The Genes of Capsicum

Deyuan Wang
Vegetable Institute, Guangdong Academy of Agricultural Sciences, Guangzhou, Guangdong, China 510640

Paul W. Bosland
Department of Plant and Environmental Sciences, New Mexico State University, Las Cruces, NM 88003

Additional index words. ají, chile pepper, genetics, vegetable breeding

Abstract. Pepper (Capsicum spp.) is one of the most cultivated vegetable and spice crops in the world. Capsicum genetics have been extensively studied, but the most recent Capsicum gene list was published more than a decade ago. Since then, new genes have been described. This updated gene list provides detailed descriptions of genes, including the genes’ characteristics, the genetic background of the mutants/lines, action mechanisms of genes, gene interactions, molecular markers, and chromosome localization when available. This new list includes 292 genes for morphological traits; physiological traits; sterility; and resistance to diseases, nematodes, and herbicides, which includes the 92 genes that have not been previously described.

Pepper (Capsicum spp.) is one of the most cultivated vegetable and spice crops worldwide, and plays an important role as a constituent in many of the world food industries (Bosland and Votava, 2000). Peppers were one of the first plants to be domesticated and cultivated in the western hemisphere. The small-fruited wild forms of peppers—chilepines—are still found growing wild in Arizona and Texas in the United States. World pepper production in 2004 reached 1.65 million ha with more than 24 million metric tons harvested (FAO, 2005). China is the world’s largest producer, with more than 53% of the total production area and nearly 50% of total world production (FAO, 2005).

Capsicum is native to the tropical and subtropical Americas and may comprise up to 30 species, among which the five major cultivated species are C. annuum L., C. frutescens L., C. chinesis Jacq., C. baccatum L., and C. pubescens Ruiz and Pavon (Bosland, 1992). Worldwide, C. annuum is the most cultivated and economically important species, and includes both sweet and hot fruits in myriad shapes and sizes.

Most Capsicum species are diploid (2n = 2x = 24), but there are a few species for which the genome is 2n = 2x = 32. Capsicum has a large genome, with the 2C DNA content ranging from 7.65 pg/nucleus in C. annuum to 9.72 pg/nucleus in C. pubescens, and with a general mean of 8.42 pg/nucleus (Belletti et al., 1998). These values correspond to the 1c genome size of 3.691 (C. annuum), 4.690 (C. pubescens), and 4.063 (general mean) Mbp. C. annuum genome is about three to four times the size of the tomato (Solanum lycopersicum L.) genome (Arumugananthan and Earle, 1991).

Capsicum genes have been studied since Webber, in 1912, investigated the inheritance of several traits (cited in Boswell, 1937). Other early contributions to Capsicum genetics were made by Atkins and Sherrard (1915) of England, Deshpande (1933) of India, Ikeno (1913, 1917) of Japan, and Halsted (1918) and Dale (1929, 1931) of the United States (all cited in Boswell, 1937). In 1933, Matsaura summarized pepper genetics, from 1910 to 1929, that consisted of 12 characters without designating any gene symbols (cited in Boswell, 1937). Boswell (1937) reviewed the inheritance of 16 characters in pepper, of which seven gene symbols were recorded for seven different traits (purple foliage and stem color, intense purple foliage and stem color, red mature fruit color, blunt fruit apex, bulged fruit base, pendent fruit position, and non-clasping fruit calyx). After Boswell’s work, inheritance studies in pepper gained more interest and included more important traits associated with greater importance of pepper production worldwide, as more inbred or spontaneous mutants being obtained.

From the late 1980s, more efforts to tag important to pepper breeding that was adapted and modified and updated from previous lists, especially the gene list of Daskalov and Poulos (1994). The current gene list contains detailed descriptions of the gene mutants and gene lines as well as the genetic background (cultivars or accessions, and species). If known, we have also added the acting mechanisms and characteristics of genes and gene interactions. In addition, molecular markers and the chromosome localization of genes have been added.

In addition to the genes listed by Daskalov and Poulos (1994), 92 new genes have been added to this gene list. The gene symbols proposed are in accordance to the rules for gene nomenclature of Capsicum for those characters that have been examined for inheritance (CENL, 1994). An attempt was made to correct errors in the gene symbols or descriptions from previous lists. This gene list (Table 1) presents the 292 known genes of Capsicum, including morphological traits, physiological traits, sterility, and resistance to diseases, nematodes, and herbicides.

