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REVIEW & INTERPRETATION

Genetics of Qualitative Traits in Pearl Millet: A Review

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ABSTRACT

Pearl millet, *Pennisetum glaucum* (L.) R. Br., is the principal food cereal on 25 million ha of the drought-prone semiarid regions of Africa and the Indian subcontinent. It is also used as a forage in Australia, southern Africa, South America, and the USA, and has shown potential as an early-maturing summer grain crop in temperate regions. Recent increased levels of breeding and genetic research, now including gene mapping, have indicated a need for a current comprehensive review of accumulated genetic information on qualitative traits in this species. The present descriptive review attempts to meet this need and reports 167 studies, since 1934, on 145 characters in 12 categories: chlorophyll deficiencies, foliage striping, leaf characters, pubescence, plant form, pigmentation, earhead characters, reproductive structures and gamete formation, sterility, seed characters, earliness and maturity, and disease resistance. Gene symbols, where assigned by authors, are listed, and the nature of genetic effects are given. Known linkages and biochemical genetic markers are also reviewed. A consistent nomenclature system should be developed and followed in the future, and a location designated for the deposition of genetic stocks.

PEARL MILLET, *Pennisetum glaucum* (L.) R. Br., is grown principally for grain in the tropical and subtropical areas of Africa and the Indian subcontinent. It is planted on ≈15 million ha in Africa and ≈11 million ha in India, yielding annually ≈10 million tons of grain. It is of importance as a high-quality forage crop in the USA, Australia, South America, and southern Africa. It tolerates drought, low soil fertility, and low soil pH, and responds well to water and favorable soil conditions (Anand Kumar, 1989). Pearl millet could become an important grain crop in the USA, as the high-quality grain has the potential to be used like corn and sorghum in rations of chicks (*Gallus gallus*), beef cattle (*Bos taurus*), and swine (*Sus scrofa*) (Hanna et al., 1991). Because of its exceptional ability to tolerate drought, pearl millet may in the near future extend feed grain production into regions too arid for sorghum (Burton, 1983).

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Information on the genetics of different traits of a crop is important for its systematic breeding and long-term improvement. With its low chromosome number, availability of an impressive range of variation for several morphological characters, ease of selfing and deliberate crossing, production of both selfed and crossed seed in good quantities, and relatively short life cycle (in most cases, <90 d) permitting three to four generations a year, pearl millet should be amenable to genetic studies (Vinchon, 1949); however, methodical attempts have not been made to study the inheritance of different characters in pearl millet. Burton and Powell (1968) cited the secondary economic importance of the crop, its restricted area of use, and the failure of geneticists to appreciate its potential as a research organism as the principal reasons for the lack of systematic genetic studies. Pearl millet is a cross-pollinated crop and is therefore highly heterozygous and heterogeneous in nature, and genetic studies necessitate the development of homozygous inbreds. The genetics of qualitative traits in pearl millet was last reviewed by Koduru and Krishna Rao (1983).

In this publication, reports on the inheritance of >140 mutant phenotypes of morphological, cytological, and biochemical marker traits are comprehensively reviewed. Studies on major genes that have contributed to or have the potential to contribute to the improvement of this crop are described.

CHLOROPHYLL DEFICIENCIES

Both lethal and nonlethal chlorophyll deficiencies have been reported in pearl millet. Based on the degree of chlorophyll deficiency, the mutant seedlings either survive or die at various stages of growth. Lethal CD mutants are normally conditioned by homozygous recessive genes and therefore can be transmitted only in a heterozygous condition. Pearl millet, because of its highly cross-pollinated nature, carries a relatively high proportion of lethal CD mutants. Though CDs are deleterious to the populations in which they are present, they provide easily identifiable seedling markers for basic and applied genetic studies (Hanna et al., 1978). Available reports are reviewed individually, as there has been no attempt

Abbreviations: CD, chlorophyll deficiencies; cms, cytoplasmic–nuclear male sterility; DES, diethyl sulfate; DM, downy mildew; EMS, ethyl methanesulfonate; gms, genetic male sterility; IVDMD, in vitro dry matter digestibility; MES, methyl ethanesulfonate; R, resistant; S, susceptible.

to uniformly describe and designate gene symbols for CDs.

The first report of CD seedlings was by Rangaswami Ayyangar (1934), who described three types of albino seedlings: pure white, white with greenish leaf tip, and cream-colored leaves. Rangaswami Ayyangar and Hariharan (1935) found albino seedlings in a population subjected to inbreeding, and reported that albinism is controlled by a single recessive gene (*c*). They also observed a pale-green factor (*e*) that showed a depressing effect on a number of characters. The F_2 of the cross green × pale green gave green, pale-green, and albino seedlings in the ratio 9:3:4 (*CE*, green; *Ce*, pale green; and *cE* and *ce*, albino). Kadam et al. (1940) reported six types of CD seedlings following inbreeding. They referred to these as albino, yellow, virescent yellow, golden yellow, virescent white, and zebra types. They observed monogenic to digenic segregation patterns for these CDs. The only exceptions were virescent white and golden yellow, which showed 15:1 and 3:1 ratios, respectively. They ascribed the "lack of regular or definite progeny behavior" to disabilities in controlling pollination effectively. They did not suggest any symbols for the genes controlling these traits. Vinchon (1949) reported a single gene pair controlling for eight lethal CDs; he considered the CD as the primary cause for the elimination of homozygotes. Krishnaswamy (1962) described a monogenically inherited pale-green seedling character, in which the homozygous recessive matures as a weak plant. Al-Fakhry et al. (1965) reported the occurrence of seven types of CDs in an accession introduced into the USA in 1955. Albino, virescent yellow, and yellow phenotypes are inherited as simple recessive traits, whereas light green and zebra types are controlled by complementary genes. Inheritance of the two other types (isolated green striped and dotted yellowish leaves) could not be studied because of insufficient population size.

Burton and Powell (1965) proposed a system of nomenclature for pearl millet CD seedling characters based on the color, degree of color, and color pattern exhibited by the first seedling leaf. This system provides for similar characters due to different genes or different alleles. In addition, it uses simple descriptive English words and selects the first letter of the word (lowercase) as a part, or all, of the gene symbol. Though this proposal was made nearly 30 yr ago, there has been no standardization in using gene symbols for CD seedlings.

Burton and Powell (1965) described six naturally occurring seedling CDs. The *w* (white) mutant produced white seedlings without any pigmentation that did not show any change until they began to brown with age. Plants homozygous for *pgw* (pale green white) produced a first leaf with little or no pale green pigmentation. First leaves of the mutant *py* (pale yellow) were uniformly yellow; a similar expression was observed in *py2* (pale yellow 2), except that the latter exhibited slightly darker color at low temperatures and under low light intensities. Gene *my* (medium yellow) in a homozygous state produced a medium yellow color on the first leaf. Plants homozygous for *y* (yellow) showed a deep yellow color on their first leaf when grown in full sun in a hot greenhouse (≈30 °C, or 85 °F). All six mutants segregated as recessives in a 3:1 ratio in selfed progenies of heterozygous plants. When heterozygous plants carrying these deficiencies were crossed, normal green plants were recovered, indicating that different loci are involved. A selfed progeny of *Yy Ww* plants gave a 9:3:4 ratio of green/yellow/white seedlings. This ratio is similar to the one obtained by Rangaswami Ayyangar and Hariharan (1935), who classified the seedlings they observed as green, pale yellow, and albino. Obviously, in addition to a standardized system of nomenclature, there is a need to use a standard chart in describing the seedling colors. Burton (1986) mentioned a single recessive gene *g* controlling golden plant color. Burton and Powell (1965) also reported that variation in the environment caused changes in the colorations of these CD mutants.

Koduru and Krishna Rao (1980) described 13 spontaneously occurring CD phenotypes and their genetic control. Ten of these showed monogenic recessive inheritance. White (*w*) was a seedling lethal, in which leucoplastids were observed in the plumules. The gene *wt* (white-tipped green) produced light yellow plumules, but soon chlorophyll development proceeded from the base of the second leaf and the tip of the lamina remained white or yellow in the second to fourth leaves and subsequent leaves were normal green. Patchy white (*pw*) seedlings exhibited dispersed and irregular white patches from the plumule stage, which in mature plants turned to light green or yellow green and the interveinal areas of green parts developed yellow streaks. Seedlings carrying the *wv* (white virescent) gene showed greening of leaves in 4 to 6 d. The first leaf was yellow, but soon turned green, starting from the tip. The seedlings were observed to be light green and weak. In seedlings carrying the white striping$_1$ (wst_1) gene, white or light yellow stripes appeared on the margins of the leaf blade and sheath. At maturity the spikelets and pericarp were yellow. In white striping$_2$ (wst_2), white to light yellow stripes were observed from the plumule stage onward and survived through the adult stage. White striping$_4$ (wst_4) appeared 15 to 20 d before flowering as dull, white, short interveinal longitudinal streaks in the middle part of the lamina. The fine striped mutant (*fst*) is greenish yellow at the plumule stage; subsequently (at the 2- to 3-leaf stage), narrow white longitudinal stripes were observed that persisted until maturity. The gene *c* (chlorina) was characterized by CD leaves at all stages of growth, beginning at the 4- to 6-leaf stage. The leaves are greenish yellow and the plants are vigorous. The gene *yv* (yellow virescent) appears as pale yellow at the plumule stage, and seedlings turned light green in ≈2 wk. Adult plants were weak, with narrow leaves and narrow thin yellowish spikes, but produced a greater number of tillers than the controls. This study found that genes *wt*, *yv*, *pw*, and wst_1 show independent assortment, and, in addition, the *w*, *wt*, and *yv* genes are inherited independently of the gene for hairy leaf margin *Hm*.

Three other genes controlling chlorophyll deficiencies show digenic recessive inheritance (Koduru and Krishna Rao, 1980). Yellow striping (yst_1, yst_2) appeared from the seedling stage as dull, interveinal longitudinal yellow stripes and persists until maturity. The yellow green mutants (yg_1, yg_2) were yellow green at the plumule stage, but subsequently chlorophyll development was observed to be normal. The light green (lg_1, lg_2) mutants were sublethals, and were characterized by narrow leaves, weak short stems, and delayed flowering.

Appa Rao et al. (1984) described six CD mutants, isolated from germplasm accessions. The color of the leaf blades was described using the color chart developed by the Royal Horticultural Society, London. This is the first study in pearl millet in which a standard color chart has been used to describe CDs. Zebra (*z*), caused transverse lemon yellow (14C) streaks alternating with normal green color on the leaf blade, leaf sheath, and internode developed under low temperatures. Zebra virescens (*zv*) developed primrose yellow (4C) transverse bands alternating with green (146C) bands from the 3-leaf to the 7-leaf stage. Bright yellow (*by*), expressed throughout the plant's life, developed a mimosa yellow (8B) color on leaf blades and was less pronounced on leaf sheath and stem. Chlorina virescens (*chv*) was distinguishable soon after emergence and showed yellowish green (146B) leaves that turned light green after flowering (Appa Rao et al., 1988c); greenish yellow (*gy*) produced pale yellow foliage (10Y) from emergence until maturity; and albino-terminalis (*at*), in which plants were normal until flower initiation, after which the top three to five leaves (including the bristles and glumes) remained amber yellow (18C) and gradually turned lighter green at maturity. Each of the mutant traits is controlled by a single recessive gene. The bright-yellow and greenish-yellow mutants described here in part resemble the yellow and light-green CDs described by Hanna et al. (1978).

Appa Rao et al. (1990) described two spontaneous CD mutant traits, white sheath and bleached leaf. Normal light-green leaf sheaths of the white sheath mutant turned white 1 wk after germination, with maximum expression occurring at flowering and persisting until maturity. This trait is controlled by a single recessive gene, for which the symbol *ws* was proposed. Normal leaves of the bleached leaf mutant tended to turn yellow from the tip toward the base 10 d after emergence, whereas the bottom third of the leaf blades, midribs, and a small portion on either side of the midribs remained green. The bleached leaf expression increased until floral initiation, when the plants turned green. The bleached trait is controlled by a single recessive gene, *bl*. Linkage studies showed that there are 43 crossover units between the bleached leaf and glossy traits (Appa Rao et al., 1987) and 45 to 54 crossover units between the white sheath and greenish-yellow leaf (*gy*) (Appa Rao et al., 1984) traits.

