology of the organism and the role of chromosomal duplication in the evolution of plant species are important among them (Khush, 1973). There are several instances to show that conclusions drawn on the nature of ploidy of different diploid species on the basis of chromosome morphology, have been revised, following trisomic analysis. For instance, Gottschalk (1954) suspected tomato to be a secondary polyploid based on morphological similarities of a few chromosome pairs. But trisomic analysis of the same by Rick and Khush (1966) revealed otherwise. Similarly, the

hypothesis of Nandi (1936) that rice is a secondary polyploid, has been repudiated by Khush (1974) and Khush et al. (1984) from the studies of rice trisomics.

Further, primary trisomics have been of great value in identification of chromosomes involved in translocation. Using trisomics, chromosomes involved in the translocation stocks of maize (Burnham, 1948), barley (Tsuchiya, 1961) and Sorghum (Hanna and Schertz, 1971 and Prabhakar, 1984), have been identified. Utilization of tertiary trisomics for maintaining genetic stocks unfruitful in homozygous state such as male sterility and in fertility restoration has been proposed in tomato by Khush and Rick (1967) and Ramage (1965) proposed a scheme for using balanced tertiary trisomics of barley in hybrid seed production.

From the foregoing discussion, although it is obvious that trisomics have extensively been used for various genetic and cytogenetic investigations in crop plants like maize, barley and tomato, not much has been done in an important crop like rice.

As may be seen from the survey of literature, whatever little has been done in rice using trisomics is restricted to chromosomal location of genetically identified marker genes, the isozyme loci and the genes for resistance to a disease or an insect.

In rice, till to-date, due attention has been paid for assigning the pigmentation genes to their corresponding linkage groups through conventional linkage analysis, but the use of trisomic series in chromosomal location of pigmentation genes has been negligible. Whatever little trisomic analysis that has been done in the past is restricted to a few pigmentation characters with an incomplete set of primary trisomics.

Keeping the above in background, the present investigation is probably a maiden attempt, to locate gene(s) governing the most complicated and fascinating group of anthocyanin pigmentation characters of almost all the plant parts at a time, using a complete set of primary trisomics of *indica* rice to arrive at a meaningful conclusion on the chromosomal position of these genes.

### 5.2.2 Anthocyanin pigmentation characters

Utilizing the primary trisomic series of *indica* rice an attempt has been made during the course of the present investigation to identify the chromosomes that carry the pigmentation genes. A total of 29 pigmentation characters were chosen for study. Results of trisomic analysis revealed that the number of critical chromosomes in many of the cases was more than expected on the basis of disomic segregation ratio. It is quite probable that pigmentation in various plant parts may be governed by one or few major Mendelian

genes in association with a number of minor modifier genes with small effects located on different chromosomes. In trisomic condition because of the additional dose of a chromosome, the minor modifier genes not recognized so far might exert more influence on this character. Keeping this in view, the results obtained are discussed in the following paragraphs.

5.2.2.1 Chromosomal location of leaf sheath (external) pigmentation genes

As may be seen from Table 20, chromosomes 1, 2, 4, 10 and 12 appear to be critical, carrying the genes conditioning leaf sheath (external) pigmentation. The results indicated that the number of critical chromosomes were more than expected on the basis of a trigenic ratio.

Based on the magnitude of chi-square deviation, it is obvious that the three major genes influencing leaf sheath (external) pigmentation are probably located on chromosomes 10, 2 and 4. With respect to the remaining two critical chromosomes 1 and 12 it may be conjectured that they might be carrying some minor genes with a modifying effect.

5.2.2.2 Chromosomal location of leaf sheath (internal) pigmentation genes

A perusal of Table 21 reveals that chromosomes 3, 4, 5, 7, 8 and 11 appear to be critical carrying the genes

conditioning leaf sheath (internal) pigmentation. Contrary to the expectation for a tetragenic segregation ratio observed in the study, six chromosomes showed critical behaviour for this character. The $F_2$ and $BC_1F_2$ populations involving triplo-4 displayed a significantly higher magnitude of chi-square deviation stressing emphatically that chromosome-4 carried a gene conditioning leaf sheath (internal) pigmentation.

Thus, as evidenced from the test of heterogeneity and the magnitude of the chi-square deviation it may be said that the four major genes governing leaf sheath (internal) pigmentation are probably located on chromosomes 4, 3, 11 and 7. As regards the remaining two critical chromosomes 5 and 8, they might be bearing some minor modifiers.

5.2.2.3 Chromosomal location of prophyllum pigmentation genes

As detailed in Table 22, chromosomes 2, 4 and 12 seem to be critical for the genes controlling prophyllum pigmentation. The results indicated clearly that the number of critical chromosomes were as per the expectation for a character under a trigenic control. As evidenced from the magnitude of chi-square deviation the three genes conditioning pigmentation in prophyllum may be assigned to chromosomes 2, 4 and 12.

5.2.2.4 Chromosomal location of pigmentation genes for leaf blade at seedling and flowering stage and for leaf midrib

It is evident from Table 23 that, chromosomes 6, 8 and 12 seem to be critical carrying the genes conditioning pigmentation in leaf blade (at seedling and flowering stage) and in leaf midrib. The results clearly revealed that the number of critical chromosomes involved agreed with the expectation for a character controlled by three interacting genes. As evidenced from the magnitude of chi-square deviation the three genes governing pigmentation in leaf blade (at seedling and flowering stage) and in leaf midrib may be allotted to chromosomes 6, 8 and 12.

5.2.2.5 Chromosomal location of pigmentation genes for leaf tip at seedling and flowering stage

As may be seen from the Tables 24 and 26, chromosomes 2, 4 and 12 seem to be critical carrying the genes conditioning pigmentation in leaf tip at seedling and flowering stage. The results clearly showed that the number of critical chromosomes involved in the operation were as per the expectation for a character under a trigenic control. The strength of calculated chi-square at 0.01 level of probability demonstrated that the three genes governing leaf tip pigmentation at seedling and flowering stage inhabit the chromosomes 2, 4 and 12.

5.2.2.6 Chromosomal location of pigmentation genes for leaf margin at seedling and flowering stages

As described in Tables 25 and 27, chromosomes 6, 8 and 12 appear to be critical possessing the genes controlling leaf margin pigmentation at seedling and flowering stage. The results revealed clearly that for a character governed by two interacting genes, there were three critical chromosomes contrary to the expectation.

As elaborated earlier (refer 4.2.1.6) the $F_2$ population of trisomic 8 showed a different behaviour for leaf margin pigmentation at seedling and flowering stages of growth. Leaf margin pigmentation at seedling stage showed a preponderance of purple plants in $F_2$ segregation, whereas at flowering stage a preponderance of green plants was realised thereby revealing a variation in the magnitude of chi-square deviation. This might have occurred due to fading of pigmentation at flowering.

The foregoing discussion compels to believe that one of the genes governing leaf margin pigmentation at seedling and flowering stages is a resident of chromosome 8. Further, as evidenced by the magnitude of chi-square deviation the other gene may be assigned to chromosome 12. It may also be said that the remaining critical chromosome 6 might be carrying some minor modifier genes with small effect.

5.2.2.7 Chromosomal location of leaf-axil pigmentation genes

A perusal of Table 28 reveals that chromosomes 2, 4 and 12 seem to be critical carrying the genes governing leaf axil pigmentation. As expected, the number of critical chromosomes tallied for a character under a trigenic control. The strength of chi-square values revealed clearly that the three genes conditioning leaf axil pigmentation are the residents of chromosomes 2, 4 and 12.

5.2.2.8 Chromosomal location of ligule pigmentation genes

As may be seen from Table 29, chromosomes, 9, 11 and 12 seem to be critical bearing genes conditioning ligule pigmentation. The results revealed that the number of critical chromosomes involved in the operation were as per the expectation for a character under a trigenic control. Based on the strength of the calculated chi-square the three genes interacting for ligule pigmentation may be assigned to chromosomes 9, 11 and 12.

5.2.2.9 Chromosomal location of auricle pigmentation genes

As described in Table 30, chromosomes 6 and 12 appear to be critical carrying genes for auricle pigmentation. The results indicated that for a character controlled by three interacting genes, only two chromosomes showed critical behaviour, suggesting the position of two of the

three independent genes on the same chromosome.

On the basis of the strength of calculated chi-square, it may be said that of the three interacting genes involved for conditioning auricle pigmentation, two genes may be the residents of chromosome-6 and the third gene of chromosome-12. It is needless to mention that the two genes residing on chromosome six may be lying more than 50 map units apart, assorting independently.

5.2.2.10 Chromosomal location of juncture (collar) pigmentation genes

As may be seen from Table 31, chromosomes 2, 4, 6 and 11 appear to be critical carrying genes for juncture pigmentation. The results revealed that the number of critical chromosomes were as per the expectation for a character under a tetragenic control.

With respect to the $F_2$ population of trisomic-4, the population comprised of a very small number of only six plants. A highly significant value of calculated chi-square in this case is likely due to the experimental error traceable to small populations.

Thus, on the basis of the magnitude of the chi-square deviation, it may be surmised that of the four genes involved in conditioning pigmentation in juncture, two genes are probably located on chromosome-6 lying more than 50 map units

and one each of the remaining two genes are probably located on chromosomes 2 and 11.

5.2.2.11 Chromosomal location of juncture back Pigmentation genes

A perusal of Table 32 indicates that chromosomes 1, 5, 9 and 12 stem to be critical sustaining the genes governing pigmentation in juncture back region. Contrary to the expectation for a character under a trigenic control, four chromosomes showed criticality for this character. The data further revealed that on the basis of the calculated values of chi-square it was difficult to assign a gene to a specific chromosome.

However, it may be approximated that the three major genes responsible for conditioning juncture back pigmentation are probably located on chromosomes 5, 9 and 12. Further, it may be said that the remaining critical chromosome 1 might be bearing some minor genes with small modifying effects.

5.2.2.12 Chromosomal location of internode pigmentation genes

As detailed in Table 33, chromosomes 4, 6, 7 and 11 appear to be critical carrying the genes controlling pigmentation in internode region. The results clearly revealed that the number of critical chromosomes involved were satisfactory to the expectation for a character under a tetragenic control.

On the basis of the magnitude of chi-square deviation it may be said that one each of these four genes may be present on chromosomes 4, 6, 7 and 11.

5.2.2.13 Chromosomal location of nodal pigmentation genes

As may be seen from Table 34, chromosomes 5, 6, 11 and 12 appear to be critical sustaining the genes conditioning pigmentation in nodal region. The results indicated clearly that the number of critical chromosomes correspond to the number of interacting genes involved. As evidenced from the magnitude of chi-square deviation, one each of these four genes may be allotted to chromosomes 5, 6, 11 and 12.

5.2.2.14 Chromosomal location of sheath pulvinus pigmentation genes

A perusal of Table 35 interestingly reveals that all the trisomic $F_2$s showed skewness of segregation in the occurrence of only green plants predominating in the population. Such observations were quite likely to occur as all the parents and the disomic and trisomic $F_1$s remained devoid of any pigmentation (Table 10).