Gene Nomenclature for Capsicum

The basic rules for Capsicum gene nomenclature have been proposed by Lippert and associates (1965), and adopted by Daskalov (1973), Cislerov (1980a, 1983), Greenleaf (1986), Daskalov and Poulos (1994), and then updated by the CENL Committee for Capsicum Gene Nomenclature (1994). In brief, the rules for assigning gene symbols adopted from others are that genes are symbolized by a maximum of three italicized Roman letters. The first letter of the symbol should be the same as that for the gene name, which should describe a characteristic feature of the mutant type in a minimum of adjectives or nouns in English or Latin. When the mutant is dominant, the first letter of the symbol is capitalized; if the mutant is
Table 1. The genes of Capsicum.

| Preferred symbol | Synonym | Character | References |
|------------------|---------|-----------|------------|
| A                | P       | Anthocyanin; the incompletely dominant gene for anthocyanin color in the foliage, flower, and immature fruit | Lippert et al., 1965; Odland, 1960; Peterson, 1959 |
| al-1 to al-8     | b,s     | Anthocyanin-less; prevents purple color on the stem, fruits, and in plants; nodes, green; anthers, yellow; only al5 shows a slight puplish marking along the lines of dehiscence; lack of purple spots on immature fruits; in some genotypes, and especially in cold and rainy weather, purple spots on the nodes and immature fruits may be observed; epistatic to A, As, and Afs; nonallelic; al6 and al7 found in C. chinense; al8 found in C. chacoense; linkage found between al2 and mos2 | Cook, 1961b; Csillery, 1980a, 1983, 1984; Daskalov and Poulos, 1994; Greenleaf, 1986; Odland, 1960 |
| Anr-1*           |         | Anthracnose resistance; resistance of C. annuum cv. Chungyong to C. dematium | Park et al., 1992 |
| Anr-2*, Anr-3*, Anr-4* |         | Resistance to C. gloeosporioides in C. annuum, Anr2 in 'BGH13077', two genes Anr3 and Anr4 in 'BGH2830' and 'BGH5085' | Fernandes and Ribeiro, 1998 |
| Anr-5*           |         | Anthracnose resistance; resistance to C. capsici | Lin et al., 2002 |
| anv              |         | Angustifolia variegada; elliptical cotyledons, long and narrow leaves | Daskalov and Poulos, 1994 |
| Ap**             | AP      | The pointed shape of the fruit apex; dominant to the indented shape | Ishikawa et al., 1998 |
| As               | P       | Anthocyanin on style; purple in the absence of A or Asf | Lippert et al., 1965 |
| Asf              | W       | Anthocyanin on style and filament; purple in the absence of A | Lippert et al., 1965; Odland, 1960 |
| aur              |         | Aurea; golden cotyledons and leaves | Daskalov and Poulos, 1994 |
| B                |         | Beta-carotene; high beta-carotene content in mature fruits, interacts with A | Lippert et al., 1965 |