Werner and Burton (1991) reported a CD zebra mutant from a breeding population that showed alternating green and yellow crossbands on leaf blades. The yellow bands turned green before maturity. Data from F_2 populations indicated that survival of the zebra plants is affected by the genetic background. The F_2 and testcross population data showed that this zebra trait is controlled by a single dominant gene. Total forage yield appeared to be reduced in this mutant, suggesting that it is not likely to be a desirable agronomic trait.

It is well known that CD mutants occur with a high frequency following treatments with both physical and chemical mutagens. Krishnaswamy and Rangaswami Ayyangar (1941) observed in x-ray irradiated seed treatments a high frequency of albinos. Chandola et al. (1963) reported the occurrence of seven types of chlorophyll mutations following treatment of seed with gamma rays. These included albina, xantha, viridoalbina, striata, xanthalba, tigrina, and maculata. The first two were observed to be lethal mutants. Joshi (1968) recorded albina and striata mutants following seed treatments with gamma rays. Albinas were lethal; striata mutants developed into stunted plants. Dev et al. (1987) obtained white seedling and yellow virescent seedling CD mutants following gamma-ray irradiation. Both CDs were found to be monogenic recessive.

Tara Mohan et al. (1973) reported induction of a high frequency of chlorophyll mutations after treatment of pearl millet seed with *N*-nitroso-*N*-methyl urea. The spectrum included types such as chlorina, xantha, viridis, albina, striata, maculata, and pale green. Singh et al. (1978a) observed albina, chlorina, xantha, and viridis CD types after treating seed with gamma rays, EMS, or a combination of these two mutagens.

Hanna et al. (1978) recovered a yellow mutant from inbred Tift 23B following seed treatment with thermal neutrons and DES, and light-green mutants from Tift 13B following treatment with neutrons and EMS. These two CDs were nonlethal and could be maintained in a homozygous recessive state. The yellow (yn_1, where *n* designates nonlethal) mutant developed yellow leaves and stems at 7 to 10 d after emergence, which persisted through maturity. The light green (lgn_1) mutant was characterized by light-green stems and leaves from seedling emergence through maturity. These two mutants were observed to be controlled by two different single recessive genes. They established a linkage group that involves the yn_1, lgn_1, and *fs* (female sterile) loci. The *tr* (trichomeless) gene was found to be independent of the yn_1 and lgn_1 loci.

Fourteen nonallelic lethal CD recessive mutants were referred to by Burton (1986). These were derived from treating seed of inbred Tift 23B with thermal neutrons and EMS (Burton and Powell, 1969). These recessive CD mutants included white (a total of 3), pale yellow, dwarf pale yellow, light yellow (2), deep yellow, very deep yellow, greenish yellow, variable green yellow, and greenish medium yellow (3).

Koduru and Krishna Rao (1980) estimated the quantity of chlorophyll *a* and *b* in the CD mutants and observed that these were less than in the corresponding controls and that reduced quantities were associated with defective chloroplasts. They also reported that CD mutants differed from their controls in leaf flavonoids. Most of these CD mutants showed a higher survival rate in the rainy season than in winter season, indicating that a higher mean day temperature and longer day light period are favorable.

Nonlethal and near-normal CD mutants have been used in examining their contribution to heterosis and as markers in genetic studies. Burton et al. (1980) used >100 induced mutants reported by Hanna et al. (1978) to determine if the forage yields of F_1 hybrids between normal inbreds and their simply inherited mutants exhibit hybrid vigor. They concluded that simply inherited mutants capable of showing high heterotic yields occur at a low frequency and that intermating unrelated inbreds is a more efficient route to developing high-yielding forage hybrids. Burton (1986) used the CD mutant golden yellow (*g*) and 14 recessive lethal CD mutants to identify heterotic blocks associated with four quantitative characters and green forage yields. Nonlethal CD inbreds could be

used in determining effects of selfed seed on yields in hybrids that involve inbred × inbred or inbred × variety crosses. This possibility was elegantly demonstrated by Burton (1989), using inbreds with yellow and red plant color.

FOLIAGE STRIPING

Ratnaswamy (1960) reported a variegated (striped) phenotype caused by a plastid mutation controlled by a recessive gene, with maternal transmission of the white plastids. Gill et al. (1969) reported that the traits yellow striping in purple foliage and yellow striping in green foliage are controlled by three complementary recessive genes (gys_1, gys_2, gys_3), while the trait white and yellow stripes in green foliage is distinguished from yellow striping by one set of genes, and the genotypes have one of the above three genes in a dominant state.

Investigations during the last 25 yr have shown that the widespread assumption that plastids and mitichondria are maternally inherited traits in plants is not correct. Pearl millet is one of the two monocotyledonous species that exhibit relatively regular biparental inheritance (>5% of progenies display paternal transmission of plastids), as opposed to uniparental maternal inheritance of plastids (Smith, 1989).

Biparental plastid inheritance in pearl millet has been reported by Krishna Rao and Koduru (1978a). A natural CD phenotype, white striping$_3$, exhibited narrow white to pale yellow stripes on leaves and sheath and the earheads were yellow to purple pink. Progenies of the reciprocal crosses between striped tillers and green plants demonstrated biparental inheritance of the striped phenotype. In most progenies, the maternal phenotype was most frequent, suggesting maternal dominance. The authors suggested that spontaneous mutation of chloroplasts and sorting out of the mutant plastids from the wild type were responsible for the striped phenotype.

Appa Rao and Mengesha (1984) reported a nonlethal CD mutant, which they named stripe. The stripe plants exhibited longitudinal yellow stripes alternating with green stripes on leaf blades, leaf sheaths, stems, peduncles, inflorescences, and spikelets. They produced yellow, green, and stripe plants (classification depending on the number and size of the yellow stripes on the leaf blade). Segregation data from crosses between green × stripe plants did not fit any Mendelian ratio. In crosses between striped plants possessing green spikelets × striped plants with yellow spikelets, the F_1 seedlings were green, while in the reciprocal they were both yellow and green. The yellow seedlings were lethal, and the segregation data in the F_2 indicated that yellow spikelet color is monogenic recessive to green spikelet color. They considered these stripe plants as sectorial chimeras, because genetically distinct yellow and green sectors are adjacent to each other, and proposed the gene symbol *sp* for this trait. Subrahmanyam et al. (1986) demonstrated a pattern of dependent maternal plastid inheritance for this mutant trait.

Reddy and Subrahmanyam (1988) reported the genetic basis of plastid alterations and their mode of transmission in the stripe mutant described above. In reciprocal crosses of striped leaf mutant with normal inbreds, they observed that the mutant phenotype carries a recessive nuclear gene, designated *vi* for abnormal plastid development. The gene *vi* showed variable penetrance and expressivity. Intraplant and interspikelet crosses disclosed maternal plastid transmission. Green and yellow progeny were obtained on crossing striped × normal inbreds; selfing striped plants or crossing them with green sibs produced yellow, striped, and green progeny. The authors suggested that, in egg cells with defective plastids, the plastids do not revert back in spite of inheriting a dominant allele from the pollen parent, while in egg cells with a mixture of green and yellow plastids, the yellow plastids could become functional under the influence of a dominant allele.

LEAF CHARACTERS

Foliar mutants normally affect the midrib, and a weak midrib leads to drooping leaves. In extreme cases, the midrib is completely absent and the plants are sterile. Krishnaswamy and Rangaswami Ayyangar (1942) reported plants with weak midribs, the leaves of which were drooping down instead of being held horizontally. They observed 13 normal : 3 mutant plants in the F_2 families, suggesting the presence of an inhibitor controlling the expression of the mutant gene. Singh et al. (1968) observed that the drooping lamina results from the involvement of two dominant genes with complementary interaction. Desai et al. (1959) reported a narrow leaf mutant characterized by small, narrow, thick, and deep green leaves. Earheads were short and bore spikelets on the opposite sides only. This trait is controlled by a single recessive gene. Appa Rao et al. (1988a) reported three spontaneous midribless mutants after screening >17 000 germplasm accessions. These midribless mutants were characterized by leafblades that tended to droop because of absence of a keel in the midrib portion of the leaf lamina. Seed set was drastically reduced in two mutants, as both the gynoecium and androecium were affected. Inheritance studies indicated that this character is controlled by a single recessive gene in each of these three mutants. Two of the mutants carried the same gene, designated mrl_1, and the third carried a different gene, designated mrl_2. The mrl_1 gene showed independent assortment with three seedling traits: bright yellow (*byby*), glossy (*glgl*), and trichomeless (*trtr*).

Appa Rao et al. (1987) reported a seedling marker, which they named glossy, in individual plants of eight germplasm accessions. The glossy plants were clearly distinguishable from emergence up to 4 wk post emergence by their shiny light-green foliage. Both the upper and lower leaf blade surfaces and leaf sheaths exhibited this trait. Hairs on leaf blades and sheaths, barbs along leaf margins, and short hairs and claws on veins on both upper and lower leaf surfaces were present on glossy lines; hence, glossiness was not due to the absence of trichomes. Water accumulated as droplets on glossy leaves, but not on normal leaves. The glossy character is inherited as a monogenic recessive. Intercrosses among the eight glossy lines indicated that three different genes control this trait. These were assigned gene symbols gl_1, gl_2, and gl_3. Genes controlling purple plant color (*P*), long bristles (*Br*), and trichomeless (*tr*) are independent of gl_1 and gl_2.

Cherney et al. (1988) induced a brown midrib mutant (*bmr*) by treating pearl millet inbreds with EMS or DES. In the M_2 generation, a single brown midrib phenotype

Table 1. Gene symbols and genetic control of morphological mutant phenotypes reported in pearl millet, 1934 to 1991.