The $F_2$ phenotypic segregation clearly revealed that the population of trisomic $F_2$ in all the 12 trisomics was of a small size and hence not a single purple plant was recovered. This character was under a tetragenic control, but two chromosomes showed criticality. Based on the extent of

chi-square deviation, chromosomes 3 and 4 appear to be critical for the genes conditioning sheath pulvinus pigmentation.

Due to inadequate size of population it is unwise to conclude that two each of the four genes controlling sheath pulvinus pigmentation may be located on chromosomes 3 and 4 with a distance of more than 50 map units. Larger population is required to be studied to arrive at a meaningful conclusion for the chromosomal position of these genes.

5.2.2.15 Chromosomal location of pulvinus ring pigmentation genes

As may be seen from Table 36, chromosomes 2, 4, 10 and 12 appear to be critical carrying genes controlling pulvinus ring pigmentation. The results clearly revealed that contrary to the expectation for a character under a trigenic control, four chromosomes showed critical behaviour for this character. Based on the magnitude of chi-square deviation, it was difficult to assign a gene to a specific chromosome. However, it may be conjectured that the three major genes governing pigmentation in pulvinus ring are probably located on chromosomes 4, 10 and 12. It may be conjectured further, that the remaining critical chromosome 2 might be carrying minor genes with small modifying effects.

5.2.2.16 Chromosomal location of glume-pigmentation genes

As described in Table 37, chromosomes 6, 8 and 12 appear to be critical sustaining the genes controlling pigmentation in glumes. The Table clearly revealed that the number of critical chromosomes corresponded to the number of operating genes responsible for pigmentation in glumes, satisfying the expectation. As evidenced from the strength of the calculated chi-square it may be surmised that one each of these three genes are the resident of chromosomes 6, 8 and 12.

5.2.2.17 Chromosomal location of lemma and palea pigmentation genes

A perusal of Table 38 revealed chromosomes 1, 3, 6, 7 and 11 to be critical carrying the genes governing pigmentation in lemma and palea. The data revealed clearly that contrary to the expectation for a character under a tetragenic control, five chromosomes exhibited a critical behaviour. It is evident that, analogous to the pigmentation in sheath pulvinus (refer 5.2.2.14), in the case of lemma and palea pigmentation also, the disomic and trisomic $F_2$ populations showed a skewed segregation in the occurrence of higher proportion of green plants thereby reflecting into deviated chi-square values. It is reasonable to study such characters in larger populations for a precise conclusion. However, on the basis of the strength of the calculated chi-square at different probability levels, one each of these four genes

may be allotted to chromosomes 3, 6, 7 and 11. Further, it may be said that some minor genes with modifying effects might be residing on chromosome-1 also.

5.2.2.18 Chromosomal location of apiculus pigmentation genes

As may be seen from Table 39, chromosomes 2, 4 and 12 appear to be critical sustaining the genes conditioning pigmentation in apiculus. The data clearly revealed that the number of critical chromosomes corresponded to the number of interacting genes conditioning apiculus pigmentation as per the expectation. As evidenced from the magnitude of the calculated chi-square one each of the three operating genes may be assigned to chromosomes 2, 4 and 12.

5.2.2.19 Chromosomal location of stigma pigmentation genes

As detailed in Table 40, chromosomes 1, 2, 4 and 12 appear to be critical carrying the genes controlling stigma pigmentation. The results indicated clearly that contrary to the expectation for a character under a trigenic control, four chromosomes revealed criticality for stigma pigmentation. Thus, it was difficult to assign a gene to a specific chromosome on the basis of the magnitude of chi-square deviation. However, it is logical to presume that the three major genes governing stigma pigmentation are probably residing on chromosomes 1, 4 and 12 and that few minor genes with small effects are also residing on the remaining critical chromosome-2.

5.2.2.20 Chromosomal location of panicle base pigmentation genes

As described in Table 41, chromosomes 4, 6, 7 and 11 appear to be critical, containing the genes controlling panicle base pigmentation. The data clearly, revealed that the number of critical chromosomes involved in the operation were as per the expectation for a character under a tetragenic control. The calculated chi-square values suggested that one each of these four genes may be located on chromosomes 4, 6, 7 and 11.

5.2.2.21 Chromosomal location of panicle pulvinus pigmentation genes

As may be seen from Table 42, chromosomes 3, 4, 6, 7 and 11 appear to be critical carrying the genes controlling panicle pulvinus pigmentation. The data revealed that contrary to the expectation for a tetragenically controlled character, five chromosomes behaved as critical. Thus, it was difficult to locate a gene to specific chromosomes on the basis of the magnitude of chi-square deviation. However, it may be inferred that the four major genes controlling panicle pulvinus pigmentation are probably residing on chromosomes 3, 6, 7 and 11. It may be inferred, further, that some minor genes with small effects may also be residing on chromosome 4.

5.2.2.22 Chromosomal location of panicle axis pigmentation genes

A perusal of Table 43 reveals chromosomes 2, 10

and 12 to be critical sustaining the genes controlling pigmentation in panicle axis. The data indicated that for a character controlled by four interacting genes, only three chromosomes revealed critical behaviour suggesting the location of two of the four genes on the same chromosome. On the basis of the magnitude of the calculated chi-square, it may be surmised that two of the four genes are probably located on chromosome 12 and they may be lying more than 50 map units apart, and one each of the remaining two genes on chromosomes 2 and 10.

5.2.2.23 Chromosomal location of nodal septum pigmentation genes

As may be seen from Table 44, interestingly chromosomes 1, 2, 3, 4, 6, 10 and 11, at a time, appear to be critical sustaining the genes governing nodal septum pigmentation. This was quite contrary to the expectation for a character controlled by three interacting genes. Thus, it was difficult to locate the genes to particular chromosomes on the basis of the magnitude of chi-square deviation.

The $F_2$ and $BC_1F_2$ populations involving chromosome-4 revealed a significantly higher value of calculated chi-square due to a preponderance of plants having purple nodal septum unlike in other critical populations, thus stressing emphatically that chromosome-4 was a critical chromosome.

Thus, it may be inferred that the three major genes conditioning purple nodal septum are probably located on chromosomes 6, 4 and 1. Further, it may be said that some minor genes with modifying effects may also be residing on the remaining critical chromosomes 2, 3, 10 and 11.

5.2.2.24 Chromosomal location of nodal ring pigmentation genes

As described in Table 45,chromosomes 1, 3, 6, 7 and 11 appear to be critical carrying the genes conditioning pigmentation in nodal ring, and thus, five chromosomes showed a critical behaviour for a character determined by four interacting genes. Although, this was contrary to the expectation on the basis of the strength of calculated chi-square, it seems plausible to surmise that the four major genes are probably residing on chromosomes 3, 6, 7 and 11. Further, it may be said that some minor genes with small effects might be residing on chromosome 1.

5.2.2.25 Chromosomal location awn colour (red awns) genes

A perusal of Table 46 indicates chromosomes 3, 5, 7, 8 and 10 to be critical sustaining the genes conditioning the presence of pigmented awns. The data revealed that contrary to the expectation for a character under a trigenic control, five chromosomes showed criticality. However, based on the magnitude of chi-square deviation the three major genes governing the presence of red awns may be

assigned to chromosomes 10, 5 and 3. Further, it may be conjectured that the remaining critical chromosomes 7 and 8 might be carrying some minor modifiers.

### 5.2.3 Morphological character

The primary trisomic series utilized in the present investigation has been isolated from the progenies of an autotriploid (3N) plant of 'Sona' - an *indica* rice variety. As may be seen from Table 10, the 3N Sona was characterized by the presence of long awns and so were the triplos 2, 3, 6, 9 and 10 also. Utilizing a complete set of primary trisomics, an attempt was therefore made in the present investigation to identify the critical chromosomes that sustain the genes for awning.

#### 5.2.3.1 Chromosomal location of genes for awning

As may be seen from Table 47, chromosomes 2, 3, 4, 5, 6, 7, 9 and 10 appear to be critical sustaining the genes conditioning awning, at a time, interestingly. The criticality of eight chromosomes, at a time, for a character governed by only two interacting genes was quite contrary to the expectation making it very difficult to locate the genes to specific chromosomes.

The presence or absence of awns in the parents as well as in the disomic and trisomic $F_1$s has been displayed in Table 10. As may be seen from the table, triplos 2, 3, 6,

9 and 10 of the female parents and their corresponding trisomic $F_1$s, except the $F_1$ of triplo-9 showed the presence of awns. This may be attributed to the additional dose of an extra chromosome. A digenic segregation ratio of 3 present : 13 absent was realized for the presence of awns, which indicated the involvement of one basic gene and one inhibitory gene. Thus, it is quite logical to presume that the dosage increase of a basic gene brought about by trisomic condition might have exerted more influence on the expression of awning character, thereby masking or nullifying the influence of an inhibitory gene to condition awning in the parental triplos 2, 3, 6, 9 and 10 as well as their corresponding $F_1$s.

Presence of awns in triploid or tetraploid rice plant derived from an awnless diploid cultivar is not an uncommon case. Awnless condition in diploid rice is due to triple recessive condition of genes $\underline{an}_1$, $\underline{an}_2$ and $\underline{an}_3$ (Takahashi, 1964; Seetharaman, 1964 and Chang, 1964). These recessive genes due to sheer increase in their dosage produce awns in triploids and tetraploids. Similar instances of dosage effect of genes are known in crops like wheat, wherein the systemic mutations and the role of complex 'Q' locus in determining free threshing character has been amply demonstrated by Swaminathan and Rao (1961) and Mackey (1966).

The foregoing discussion leads to believe that of the eight chromosomes revealing a critical behaviour, only chromosomes 3 and 10 might be carrying one each of the two

major genes conditioning awning as indicated by the magnitude of chi-square deviation. The remaining critical chromosomes 2, 4, 5, 6, 7 and 9 might be sustaining minor modifiers that exert more influence on the character due to triplex condition of the respective chromosomes.

### 5.2.4 Atypical behaviour of trisomic $F_1$s and $F_2$s vis-a-vis their disomic counterparts in respect of pigmentation in some plant parts

In the present investigations, with regard to pigmentation in leaf margin (seedling and flowering stage), auricles, juncture, nodal septum and nodal ring; and presence of red awns, the trisomic $F_1$s and their $F_2$s, showed unusual behaviour contrary to the expectation. Such atypical behaviour of trisomic $F_1$s and $F_2$s vis-a-vis their disomic counterparts has been discussed hereunder.