| bc*              |         | Beta-carotene; high beta-carotene content conferred by preventing hydroxylation of beta-carotene to beta-cryptoxanthin; mutant from ‘Pasardzhishka Kapta’ | Chalukova et al., 1993; Daskalov et al., 1995 |
| bl               |         | Branchless; plants grow normally to the stage of first stem bifurcation, then stem development stops; found in PI 169113 | Bergh and Lippert, 1964 |
| brl              |         | Braquatica latifolia; shortened stem internodes; leaf blades wide, large, round, and dark green with short, thickened petioles | Daskalov and Poulos, 1994 |
| Bs-1             |         | Bacterial spot resistance; hypersensitive resistance in PI 163192 to race 2 of X. campestris pv. vesicatoria | Cook and Stall, 1963 |
| Bs-2             |         | Bacterial spot resistance; hypersensitive resistance in PI 260450 to both race 1 and race 2 of X. campestris pv. vesicatoria | Hibberd et al., 1987 |
| Bs-3             |         | Bacterial spot resistance; hypersensitive resistance in PI 271322 to race 1 of X. campestris pv. vesicatoria | Hibberd et al., 1987 |
| Bs-4*            |         | Bacterial spot resistance; hypersensitive resistance in PI 235047(C. pubescens) to race 6 of X. campestris pv. vesicatori. | Sahin and Miller, 1997, 1998 |
| bs-5*, bs-6*     |         | Bacterial spot resistance; nonhypersensitive resistance in ECW-12346 to race 6 of X. campestris pv. vesicatoria | Jones et al., 2002 |
| bv               | mutant-2| Buzy variegated; plants with creamy white apical leaves spotted with small green areas; extensive lateral shoot development provides a bushy appearance | Cook, 1962; Lippert et al., 1964 |
| Bzt              |         | Bentazon tolerance; confers a high level of tolerance to herbicide bentazone in C. annuum cv. ‘Santaka’; modifying genes may affect the Bzt gene in ‘Bohemian Chili’ | Fery and Harrison, 1990; Wolff et al., 1992 |
| c-1              | c       | Carotenoid pigment inhibitors; reduce 10% in red color of mature fruits; c1 and c2 reduced pigmentation of y’ and y by the inhibition of beta-carotene | Hernandez and Smith, 1985; Lippert et al., 1965 |
| c-2              | cl      | Carotenoid pigment inhibitors; much stronger red color reduction than c1 | Hernandez and Smith, 1985; Lippert et al., 1965 |
| ca               |         | Carotenoid pigment inhibitors; much stronger red color reduction than c1 | Csillery, 1980a |
| call             |         | Callus proliferations; tiny wartlike structures scattered on cotyledon, stem, and leaf; more pronounced on the abaxial surface | Csillery, 1983 |
| ce               | e       | Callus enclosed; calyx-enclosed fruit base; loci ce and fb are linked at 4.7 cM | Miller and Fineman, 1938 |