Character or mutant phenotype	Gene symbol	Genetic effect†	References
		Chlorophyll deficiencies (CD)‡	
Albina or white	*w*	1 r	Rangaswami Ayyangar, 1934 Rangaswami Ayyangar and Hariharan, 1935 Kadam et al., 1940 Krishnaswamy and Rangaswami Ayyangar, 1941 Al-Fakhry et al., 1965 Burton and Powell, 1965 Koduru and Krishna Rao, 1980 Burton, 1986 Dev et al., 1987
Albino-terminalis	*at*	1 r	Appa Rao et al., 1984
Patchy white	*pw*	1 r	Koduru and Krishna Rao, 1980
White virescent	*wv*	1 r	Koduru and Krishna Rao, 1980
Virescent white		2Dde	Kadam et al., 1940
White-tipped green	*wt*	1 r	Koduru and Krishna Rao, 1980
Light green		2 r	Al-Fakhry et al., 1965
	lgn_1	1 r	Hanna et al., 1978
	lg_1 lg_2	2 r	Koduru and Krishna Rao, 1980
Chlorina	*c*	1 r	Koduru and Krishna Rao, 1980
Chlorina virescens	*chv*	1 r	Appa Rao et al., 1984
Pale green		2 re	Rangaswami Ayyangar, 1934 Rangaswami Ayyangar and Hariharan, 1935
		1 r	Krishnaswamy, 1962
Yellow green	yg_1 yg_2	2 r	Koduru and Krishna Rao, 1980
Greenish yellow	*gy*	1 r	Appa Rao et al., 1984 Burton, 1986
Green yellow		1 r	Burton, 1986
Greenish medium yellow		1 r	Burton, 1986
Pale green yellow	*pgw*	1 r	Burton and Powell, 1965
Pale yellow	*py*	1 r	Burton and Powell, 1965 Appa Rao et al., 1984 Burton, 1986
Pale yellow 2	*py*		Burton and Powell, 1965
Light yellow		1 r	Burton, 1986
Medium yellow	*my*	1 r	Burton and Powell, 1965
Yellow	*y*	1 r	Burton and Powell, 1965
		1,2 r	Kadam et al., 1940
		1 r	Al-Fakhry et al., 1965
	yn_1	1 r	Hanna et al., 1978
Bright yellow	*by*	1 r	Appa Rao et al., 1984
Deep yellow		1 r	Burton, 1986
Very deep yellow		1 r	Burton, 1986
Virescent yellow		1,2 r	Kadam et al., 1940
		1 r	Al-Fakhry et al., 1965
Yellow virescent	*yv*	1 r	Koduru and Krishna Rao, 1980 Dev et al., 1987
Dwarf pale yellow		1 r	Burton, 1986
Golden yellow		1 r	Kadam et al., 1940
Golden	*g*	1 r	Burton, 1986
Zebra	*z*	1,2 r	Kadam et al., 1940
		2 r	Al-Fakhry et al., 1965
		1 r	Appa Rao et al., 1984
		1 D	Werner and Burton, 1991
Zebra virescens	*zv*	1 r	Appa Rao et al., 1984
White striping$_1$	wst_1	1 r	Koduru and Krishna Rao, 1980
White striping$_2$	wst_2	1 r	Koduru and Krishna Rao, 1980
White striping$_4$	wst_4	1 r	Koduru and Krishna Rao, 1980
Yellow striping	yst_1 yst_2	2 r	Koduru and Krishna Rao, 1980
Fine striping	*fst*	1 r	Koduru and Krishna Rao, 1980
Bleached leaf	*bl*	1 r	Appa Rao et al., 1990
White sheath	*ws*	1 r	Appa Rao et al., 1990
		Foliage striping	
Striping (variegated)		1 r	Ratnaswamy, 1960
Yellow striping in purple foliage	gys_1 gys_2 gys_3	3 r	Gill et al., 1969
Yellow striping in green foliage	gys_1 gys_2 gys_3	3 r	Gill et al., 1969
White striping$_3$		Plastid inheritance	Krishna Rao and Koduru, 1978a
Stripe	*sp*	Sectorial chimera	Appa Rao and Mengesha, 1984
Abnormal plastid development	*vi*	1 r	Reddy and Subrahmanyam, 1988
		Leaf characters§	
Weak midribs		2 Dre	Krishnaswamy and Rangaswami Ayyangar, 1942
Drooping lamina		2 Dc	Singh et al., 1968
Narrow leaf		1 r	Desai et al., 1959
Midribless	mrl_1 mrl_2		Appa Rao et al., 1988a
Glossy	gl_1 gl_2 gl_3	1 r	Appa Rao et al., 1987

(continued next page)

Table 1. *Continued.*

Character or mutant phenotype	Gene symbol	Genetic effect†	References
		Pubescent mutants	
Hairy leaf	*hl*	1 r	Singh et al., 1967 Burton and Powell, 1968 Singh et al., 1968 Gill et al., 1971 Khan and Bakshi, 1976 Krishna Rao and Koduru, 1979 Appa Rao et al., 1988c
Hairy node	*Hn*	1 D	Gill et al., 1971
Hairy lamina	*hl*	1 r	Krishna Rao and Koduru, 1979
Hairy leaf sheaths and blades		1 r	Appa Rao et al., 1988c
Hairy leaf surface	*Hr*	2 Dre	Lal and Singh, 1971
Hairy sheath	*hs*	1 r	Krishna Rao and Koduru, 1979
Hairy stem	*hst*	1 r	Krishna Rao and Koduru, 1979
Hairy leaf margin	*Hm*	1 D	Krishna Rao and Koduru, 1979
Trichomeless	*tr*	1 r	Powell and Burton, 1971
		Plant form	
Wavy stem	*ws*	1 r	Krishna Rao and Koduru, 1979 Burton, 1981
Thick stem		1 D	Al-Fakhry et al., 1965
Dwarf plant stature	d_1	1 r	Burton and Fortson, 1966
Dwarf plant stature	d_2	1 r	Kadam et al., 1940 Burton and Fortson, 1966
Dwarf plant stature (other than d_2)		>2 r	Burton and Fortson, 1966
Dwarf plant stature	d_3	1 r	Appa Rao et al., 1986
Dwarf plant stature	d_4	1 r	Appa rao et al., 1986
Dwarf plant stature		2 Dc	Al-Fakhry et al., 1965
Semidwarf (translocation dwarf)		1 r	Koduru and Krishna Rao, 1984
Brachytic dwarf	d_b	2 re	Gupta et al., 1985
		Plant pigmentation	
Purple coleoptilar leaf		1 D	Yadav, 1976
Purple seedling base	Pb_1 Pb_2	2 Dc;	Koduru and Krishna Rao, 1979
Golden plant color		1 r	Burton, 1968
Red plant color		1 D	Burton, 1968
Purple plant color	*P*	1 D	Burton, 1968 Lal and Singh, 1971
Purple plant color	*PP*	1 D	Appa Rao et al., 1988c
Purple foliage	Pp_1 Pp_2	2 Dc	Athwal and Gill, 1966 Gill, 1969 Gill and Athwal, 1970
Purple midrib, margin, and sheath		1 D	Manga et al., 1988
Brownish leaf type-1		1 D	Al-Fakhry et al., 1965
Brownish leaf type-2		2 D	Al-Fakhry et al., 1965
Purple apicule		1 r	Manga et al., 1988
Purple leaf sheath		1ic	Singh et al., 1967
Purple leaf junction		1 D	Singh et al., 1968
Red stem	*Rp*	2 Dre	Gill, 1969
Purple stem	Ps_1 Ps_2	2 Dc;	Koduru and Krishna Rao, 1979
Purple internode		1 D	Al-Fakhry et al., 1965
		1 D	Manga et al., 1988
Purple (red) node	*Rn*	1 D	Krishnaswamy, 1962 Al-Fakhry et al., 1965 Singh et al., 1968 Koduru and Krishna Rao, 1979
	Pn_1 Pn_2	2 Dc	Appa Rao et al., 1988c Manga et al., 1988
Red node	Rn_1 Rn_2	2 Dc	Gill, 1969
Orange node	*on*	1 r	Degenhart et al., 1991
Purple node and auricle	*Pna*	1 D	Appa Rao et al., 1968c
Purple auricle	Par_1 Par_2	2 Dc	Manga et al., 1988
Yellow spikelets		1 r	Appa Rao and Mengesha, 1984
Purple glumes	Pg_1Pg_2	2 D	Gill, 1969
Purple glume tip	*pap*	1 r	Manga et al., 1988
Purple lemma, palea, bristle		1 D	Singh et al., 1967
Purple bristle	Bep_1 Bep_2	2 Dc	Gill, 1969
		1 D	Singh and Pandey, 1973
Purple anther		1 Dc	Athwal and Gill, 1966

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was isolated in the DES treatment. Lignin concentration in the *bmr* mutant was 40 g kg^{-1}, compared with 50 g kg^{-1} for the normal genotype, and IVDMD was 726 g kg^{-1} for the *bmr* mutant, compared with 659 g kg^{-1} for the normal. Cherney et al. (1990) reported in studies with wether sheep that dry matter digestibility, neutral-

Table 1. *Continued.*

Character or mutant phenotype	Gene symbol	Genetic effect†	References
		Earhead characters	
Exsertion of nodes from leaf sheath		1 D	Al-Fakhry et al., 1965
Exsertion of head from flag leaf		1 D	Al-Fakhry et al., 1965
Flag leaf interlocking		3 r	Singh et al., 1969
Curly spike	*Cl*	2 Dre	Singh and Pandey, 1972
Goose-neck peduncle		1 r	Krishnaswamy and Rangaswami Ayyangar, 1942
Complete sterility		2 Dde	Kadam et al., 1940
Spikeletless	*sl*	1 r	Rai et al., 1987
Basal branching		1 r	Rangaswami Ayyangar et al., 1935a
Branched earhead base	*Beb*	1 D	Gill and Athwal, 1970 Gill et al., 1971
Tapering earhead tip	Te_1Te_2	2 Dc	Gill and Athwal, 1970 Gill et al., 1971
Tufted earhead tip	*tet*	1 r	Gill and Athwal, 1970 Gill et al., 1971
Branched earhead tip	*bet*	1 r	Krishnaswamy and Rangaswami Ayyangar, 1942 Gill and Athwal, 1970 Gill et al., 1971
Bizarre earhead	be_1 be_2	2 rd	Dev et al., 1987
Curved earhead tip		1 D	Singh et al., 1968
		3 r	Singh et al., 1969
Naked earhead tip		2 Dre	Singh et al., 1969
	Net_1 Net_2	2 D	Gill et al., 1971
Bristling	*Br*	1 D	Rangaswami Ayyangar and Hariharan, 1936 Krishnaswamy, 1962 Ahluwalia and Shankar, 1964 Athwal and Gill, 1966 Lal and Singh, 1967 Gill and Athwal, 1970 Gill et al., 1971
		1 ic	Yadav, 1974a
		>1+	Appa Rao et al., 1988c
Semicompact earheads		1 D	Singh and Pandey, 1973
Floret-bearing bristles	*Fbb*+*Br*	1 D	Gill et al., 1971
Rough spike surface		2 Dre	Singh et al., 1969
Earhead tip sterility		2 Dc	Krishnaswamy and Rangaswami Ayyangar, 1942 Singh et al., 1969
		1 D	Singh et al., 1967 Singh et al., 1968
Gappiness in earheads		1 r	Krishnaswamy and Rangaswami Ayyangar, 1942
		Reproductive structures	
Multiple carpels		1 r	Manga, 1977
Pistilless		1 r	Pokhriyal et al., 1967
Large pollen grains		1 D/2 Dc	Verma et al., 1969
Dummy pollen		1 r	Rangaswami Ayyangar and Panduranga Rao, 1935
Stubby head		1 r	Hanna and Powell, 1973
		Sterility	
Cytoplasmic male sterility	*ms, ms, ms*	1 r	Burton and Athwal, 1967
Restoration of pollen fertility (A_1 cytoplasm)		2 Dc	Siebert, 1983
Restoration of pollen fertility (A_2 cytoplasm)		2 Dde	Siebert, 1983
Genetic male sterility		1 r	Krishnaswamy and Rangaswami Ayyangar, 1942 Gill et al., 1973
	ms_1 ms_2	1 r	Krishna Rao and Koduru, 1978a,b
Triggering of male-sterile genes, C-2 cytoplasm	R_1 R_2	2 Dde	Krishna Rao and Uma devi, 1989
Female sterility	*fs*	1 r	Hanna and Powell, 1974
		Seed characters¶	
Yellow grain color	*Y*	1 D	Patel, 1939 Burton and Powell, 1968 Lal and Singh, 1971 Madhava Rao and Kulliaswamy, 1975
Purple seed color		1 D	Al-Fakhry et al., 1965
		2 Dc	Al-Fakhry et al., 1965
Pearly amber grain		1 D	Khan and Bakshi, 1976
Yellow endosperm	*y*	1 r	Phul et al., 1969
Amber endosperm	y^a	1 r	Phul et al., 1969
Deep slate endosperm	y^{ds}	1 r	Phul et al., 1969
Light slate endosperm	y^s	1 r	Phul et al., 1969
Purple pericarp	*Prp*	1 D	Phul et al., 1969
Sugary grain	*su*	1 r	Patel, 1941

(continued next page)