#### 5.2.4.1 Leaf margin at seedling and flowering stages

The leaf margin pigmentation at seedling and flowering stages in the parents as well as in the disomic and trisomic $F_1$s is detailed in Table 10. It reveals interestingly, that the $F_1$ of trisomic 8 had pigmented leaf margin at both the seedling and flowering stages of growth. This might be attributed to the presence of an extra dose of chromosome-8, presumably possessing a basic gene to exert more influence on the expression of the character, thereby nullifying or diluting the influence of an inhibitory gene

leading to purple pigmentation in $F_1$ itself. This argument is substantiated by the fact that the $F_2$ population of trisomic-8 exhibited a significant departure from the expected 3:13 digenic ratio for leaf margin pigmentation at seedling and flowering stages (Tables 25 and 27), as seen by the differential proportion of purple or green plants at both these stages. Leaf margin pigmentation at seedling stage exhibited a skewed segregation with the occurrence of more than expected proportion of purple plants, whereas, at flowering stage, the skewness of segregation was due to the occurrence of green plants predominating the population. A differential expression of the same character was observed at different timings of growth, providing an evidence that the biosynthetic pathway of anthocyanin production is regulated with respect to both developmental time and pattern. Such an observation of fading of pigmentation at maturity is not uncommon as it has been reported earlier in crop plants like rice (Nagai, 1958), maize (Coe, 1957, 1985) and in soybean (Groose and Palmer, 1991).

5.2.4.2 Auricles

The auricle pigmentation in the parents as well as in the disomic and trisomic $F_1$s is presented in Table 10 which reveals interestingly, that the $F_1$s involving chromosomes 2, 3, 6 and 8 exhibited pigmented auricles. This might be attributed to the presence of an extra dose of respective chromosomes. For auricle pigmentation, a trigenic

interaction involving two complementary genes along with an inhibitory gene has been realized. It is quite probable that the extra dose of the chromosome might have carried the additional dose of regulatory genes responsible for triggering the reaction leading to the production of anthocyanin pigmentation in the auricles thereby masking or nullifying the effect of the inhibitory gene. Of these, remarkable was the case of triplo-6 $F_1$, which consistently exhibited dark purple auricles till maturity, without any dilution or fading.

In respect of $F_2$ behavioural pattern (Table 30), skewed segregation was observed with the preponderance of plants with purple auricles, only in the case of triplo-6 which was reflected in the higher magnitude of Chi-square deviation, while segregation in the $F_2$ of trisomics 2, 3, and 8 appeared non-significant suggesting that the expression of pigmented auricles in the $F_1$s of trisomics 2, 3 and 8 could also be due to the pleiotropic effect of pigmentation gene/s residing in other chromosomes, which is not uncommon in a crop like rice (Dhulappanavar, 1981; Nadaf, 1989).

5.2.4.3 Juncture

The behaviour of juncture pigmentation in the parents as well as in the disomic and trisomic $F_1$s is detailed in Table 10. A perusal of the table reveals that, the $F_1$s involving chromosomes 6 and 8 had pigmented juncture.

As detailed in the previous cases, this might be attributed to the dosage increase of genes brought about by trisomic condition. For juncture pigmentation a basic gene and three duplicate inhibitory genes were involved. Therefore, it is logical to presume that certain of these genes may behave differentially depending upon their dosage, resulting in an altogether different phenotype from the one normally obtained in disomic condition.

As may be seen from Table 31, analogous to auricle pigmentation, trisomic-6 showed a remarkably consistent dark purple juncture till maturity without any dilution and in this case also the observation was substantiated with the pattern of $F_2$ segregation. In $F_2$, the skewness of segregation was towards a very high proportion of plants with purple juncture, thereby showing a very high value of calculated chi-square.

Although the $F_1$ of trisomic-8 showed a pigmented juncture (Table 10), a non-significant value of calculated chi-square was realized in the $F_2$ segregation unlike the $F_1$ and $F_2$ of trisomic-6, indicating the absence of the genes controlling juncture pigmentation on the eighth chromosome. However, expression of purple pigmentation in $F_1$ of triplo-8 could be attributed to the pleiotropic effect of pigmentation gene/s located in other chromosomes, which is not uncommon in rice (Dhulappanavar, 1981; Nadaf, 1989).

5.2.4.4 Nodal septum

The behaviour of nodal septum pigmentation in the parents as well as in the disomic and trisomic $F_1$s is detailed in Table 10. It revealed that the $F_1$s of trisomics 1 and 3 and the $BC_1F_1$ of trisomic 4 had pigmented nodal septum contrary to the expectation. The additional dose of an extra chromosome, as conjectured in the previous cases might have influenced to alter the phenotype. A trigenic segregation ratio of 21 pigmented:43 non-pigmented was realized for nodal septum pigmentation indicating an operation of three interacting genes, namely, a basic gene and two complementary inhibitory genes. It may be surmised that the basic gene in trisomy condition might have triggered the reaction leading to anthocyanin production, thereby blocking the influence of other genes involving the operation.

As may be seen from Table 44, trisomics 1, 3 and 4 showed a highly significant values of calculated chi-square in the $F_2$ generation also thereby agreeing with the atypical behaviour of their corresponding trisomic $F_1$s.

5.2.4.5 Nodal ring

The nodal ring pigmentation in the parents as well as in the disomic and trisomic $F_1$s has been presented in Table 10. It indicated that the $F_1$s of triplos-1, 2, 3, 4, 6, 7 and 12 showed pigmented nodal ring. Probably, this may

be due to the dosage increase of genes brought about by trisomic condition. For nodal ring pigmentation, a tetragenic interaction involving three complementary genes and one inhibitory gene was realized in a segregation ratio of 27 purple:229 green. It is quite likely that with the addition of an extra chromosome one of the complementary genes might have functioned differentially as an anti-inhibitory gene to mask the effect of inhibitory gene thereby conditioning pigmentation of nodal ring in trisomic $F_1$s itself.

Interestingly, trisomics 1, 3, 6 and 7 showed a highly significant values of calculated chi-square in the $F_2$ generation also (Table 45) thereby, substantiating the observation made in their respective trisomic $F_1$s.

However, contrary to the expectation, the $F_2$ segregation of the remaining trisomics 2, 4 and 12 revealed non-significant values of calculated chi-square despite their $F_1$s expressing purple pigmentation in nodal ring. This might be due to pleiotropic effect of some other genes located in other chromosomes.

5.2.4.6 Awn colour (red awns)

The presence or absence of red awns in the parents as well as in the disomic and trisomic $F_1$s has been presented in Table 10. As detailed in the table, the $F_1$s of trisomics 2, 3, 6 and 10 showed the presence of red awns. The parental

trisomics 2, 3, 6 and 10 are characterized by the presence of awns, just like their parent Sona (3N). Thus, it is evident that the additional dose of an extra chromosome is responsible for awnlated condition in the $F_1$s of these trisomics.

Table 46 reveals that the presence of red awns was under a trigenic control indicating the involvement of two complementary and one inhibitory gene. It is quite logical to presume that with the dosage increase, the additional complementary gene(s) might have functioned differentially as anti-inhibitory gene to alter the effect of inhibitory gene thereby conditioning pigmentation in awns in the $F_1$s of these trisomics. It further reveals that contrary to the expectation the $F_2$ populations involving triplos-2 and 6 showed non-significant values of calculated chi-square which could be attributed to the pleiotropic effect as deduced in earlier cases, whereas the $F_2$ of triplos-3 and 10 showed a highly significant values of calculated chi-square as per expectation.

## 5.3 Transmission of the extra chromosome

In different trisomics, the transmission rates of the extra chromosome through male and female gametes are normally different in rice (Misra et al., 1986). The studies on transmission of trisomics are essential for computation of expected genetic segregation ratios.

In the present investigation, the transmission frequency of the extra chromosome was critically studied from the selfed progenies of parental trisomics as well as from the $F_2$ and backcross progenies of trisomics and marker crosses.

### 5.3.1 Selfed progenies of parental triplos

The transmission of the extra chromosomes varied from 17.58 per cent in triplo-5 to 45.52 per cent in triplo-12 (Table 48). Similar transmission studies have been made by Sadananda (1981) in IR-36 trisomics and Misra et al. (1986) in Sona trisomics. A comparison of the results of these two studies with those of the present study (Table 51) shows that the transmission frequencies varied from one study to another. It is possible that the transmission frequencies for a particular chromosome are affected by the genotypic background as was suggested by Khush (1973). However, Misra et al.(1986) studied the transmission frequency of the extra chromosome in the same variety Sona as is the case with this study. Here, again, there is no general agreement between the two studies for most of the chromosomes. For some of the chromosomes like triplos-3, 6, 7, 8 and 10, the differences between the transmission frequencies arrived at, in the two studies are too obvious to ignore. Further investigations are necessary wherein, along with phenotypic data, cytological analyses of the deviant phenotypes should also be made.

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Table 51. A comparative study of the percentage transmission frequency of extra chromosome in primary trisomics upon selfing

| New designations of triplos | Trisomics | Sadananda (1981) (IR-36 background) | Misra et al. (1986) (Sona background) | Present study (Sona background) |
|---|---|---|---|---|
| Triplo -1 | Triplo- 1 | 14.30 | 16.00 | 19.44 |
| Triplo- 2 | Triplo- 2 | 33.00 | 4.50 | - |
| Triplo- 6 | Triplo- 3 | 30.99 | 38.70 | 30.09 |
| Triplo- 3 | Triplo- 4 | - | - | - |
| Triplo- 5 | Triplo- 5 | 17.19 | 21.40 | 17.58 |
| Triplo-12 | Triplo- 6 | 27.15 | 17.70 | 33.33 |
| Triplo- 7 | Triplo- 7 | 14.30 | 39.00 | 29.47 |
| Triplo- 8 | Triplo- 8 | 2.03 | 33.30 | 26.73 |
| Triplo- 9 | Triplo- 9 | 27.27 | 21.00 | 18.18 |
| Triplo-10 | Triplo-10 | 22.59 | 17.70 | 26.32 |
| Triplo-11 | Triplo-11 | 13.40 | 28.30 | 22.12 |
| Triplo- 4 | Triplo-12 | 28.28 | 50.00 | 45.52 |

(-) Not studied

Misra et al. (1986) reported the appearance of only one non-parental trisomic (0.7%) in the selfed progenies of triplo-11. As pointed out by Khush (1973) one of the unique and useful features of the breeding behaviour of all kinds of trisomics is that, they produce related non-parentals and a few unrelated trisomics in their progenies in addition to parental trisomics.

In the present investigation, in the selfed progenies of triplos-3, 5, 6, 7, 8 and 12, one to four nondisomic-like plants appeared with an average frequency of 2.12 per cent (Table 48). However, it was difficult to distinguish between possible related non-parentals, unrelated trisomics and possible tetrasomic plants. The cytological details of such unusual plants need to be further investigated.

Khush (1973) has pointed out that the trisomics also appear spontaneously in the progenies of disomics at a very low frequency. Primary trisomics of Datura (Blakeslee and Avery, 1938) and Lotus pedunculatus (Chen and Grant, 1968) have been reported to produce some unrelated trisomics in their progenies with a very low frequency. Recently, Ashu Bhan et al.(1989) have reported the production of trisomics for a nucleolar chromosome in the progeny of a primary trisomic of Plantago lagopus for a non-nucleolar chromosome.