(Continued on next page)
Table 1. (Continued) The genes of Capsicum.

| Preferred symbol | Synonym | Character | References |
|------------------|---------|----------|------------|
| cf*-1            | cf      | Closed flower; petals remain attached to one another at anthesis; allelic to the gene governing the closed-flower trait in nine Hungarian mutants; found in ‘UFBG 8209-1’ | Subramanya and Ozaki, 1984; Zeweite and Bosland, 2001 |
| cf*-2*           |         | Closed flower; calyx enlarged covering half portion of the corolla, which remains rolled and never opens; plant dwarf and bushy with many branches; most flowers with protruded style; allelism to cf | Pathak et al., 1983a |
| cf**             |         | Conditional female sterile; vigorous plants with compact habits; normal developed flowers containing fertile pollen grains; obtained from an M2 population of the cv. ’Borjana’ | Daskalov and Mihailov, 1988 |
| chl              |         | Chlorina; greenish yellow variegation or chlorophyll deficiency | Lippert et al., 1965 |
| ci               |         | Compound inflorescences; dichasial structure formed with repeated branching as in a pseudo umbel at the end of the sympodia | Csillery, 1983 |
| cl               |         | Chlorophyll retainer; in mature fruit; combines with y’ (red) or y (yellow) to produce brown and olive green mature fruit color, respectively | Hurtado-Hernandez and Smith, 1985; Shifriss and Pilovsky, 1992; Smith, 1950 |
| cm*              |         | Cucumber mosaic virus (CMV) resistance; resistance to CMV in ‘Perennal’ | Singh and Thakur, 1977 |
| ct               |         | Compact; controls the extent of lateral axillary shoot development before the first bifurcation; plants have more numerous and erect axillary shoots at maturity, and are about half as tall as normal, but internodes are not as short as in dwarfs | McCammon and Honna, 1984; Shifriss and Hakim, 1977 |
| cw*              |         | Curved leaf; leaves have curved leaf blades with long petioles; fewer branches, flowers, and fruits; seed set poor; induced from ‘PC 1’ | Aniel Kumar et al., 2001 |
| cy               |         | Chappy; at maturity the exocarp is covered with transversally oriented, small suberized cracks | Csillery, 1983 |
| div              |         | Diversa; deformed leaves, yellow-green virescnet leaves | Daskalov and Poulos, 1994 |
| dm               |         | Diminished morphology; extremely small (2 cm in length, 1 cm in width) leaves with normal shape; main stem before the first cyme has 18 to 20 internodes; equally tiny stem and flowers; moderate water stress causes wilting as in small | Csillery, 1983; Daskalov and Poulos, 1994 |
| Dms              |         | Dominant genetic male sterility; mutation from m5; Desynapsis; univalents occur at a high frequency despite regular pachytene pairing at diakinesis and metaphase I; causes a high degree of sterility; modifying genes may affect ds | Daskalov and Poulos, 1994 |
| ds               |         | Determinate growth; determinate growth habit; Dt and Ct condition indeterminate growth, and are epistatic to one another | McCammon and Honna, 1984 |
| dt               |         | Determinate growth; determinate growth habit; Dt and Ct condition indeterminate growth, and are epistatic to one another | McCammon and Honna, 1984 |
| dtr              |         | Datura leaves; normal cotyledons; leaves are irregularly dentate like leaves of Datura spp. with maximal expression around the 10th leaf | Csillery, 1983; Daskalov and Poulos, 1994 |
| dvg              |         | Deforme variegata; deformed and undulated green virescnet variegated leaves | Daskalov and Poulos, 1994 |
| dw-1             |         | Dwarf; plant height, 12 to 15 cm; very short internodes; very thick, curly, dark-green, glossy leaves; normal flowers; reduced female fertility; mutant in ‘Zlaten Medal’ | Daskalov, 1973b, 1974 |
| dw-2             |         | Dwarf; 15 ot 20 cm in height; short internodes; thick, dark-green leaves; mutant in ‘Zlaten Medal’ | Daskalov, 1974 |
| dw-3             |         | Dwarf; 10 to 15 cm height; found in the interspecific hybridization between C. baccatum and C. annuum | Csillery, 1980a |
| dw-4             |         | Dwarf; 8 to 10 cm in height, shorter than dw; found in the interspecific hybridization between C. frutescens and C. annuum | Csillery, 1983 |
| dw-3*            |         | Dwarf; induced mutant in the Italian variety ‘Friariello’ | Restaino, 1991 |
| dw-6*, dw-7*     |         | Dwarf, controlled by two complementary dominant genes; found in the interspecific hybridization between C. annuum and C. chinense | Yazzawa et al., 1991 |
| dw-8*            |         | Dwarf; 10 to 20 cm height; reduced size of leave, flowers, fruits, and seeds; mutant from ‘PC 1’; early flowering; flowering 20 to 25 d earlier than ‘PC 1’; seed set poor; induced from ‘PC 1’ | Aniel Kumar et al., 2001 |
| cf               |         | Early flowering; flowering 20 to 25 d earlier than ‘PC 1’; seed set poor; induced from ‘PC 1’ | Aniel Kumar et al., 2001 |
| ep               |         | Elongated petiole; narrow and long petiole; irregular flower opening; induced from ‘PC 1’ | Aniel Kumar et al., 2001 |
Table 1. (Continued) The genes of Capsicum.