Table 1. *Continued.*

Character or mutant phenotype	Gene symbol	Genetic effect†	References
		Earliness and maturity#	
Early maturity	e_1	1 r	Burton, 1981
Early maturity	e_2	1 r	Hanna and Burton, 1985
		Resistance to diseases	
Downy mildew resistance		1/2 D	Appadurai et al., 1975
	DM_1 DM_2	2 Dde	Gill et al., 1978
Smut resistance		1 ic	
		1 D	
		2 Dde	Yadav, 1974b
Rust resistance	Rpp_1	1 D	Andrews et al., 1985
	Rr_1	1 D	Hanna et al., 1985
		1 D	Sokhi et al., 1987
		2 Dc	Sokhi et al., 1987
Pyricularia grisea resistance		2 D	Wilson et al., 1989
		3D	Hanna and Wells, 1989
Bipolaris setariae resistance	Bp_1 Bp_2 bp_3 bp_4	4 gs	Wells and Hanna, 1987, 1988
		Gamete formation	
Desynapsis	*ds*	1 r	Minocha et al., 1975 Pantulu and Rao, 1976 Subba Rao, 1980
Desynapsis and fragmentation	*ds ds*	2 rd	Lakshmi et al., 1979
Multiploid sporocytes	*mu*	1 r	Pantulu and Manga, 1971

† 1 r = single recessive gene; 2 r = two recessive genes; 3 r = three recessive genes; 1 D = single dominant gene; 2 D = two dominant genes; 3 D = three dominant genes; 1 ic = single gene, incomplete dominance; 2 Dc = two dominant genes with complementary action; 2 rd = two recessive duplicate genes; >1+ = more than one gene with additive action; 2 re = two genes, recessive epistasis; 2 Dde = two genes, duplicate dominant epistasis; 2 Dre = two genes dominant and recessive epistasis; 4 gs = two duplicate genes, 1 inhibitory gene, 1 antiinhibitory gene.
‡ Other CDs reported include: maculata (Chandola et al., 1963; Tara Mohan et al., 1973); striata (Chandola et al., 1963; Joshi, 1968; Tara Mohan et al., 1973); viridis (Tara Mohan et al., 1973; Singh et al., 1978); xantha (Chandola et al., 1963; Tara Mohan et al., 1973; Singh et al., 1978); and virido-albina, xantha-alba, and tigrina (Chandola et al., 1963).
§ Brown-midrib trait (Cherney et al., 1988) not included, as genetics of the trait not worked out.
¶ Endosperm colors described by Phul et al. (1969) form multiple allelic series.
# Response to photoperiod (Burton, 1966) and studies on earliness and lateness of landraces (Bilquez, 1963; Bilquez and Clement, 1969; Bharadwaj and Webster, 1971; Appa Rao et al., 1988b) not included.

detergent fiber digestibility, and acid-detergent fiber digestibility were consistently higher in the *bmr* genotype than in the normal genotype. The nature of inheritance and any pleiotropic effects of this trait need to be studied, particularly as this trait has an excellent potential for improving the quality of forage pearl millet.

Degenhart et al. (1991) identified an orange node trait (*on*), conditioned by a single recessive gene, that resembles the brown midrib trait. A preliminary evaluation indicated that earhead, stem, and leaf sheath components are more digestible in the *onon* plants than in normal plants, and that leaf blade digestibility did not differ; however, *onon* plants yielded less dry matter than *ONON* or *ONon* plants. Results from crosses between orange node × brown midrib traits indicated that both traits are affected by the same gene.

PUBESCENCE

From the seedling stage onward, pearl millet has hairiness (pubescence) on several plant parts. Hairiness in leaves, particularly in seedling leaves, can be easily recognized and is useful as a genetic marker; however, there is considerable variation for hairiness both within a plant and between landrace accessions. Burton and Powell (1968) discerned that pearl millet plants may (i) be completely smooth, (ii) have only hairy nodes, (iii) have hairy nodes and leaves, or (iv) have hairy nodes, leaves, and stems. They found that smooth plant parts are dominant over hairy parts, with F_2 populations segregating in a 3 smooth : 1 hairy ratio.

Singh et al. (1967) reported that smooth leaf character is dominant over hairy leaf and is controlled by a single gene. Identical results, indicating that leaf hairiness is controlled by a single recessive gene, were reported by Burton and Powell (1968), Singh et al. (1968), and Khan and Bakshi (1976). Gill et al. (1971) proposed the symbol *hl* for hairy leaf, which is characterized by dense hairs on both surfaces of the lamina from the early seedling stage onward. They also observed that hairy node (*Hn*) is dominant over nonhairy node and is controlled by a single gene. In crosses between plants with hairy node (bristlelike, long stiff hairs in whorls at the nodes) and plants with hairless node, monofactorial dominant inheritance of the hairy node has also been observed (Krishna Rao and Koduru, 1979; Appa Rao et al., 1988c). Hairy node (*Hn*) and hairy leaf (*hl*) segregated independently. Krishna Rao and Koduru (1979) reported that hairy lamina (*hl*) is inherited as a monogenic recessive. Appa Rao et al. (1988c) observed that hairy leaf sheaths and blades were monogenic recessive to glabrous leaf sheaths and blades. Al-Fakhry et al. (1965) reported that hairy ligule is controlled by two dominant genes.

In contrast to these studies, Lal and Singh (1971) observed that F_2 populations of scabrous leaf surface × hairy leaf surface inbred segregated in the ratio of 13 scabrous : 3 hairy types. A factor *Hr* was responsible for lamina hairiness, and its allele, *hr*, was responsible for scabrous lamina. A nonallelic inhibitor, *I*, inhibited the expression of *Hr*. No linkage was observed between the genes for purple plant pigmentation (*P*), *Hr*, and spike bristling *Br*.

Krishna Rao and Koduru (1979) made a detailed study of five hairy phenotypes (including hairy lamina, mentioned above). In crosses between plants with hairy lamina, hairy sheath, hairy stem, hairless (glabrous) sheath, and hairy node, they observed that glabrous sheath is dominant over hairy sheath and that the presence of hairs on the lamina and sheath is controlled by different nonallelic genes. Results indicated that hairy sheath is inherited as a monofactorial recessive. They proposed the symbol *hs* for this trait. In hairy stem (*hst*), dense hairs are present on the exposed internodes, with increased density toward the node. This phenotype was found to be controlled by a single recessive gene. For the trait hairy margin, the leaves have long, bristlelike hairs, with a bulbous base along the margin of the basal part of the lamina. Hairy leaf margin (*Hm*) was observed to be controlled by a single dominant gene. The gene for hairy lamina (*hl*) showed independent assortment from the gene for hairy node (*Hn*) and showed linkage with hairy stem (*hst*), hairy sheath (*hs*), and hairy leaf margin (*Hm*). The gene *Hl* was observed to have an epistatic effect on the expression of *hs*. The recombination percentages between the gene pairs *hl–hst*, *hl–hs*, and *hl–Hm* were 0, 8.3, and 19.8, respectively. The genes were assigned to one linkage group, *hl–hst–hs–Hm*.

Of all the genes conditioning leaf pubescence, the trichomeless (*tr*) gene reported by Powell and Burton (1971) has been most investigated and utilized. They observed a trichomeless mutant in the pubescent pearl millet inbred Tift 23B. When homozygous, this single recessive gene not only suppresses all trichomes, stigma branches, and tufts of hairs normally present on the apex of anthers, and imparts a smooth waxlike surface to the cuticle, but also affects several other characters.

Comparing near-isogenic Tift 23S (*trtr*) with pubescent Tift 23H (*TrTr*), Burton et al. (1977) found that the *tr* gene reduces transpiration without altering stomatal length and frequency. The *tr* gene imparts a smooth waxlike surface to the cuticle, which causes the leaf blades to accumulate and retain dew longer, thus increasing the rate at which rust developed on them. It significantly reduced oviposition by the fall armyworm, *Spodoptera frugiperda* (J.E. Smith), and the corn earworm, *Helicoverpa zea* (Boddie) (syn. *Heliothis zea*), and reduced foliar feeding of first- and second-instar larvae of *S. frugiperda* fourfold. Hanna et al. (1974) found that normal leaves with trichomes were digested more quickly in rumen fluid than were trichomeless leaves. Hanna and Akin (1978) attributed the reduced penetration of rumen microbes and reduced transpiration to a 1.3- and 3-fold reduction, respectively, in the number of cuticle cracks on the upper and lower surfaces of the trichomeless leaves. The *tr* gene increased palatability of forage for cattle and favored increased intake in the first few days in a green feeding test. It did not increase overall livestock gains, because the slower digestion rate of *tr* forage slowed its passage through the gastrointestinal tract and reduced intake, thus countering its greater palatability. The loss of IVDMD associated with the *tr* gene tends to be compensated by associated drought tolerance and better palatability (Burton et al., 1977, 1988). Based on results of forage and dry matter production and associated beneficial effects of the *tr* gene, they concluded that the *tr* gene can be used to increase forage yields.

PLANT FORM

Al-Fakhry et al. (1965) found that thick stems are dominant over thin stems, and a single recessive gene is involved. The stems of pearl millet are erect and straight. Koduru and Krishna Rao (1979) observed in the wavy stem (*ws*) mutant that the internodes arch slightly at the nodes in an opposite direction at successive internodes, producing a zig-zag appearance of the stem; this trait is accentuated at the basal part of the stem and is inherited as a monogenic recessive. Burton (1981) reported that an early-maturing plant from the cultivar Katherine had one or two zig-zag stems that fell over before the seeds were mature. He noted linkage between genes for earliness and the spindly character was so close that "only after repeated extensive efforts were we able to separate them."

Dwarfing genes have been successfully utilized in wheat, rice, barley, and sorghum in the breeding of improved dwarf varieties. Dwarf varieties offer the advantages of reduced lodging, improved harvest index, better leaf-to-stem ratio, and easier mechanical harvesting. Dwarf plants in pearl millet were discovered almost simultaneously in the USA and India.

Kadam et al. (1940) described dwarf plants in a local inbred pearl millet that showed shorter internodes, overlapping leaf sheaths, and poor exsertion of the earhead as a result of reduced peduncle length. They ascribed the occurrence of these recessive dwarf-type plants as probably due to mutation.

In 1940, in the progeny of one of the five tall and late-maturity introductions from Russia, an unusually short plant with a mass of leaves due to extremely shortened internodes was isolated (Burton and DeVane, 1951). This plant bred true, but was not productive enough to have practical value. This short line was crossed with a broad-leafed, highly palatable, common pearl millet inbred to combine desired qualities of both lines. Plants carrying the favorable characters were combined to form a synthetic variety called Starr (Hein, 1953); this synthetic variety was <1.8 m in height, due to its shorter internodes. Though Burton and DeVane (1951) did not use the term dwarf for the lines or the synthetic, their description satisfactorily fits dwarf genotypes.

Burton and Fortson (1966) reported the inheritance of five different dwarfs, named D_1 to D_5, in pearl millet. Dwarfness in D_1 and D_2 is controlled largely by one or two recessive genes. When transferred to a near-isogenic background, dwarfness in D_1 and D_2 is controlled by single but different recessive genes, designated as d_1 and d_2. In lines D_3, D_4, and D_5, near-normal F_2 distribution curves and minimum-gene-number estimates suggest that dwarfness in these three lines is controlled by more than two recessive genes. The d_2 dwarf gene in pearl millet has several pleiotropic effects on the plant phenotype.

Principally, it reduces plant height by 50% through a reduction in the length of the stem internode, except the peduncle (Burton and Fortson, 1966), leading to higher proportion of leaves. The leaves have a higher digestible nutrient content than the stems (Johnson et al., 1968). The higher leaf percentage of the dwarf significantly increases the IVDMD (Hanna et al., 1979). Although dwarfs yield less dry matter than their tall counterparts in clipping trials, they yield more animal products in grazing and feeding experiments (Burton et al., 1969). Based on the composition and digestibility of dwarf millet forage, Johnson et al. (1968) concluded that the d_2 gene could be used in improving the nutritive value of forage millets.