Why the presence of an extra chromosome increases the rate of spontaneous occurrence of other trisomics is not

clear. The increase probably is brought about by the extra chromosome causing interference in the regular mechanics of meiotic division to increase the rate of non-disjunction (Khush, 1973).

### 5.3.2 $F_2$ or backcross progenies

The transmission frequency of the extra chromosome varied from 5.56 per cent in triplo-4 to 38.27 per cent in triplo-12. In general, triplos-6, 8, 10 and 12 transmitted the extra chromosome at a high frequency, whereas triplos-1, 2, 4, 5, 9 and 11 at a lower frequency. Similar transmission studies have been made by Jena (1982) in Sona trisomics and Khush et al. (1984) in IR-36 trisomics. Table 52 details a comparison of the results of these two studies with those of the present study. The table reveals that the IR-36 trisomics-6, 7, 8, 10 and 12 transmitted the extra chromosome at a high frequency and trisomics-1 and 4 at a lower frequency. Thus, the present findings are in close agreement with those of Khush et al. (1984) with respect to the transmission frequency of trisomics 1, 4, 6, 7, 8, 10 and 12. However, in case of triplos-2, 3, 5, 9 and 11 the transmission frequencies revealed in the present study are at variance, and need further confirmation.

Further, Khush et al. (1984) observed that as the female transmission rates were calculated from the highly heterozygous $F_2$ or backcross populations, the heterozygosity

Table 52. A comparative study of the percentage transmission frequency of extra chromosome in primary trisomics from the $F_2$ or backcross progenies

| New designations of triplos | Trisomics | Jena (1982) | Khush et al. (1984) | | Present study |
|---|---|---|---|---|---|
| | | Male transmission | Female transmission | Male transmission | |
| | | 2n x (2n+1) | (2n+1)=2n | 2nx(2n+1) | (2n+1)$F_1$ selfed |
| Triplo- 1 | Triplo- 1 | 0.00 | 15.50 | 0.00 | 12.27 |
| Triplo- 2 | Triplo- 2 | - | 31.60 | 0.00 | 15.46 |
| Triplo- 6 | Triplo- 3 | 0.00 | 37.90 | 0.00 | 24.53 |
| Triplo- 3 | Triplo- 4 | - | 17.80 | - | 5.56[a] |
| Triplo- 5 | Triplo- 5 | 0.00 | 32.70 | 1.60 | 16.57 |
| Triplo-12 | Triplo- 6 | 4.00 | 37.10 | 5.60 | 32.92 |
| Triplo- 7 | Triplo- 7 | - | 31.10 | 0.00 | 25.08 |
| Triplo- 8 | Triplo- 8 | 0.00 | 25.50 | 14.30 | 34.34 |
| Triplo- 9 | Triplo- 9 | - | 35.50 | 27.30 | 13.41 |
| Triplo-10 | Triplo-10 | 0.00 | 27.40 | 1.50 | 28.13 |
| Triplo-11 | Triplo-11 | 3.10 | 39.70 | 2.70 | 17.33 |
| Triplo- 4 | Triplo-12 | 0.00 | 43.90 | 0.50 | 38.27 |

(-) Not studied

[a] $BC_1F_2$ population data

might have favoured the higher transmission rates of the extra chromosome. It is evident that transmission rates are also affected by genetic background (Khush, 1973). In the present study also, the transmission rates were calculated from the highly heterozygous $F_2$ or backcross populations, but such an impact of heterozygosity was not revealed. However, it should be noted here that Khush et al. (1984) studied a very large $F_2$ population with an average of 1270 plants, whereas in the present study the average $F_2$ population consisted of 171 plants only.

As regards male transmission of the extra chromosome, IR-36 trisomics-5, 6, 8, 9, 10, 11 and 12 transmitted their extra chromosomes through pollen (Khush et al., 1984) as against only trisomics-6 and 11 of Sona series (Jena, 1982). In case of IR-36 series, higher male transmission was observed in triplo-8 (14.30%) and triplo-9 (27.30%). This was as per expectation, because in the selfed progenies of these triplos a few tetrasomic plants were consistently observed (Khush et al., 1984). Here again, the behaviour of triplo-8 in Sona trisomics differs, suggesting the need for further investigations.

In the present study, the $F_2$ populations involving triplos-6 and 7 showed a few non-disomic like plants with an average frequency of 1.64 per cent (Table 49). The cytological details of such unusual plants need to be probed further.

The overall comparison of the studies on the transmission rate of the extra chromosome in indica trisomes does not reveal any specific trend, suggesting that further investigations combining phenotypic data and cytological analysis should be carried out.

## 5.4 Chromosomal location of genes for pigmentation - an overview

The segregation pattern for different anthocyanin pigmentation characters in $F_2$ generation of crosses between trisomics and the marker parent has been summarised in Table 53. It reveals that when all the 29 pigmentation characters studied in the present investigation were considered together, the genes conditioning anthocyanin pigmentation in different plant parts were found to be distributed on all the 12 chromosomes of the genome. Of these, chromosomes 12, 6, 4 and 2 were found to carry more number of genes for pigmentation. The chromosome-12 was found to carry genes for pigmentation in 19 plant parts namely, leaf sheath (external), prophyllum, leaf blade (seedling and flowering stage), leaf tip (seedling and flowering stage) leaf margin (seedling and flowering stage), leaf midrib, leaf axil, ligule, auricles, juncture back, node, pulvinus ring, glumes, apiculus, stigma and panicle axis. Chromosome-6 on the other hand carried genes for pigmentation in 15 plant parts namely, leaf blade (seedling and flowering stage), leaf margin (seedling and flowering stage), leaf midrib, auricles,

Table 53. Summary of primary trisomic segregation tests** in rice

| Primary trisomics Designation New | Old | Confirmed genetic ratio | Leaf sheath (external) | Leaf sheath (internal) | Prophyllum | Leaf blade (seedling stage) | Leaf tip (seedling stage) | Leaf margin (seedling stage) | Leaf blade (flowering stage) | Leaf tip (flowering stage) | Leaf margin flowering stage | Leaf midrib | Leaf axil | Ligule | Auricle | Juncture | Juncture back | Internode | Node | Sheath pulvinus | Pulvinus ring | Glumes (empty) | Lemma and palea | Apiculus | Stigma | Panicle base | Panicle pulvinus | Panicle axis | Nodal septum | Nodal ring | Red awn(dawn colour) | Awning |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Triplo-1 | Triplo-1 | 45:19 | T | D | D | D | D | D | D | D | D | D | D | D | D | D | T | D | D | D | D | D | T | D | T | D | D | D | T | T | D | D |
| Triplo-2 | Triplo-2 | 27:229 | T | D | T | D | T | D | D | T | D | D | T | D | D | D | T | D | D | D | D | T | D | D | I | T | D | D | I | T | D | T |
| Triplo-6 | Triplo-3 | 45:19 | D | T | D | D | D | D | D | D | D | D | D | D | D | D | D | D | D | T | D | D | T | D | D | T | D | D | D | T | T | T |
| Triplo-4 | Triplo-4 ($F_2$) | 9:55 | D | T | D | D | D | D | D | D | D | D | D | D | D | T | D | D | D | D | D | D | D | D | D | D | D | D | T | D | D | D |
| Triplo-3 | Triplo-4 ($BC_1F_2$) | 3:13 | T | T | T | D | T | D | D | T | D | D | T | D | D | D | D | T | D | T | T | D | D | T | T | T | T | D | T | D | D | T |
| Triplo-5 | Triplo-5 | 45:19 | D | T | D | D | D | D | D | D | D | D | D | D | D | D | T | D | T | D | D | D | D | D | D | D | D | D | D | D | T | T |
| Triplo-12 | Triplo-6 | 9:55 | D | D | D | T | D | T | T | D | T | T | D | D | T | T | D | T | T | D | D | T | T | D | D | T | T | D | T | T | D | T |
| Triplo-7 | Triplo-7 | 9:55 | D | T | D | D | D | D | D | D | D | D | D | D | D | D | D | T | D | D | D | D | T | D | D | T | T | D | D | T | T | T |
| Triplo-8 | Triplo-8 | 3:253 | D | T | D | T | D | T | T | D | T | T | D | D | D | D | D | D | D | D | D | T | D | D | D | D | D | D | D | D | T | D |
| Triplo-9 | Triplo-9 | 9:55 | D | D | D | D | D | D | D | D | D | D | D | T | D | D | T | D | D | D | D | D | D | D | D | D | D | D | D | D | D | T |
| Triplo-10 | Triplo-10 | 9:247 | T | D | D | D | D | D | D | D | D | D | D | D | D | D | D | D | D | T | D | D | D | D | D | D | T | T | D | T | T | T |
| Triplo-11 | Triplo-11 | 45:19 | D | T | D | D | D | D | D | D | D | D | D | T | D | T | D | T | T | D | D | D | T | D | D | T | T | D | T | T | D | D |
| Triplo-4 | Triplo-12 | 9:247 | T | D | T | T | T | T | T | T | T | T | T | T | T | D | T | D | T | D | T | T | D | T | T | D | D | T | D | D | D | D |

** D = Disomic segregation

T = Trisomic segregation

junctura, node, internode, glumes, lemma and palea, panicle base, panicle pulvinus, nodal septum and nodal ring; while chromosomes-4 and 2 were found to carry genes for pigmentation in 14 and 11 plant parts respectively. With respect to chromosome-4 the genes conditioning pigmentation in leaf sheath (external), leaf sheath (internal), prophyllum, leaf tip (seedling and flowering stage), leaf axil, internode, sheath pulvinus, pulvinus ring, apiculus, stigma, panicle base, panicle pulvinus and nodal septum were found to be located on it whereas the genes for pigmentation in leaf sheath (external), prophyllum, leaf tip (seedling and flowering stage), leaf axil, junctura, pulvinus ring, apiculus, stigma, panicle axis and nodal septum were found to be present on chromosome-2. The remaining chromosomes-1, 3, 5, 7, 8, 9, 10 and 11 were found to carry genes for pigmentation in 2 to 10 plant parts.

Further, it is quite evident from Table 53, that among a total of 29 characters studied in the present investigation, only in 14 characters the number of critical chromosomes were as expected on the basis of disomic ratios. The characters namely, pigmentation in prophyllum, leaf axil, ligule, glumes, apiculus, midrib, leaf blade (at seedling and flowering stage) and leaf tip (at seedling and flowering stage), were found to be controlled each by three interacting genes and three chromosomes were found to be critical as per expectation. Similarly, the characters namely, pigmentation in junctura, node, internode and panicle base were under a

tetragenic control and as per expectation four chromosomes revealed criticality. The number of critical chromosomes on the basis of disomic ratios were less than expected in three characters namely, pigmentation in auricles, sheath pulvinus and panicle axis, thereby suggesting the possibility of residence of two genes on one and the same chromosome at a distance of more than 50 map units.