| Preferred symbol | Synonym                  | Character                                                                                           | References                        |
|------------------|--------------------------|-----------------------------------------------------------------------------------------------------|-----------------------------------|
| fa               | Fascication; compact, bushy plants with determinate tendency, short internodes; flowers and fruits borne in clusters; minor genes involved in the expression of fascication | Greenleaf, 1986; Lippert et al., 1965; Van der Beek and Ltiifi, 1990 |
| fb               | Fruit base nonbulging; nonbulged fruit base                                                                 | Miller and Fineman, 1938          |
| Fc               | Filament color; filament color in anthers                                                                 | Prince et al., 1993              |
| fcf**            | Fasciflora; female sterile plants with 27% to 55% pollen fertility, seedless fruits                      | Daskalov and Poulos, 1994        |
| fems*            | Female and male sterility; pollen shrunked, containing no starchy; many small fruits with no seeds; mutant in line '4526' | Martin and Crawford, 1951        |
| fi-1             | Filiiform; threadlike leaves, blossom irregularities; female sterility                                      | Cook, 1961b; Lippert et al., 1965 |
| fi-2             | Filiiform; similar to fi-1; narrow cotyledons and leaves (3–4 mm); threadlike petals; carpels usually not fused to pistil except in particularly small fruited varietal background; incomplete female sterile | Csillery, 1980a                  |
| ffl*             | Folded leaf; leaves folded upward, giving boat-shaped appearance; prominent in seedlings; partially fertile; induced from 'PC 1' | Aniel Kumar et al., 2001         |
| ffc              | Flaccid; cotyledons and leaves exhibit flaccid condition; the mutant has a normal leaf phenotype, but under typical field and greenhouse conditions it becomes flaccid; induced mutant from 'Keystone Resistant Giant No. 3' | Bosland, 2002                    |
| ffs              | Female sterile; complete female sterility with no other primary phenotypic effects; mature plants with a twiggy, multilobed periphery; mutant from PI 159276 | Yuan and Li, 2000                |
| fll*             | Flowerless plant; profuse vegetative growth, induced from 'PC 1'                                        | Aniel Kumar et al., 2001         |
| Flv              | Flavi; yellow-green leaves, shorter and less vigorous plants                                             | Daskalov and Poulos, 1994        |
| fms              | Functional male sterility; degenerated corolla; shriveling stamens develop incomplete anther and stigma; anthers closed by longer calyces; mutant from 'Fudjian' |                                   |
| frf              | Frilly; leaf margins are marked by undulation                                                            | Csillery, 1980a                  |
| fs               | Female sterile; complete female sterility with no other primary phenotypic effects; mature plants with a twiggy, multilobed periphery; mutant from PI 159276 | Bergh and Lippert, 1964          |
| fV               | Fan vein; veins of true leaves are branching like a fan; the leaf blade is often dissected, segments curled like tendrils, corolla, and other parts of the flower are irregularly reduced, but fruits may develop; found in C. chacoense | Csillery, 1980a                  |
| gd               | Glossy diminutive; small leaves with an unusual, pale glossy appearance; plants vary from tiny to as large as one-sixth of the normal plant size; mutant from PI 159226 | Bergh and Lippert, 1964          |
| gds              | General defense system; provides resistance to Xanthomonas campestris pv. vesicatoria in PI 163192; promotes cell growth and cell wall thickening | Csillery et al., 2004; Szarka and Csillery, 1995 |
| Gi               | Graft incompatible; manifests when grafted to other Solanaceae                                           | Lippert et al., 1965             |
| H                | Harry or pubescent leaf surface; dominant gene; H_ is epistatic to Sm_ whereas smsm is epistatic to hh. | Holmes, 1934; Shuh and Fontenot, 1990 |
| ht               | Hungarian tricolor; the basal one-fifth part of the fruit less pigmented, like sw before maturity, whereas more or less dark-green on top four-fifths portion; at full maturity a red-white-green fruit color results from the top to the calyx | Csillery, 1983                  |
| im               | Intermediate; purple color at intermediate maturity in originally nonpurple immature fruit; relation to A unknown | Lippert et al., 1965             |
| k                | Easy pedicle detachment from node; related to flower and young fruit abscission                           | Uzo, 1984                       |
| L1               | Localization; localization of P0 strain of tobacco mosaic virus (TMV) in 'Brumema Wonder' and 'Verbeterde glas' | Boukema et al., 1980b; Holmes, 1934, 1937 |
| L1'              | Localization; localization of TMV resistance at high temperature; 'C' for 'Crisillo de Moralos-334', allele at L locus | Daubeze et al., 1990             |
| L1**             | Localization; localization of both P0 and P1 strains of TMV in C. frutescens cv. Tabasco                 | Boukema, 1980; Holmes, 1934, 1937 |
### Table 1. (Continued) The genes of Capsicum