Soman et al. (1989) studied the effect of the d_2 gene on the length of the coleoptile, mesocotyl, and plumule in pairs of isogenic tall and dwarf inbreds and hybrids. They found that, although the culm length differed significantly between the tall and dwarf genotypes, the d_2 gene did not affect coleoptile or mesocotyl length, as reported for dwarfing genes in other cereals. Tongoona et al. (1984), using near-isogenic pairs differing in plant height, investigated the effect of the d_2 gene on root development. They observed that the overall mean root-to-shoot ratio of the dwarf hybrids was greater than that of tall hybrids, and in two out of three comparisons the dwarf line had a higher root-to-shoot ratio at two or more harvests. They concluded that there was no relationship between plant height and root development and that in certain genetic backgrounds the dwarfing gene is, among its other pleiotropic effects, associated with a large root system.

Rai and Hanna (1990) studied the effects of the d_2 gene on several morphological characters by comparing tall and dwarf near-isogenic lines. Results indicated that the dwarf isogenic lines were shorter than their tall counterparts, but had larger peduncles, longer and narrower panicles, thicker culms, wider leaves, and smaller seeds. Differences for days to 50% bloom, total and effective tillers plant^{-1}, and leaf sheath length were nonsignificant or were inconsistent across locations. Bidinger and Raju (1990), comparing tall and dwarf near-isogenic hybrids, found that dwarf hybrids on an average yielded less (by 9%) than their tall counterparts because of smaller average seed size (8.40 mg grain^{-1} for talls vs. 7.51 for the dwarfs). The inferior ability of the dwarfs to fill grains was indicated by a grain number per unit area similar to that of the tall hybrids. Both these reports concluded that the d_2 gene could be used to advantage by incorporating it into diverse genetic backgrounds and developing specific parental combinations.

Because it can be introduced into normal tall backgrounds through backcrossing, the d_2 gene has been extensively used in the development of dwarf open-pollinated varieties, male steriles, and hybrids in the USA, India, and Africa. In the USA, this gene has been used in the development of several male steriles, inbred lines, and dwarf hybrids (Burton and Powell, 1968; Burton. 1983). In India, Bakshi et al. (1966) incorporated the d_2 gene into tall phenotypes and derived a dwarf-statured inbred that yielded >2 t ha^{-1} of grain. From West Africa, Chantereau and Etasse (1976) described the development, using this gene with three local tall landraces, of three dwarf populations, 3/4 Souna, 3/4 Haini-Kirei, and 3/4 Ex Bornu (with 3/4 indicating the first backcross stage in which dwarf F_2 plants are constituted into a population).

Appa Rao et al. (1986) described new sources of dwarfing genes in pearl millet. Thirteen naturally occurring dwarf lines identified in the world collection were tall when crossed with a tall inbred, indicating that dwarfness is a recessive trait. The height differences in three dwarfs were controlled by more than one gene, as they showed continuous variation for plant height in the F_2. In 10 crosses, the F_2 segregation ratio was 3 tall : 1 dwarf, indicating that dwarfness is controlled by a single recessive gene. When these dwarfs were crossed with either d_1 or d_2 dwarfs (Burton and Fortson, 1966), only two crosses produced tall F_1 hybrids and segregated in the F_2, indicating that they were nonallelic to d_1 and d_2. Reciprocal crosses of these two dwarfs produced tall F_1 hybrids and showed a dihybrid segregation of 9:3:4 in the F_2, showing that the factors for dwarfing in these two parents were nonallelic. They were assigned the gene symbols d_3 and d_4. Some of these dwarf sources have desirable attributes, such as early maturity, long earheads, dense bristles, and abundant and synchronous tillering.

In a study reported by Al-Fakhry et al. (1965), complementary genes (9 dwarf : 7 nondwarf) best accounted for the data on the inheritance of dwarfism; however, this could have been due to the small population size (30 plants) used in the F_2 generation. Koduru and Krishna Rao (1984) isolated a semidwarf designated TLD (translocation dwarf) as a segregate in the selfed progeny of a spontaneously produced interchange heterozygote. This mutant was characterized by dark green, erect, and narrow leaves, early maturity, and a sterile earhead tip. The number of internodes in the mutant was almost equal to that of the tall parent, and the internode length was reduced. The mutant was shown to be an interchange homozygote. The homozygosity for the interchanged chromosomes and the mutant phenotype were tightly linked and the chromosomes involved were identified as 3 and 6. This phenotype is inherited as a monogenic recessive. They also reported that the viability of the gametes carrying the interchange chromosomes and of the zygotes of the three classes was normal. Krishna Rao et al. (1981) found that both this semidwarf phenotype and Tift 23DB (d_2) are defective in the utilization of gibberellic acid. They observed that coleoptile length and mature plant height were not correlated in either dwarf, suggesting that the factors controlling the two phenotypes are not identical.

Dwarf phenotypes have also been induced in pearl millet. Venkateswarulu and Mani (1973) reported induction by MES of two true-breeding dwarf mutants, one with long and thick earheads and synchronous tillering and the other with narrow and erect leaves and thin and long earheads. Gupta et al. (1982) reported isolation of monogenic recessive brachytic dwarf mutants following treatments with EMS. One of these mutants, JMB-D-24-3-1, was found to be promising, owing to its profuse tillering, high number of nodes, dark and broad green leaves, and high leaf-to-stem ratio (Gupta and Premachandran, 1983). In crosses involving JMB-D-24-3-1 × ICMB 81, a downy mildew resistant d_2 dwarf mutant induced in Tift 23DB (Anand Kumar and Andrews, 1984)

segregated in the F_2 generation, which suggests that recessive epistasis among two nonallelic genes was involved, resulting in three discrete classes: tall, d_2 type, and brachytic dwarf type in a 9:3:4 ratio. This indicates that the gene conditioning brachytic dwarfism is at a different locus than the d_2 gene, and a different gene symbol (d_b) was assigned to this trait (Gupta et al., 1985). Analysis of allelic relationships of seven dwarf mutants obtained by gamma irradiation and chemical mutagenesis revealed each of them to be controlled by a single recessive gene (Sukhadev et al., 1987). Six of these dwarfs were allelic to the d_2 gene, and the seventh was allelic to d_1.

PIGMENTATION

In pearl millet, pigmentation on several plant parts occurs. Figure 1 illustrates the different plant parts in which CDs and plant pigmentation occur.

Inheritance of purple pigmentation of the coleoptilar leaf was reported to be controlled by a single dominant gene (Yadav, 1976). Koduru and Krishna Rao (1979) reported that the purple seedling base, which is recognizable from the 1-leaf or 2-leaf stage, is controlled by the complementary interaction of two dominant genes, Pb_1 and Pb_2.

Burton and Powell (1968) mentioned genetic stocks that exhibited a wide range of plant pigmentation (golden, green, red, and purple) in most of the plant parts. Studies indicate that green is dominant over golden and recessive to red and purple. Segregation in the F_2 generation suggests that each of these traits is conditioned by a single gene. From studies involving green × purple plants, Lal and Singh (1971) also reported that purple plant pigmentation is controlled by a single dominant gene; they assigned the gene symbol *P* for purple plant pigmentation. Appa Rao et al. (1988c) reported that purple pigmentation on all plant parts is controlled by a single dominant gene. They observed that purple coloration of leaf sheaths, leaf blades, internodes, bristles, and glumes is inherited as a single unit, indicating pleiotropic effect of a single gene. They proposed the symbol *PP* for this trait.

Based on observations on several genetic stocks, Gill (1969) reported that plants showing anthocyanin pigmentation on any part invariably exhibited purple involucre, indicating that genes for purple involucre are primary anthocyanin genes and that localization of pigmentation in other plant parts is probably controlled by an additional set of genes. Athwal and Gill (1966) and Gill and Athwal (1970) reported that purple foliage is under the control of two dominant complementary genes, to which Gill (1969) assigned the gene symbols Pp_1 and Pp_2. Gill and Athwal (1970) observed that purple foliage and branched eartip are inherited independently. Al-Fakhry et al. (1965) mentioned two brownish leaf types controlled by a single dominant gene and two dominant genes, respectively.

Singh et al. (1967) observed that purple leaf sheath is governed by a single gene exhibiting incomplete dominance. Singh et al. (1968) reported that the trait purple leaf junction (auricle) is dominant over white leaf junction and is controlled by a single gene.

Gill (1969) observed that red pigmentation of the stem behaves as a recessive character. Red stem pigmentation is due to the presence of a gene *Rp*, which is inhibited by a nonallelic gene *I*. Koduru and Krishna Rao (1979) reported two dominant complementary genes as responsible for purple stem trait (Ps_1 and Ps_2).

Al-Fakhry et al. (1965) reported that purple node and

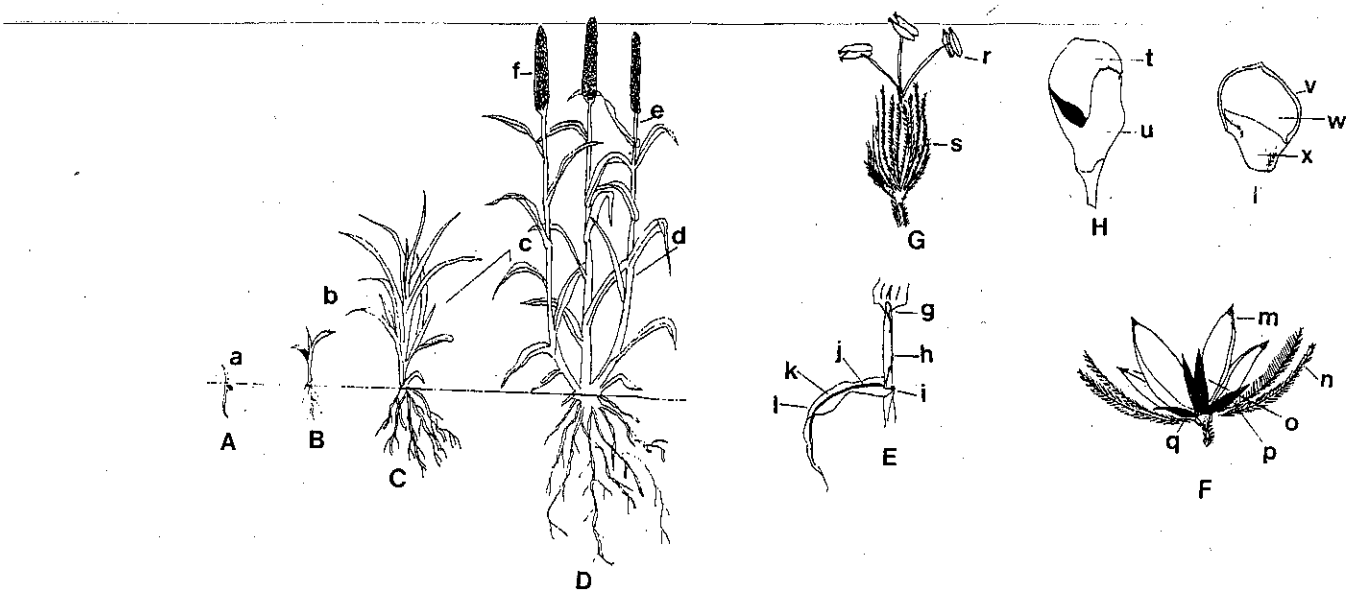

Fig. 1. Diagrammatic stages of the life cycle of a pearl millet plant, illustrating plant parts in which chlorophyll deficiencies and pigmentation can occur. A = seedling, B & C = vegetative stages; D = adult plant; E = part of stem; F = spikelet; G = single flower; H = spikelet and grain; I = grain; a = coleoptile; b = foliage at vegetative stage; c = foliage at vegetative and adult plant stage; d = stem; e = peduncle; f = earhead; g = ligule; h = leaf sheath; i = node; j = midrib; k = leaf blade; l = leaf margin; m = upper perfect flower; n = bristles subtending group of spikelets; o = upper glume; p = lower staminate flower; q = lower glume; r = penicilled anther; s = group of two spikelets subtended by several bristles; t = grain; u = glume; v = pericarp; w = endosperm; x = embryo.

purple internode traits are inherited as simple dominant traits. Singh et al. (1968) reported that purple stem nodes are dominant over normal green nodes, and that this trait is controlled by a single gene. Purple node (red node) is governed by a dominant gene (*Rn*) (Krishnaswamy, 1962; Koduru and Krishna Rao, 1979). Gill (1969) reported that red nodes are controlled by two dominant complementary genes (Rn_1 and Rn_2). Appa Rao et al. (1988c) observed complementary gene interaction for purple node color (9 purple : 7 green) and proposed the gene symbols Pn_1 and Pn_2. Purple nodes and auricles were inherited as a monogenic dominant trait to green nodes and auricles. As this trait was different from those reported by Koduru and Krishna Rao (1978) and Gill (1969), the gene symbol *Pna* was proposed (Appa Rao et al., 1988c).