Interestingly, in 12 cases the number of critical chromosomes identified were more than expected on the basis of disomic ratios. Although a trigenic segregation ratio was realized for juncture back, stigma and pulvinus ring, there were four chromosomes showing a critical behaviour. With respect to leaf margin pigmentation at seedling and flowering stage three chromosomes revealed critical behaviour inspite of the involvement of only two interacting genes. Similarly, for pigmentation in lemma and palea, nodal ring and panicle pulvinus four genes were involved and yet there were five chromosomes showing a critical behaviour. In the remaining four cases that is, in leaf sheath (external), leaf sheath (internal), nodal septum, and awn colour (red awn) the situation was still more complex. Pigmentation in leaf sheath (external), nodal septum and awn (red awn) was controlled by three interacting genes, but surprisingly, the number of critical chromosomes involved was 5, 7 and 5 respectively. With respect to pigmentation in leaf sheath (internal) a tetragenic control was realized but six chromosomes revealed a critical behaviour.

Thus, the foregoing paragraphs clearly illustrate that genetics of anthocyanin pigmentation may be still more complex than what is hitherto believed. However, the number of critical chromosomes being much more than what is expected on the basis of disomic ratios, is not an altogether uncommon phenomenon; as Siddiq et al. (1986) had also observed a similar trend for pigmentation characters in an attempt to locate the genes utilizing primary trisomic series of indica and japonica races.

A comparison of the present study (1992) has been made with that of Khush et al. (1984) and Siddiq et al. (1986) with regard to the chromosomal location of pigmentation genes in indica race (Table 54). The findings of the present investigations are closely corroborated with that of the only available report of Siddiq et al. (1986), suggesting that genes for pigmentation are distributed on almost all the chromosomes of the genome. But, it may be noted here that Siddiq et al. (1986) dealt with an incomplete set of nine trisomics. This thesis is the only report so far to deal with a large number of complex pigmentation characters with a complete set of primary trisomics of indica rice.

As in the case of Siddiq et al. (1986) the number of critical chromosomes in many cases was more than expected on the basis of disomic segregation ratio, which seems to be in direct conflict with genetic and linkage findings.

Table 54. A comparative study of the chromosomal location of genes for pigmentation

| | Pigmentation character | Khush et al.[a] (1984) Critical chromosome | Siddiq et al. (1986)[b] | | Present study[c] | |
|---|---|---|---|---|---|---|
| | | | Disomic ratios | Critical chromosome | Disomic ratios | Critical chromosomes |
| 1 | Leaf axil | - | 39: 25 | 5,6,7,10,11 and 12 | 45: 19 | 2,4, and 12 |
| 2 | Juncture | - | 21: 43 | 2,3,6,7,9,10 and 12 | 3:253 | 2,4,6 and 11 |
| 3 | Internode | - | 117:139 | 5 and 10 | 9:247 | 4,6,7 and 11 |
| 4 | Node | - | 21: 43 | 3,10,11 and 12 | 9:247 | 5,6,11 and 12 |
| 5 | Glumes (sterile lemma) | - | 27: 37 | 3,6 and 9 | 9: 55 | 6,8 and 12 |
| 6 | Apiculus | - | 189: 67 | 2,3,5,6,7,11 and 12 | 45: 19 | 2,4 and 12 |
| 7 | Stigma | 3 | 45: 19 | 2,3,5,7,9,11 and 12 | 45: 19 | 1,2,4 and 12 |
| 8 | Septum | - | 39: 25 | 5,7,9,11 and 12 | 21: 43 | 1,2,3,4,6,10 and 11 |
| 9 | Awn colour | - | 39: 25 | 5,6,7,10,11 and 12 | 9: 55 | 3,5,7,8 and 10 |

a IR-36 primary trisomics 3,5,7,8,9,10 and 12 only, were studied/tested

b IR-36 primary trisomics 2,3,5,6,7,9,10,11 and 12 only, were studied/tested

c Sona primary trisomics complete set was studied/tested

- Not studied

Assigning a character to a specific chromosome, as is customarily done in the study of independence of linkage groups using marker genes, is understandable when it follows a monogenic inheritance. Where a character is controlled by more than one gene and still a single chromosome is found critical, one has to assume that all influential genes including structural and regulator-modifier components are located on the same chromosome (Siddiq et al., 1986). With more and more understanding at the molecular level of the gene and its complex mode of functioning, the observed character-chromosome relationship warrants a satisfactory explanation.

Of the several possibilities, the dosage effect of the genes brought about in the trisomic state is one. Phenotype is the result of a series of regulated biochemical reactions. Alteration at any step in the biochemical pathway due to impaired enzymes and their concentration resulting out of change in genetic architecture, at any stage of development, may lead either to non-expression of a character or to an altered phenotype (Siddiq et al., 1986). The biosynthetic pathway of anthocyanin production is regulated with respect to developmental time and pattern. The regulation of anthocyanin biosynthesis being under control of a battery of genes acting in a variety of ways, is also very complex and multifold. The inheritance pattern of anthocyanin pigmentation characters have also been reported by various

workers to be complex with an involvement of genes of varying actions. Mode of action of these genes in a classical sense may be basic, complementary, duplicate, additive, supplementary, inhibitory or anti-inhibitory. Increase in dosage of genes brought about by trisomic condition might change the existing relationship between mutants and act either in complementary, additive, or in inhibitory fashion for making or masking the phenotype.

Similarly, involvement of pleiotropic genes and a number of regulatory genes acting in an unknown fashion might be yet another reason for the manifestation of skewed segregation of many trisomics at a time even for qualitatively inherited anthocyanin pigmentation characters. The involvement of common genes have been reported in the past by many workers for various pigmentation characters (Ramiah and Rao, 1953; Takahashi, 1957; Richharia et al., 1960; Dhulappanavar, 1973a, b, c; Manjunath, 1973; Setty and Misro, 1973; Thimmappaiah, 1975; Dhulappanavar 1980-81; Pavithran, 1985; Siddiq, et al., 1986; Nadaf, 1989 and Goud and Nadaf, 1990b).

Another possibility could be that the structural gene(s) and regulator gene (s) are located on different chromosomes. There are instances of this kind in maize in respect of controlling elements (McClintock, 1941; Doe, 1985) and anthocyanin pigmentation (Reddy and Reddy, 1975), where genes controlling a same pathway have been reported to occur on different chromosomes.

The existence of certain genes or sequences of genes on more than one chromosome, probably due to selective polyploidization, of a few chromosomes of the genome and distribution of the same in small segments over chromosomes in the course of karyotypic evolution of rice - could be yet another factor, although there is no direct evidence in its support. The linkage analysis in indica rice has clearly revealed that the three basic genes C, A and P responsible for the anthocyanin pigmentation, or their multiple alleles are supposed to be located in not more than three linkage groups. But, in fact, genes for anthocyanin pigmentation are found in eight linkage groups namely, I, II, III, IV, V, VII, IX and X (Refer Figure 3). Further, it is evident that the localisation genes governing purple pigmentation of the same plant parts are located in more than one linkage group or chromosome (Goud and Nadaf, 1990a). This lends an indirect evidence to such a possibility. Further, the thesis of a gene or a set of genes to remain in duplicated forms in more than one chromosome of the genome is particularly indicative of the presence of segmental homology between some of the chromosomes of the complement. Here, it is relevant to mention the studies of Sur (1975), Jena (1982) and Chung and Wu (1988) which also lends some support to this view. Cytological analysis of the different primary trisomics by the authors revealed non-homologous pairing at pachytene stage. In the absence of any meaningful explanation to this

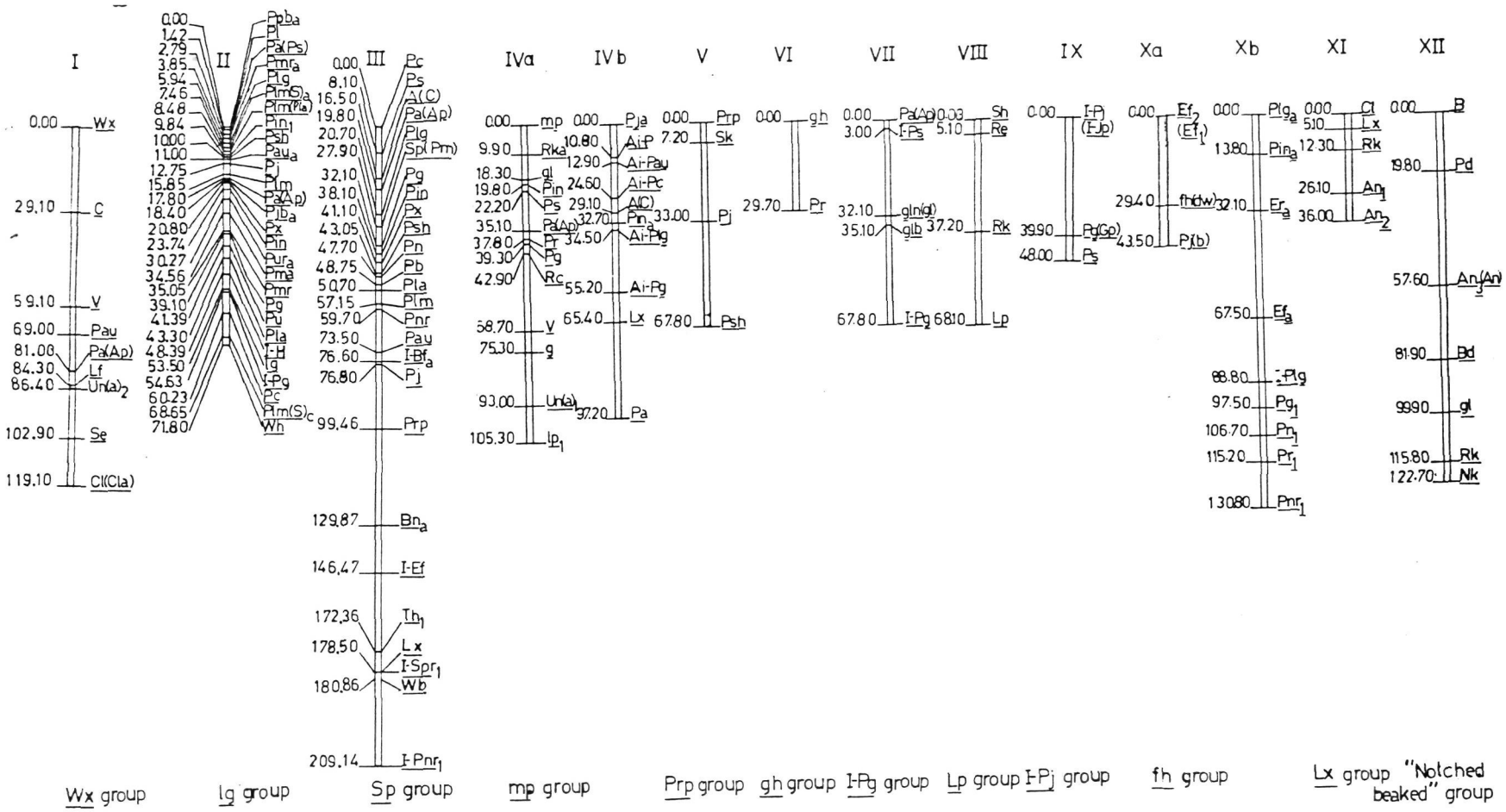



FIG. 3 LINKAGE MAPS IN INDICA RICE [GOUD AND NADAF, 1990a].

unusual pairing phenomenon, it could be inferred as presumably due to cryptic structural similarities between different chromosomes of the genome.