| Preferred symbol | Synonym | Character | References |
|------------------|---------|-----------|------------|
| $L^\alpha$        |         | Localization; this allele in 'Greygo' behaves like the allele $L^\alpha$ characteristic of 'Tabasco'; identity of the allele $L^\alpha$ relative to $L^\alpha$ is undetermined | Salamon et al., 2001 |
| $L^\beta$        |         | Localization; localization of $P_a$, $P_t$, and $P_{t2}$ strains of TMV in C. chinense | Boukema, 1980; van den Berkmortel, 1997 |
| $L^\gamma$        |         | Localization; localization of $P_a$, $P_t$, $P_{t2}$ and $P_{t2,3}$ strain of TMV in C. chinense accessions P1260429 and SA185; allelic series $L^\gamma > L^\delta > L^\alpha > L^\beta > L^\gamma$ | Boukema, 1984 |
| $Ld^*$           |         | Leaf dimorphism; small, narrow, thin, light-green leaves at top of plant; long, robust, thick, dark-green leaves at lower portions of plant; induced from 'PC 1' | Aniel Kumar et al., 2001 |
| $l f^*$          |         | Late flowering; flower initiation 20 to 30 d later than in 'PC 1' | Aniel Kumar et al., 2001 |
| $l^m r-1^*$, $l^m r-2^*$, $l^m r-3^*$ |         | Leveillula mildew resistance; three pairs of genes control the resistance to powdery mildew incited by Leveillula taurica (Oidiopsis taurica) in C. annuum 'H-V-12' | Shifris et al., 1992 |
| $lov$            |         | Loss of vesicle; nonpungency in C. chinense accession 'NMCA 30036' and PI 543190; complementation with pun1 results in pungency | Votava and Bosland, 2002 |
| $l sm$           |         | Light-sensitive mosaic; green cotyledons in normal planting; when seeds germinate in light, cotyledons and leaves are variegated; stem and fruits may develop chlorophyll-less sections and stripes; loss of chlorophyll is related to the length of light exposure; 3 to 6 d of light exposure before the full expansion of the cotyledons is sufficient to produce plantlets like xantha | Csillery, 1980a |
| $lut-1$ to $lut-18$ |         | Lutescens; cotyledon and leaves are uniformly yellowish, lighter than normal green, however, distinct accessions may represent different intensities of color; nonallelism among $lut-1$ to $lut-18$ | Csillery, 1980a, 1983, 1985 |
| $lut-19^*$       |         | Lutescens; cotyledon and leaves are uniformly yellow-green; mutant from C. annuum; allelism of $lut-19$ and $lut-1$ to $lut-19$ unknown | Ma et al., 2001 |
| $m-1$            |         | Marbled; the first true leaf develops isolated zones of white, light-green, and normal green, suggesting a marbled appearance; the boundaries of the zones are distinct; leaves are puckered with irregular margins where marbling occurs | Lippert et al., 1964 |
| $m-2$            |         | Marbled; all leaves have zones of white, light-green and green when grown in the greenhouse; when grown in the field, only the first true leaf has the marbled pattern; mutant found in 'Pasardjishka kapia 794' | Daskalov, 1974 |
| $m-3$            |         | Marbled; marbled leaves well pronounced in the open field and the greenhouse; mutant plants are reduced in size; found in 'Zlaten Medal' | Daskalov, 1974 |
| $m-4$            |         | Marbled; distinct green and white zones on foliage and immature fruits | Daskalov and Poulos, 1994 |
| $Me-1$           |         | Meloidogyne spp. resistance; M. arenaria, M. incognita, and M. javanica; resistance, but not to the 'Seville' isolate of M. species in 'PM 217' | Souza-Sobrinho et al., 2002 |
| $Me-2$           |         | Meloidogyne spp. resistance; resistant to M. javanica and the 'Seville' isolate of M. species in 'PM 217' | Souza-Sobrinho et al., 2002 |
| $Me-3$           |         | Meloidogyne spp. resistance; resistant to M. incognita, M. javanica, and most M. arenaria isolates (except M. arenaria Ain Toujdate isolate) in PI 322719 | Djian-Caporalino et al., 2001; Souza-Sobrinho et al., 2002 |
| $Me-4$           |         | Meloidogyne spp. resistance; resistant to M. arenaria Am Taoudjate isolate in PI 322719 | Souza-Sobrinho et al., 2002 |
| $Me-5$           |         | Meloidogyne spp. resistance; resistant only to M. javanica in 'Yolo Wonder' | Souza-Sobrinho et al., 2002 |
| $Me-6$           |         | Meloidogyne spp. resistance; specifically resistance to M. arenaria and M. javanica (French population) in 'Yolo Wonder' | Djian-Caporalino, personal communication |
| $Me-7$           |         | Meloidogyne spp. resistance; conferring a high level of resistance to M. arenaria, M. incognita, and M. javanica in 'CM 334' | Pegard et al., 2005 |
| $Mech-1$, $Mech-2$ |         | Meloidogyne chitwoodii resistance; Mech1 found in 'PM217'; Mech2 found in 'CM 334' | Djian-Caporalino et al., 2004 |
| $Mo A$           |         | Modifier of A; second locus for anthocyanin color, intensifies the purple color in the presence of A | Lippert et al., 1965 |