Singh et al. (1967) reported that the purple color of lemma, palea, and bristles (scored together) is a monogenic dominant trait. Gill (1969) reported that purple bristling (Bep_1 and Bep_2) and purple glume (Pg_1 and Pg_2) traits are controlled by two sets of dominant complementary genes. They explained the observed monogenic segregation in some crosses of purple foliage, purple glumes, and red nodes on the supposition that the corresponding testers were carrying one of the two factors as a homozygous dominant. Singh and Pandey (1973) observed that purple bristle is inherited as a simple dominant trait over white bristle. Minocha et al. (1980) mentioned that purple anther color is dominant over green/yellow colored anthers and is controlled by two complementary genes (Athwal and Gill, 1966).

Manga et al. (1988) studied the mode of inheritance and linkage relationships of genes governing purple coloration of seven plant parts. The F_2 segregation data for purple node (Pn_1Pn_2; similar to Appa Rao et al., 1988c) and purple auricle (Par_1Par_2) showed a good fit to a 9:7 ratio, indicating that these two characters are governed by a pair of complementary genes. Purple apicule is controlled by a single recessive gene (*pap*) and purple internode by a single dominant gene (Ps_1). Purple midrib, leaf margin, and leaf sheath are inherited together and are under the control of a single dominant gene. Linkage was observed between (i) purple node and purple auricle and (ii) purple midrib, margin, and sheath with both purple internode and purple apicule.

EARHEAD CHARACTERS

The earhead (inflorescence) of pearl millet is a compact cylindrical spike that varies in landraces from 4 cm to 2 m long. Earhead shapes also differ greatly, and range from lanceolate, cylindrical, conical, club, and fusiform to globular types (IBPGR, 1981). The involucre is borne on a stalk and has a cluster of bristles that are generally inconspicuous in mature earheads. Sometimes these bristles may exceed the spikelet length, giving the appearance of a cat's tail. The spikelets are 4 to 7 mm long in each involucre and range from one to five in number, but usually occur in pairs, with a sessile male and a bisexual floret with a short pedicel (Burton and Powell, 1968). Variation exists for head circumference, density and length of bristles, peduncle length, and grain size, color, and shape. Singh and Pandey (1973) observed that the semicompact earhead trait is dominant over compact earhead and is controlled by a single gene.

Al-Fakhry et al. (1965) observed that exsertion of nodes from the leaf sheath and of head from the flag leaf are controlled by single dominant genes. In pearl millet, the sheath of the boot leaf is generally longer than the lamina. Singh et al. (1969) observed plants in which the flag leaf was interlocked with the laminae of the lower leaves. They reported that this flagleaf interlocking trait is controlled by three recessive genes.

Singh and Pandey (1972) described curly spike, where whole spikes were curved and emerged late from the boot leaf. In the F_2 population, a ratio of 13 normal : 3 curly spike plants was recorded, suggesting that an inhibitor *I* inhibits the expression of the dominant curly gene *Cl*.

Krishnaswamy and Ayyangar (1942) observed plants with goose-necking of peduncles in progenies obtained following x-ray treatments. In these mutants, part of the peduncle just below the panicle bends, resulting in the panicle being held horizontally or hanging downwards, instead of the normal vertical position. This trait was found to be inherited as a monogenic recessive.

Kadam et al. (1940) reported sterility characterized by earheads bearing only bristles, and spikelets containing only rudimentary anthers devoid of pollen. From the behavior of plants heterozygous for this trait, they concluded that complete sterility is controlled by two duplicate genes.

Rai et al. (1987) described a spikeletless mutant, from an S_2 progeny, that was characterized by absence of spikelets from the entire spike, although involucral bristles were present. Segregation ratios observed in the S_2 and S_3 progenies derived from normal plants of the S_2, and F_2 progenies derived from crosses between normal inbreds and plants heterozygous for this trait, indicate that this character is inherited as a monogenic recessive; the symbol *sl* was proposed.

In an African variety named Jamnagar Giant, Likhite and Patil (1936) reported earheads that exhibited abnormalities such as basal branching and apical twining, twisting, and splitting. Rangaswami Ayyangar et al. (1935a) observed the occurrence of basal branching of the earheads in plants raised from pearl millet seed from Nigeria. They reported that this trait is governed by a single recessive gene. On the other hand, Gill and Athwal (1970) and Gill et al. (1971) reported that the branched ear base trait is controlled by a single dominant gene (*Beb*). They also reported that tapering eartip is controlled by two dominant genes, designated Te_1 and Te_2, with complementation, and the tufted eartip is controlled by a single recessive gene designated as *tet*.

Chandrasekharan and Sundararaj (1950) observed two branched (forked) earheads, but seed from these when planted again did not exhibit this characteristic; they did not offer any explanation. Chaudhuri and Gupta (1963) observed similar forking of earheads; plants from seed of forked-earhead plants showed this trait at a very low frequency (0.001%), and the authors concluded that forking was caused by external influences. Such unusual branched earheads were also reported by Thakare and Murty (1974). The branched eartip trait, where the apex of the earhead is split into two or more branches, was found to be controlled by a single recessive gene, *bet* (Krishnaswamy and Rangaswami Ayyangar, 1942; Gill and Athwal, 1970; Gill et al., 1971). No linkage between purple foliage and branched eartip, branched ear

base and branched eartip, or bristling and branched eartip traits was observed (Gill and Athwal, 1970). Dev et al. (1987) obtained a mutant, from gamma ray treatments, that they called bizarre earhead. This mutant trait was found to be controlled by duplicate recessive genes, for which the symbols be_1 and be_2 were proposed.

In ear-tip curving plants, the rachis is weak and the earhead tips become curved as they emerge out of the flag leaf. Singh et al. (1968) described a curved eartip trait that is controlled by a single dominant gene, whereas Singh et al. (1969) reported that this trait is governed by three recessive genes.

Singh et al. (1969) reported that inhibitory factors (13 normal : 3 naked eartip) were involved in the expression of the naked eartip trait, which is characterized by earheads with prolonged rachis devoid of spikelets and bristles at the apex. Gill et al. (1971), however, observed that this trait is controlled by two dominant genes (Net_1 and Net_2) with complementation.

Rangaswami Ayyangar and Hariharan (1936) mentioned that an African race, *Pennisetum echinurus*, which has bristled earheads in crosses with *P. leonis* without bristles, showed an F_2 segregation with a wide range of bristled and nonbristled forms. Grouping all the bristly forms together, they obtained a ratio of 3 bristled : 1 nonbristled types. Ahluwalia and Shankar (1964) reported that bristling (*Br*) is governed by a single dominant gene and variation in the density of bristling is possibly through the influence of modifying factors. Several other authors reported identical results (Krishnaswamy 1962, quoted by Ahluwalia and Shankar, 1964; Athwal and Gill, 1966; Lal and Singh, 1971; Singh and Pandey, 1973; Khan and Bakshi, 1976; Singh et al., 1967; Gill and Athwal, 1970; Gill et al., 1971). A conflicting report by Yadav (1974a) noted monogenic incomplete dominance for bristling. In crosses between long- and short-bristled plants, however, the bristle length was intermediate in the F_1 and continuous variation was observed in the F_2, indicating the additive action of more than one gene (Appa Rao et al., 1988c).

In samples of short-bristled pearl millet received from Africa, Rangaswami Ayyangar et al. (1935b) noticed spikelet bearing bristles that were the prolongation of the fascicle axis. Some of these spikelets bore normal grain. Compact spikes in pearl millet generally have spikelets with short rachillae, whereas in loose spikes the rachillae are long. Gill et al. (1971) observed that the *Br* gene in combination with the dominant gene for floret-bearing bristles (*Fbb*) produces floret-bearing bristles. In the absence of *Br*, *Fbb* was found to be ineffective. In segregating populations involving compact and loose spike types, Singh et al. (1969) observed plants bearing spikes in which a few rachillae were longer than the rest, giving an uneven and rough spike surface. They reported that inhibitory factors are involved in the expression of this trait. The rough spike trait is apparently different from the easily distinguishable spikelet-bearing bristled trait.

In tip-sterile plants, the tip of the rachis bears sterile bristles without spikelets. Krishnaswamy and Rangaswami Ayyangar (1942) reported that eartip sterility is a result of complementary action of two genes segregating in a 9 ear-tip sterile : 7 ear-tip fertile types. Singh et al. (1969) observed a similar ratio in the F_2 generation, but could not confirm it in the backcross generations. In other studies, however, Singh et al. (1967, 1968) reported that eartip sterility is controlled by a single dominant gene.

Krishnaswamy and Rangaswami Ayyangar (1942) reported that gappiness in earheads, characterized by the absence of spikelets, is conditioned by a single recessive gene. In crosses that involved pearl millet and wild millet [*P. glaucum* subsp. *monodii*; syn. *P. violaceum* (L.) Rich.], Bilquez and LeComte (1969) found that shattering of mature spikelets is conditioned by three recessive genes.

REPRODUCTIVE STRUCTURES AND GAMETE FORMATION

Reproductive Structures

In pearl millet, the ovary in the hermaphroditic flowers is obovate, smooth, and with two styles (branched) that are generally connate at the base. Each of the two styles has a single plumose stigma at its end and the styles have short hairs that are uniformly distributed along the entire length of the styles. Ratnaswamy (1954) reported the occurrence of pistils having three, four, and five stigmas, all of which were normal. Kajjari and Patil (1956) reported abnormal spikes with shriveled anthers lacking pollen grains but with normal stigmas. When pollinated, these spikes set adequate seed. Goyal (1962) observed a plant in which the spikelets did not possess any anthers, and ovaries exhibited abnormalities ranging from bulbous ovaries, thick styles, unbifurcated styles, two to five ovaries per spikelet, and few stylar hairs. The earhead did not set any seed following pollination with normal lines. Manga (1977) obtained two mutant plants in the progeny of desynaptic plants. These plants showed complete sterility. Some of the spikelets had three extra carpels, and normal carpel and stamens were absent. In others, up to two extra carpels were observed, and the number of extra carpels corresponded to the number of absent stamens. Partial to complete transformation of the stamens into carpels was observed. Infrequent formation of two seeds from the same spikelet indicated that some of the extra carpels were functional. This mutant condition is controlled by a single recessive gene.

Pokhriyal et al. (1967) observed in a selfed progeny both pistilless and partially pistilless plants. This mutant character is similar to that observed by Sikka et al. (1957). Pollen production in pistilless mutants was abundant and fertile. This character was governed by a single recessive gene.

Rangaswami Ayyangar and Panduranga Rao (1935) observed in sorghum and pearl millet a few dummy pollen grains, grains that were smaller in size than normal grains, devoid of solid contents and without the ability to germinate. They observed that nondehiscence of anthers was associated with an extreme paucity of dummy pollen. Dehisced anthers contained 7% dummy pollen, as compared with 1% in nondehisced anthers. This trait is inherited as a simple recessive. They concluded that dummy pollen may have a role in ensuring dehiscence of anthers in night-flowering millets. In pearl millet, large pollen grain size is dominant over small and is reported

to be controlled by one dominant gene or two complementary genes (Verma et al., 1969).