Overall examination of the present findings from trisomic analysis reveals that though all the 12 chromosomes are involved, majority of pigmentation genes are located on chromosomes 12, 6, 4, 2 and 11 in order of their magnitude. Although Siddiq et al. (1986) have studied only a part of the indica trisomics (triplos-2, 3, 5, 6, 7, 9, 10, 11 and 12) for anthocyanin pigmentation in nine plant parts, the authors also reported chromosomes 12 and 11 as carrying majority of the pigmentation genes.

Thus, it appears to be more likely that major basic genes such as A, C and P are located on the critical chromosomes 12, 6, 4 and 2. It is evidenced from the observation that genes for same set of characters are located on these chromosomes (Table 53). However, the exact location of each gene is possible only through very extensive genetic analysis coupled with biochemical studies. Indication of the involvement of rest of the critical chromosomes 11, 8, 7, 3, 1, 10, 5 and 9 in anthocyanin pigmentation may be pertaining to the localization genes and/or a battery of regulatory/modifying genes.

By following linkage analysis, it has been indicated that genes conditioning anthocyanin pigmentation in different

plant parts are located on almost all the linkage groups in *indica* rice, as compared to only three linkage groups namely, I, II and III in *japonica* rices (Nagao and Takahashi, 1963; Misro *et al*., 1966; Goud and Nadaf, 1990a and Kinoshita and Takahashi, 1991). In close agreement with the only available report of Siddiq *et al*. (1986), the trisomic analysis in *indica* rice in the present investigation also revealed that the genes conditioning anthocyanin pigmentation in different plant parts are distributed on almost all the chromosomes of the genome. The present findings appear to be agreeable with the recent review on the present status of linkage groups in *indica* rice (Goud and Nadaf, 1990a) and the updated linkage maps of *indica* rice (Figure 3) presented by the authors. Further, it appears that the linkage group II could be assigned to triplo-12 with near certainty because this trisomic has revealed criticality for majority of the characters mapped in this group. Similarly, linkage groups III and IV could be assigned to triplos-6 and 4, respectively, with near certainty because these trisomics have also revealed critical behaviour for many characters mapped in these groups. However, this needs further confirmation.

Kinoshita and Takahashi (1991) have presented the current linkage maps (Anonymous, 1990a) with up-to-date information on the recombination frequencies which also include the unlocated genes belonging to each group (refer Figure 4). Further, they have presented an agreed system of numbering rice

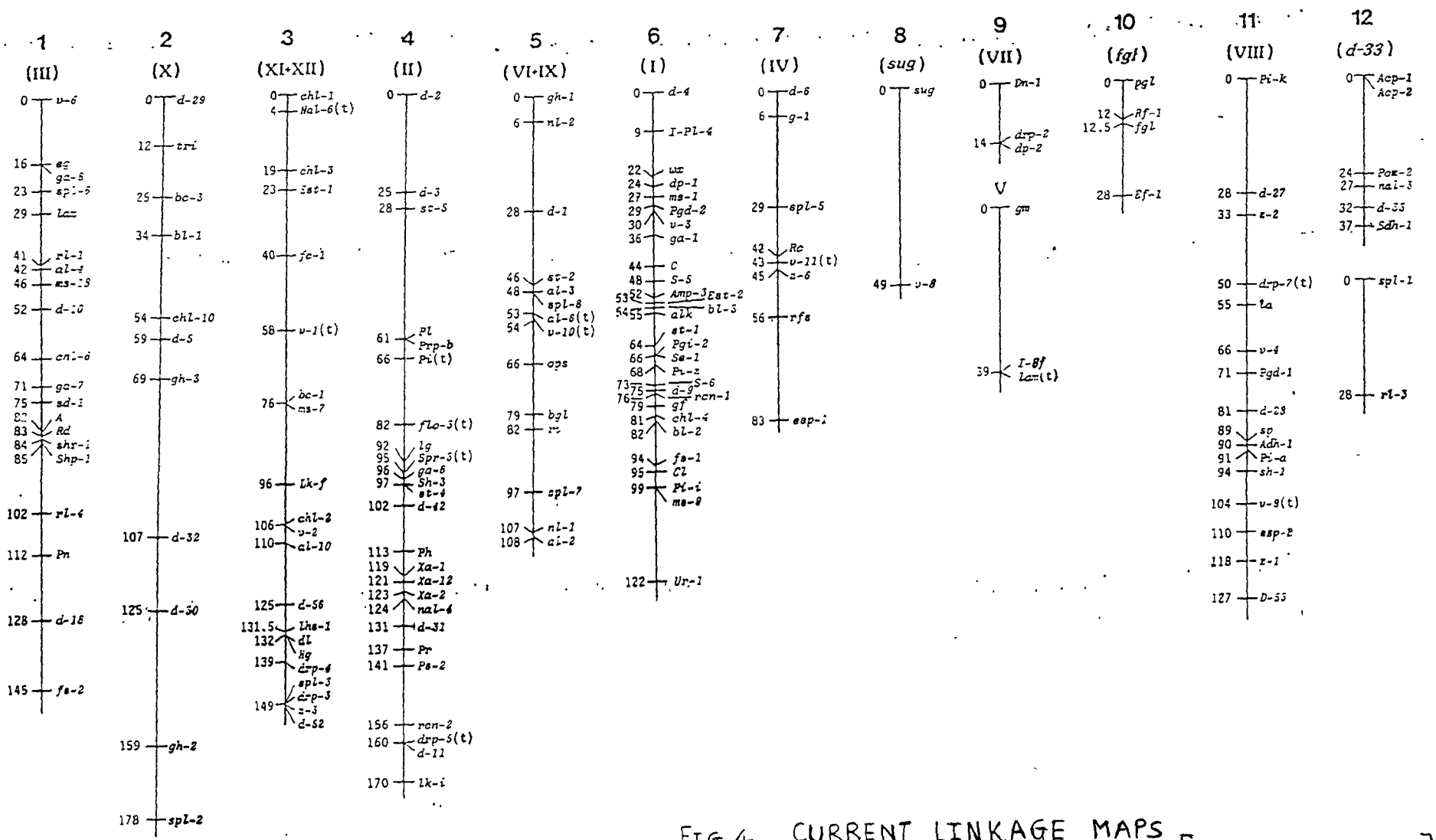



FIG. 4 CURRENT LINKAGE MAPS [ANONYMOUS, 1990a]

315.5.16

chromosomes (Anonymous, 1990b) and their relationships with linkage groups constructed by Nagao and Takahashi (1963) and Kinoshita (1984) (Refer Table 55).

A perusal of Figure 4 reveals that of the total genes allotted to the 12 linkage groups (chromosomes), only eight genes are pertaining to anthocyanin pigmentation characters and the remaining marker genes belong to different character groups namely, chlorophyll aberration characters, morphological characters, sterility characters, fungal and bacterial disease resistance characters, insect resistance characters and isozyme characters. Further, the figure clearly reveals that the linkage groups X (chromosome-2), XI + XII (chromosome-3), VI + IX (chromosome-5), IV (chromosome-7), sug (chromosome-8), VII and V (chromosome-9), fgl (chromosome-10), VII (chromosome-11) and d-33 (chromosome-12) do not possess any pigmentation gene at all whereas only linkage groups I (chromosome-6), II (chromosome-4) and III (chromosome-1) possess a total of eight pigmentation genes.

As may be seen from Figure 4, linkage group I (chromosome-6) possess two pigmentation genes namely I-pl-4 (Inhibitor for purple leaf-4) and C (Chromogen for anthocyanin), linkage group II (chromosome-4) possess four pigmentation genes namely, Pl (purple leaf), Prp-b(Pb), purple pericarp, Pr (purple hull) and Ps-2 (purple stigma-2) and linkage group III (chromosome-1) possess the remaining two genes namely, A (anthocyanin activator) and Pn (purple node).

**Table 55.** An agreed system of numbering rice chromosomes and their relationships with linkage group**

| New system agreed chromosome number | Linkage groups Kinoshita (1984) | Trisomics | | Chromosomes Kurata et al. (1981) | Translocations Nishimura (1961) |
|---|---|---|---|---|---|
| | | Khush et al. (1984) | Iwata & Omura (1975,1984 a and b) | | |
| 1 | III | 1 | O | $K_1$ | 3 |
| 2 | X | 2 | N | $K_2$ | 8 |
| 3 | XI + XII | 4 | M | $K_3$ | 5 |
| 4 | II | 12 | E | $K_4$ | 11 |
| 5 | VI + IX | 5 | L | $K_9$ | 2 |
| 6 | I | 3 | B | $K_6$ | 6 |
| 7 | IV | 7 | F | $K_{11}$ | 10 |
| 8 | Sug | 8 | D | $K_7$ | 12 |
| 9 | V + VII | 9 | H | $K_{10}$ | 1 |
| 10 | fgl | 10 | C | $K_{12}$ | 7 |
| 11 | VIII | 11 | G | $K_8$ | 9 |
| 12 | d-33 | 6 | A | $K_5$ | 4 |

** (Anonymous, 1990b)

Thus, it is evident that the anthocyanin pigmentation characters have not been studied intensively and extensively by the Japanese School and the IRRI School like that of the Indian workers. Further, these two schools strongly feel that the linkage maps of *indica* and *japonica* ecotypes are fairly similar. It is however, astonishing that the wealth of literature pertaining to anthocyanin pigmentation characters available through linkage analysis in *indica* rices could not be consolidated in the current linkage maps presented (Anonymous, 1990a). Kinoshita and Takahashi (1991) admitted that it was difficult to consolidate the Indian linkage groups published by Misro (1981) in the maps because of the shortage of common markers.

There have been two different genetic propositions on the anthocyanin colouration in different organs of plant in *japonica* and *indica* rices. According to the scheme of Takahashi (1982), there was multiple differentiation of alleles at the three loci, C, A and P to account for the intensity of pigmentation and colour hues, depending on the various combinations of the three genes. The colouration of parts other than the apiculus can be partly explained by the pleiotropic action of the basic genes. However, the majority of colouration patterns was the end product of the pleiotropic effect of distributing and localizing the pigment into respective parts in co-existence with the basic colour-producing genes. In contrast with this, the genes were allotted to the

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coloured localized in only one or two organs and the linkage relationships were considered to be among genes in indica rice (Misro, 1981). In addition, at least six kinds of inhibitors inserted their actions on the colouration of various parts in Takahashi's scheme, while the interactions of inhibitors (I) and anti-inhibitors (Ai) were postulated to describe the various segregation ratios in the colouration by Dhulappanavar's system (Dhulappanavar, 1977). Thus, further experiments are needed for the identification of colour genes by exchanging materials between Japanese and Indian workers (Kinoshita, 1986a).