Hanna and Powell (1973) described a stubby head mutant, an induced facultative apomict with multiple ovaries, ovules, and embryo sacs. This mutant was characterized by reduction in the length of the peduncle and the top two internodes and produced multiple flag leaves around a knoblike inflorescence. The florets produced both single and multiple pistils per floret which gave single and twin caryopses. This characteristic stubby head is a recessive character. Testcross results indicated that this is a facultative apomict.

Gamete Formation and Development

Pearl millet has a chromosome number of $2n = 14$. The cytogenetics of this crop was reviewed by Jauhar (1981) and Pantulu and Krishna Rao (1982).

In pearl millet, both asynaptic and desynaptic mutants are reported. Asynaptic mutants are those in which there is complete failure of pairing between chromosomes at meiotic prophase; in desynaptic mutants, maintenance of pairing between initially synapsed chromosomes at meiosis is disrupted. The desynaptic condition in pearl millet is controlled by a monogenic recessive gene, *ds* (Minocha et al., 1975; Pantulu and Krishna Rao, 1982; Subba Rao, 1980). Pantulu and Subba Rao (1976) reported that *ds* reduces both the association and the chiasma frequency of B-chromosomes, similar to A-chromosomes. Lakshmi et al. (1979) suggested, based on observations on a single F_2 family of desynaptic × normal plants, that desynapsis accompanied by chromosome fragmentation results from the action of duplicate recessive factors. Jauhar (1981) noted that the degree of expression of the desynaptic gene could be under the control of modifiers or perhaps polygenes. Pantulu and Manga (1971) described a multiploid sporocyte condition in which pollen mother cells in the anther locules were devoid of individual boundaries and aggregated into plasmodiumlike masses. This is inherited as a recessive trait; the gene symbol *mu* was assigned.

STERILITY

Cytoplasmic–Nuclear Male Sterility

Cytoplasmic male sterility in pearl millet has been reviewed by Anand Kumar and Andrews (1984).

As early as 1940, Kadam et al. observed cases of complete sterility (rudimentary anthers without any pollen and complete absence of stigmas) and partial sterility (characterized by partial grain set). Kajjari and Patil (1956) reported male-sterile plants and suggested that the observed sterility could be of cytoplasmic type. Burton (1958, 1965) reported the discovery of cytoplasmic male sterility, and the first male-sterile Tift 23A and its maintainer Tift 23B were released. Since its discovery, cytoplasmic male sterility has been incorporated into tall and dwarf genetic backgrounds and used in the production of commercial F_1 hybrids.

Burton and Athwal (1967) proposed that in an A-line (male sterile), cytoplasmic male sterility results from an interaction of a homozygous recessive gene *ms* with its sterile cytoplasm. The sterility maintainer (B-line) is also homozygous for this gene, but is fertile because of its normal cytoplasm. Based on genetic–cytoplasmic interactions for restoration of fertility, they classified sterile cytoplasms into A_1 (in cms Tift 23A), A_2 (in cms line 66A), and A_3 (cms line 67A) types. According to studies on these three cms lines, fertility or sterility results from an interaction of three nuclear genes (ms_1, ms_2, and ms_3) with normal or sterile cytoplasm. The restoration of pollen fertility in three F_1 hybrids with male-sterile cytoplasms gave a continuous pattern of variation, indicating multigenic inheritance (Siebert, 1983). It was suggested that male fertility restoration in the A_1 cytoplasm is controlled by two major dominant complementary genes with at least one modifier. For the A_2 cytoplasm, segregation for restoration is characteristic of two major dominant genes with duplicate action. Male-sterile cytoplasm from *P. glaucum* subsp. *monodii* was found to be different from the A_1, A_2, and A_3 cytoplasms and has been designated as A_4 cytoplasm (Hanna, 1989). Dominant male fertility–restoring mutations, cytoplasmic mutations, and environmental factors such as temperature influence the expression and stability of male sterility (Anand Kumar and Andrews, 1984; Rai and Hash, 1990).

Genetic Male and Female Sterility

Krishna Rao et al. (1990) recently reviewed the applications of genetic male sterility in crop plants and indicated that it could be used in the production of hybrid varieties and in intra- and inter-specific hybridization to introduce new genetic diversity. In pearl millet, because of the protogynous nature of flowering and the availability of stable cms lines, use of gms has limited potential; however, it could be of use in studies that require expression of male sterility in normal cytoplasm.

Genetic male sterility controlled by a single recessive gene was reported by Krishnaswamy and Rangaswami Ayyangar (1942) in segregating generations following x-ray irradiation. Gill et al. (1973) observed a case of gms that was controlled by three recessive genes with complementary action.

Krishna Rao and Koduru (1978b,c) reported gms controlled by single recessive genes in two inbred lines Vg272 and IP 482. In Vg272 (ms_1), the anthers emerge but remain indehiscent and contain aborted pollen grains because the microspores degenerate before the first mitosis (PGM-type gms). In later generations, however, sterility resulted from failure of archeospore differentiation or function (Arc-type gms). The gene ms_2 from IP 482 showed fusion of pollen mother cells in different numbers at pachytene to form syncytes that later formed pollen grains of varying sizes. The expression of gms was influenced by genetic background and environment (Krishna Rao and Uma Devi, 1983).

Krishna Rao and Uma Devi (1989) reported that the male sterility genes in four inbred lines are allelic and are inherited as simple recessives. Presence of a dominant gene (*Ms*) resulted in male fertility, whereas homozygous recessive alleles (*ms ms*) resulted in male sterility. Type of cytoplasm and nuclear factors influenced the stage at which male sterility was expressed during the development of the male gametophyte. In C-1 type of cytoplasm, presence of male sterile genes in a homozygous state led to Arc-type sterility. In C-2 cytoplasm, degeneration started during meiosis with fusion of meiocytes and syncyte formation (Syn-type) or in PGM-type.

The triggering activity of recessive male sterility genes in C-2 cytoplasm seemed to be regulated by two nuclear factors R_1 and R_2 with duplicate gene action. In a recessive state, the R factors in C-2 cytoplasm resulted in PGM-type of expression.

A thermal neutron–induced female-sterile mutant was reported by Hanna and Powell (1974). The mutant produced abundant viable pollen and exserted stigmas but no seed. Female sterility was conditioned by a single recessive gene (*fs*) and the mutant was maintained in a heterozygous condition. The ovules aborted soon after anthesis; at anthesis, they appeared immature and malformed. This mutant has potential as a male parent in hybrid production and to derive obligate apomicts from facultative apomicts.

SEED CHARACTERS

Consumers associate grain quality with specific colors, sizes, and hardness (Hoseney et al., 1987). Of these, seed color presents considerable variability in pearl millet and its inheritance has received relatively more attention. Grain color in pearl millet is dependent on the endosperm and pericarp colors, the expression of the endosperm color being distinct in cases only where the pericarp is colorless. The IBPGR descriptors for pearl millet (IBPGR, 1981) lists nine seed colors, ranging from ivory to purplish black.

Patel (1939), Burton and Powell (1968), and Madhava Rao and Kullaiswamy (1975) reported that yellow grain color is inherited as a monogenic dominant to bluish green (slate-color) grains. Lal and Singh (1971) reported similar findings and assigned the gene symbols *Y* for yellow color and *y* for bluish green. Khan and Bakshi (1976) reported that pearly amber color is dominant to slate (bluish-green) and is controlled by a single dominant gene.

Al-Fakhry et al. (1975) observed that purple seed color is inherited as a simple dominant character over gray seed color; control in another purple seed color mutant (not described) was by dominant complementary genes. Phul et al. (1969) reported the inheritance of four endosperm colors (light slate, deep slate, amber, deep yellow). In all cross-combinations, a monogenic ratio was obtained and endosperm color appeared to be determined by different allelomorphs at the same locus. They assigned the gene symbols *y* (yellow), y^a (amber), y^{ds} (deep slate) and y^s (light slate) for the endosperm colors. They observed that purple pericarp color is determined by a single dominant gene and assigned the symbols *Prp* to purple pericarp and *prp* to colorless pericarp.

Burton (1952) found that semi-self-fertile lines and their progenies produced heavier seeds when topcrossed than when selfed, indicating the influence of foreign pollen on seed weight. The percentage increase in seed weight because of foreign pollen influence showed a tendency to be inherited.

Patel (1939) and Lal and Singh (1971) observed xenia effect of golden yellow over bluish-green grain color. Similar xenia effect was observed by Phul et al. (1969) when plants possessing slate colored grains were crossed with yellow and amber grain color stocks.

Patel (1941) observed sugary and starchy grains in the same earhead. The sugary trait bred true and was designated *su*, recessive to normal starchy (*Su*). Appa Rao et al. (1982) described sweet-stalk types in pearl millet. At maturity, on average, the soluble sugar content in the stalks of the sweet-stalk types was 10.6%, compared with 3.7% in normal types. They did not report any data for grains.

EARLINESS AND MATURITY

In pearl millet maturity is recorded as time (in days) from planting to 50% stigma emergence or anthesis, because the variation for the period from fertilization to maturity is generally constant across genotypes.

Photoperiod sensitivity in pearl millet is a well-recognized trait. In West Africa, the early souna and gero millet types are relatively day-neutral and can flower irrespective of daylength, whereas the sanio and maiwa types head only when the days are short (<12h). Bilquez (1963) concluded that two alleles (L_1 and L_2) of a major gene condition earliness in souna (L_1L_1) and sanio (L_2L_2) millet, without dominance. The variation within the souna group was attributed to multiple genes, with two alleles each. The dominant allele conditions earliness and the recessive allele, lateness (Bilquez and Clement, 1969).

In Nigeria, observations on F_1's between photoperiod-sensitive maiwa populations and early Tift 23A suggest a high degree of dominance for earliness (Bharadwaj and Webster, 1971). Burton (1966) considered that photoperiodism exhibited by those lines that failed to flower when daylengths were >12h may be a characteristic of pearl millet. He estimated the minimum gene number to be between two and four, and also observed lack of dominance in the action of genes controlling photoperiodism. Begg and Burton (1971) reported that the five genotypes they studied behave as facultative short-day plants; i.e., they were capable of flowering under long days, but flowered much earlier under short days.

Wilson et al. (1990) observed that in crosses between tall and late-maturing landraces from Burkina Faso, in West Africa, and Tift 85DB, the F_1's were intermediate between the parents for time in days from planting to anthesis. The average number of loci governing maturity was estimated at 3.4, and additive gene effects were noted to be predominant. Working with early (74.8 d to 50% heading), intermediate (89.6), and late (98.6) inbreds, Ram and Singh (1975) observed that earliness is primarily due to dominant genes and heading appears to be simple in inheritance.

Burton (1981) isolated an early gene from a weak, spindly, very early-maturing plant from the cultivar Katherine. Results indicated that early maturity (45–66 d to anthesis) was recessive to normal maturity (80–100 d) and is conditioned by a single gene designated e_1. This gene, when homozygous, removed photoperiod sensitivity in pearl millet and provided early maturity. This early gene was transferred to Tift 23DB, and an early version, Tift 23DBE, was developed. At Tifton, GA (31° N), inbred Tift 23DBE flowered in 45 to 55 d regardless of season planted, whereas Tift 23DB flowered in 75 to 85 d if planted in May and in 55 to 65 d if planted in August.

Hanna and Burton (1985) reported the induction of two early-maturity mutations following seed treatment of inbred Tift 23B with EMS and thermal neutrons. Both mutants showed shorter plant height, shorter head length, and narrower stem diameter than the parent. Reduction

of internode number was observed to be a pleiotropic effect of the early mutant loci. Mutants 1 and 2 flowered in 49 and 38 d, respectively, after planting on 12 June, compared with 76 d for Tift 23B. Inheritance studies indicated that earliness is controlled by single recessive genes. In Mutant 1, earliness is controlled by the e_1 gene described above and was not associated with spindly characteristics of the natural mutant; in Mutant 2, earliness is controlled by e_2. Linkage analysis showed that e_1 and the d_2 gene segregate independently.