Here it is relevant to recall the singular available report of Sadanand (1981). The author made an attempt to locate the genes conditioning pigmentation in leaf axil, internode, awn juncture, septum, stigma, apiculus, node and sterile lemmas using an incomplete set of primary trisomics of japonica in CS-M3 background (Khan and Rutger, 1973 and Khan, 1974). Although only trisomics 4, 5, 7, 8, 9, 10, 11 and 12 were tested for segregation of pigmentation genes, interestingly, all the chromosomes were found to be critical carrying genes conditioning anthocyanin pigmentation. When pigmentation in all the nine different plant parts was considered together, it was found that triplo-12 was critical for pigmentation in a maximum of seven plant parts. Thus, the findings of Sadananda (1981) showing anthocyanin pigmentation genes distributed on all the eight chromosomes under

investigation do not corroborate with the current linkage maps (Anonymous, 1990a) showing position of pigmentation genes on only chromosomes 6, 4 and 1. Further, interestingly, the segregation ratios of 3:13, 27:37, 117:139, 135:121, 27:229 and 243:781 obtained by the author for different pigmentation characters have never been reported by the earlier Japanese workers. This might be due to the differences in the gene constellations of the parental material used by various workers.

Thus, the foregoing discussion provides an appraisal of the situation for the intricate inheritance patterns involved in anthocyanin pigmentation characters and the chromosomal position of the pigmentation genes. It appears from the genetic architecture of anthocyanin pigmentation that there prevail substaintial differences between the *indica* and *japonica* sub species in the sequence of gene arrangement on different linkage groups. Further, the linkage maps of *indica* (Figure 3) and that of *japonica* (Figure 4) reveal that linkage group III has a major role in determining anthocyanin pigmentation in both the varietal groups. Yet they differ from each other to a greater extent. The *japonica* maps show only the basic gene A for anthocyanin activator and a gene Pn for purple node on linkage group III, whereas there are as many as 23 different localisation genes on linkage group III of *indica* thereby indicating how strong the differences are between the two racial groups in respect of gene sequences

governing even one single character like anthocyanin-pigmentation.

The findings in the present investigations regarding chromosomal location of pigmentation genes using *indica* trisomics also reveal that the gene sequences of the chromosomes do not appear to agree with *japonica* linkage maps (Anonymous, 1990a) whereas it appears to be agreeable with the *indica* linkage maps (Goud and Nadaf, 1990a).

Thus, it may be concluded that comprehensive genetic resolution of the complex patterns of inheritance of pigmentation characters in a crop like rice appears possible only through intensive and extensive genetical studies in a common genetic background. Further, to resolve the discrepancy of the pigmentation genes postulated in *japonica* and *indica* rices, identity of the chromosomes has to be established utilizing translocation stocks and primary trisomics in both the varietal groups and chromosomal location of pigmentation genes has to be carried out with the help of these stocks using common markers.

Restriction fragment length polymorphisms (RFLPs) are useful for a tool of plant breeding as well as genetic markers of linkage analysis. The use of RFLP and other biochemical markers has made it possible to construct new maps rapidly. Integration of conventional linkage maps of *japonica* and *indica* rices and the molecular linkage maps is

imperative for any meaningful exploitation of the rice crop in the future by means of biotechnological techniques.

## 5.5 Application of results of present investigations in retrospect and future prospects

In the present investigation, the non-critical disomic $F_2$ populations obtained from $F_1$ plants of indica trisomics 1 to 12 x Crossa were also raised to study their behaviour. It is expected that the non-critical disomic $F_2$ plants obtained from the crosses of Sona trisomics 1 to 12 x Crossa would be quite similar to that of the normal disomic $F_2$ plants of Sona disomic (control) x Crossa. But it did not hold true in the non-critical disomic $F_2$ populations involving trisomics 1, 4, 8, 10 and 12. Many trisomic like plants with a phenotype of their respective trisomic parent/s appeared surprisingly. Such plants were identified on the basis of the phenotypic expressions of marker characters. The $F_1$s of these populations showed 12 II only without an extra chromosome, yet these non-critical disomic $F_2$ populations were remarkably different, in general, from that of the normal disomic $F_2$ population. The phenotypic segregation revealed trisomic like plants as if there was an impact of the concerned parental trisomic. A total of 3519 plants were critically observed for all the characters studied in the present investigations. However, it was not possible to study the cytological details of such characteristic plants.

Zhang (1988), while observing the progenies of the trisomic series of indica cultivar Guangluai-4 found a similar unique phenomenon that in a few of diploid plants with 2n=24, a part or whole of the extra chromosome was duplicated and attached longitudinally to its corresponding bivalent. Such plants showed characteristics similar to those of the trisomic plants. Pachytene analysis revealed that the chromosomes with repeated segments were longer and had more Giemsa bands and chromomeres than normal ones. The author reported that chromosomes 9 and 10 were easily distinguishable as they organize nucleoli. Similarly, in triplos-5, 8 and 9, plants with repeated segments had morphological characteristics of corresponding trisomics. Namely, the diploid with repeated segments of chromosomes-5 had fine hairs on leaf surface, similarly as triplo-5, that with chromosome-8 had rolled leaves similarly as triplo-8, and that with chromosome-9 appeared stout similarly as triplo-9. Probably, the segmental repetition was due to the occurrence of breaks at different points in two of the three homologous chromosomes, their reciprocal translocation resulting in tandem fusion of partly homologous segments, and their duplication.

In the present investigation, although it was not possible to study the cytological details of such remarkable trisomic like plants, the morphological characteristics of corresponding trisomics was observed in a similar fashion; and hence the speculation of such segmental repetition is not

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unjustified. The cytological details of such plants need to be probed. Further as mentioned earlier, all the 3519 $F_2$ plants involving trisomics 1, 4, 8, 10 and 12 were critically observed for all the characters studied, in the present investigation. These populations are also expected to reveal a modified ratio due to distorted segregation which through critical genetic and cytological analysis, in comparison with the behaviour of their disomic counterparts, would throw light on the genic constellation with respect to the chromosomes carrying them towards the expression of trisomic phenotypes.

Transposable elements are important in the generation of mutations. They have proved useful in facilitating genetic analysis of the plant genome (Wu and Xie, 1990). Presence of such biological "mutagenising agents", transposable elements, in two of the structural genes of the anthocyanin biosynthetic pathway has been reported in Antirrhinum majus for the control of floral pigmentation (Martin et al., 1987). In maize, a large number of genes involved in anthocyanin biosynthesis have unstable alleles due to transposable element insertions (McClintock, 1948; and Emerson, 1921). Anthocyanin genes are useful genetic markers in soybean (Glycine max (L.) Merr.) also. Groose and Palmer (1991) have suggested that further elucidation of genetic control of anthocyanin biosynthesis may facilitate the characterization of putative transposable elements in mutable alleles of anthocyanin genes in this species.

Recently, Reddy et al. (1991) have presented the report on the progress of the Rockefeller Foundation (RF) Project on "Genetic and molecular biological approaches for identification and isolation of transposable elements in rice". About six selected lines with their purple leaf phenotype were utilized for isolation and identification of certain anthocyanin pigmentation genes for transposon tagging. A few genes that were active in anthocyanin pigmentation were characterized from different rice lines using maize specific probes. Maize probes that were used for this purpose included regulatory and structural genes of the anthocyanin pathway such as C1 and P (regulatory) and A1, C2, A2, $BZ_1$ and $BZ_2$ (structural). cDNA sequences of these genes have been used to screen a rice cDNA library constructed from a coloured indica rice line Purple Puttu. The authors reported that several cDNA clones homologous to each of the probes used have been isolated and will be further characterized and sequenced. The genomic analysis of different rice lines and mutants using these probes will eventually determine genes with line specific transposable element sequences. The authors have further reported that in addition to cDNA cloning. Northern, southern and western experiments have been carried out to identify the genomic structure of anthocyanin genes in rice as well as their expression.

During the course of present investigations completely purple, morphologically parental trisomic like

derivatives, were isolated unprecedentally from the $F_2$ generation plants of trisomics and marker crosses in 11 of the 12 trisomics. Thus, a complete set of trisomics in purple background could be isolated by identifying the extra chromosome cytologically. Such derivatives are of immense value for basic genetic and biochemical studies for chromosomal location of genes as well as for exploring putative transposable elements in mutable alleles of anthocyanin genes.

# CHAPTER - VI

# SUMMARY

## VI. SUMMARY

For carrying out trisomic analysis in rice (Oryza sativa L.) 13 crosses involving a complete set of primary trisomics (triplos-1 to 12) in the background of an indica cultivar 'Sona', its disomic control 'Sona' and an indica marker 'Crossa', were attempted. The primary trisomics and the disomic control Sona were completely green (acyanic) and were kept as female parents, whereas the male parent Crossa was completely purple, having anthocyanin pigmentation in almost all the plant parts. The disomic control - $F_1$, $F_2$ and $F_3$; the trisomics 1 to 12 $F_1$s and $F_2$s as well as the trisomic 4 $BC_1F_1$ and $BC_1F_2$ progenies were raised at the Central Rice Research Institute, Cuttack, Orissa state, during the years from 1989 to 1991. The cytological preparations of all the trisomic parents, $F_1$s and $BC_1F_1$ under study were made and the presence of the extra chromosome was confirmed at metaphase I or metaanaphase stages of meiosis, as 12 II + 1 I or 11 II + 3 I. The disomic $F_2$ population consisting of 900 plants, the trisomics 1 to 12 $F_2$ populations consisting of 1991 plants, the trisomic-4, $BC_1F_2$ population of 72 plants and 29 $F_3$ families each consisting of 100 to 140 plants were studied. A total of 30 characters including 29 pigmentation and one morphological character were included in the study. The main objectives of the investigation were:

1. To study the inheritance pattern of the anthocyanin pigmentation and one morphological characters.

ii. to identify and locate these genes to specific chromosomes so as to establish gene-chromosome relationship, and

iii. to study the transmission rate of the extra chromosome of each trisomic through hybridization and selfing. The salient features of the investigations are as follows:

Inheritance studies revealed the involvement of two to four pairs of genes for the characters under investigation. Digenic ratio of 3 purple:13 green was obtained for leaf margin pigmentation at seedling and flowering stages and for presence or absence of awning. Trigenic $F_2$ ratio of 45 purple: 19 green was postulated for pigmentation in leaf sheath (external), culm base, prophyllum, leaf tip at seedling and flowering stages, leaf axil, pulvinus ring, apiculus and stigma. Trigenic $F_2$ ratio of 9 purple:55 green was realized for pigmentation in leaf blade at seedling and flowering stages, leaf midrib, ligule, auricles, juncture back, glumes, and for presence or absence of red awn. A trigenic interaction was also realized in a $F_2$ segregation ratio of 21 purple:43 green for pigmentation in nodal septum. Tetrahybrid $F_2$ ratios were observed for the presence or absence of pigmentation in node, internode, lemma/palea, panicle base and panicle pulvinus (9:247), for leaf sheath (internal), panicle axis and nodal ring (27:229) and also for juncture and sheath pulvinus (3:253). All the $F_2$

segregation ratios revealed a breeding behaviour that was in confirmation with the $F_3$ generation. The culm base pigmentation was not studied in $F_3$ families. As per the information obtained for inheritance pattern of the genes of all the characters under study, the genotypic constitution was tentatively assigned for each character as observed in the two parents Sona and Crossa.

Trisomic analysis was done for 30 characters and critical chromosomes were identified with the help of modified ratio technique. For 14 characters, the number of critical chromosomes were as expected on the basis of disomic ratios. The genes conditioning pigmentation in prophyllum, leaf tip at seedling and flowering stages, leaf axil and apiculus were found to be located on chromosomes 2, 4 and 12; whereas in case of leaf blade at seedling and flowering stage, leaf midrib and glumes, the three genes conditioning pigmentation were located on chromosomes 6, 8 and 12. The three genes responsible for a pigmented ligule were assigned to chromosomes 9, 11 and 12, whereas the four genes responsible for a pigmented internode and panicle base were on chromosomes 4, 6, 7 and 11. Chromosomes 5, 6, 11 and 12 were found to carry one each of the four genes conditioning pigmentation in nodal region, whereas in case of junctura, of the four genes conditioning pigmentation, two independent genes were located on chromosome-6 and one each of the remaining two genes appeared to be on chromosomes 2 and 11.

The number of critical chromosomes on the basis of disomic ratios were less than expected in three characters namely, pigmentation in auricles, sheath pulvinus and panicle axis, thereby suggesting the possibility of residence of two genes on the same chromosome at a distance of more than 50 map units. Thus, of the three genes conditioning auricle pigmentation, two genes were located on chromosome-6 and the third gene on chromosome-12, whereas two of the four genes responsible for a pigmented panicle axis were located on chromosome-12 and one each of the remaining two genes on chromosomes-2 and 10. With respect to pigmentation in sheath pulvinus, the size of all the 12 trisomic $F_2$ populations was inadequate and the genes could not be assigned to respective chromosomes clearly.

The number of critical chromosomes were more than expected on the basis of disomic ratios in the remaining 12 pigmentation and one morphological characters. In case of pigmentation in leaf margin at seedling and flowering stages two major genes involved appeared to be located on chromosomes-8 and 12, whereas chromosome-6 appeared to carry minor genes with modifying effect. The three major genes conditioning pigmentation in juncture back region seemed to be on chromosomes-5, 9 and 12 and some minor genes appeared to be on chromosome-1. As regards pigmentation in stigma, the three major genes seemed to be located on chromosomes-1, 4 and 12 and some minor genes seemed to be on chromosome-2.

In case of pigmentation in pulvinus ring, chromosomes-4,10 and 12 appeared to carry one each of the three major genes and chromosome-2 appeared to possess some minor modifiers. Chromosomes-3, 6, 7 and 11 were found to carry the major genes conditioning pigmentation in lemma/palea and nodal ring region and the minor genes involved in the operation were carried by chromosome 1, whereas in case of panicle pulvinus pigmentation the major genes appeared to be located on chromosomes-3, 6, 7 and 11 and minor modifier genes on chromosome-4. With respect to leaf sheath (external) pigmentation, the three major genes were found to be located on chromosomes-10, 2 and 4 and some minor genes on chromosomes-1 and 12, whereas the major genes influencing leaf sheath (internal) pigmentation were found to be located on chromosomes-4, 3, 7 and 11 and some minor genes were also found to be on chromosomes-5 and 8. In case of nodal septum pigmentation, chromosomes-6, 4 and 1 appeared to possess the major influencing genes and chromosomes-2, 3, 10 and 11 were found to carry some minor modifiers. As regards the presence of red awn, the three major genes appeared to be located on chromosomes-10, 5 and 3 and the minor modifying genes on chromosomes-7 and 8. In case of the morphological character that is, awning, chromosomes-3 and 10 were found to possess the major genes, whereas, chromosomes-2, 4, 5, 6, 7 and 9 were found to possess minor genes.

The studies on the transmission rate of the extra chromosome revealed that upon selfing the transmission frequency

of the extra chromosome varied from 17.58 per cent in triplo-5 to 45.52 per cent in triplo-12. Triplo-1 showed 19.44 per cent transmission frequency. Non-disomic like plants appeared in the selfed progenies of triplos-3, 5, 6, 7, 8 and 12 with a frequency ranging between 0.97 per cent in triplo-3 to 3.96 per cent in triplo-8. The studies on the transmission frequency of the extra chromosome from the $F_2$ and/or back-cross progenies of trisomic and marker crosses revealed that transmission frequency of the extra chromosome varied from 5.56 per cent in triplo-4 to 38.27 per cent in triplo-12. Triplo-1 showed 12.27 per cent transmission frequency. The non-disomic like plants also appeared in the progenies of triplos-6 and 7 with a frequency of 1.25 per cent and 2.03 per cent respectively.

The present investigations have clearly revealed that the anthocyanin pigmentation genes are distributed on almost all the chromosomes of the genome; which is in agreement with the linkage maps of indica rice.

Thus, the present investigations have provided a possibility of assessing the non-traditional approach for assigning the intricately inherited anthocyanin pigmentation genes to the corresponding chromosomes. An understanding of the position of genes and the neighbouring genes facilitates the transfer of characters of interest to cultivated rice. Further, the exact location of genes may also prove helpful for future chromosome engineering studies.

## CHAPTER - VII

# REFERENCES

## VII. REFERENCES

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# APPENDIX

Appendix - I. Genetic explanation of various segregation ratios reported for different characters

| Sl. No. | Ratios | Genetic explanations |
|---|---|---|
| 1 | 2 | 3 |
| 1 | 1: 3 | One inhibitory gene |
| 2 | 3: 1 | Single gene |
| 3 | 1:15 | Two inhibitory duplicate genes |
| 4 | 3:13 | One basic gene and one inhibitory gene |
| 5 | 9: 7 | Two complementary genes |
| 6 | 15: 1 | Two duplicate genes |
| 7 | 1:63 | Three inhibitory duplicate genes |
| 8 | 3:61 | One basic gene and two inhibitory duplicate genes |
| 9 | 9:55 | Two complementary genes and one inhibitory gene |
| 10 | 15:49 | Two duplicate genes and one inhibitory gene |
| 11 | 21:43 | One basic gene and two inhibitory complementary genes |
| 12 | 27:37 | Three complementary genes |
| 13 | 39:25 | One basic gene, one inhibitory gene and one anti-inhibitory gene |
| 14 | 45:19 | One basic gene and two complementary duplicate genes |
| 15 | 54:10 | Any two of the three complementary duplicate genes |
| 16 | 57: 7 | One independent dominant gene and two complementary genes |
| 17 | 63: 1 | Three duplicate genes |
| 18 | 3:253 | One basic gene and three inhibitory duplicate genes |

Appendix-I continued

| 1 | 2 | 3 |
|---|---|---|
| 19 | 9:247 | Two complementary genes and two inhibitory duplicate genes |
| 20 | 15:241 | Two duplicate genes and two inhibitory duplicate genes |
| 21 | 21:235 | One basic gene, one independent inhibitory gene and two inhibitory complementary genes |
| 22 | 27:229 | Three complementary genes and one inhibitory gene |
| 23 | 30:226 | One basic gene and three inhibitory complementary genes |
| 24 | 63:193 | Three duplicate genes and one inhibitory gene |
| 25 | 81:175 | Four complementary genes |
| 26 | 105:151 | Two duplicate genes and two inhibitory complementary genes |
| 27 | 108:148 | One inhibitory and three complementary genes wherein one complementary gene functioned as anti-inhibitory also |
| 28 | 111:145 | One basic gene and three inhibitory complementary genes |
| 29 | 117:139 | Two complementary genes, one inhibitory gene and one anti-inhibitory gene |
| 30 | 129:127 | One basic gene, one inhibitory gene and two anti-inhibitory complementary genes |
| 31 | 135:121 | Two basic complementary genes and two complementary duplicate genes |
| 32 | 147:109 | One basic gene, two inhibitory duplicate genes and one anti-inhibitory gene |
| 33 | 162: 94 | One basic gene and any two of the three complementary duplicate genes |
| 34 | 165: 91 | One basic gene, two inhibitory complementary genes and one anti-inhibitory gene |
| 35 | 183: 73 | One basic gene, one inhibitory gene and two anti-inhibitory duplicate genes |

Appendix-I continued

| 1 | 2 | 3 |
|---|---|---|
| 36 | 189: 67 | One basic gene and any one of the three complementary duplicate genes |
| 37 | 195: 61 | Two duplicate genes, one inhibitory gene and one anti-inhibitory gene |
| 38 | 207: 49 | Two duplicate sets of complementary genes with two genes in each set |
| 39 | 219: 37 | One independent dominant gene and three complementary genes |
| 40 | 243: 13 | Any two of the four complementary duplicate genes |
| 41 | 246: 10 | Two independent dominant genes and two complementary genes |
| 42 | 249: 7 | One independent dominant gene and any two of the three complementary duplicate genes |
| 43 | 255: 1 | Four duplicate genes |
| 44 | 3:1021 | One basic gene and four inhibitory duplicate genes |
| 45 | 21:1003 | One basic gene, two inhibitory duplicate genes and two inhibitory complementary genes |
| 46 | 54:970 | Three complementary genes and two inhibitory duplicate genes |
| 47 | 63:961 | Three duplicate genes and two inhibitory duplicate genes |
| 48 | 171:853 | Two complementary genes, one inhibitory basic gene and two inhibitory complementary duplicate genes |
| 49 | 243:781 | Five complementary genes |
| 50 | 255:769 | Four duplicate genes and one inhibitory gene |
| 51 | 378:646 | Three complementary duplicate genes and two inhibitory complementary genes |

Appendix-I continued

| 1 | 2 | 3 |
|---|---|---|
| 52 | 387: 637 | Two complementary genes, one inhibitory gene and two anti-inhibitory complementary genes |
| 53 | 406: 619 | Three basic complementary genes and any one or both of the remaining two complementary duplicate genes |
| 54 | 567: 457 | One basic gene and any three of the four complementary duplicate genes |
| 55 | 606: 418 | One basic gene, three inhibitory complementary genes and one anti-inhibitory gene |
| 56 | 702: 322 | Three complementary genes, one inhibitory gene and one anti-inhibitory gene |
| 57 | 3:4093 | One basic gene and five inhibitory duplicate genes |

Other ratios

| | | |
|---|---|---|
| 1 | 9:3:4 | Two complete dominant genes with one gene, when homozygous recessive is epistatic to the other |
| 2 | 9:6:1 | Two complete dominant genes affecting different expressions, interaction between both dominant gives new phenotype |
| 3 | 12:3:1 | Two complete dominant genes with one gene, when dominant, is epistatic to the other |

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