Contrary to Burton (1981), Appa Rao et al. (1988b) observed that Tift 13E carrying the early-maturity gene e_1 flowered in 49 to 67 d at Patancheru, India (17°27′ N), when sown on five different dates. From the world germplasm accessions, seven accessions were identified that flower earlier than Tift 13E. Of these, IP 4021 (Bhilodi), classified as day-neutral, flowered in 33 d irrespective of planting date. Crosses between IP 4021 and Tift 13E indicated that the former carries recessive and nonallelic genes for earliness. Crosses among other lines with IP 4021 indicated that lateness is partially dominant, or overdominant, over earliness and the differences in flowering are controlled by one or two genes with modifiers.

DISEASE RESISTANCE

The four fungal diseases that cause major damage to pearl millet in both Africa and India include downy mildew [caused by *Sclerospora graminicola* (Sacc.) J. Schröt.], smut [*Moesziomyces bullatus* (J. Schröt.) K. Vánky; syn. *Tolyposporium penicillariae* Bref.], ergot (*Claviceps fusiformis* Loveless), and rust (*Puccinia substriata* Ellis & Barth. var. *indica* Ramachar & Cummins; syn. *P. penniseti* Zimmerm.). Rusts and leaf spots cause severe damage on pearl millet in the southern USA, where it is grown for forage. Resistances to diseases have been identified in pearl millet, and also in the wild species *P. glaucum* subsp. *monodii*, which belongs to the primary gene pool of *Pennisetum*.

Kumar et al. (1983) evaluated >2900 A- and B-lines, employing an effective field screening technique for downy mildew reaction. Lines carrying male-sterile cytoplasm were no more susceptible than those with normal cytoplasm, indicating that in pearl millet the sterile cytoplasm is not involved in determining susceptibility to DM.

Working with two groups of inbreds under natural levels of infection, Appadurai et al. (1975) observed that resistance to DM is governed by one or two single dominant genes. Mehta and Dang (1978), using inbreds resistant and susceptible to DM and a DM sick-plot, found that resistance to downy mildew shows dominance. Singh et al. (1978b), using inbreds and a sick-plot, recorded resistant and susceptible reactions in the F_1 for R × R inbreds and S × S inbreds, respectively. For R × S inbreds, the F_1's were susceptible, indicating that susceptibility is dominant over resistance. In the F_2, the frequency of resistant plants was higher in R × R crosses than in the other two classes, and the plant-to-plant variation was high, indicating polygenic inheritance. Inheritance of resistance to DM was studied under artificial epiphytotic conditions in 10 crosses by Gill et al. (1978). They found duplicate dominant factors controlling resistance and proposed the gene symbols DM_1DM_2, DM_1dm_2, and dm_1DM_2 for the resistant and dm_1dm_2 for the susceptible genotypes.

In a study that involved six cultivars (Yadav, 1974b), inheritance of resistance to smut ranged from monogenic incomplete dominance to monogenic complete dominance and duplicate action of two genes; however, Mehta and Dang (1978) determined resistance to ergot and smut to be under polygenic control.

Andrews et al. (1985) identified a single dominant gene for rust resistance in an S_2 progeny selected from an accession from Chad. They proposed the gene symbols Rpp_1 and rpp_1 for the dominant gene governing resistance and its recessive allele conditioning susceptibility. Hanna et al. (1985) identified a single dominant gene (Rr_1) for rust resistance in a *P. glaucum* subsp. *monodii* from Senegal. This gene was successfully transferred to pearl millet. Sokhi et al. (1987) observed that resistance to rust is governed by a single dominant gene in line P-1564 and by two dominant genes exhibiting complementary action in line 700481-23-2.

Wilson et al. (1989) reported that in crosses between landraces from Burkina Faso and Tift 85DB, resistance to the leaf spot caused by *Pyricularia grisea* (Cooke) Sacc. [teleomorph: *Magnaporthe grisea* (T.T. Hebert) Yaegashi & Udagawa] is controlled by two independent dominant genes. Hanna and Wells (1989) observed that resistance to this leaf spot in a *P. glaucum* subsp. *monodii* accession is controlled by three independent dominant genes and that these were transferred to a male-sterile line and a cultivar. Wells and Hanna (1987, 1988) observed that resistance to a severe leaf spot caused by *Bipolaris setariae* (Sawada) Shoemaker [teleomorph: *Cochliobolus setariae* (Ito & Kuribayashi in Ito) Drechs. ex Dastur] in pearl millet × *P. glaucum* subsp. *monodii* crosses is controlled by dominant gene action. This ratio is characteristic of a four-independent-gene system, with duplicate dominant genes, one inhibitory gene, and one anti-inhibitory gene. The gene symbols $Bp_1Bp_1Bp_2Bp_2$ (duplicate genes), bp_3bp_3 (inhibitory gene), and bp_4bp_4 (anti-inhibitory gene) were assigned. Khan and Bakshi (1976) reported that resistance to a leaf spot of unknown etiology (*Curvularia penniseti*?) was inherited as a dominant trait.

BIOCHEMICAL GENETIC MARKERS

Genetically controlled variation in electrophoretic properties of enzymes has been reported in pearl millet. Some of the variants have been found to be codominant (heterozygotes produce both of the variants, rather than only one), suggesting that the locus coding for the enzyme is the structural gene.

Sandmeier et al. (1981) found a carboxylic esterase with nine different alleles that is inherited as a Mendelian trait, with a ratio of 1:2:1. They also characterized eight anodic peroxidases and found that one of them (P_5) is controlled by two genes. Leblanc and Pernes (1983) noted that alcohol dehydrogenase, phosphoglucomutase, and phosphoglucoseisomerase are coded by three loci. Banuett-Bourrillon (1982) reported that two structural genes for alcohol dehydrogenase (Adh_1 and Adh_2) are closely linked. Tostain and Riandey (1984) found that catalases in pearl millet are coded by only one gene, *Cat A*, and that esterases in dry grains having α-naphthyl acetate as the preferential substrate are controlled by one gene, *Est*

B, and its two alleles. Endopeptidases (*EP A*) showed a simple mode of inheritance. Malate dehydrogenase is composed of several dimeric enzymes under complex genetic control: four structural genes (*Mdh A*, *Mdh B*, *Mdh C*, *Mdh D*) and a migration modifier (*Mmm*) (Tostain and Riadney, 1985). Tostain and Lavergne (1986) found that two dimeric isozymes of glutamate oxaloacetate transaminase were coded for by unlinked nuclear genes, *Got A* and *Got B*, each occurring in three codominant allelic forms.

Tostain (1985) evaluated linkage relationships between the dwarfing gene d_2 and seven enzymic marker genes. He has shown linkage between *Pgi A* (phosphoglucose isomerase) and *Pgm A* (phosphoglucomutase); between *Skdh A* (shikimate dehydrogenase) and *Adh A* (alcohol dehydrogenase); between D_2 and *Skdh A*; and between D_2 and *Adh A*. The latter three genes were linked in the order *Adh A*–11cM–*Skdh A*–9cM–D_2. Selection of heterozygotes (D_2d_2) was suggested as possible at the seedling stage, using the close linkage between the d_2 and *Skdh A* genes.

These enzymatic marker genes were further used by Tostain et al. (1987) and Tostain (1992) to identify patterns of diversity within and between cultivated and wild West African pearl millet, and to draw conclusions about breeding utility and domestication.

Subba Rao et al. (1989) observed that, of the five seed esterase isozymes, four are under the control of a single gene and the fifth is controlled by three independent loci with duplicate gene action. Loci Est_1, Est_3, and Est_4 are linked, while Est_2 is independent of this linkage group. These esterases appear to be genetically different from the carboxylic esterases reported by Sandmeier et al. (1981).

Genetic analysis of F_1 and F_2 populations derived from crosses between a purple foliage and green foliage inbreds indicated that 17 flavonoid syntheses are controlled by 42 genes. One flavonoid showed monogenic inheritance, four digenic and eleven trigenic. Presence of inhibitory and duplicate genes was also indicated (Singh and Gill, 1980).

CONCLUSION

In the literature reviewed here, the number of morphological mutants reported in pearl millet is 145. The major categories include chlorophyll deficiencies (26%), plant pigmentation (18%), and earhead characters (14%) (Fig. 2). This compares well with maize, where >250 morphological mutants have been reported (Neuffer et al., 1968).

Notwithstanding the progress made in understanding the genetic control of several qualitative traits, linkage relationships are known for only some of them. These include the linkage group involving the yn_1 (yellow mutant), lgn_1 (light-green mutant), and *fs* (female sterile) loci (Hanna et al., 1978), and the *hl* (hairy lamina), *hst* (hairy stem), *hs* (hairy sheath), and *Hm* (hairy leaf margin) loci (Krishna Rao and Koduru, 1979). Linkage between bleached leaf (*bl*) and glossy trait (gl_1) and white sheath (*ws*) and yellow leaf (*y*) traits has been observed (Appa Rao et al., 1990). Manga et al. (1988) observed linkage (i) between purple node (Pn_1Pn_2) and purple auricle (Par_1Par_2) and (ii) between purple midrib, margin, and sheath; purple internode; and purple apicule. Linkage between the d_2 gene and *Skdh A* (shikimate dehydrogenase) could be used in the separation of D_2D_2 and

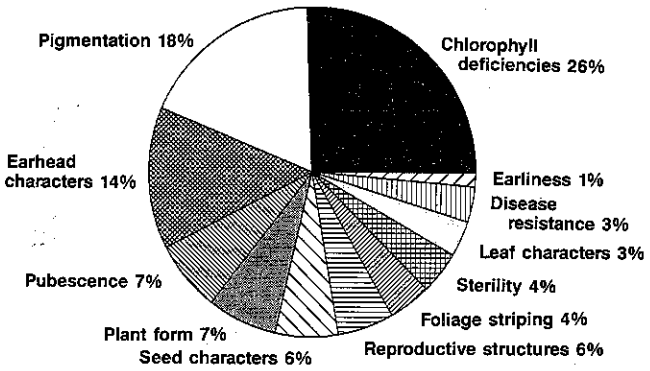

Fig. 2. Proportion of different categories of morphological mutants reported (a total of 145) in pearl millet. Mutations controlling gamete formation and development and those affecting reproductive structures are shown together.

heterozygote D_2d_2 at the seedling stage, as suggested by Tostain (1985). All other morphological traits (excluding enzymic markers) tested exhibit independent assortment. In contrast, linkage between several isozymes has been demonstrated.

Minocha and Sidhu (1979) determined the location of 10 genes affecting morphological traits to chromosomes using a primary trisomic series. The study showed that chromosome 1 carries the genes *hl* (hairy leaf) and Pg_1 (purple glume); chromosome 2, *Br* (bristled earhead), *yst* (yellow foliage striping), and Pp_1 (purple pigmented foliage); chromosome 4, Pp_2 (purple pigmented foliage); chromosome 5, *Beb* (branched ear base) and Rn_1 (purple node); and chromosome 6, *Bet* (branched eartip) and Pg_2 (purple glumes).

Obviously only a few mutant traits reported are of significance in the improvement of grain and forage production. The others have potential uses in basic and applied genetic studies. Some major genes have formed the basis for increasing both grain and forage yield and quality. These include cytoplasmic–nuclear male sterility, the dwarfing gene d_2, and the trichomeless gene *tr*. Other major mutant traits, such as the brown midrib, have potential for increasing forage quality.

Several reports that describe similar mutant phenotypes and allelic relationships of genes controlling these similar phenotypes are inaccessible. Several publications do not provide adequate description of the mutant phenotype to permit comparison of phenotypes by other researchers. For the CD mutants, use of a standard color catalog for accurate description and classification, in addition to an indication of the growth stage, is essential. Most reports do not describe the extent of inbreeding of the genetic stocks. Inadequate inbreeding may explain different genetic control observed for the same trait.

It would be useful if a color catalog illustrating all the mutant phenotypes available in pearl millet were brought out. Such a catalog would help in recognition and proper classification of mutant phenotypes and would help put the study of the genetics of qualitative characters in pearl millet on a sound basis.

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