(Continued on next page)
Table 1. (Continued) The genes of Capsicum.

| Preferred symbol | Synonym | Character | References |
|------------------|---------|-----------|------------|
| Ms-1, Ms-2, Ms-3 | M1, M2, M3 | Multiple flowers per node; controls multiple flower expression; recessive homozygosity at any two loci is epistatic to the dominant allele present at the third locus | Daskalov and Poulos, 1994; Shuh and Fontenot, 1990 |
| mos-1 to mos-52 | Mosoax; cotyledons are either normal or display mosaic variegation of the leaves; mos-1 to mos-51 found in C. annuum; mos-52 in C. baccatum; nonallelism among mos-1 to mos-9 | Ceillery, 1980a, 1983 |
| ms-1 | Genic male sterility; anthers are small and shrunken, devoid of pollen grains; ms-1 gene may be linked with one of the genes involved in pigmentation; mutant from 'All Big' | Shifriss and Frankel, 1969; Shifriss and Eidelman, 1987 |
| ms-2 | Genic male sterility; shrunk anthers that release numerous, aborted pollen grains; mutant from 'California Wonder' | Shifriss and Rylski, 1972 |
| ms-3 | Genic male sterility; shrunk anthers, in some cases only a very small amount of fertile and sterile pollen is formed; irradiation induced mutant from 'Pasardjshka Kapia 794' | Daskalov, 1968 |
| ms-4 | Genic male sterility; anthers are not reduced severely, contain a small amount of fertile and sterile pollen grains; irradiation induced mutants from the variety 'Pasardjshka Kapia 794' | Daskalov, 1971, 1974 |
| ms-5 | Cytoplasm male sterility; anthers are very severely reduced, contain no pollen grains in field; under greenhouse conditions certain plants may produce a small amount of fertile pollen grains; irradiation induced mutant from 'Kalinkov 8007' | Daskalov, 1974 |
| ms-6, ms-7, ms-8 | Genic male sterility; shrunk anthers with reduced anther sizes; sometimes only a very small amount of fertile pollen is formed; nonallelic among ms-1 to ms-8; irradiation induced mutant from 'Zlaten Medal' | Daskalov, 1973b |
| ms-9 | m9 | Genic male sterility; y-irradiation induced male sterile mutants | Daskalov and Poulos, 1994; Greenleaf, 1986 |
| ms-10 | mc-509 | Genic male sterility; ethyl methansulphonate (EMS)-induced male sterile mutant; ms-10 was found allelic to the msk allele isolated spontaneously in Korea | Daskalov and Poulos, 1994; Greenleaf, 1986; Shifriss, 1995 |
| ms-11 | mc-705 | Genic male sterility; EMS-induced male sterile mutants | Daskalov and Poulos, 1994; Greenleaf, 1986 |
| ms-12* | Genic male sterility; small and shrunk anthers without pollen grains; postmeiotic breakdown of microspores; nonallelism to ms-1 and ms-2; allelism with Daskalov's ms-alleles unknown; mutant from 'Gambo' | Shifriss, 1973 |
| ms-13* | ms | Genic male sterility; complete pollen sterility; the postmeiotic breakdown of microspores; mutant from 'CA452-1' | Meshram and Narkhede, 1982 |
| ms-14* | Genic male sterility; androecium transformed into petaloid structures; mutant from 'Kalyanpur selection' | Pathak et al., 1983b |
| ms-15* | ms | Genic male sterility; anthers are dark blue, and reduced 50%; postmeiotic breakdown of the microspore during the formation of male gametes; mutant from 'CA-960' | Meshram et al., 1992 |
| msc-1* | Genic male sterility; spontaneous mutant found in China; allelism with ms-1 to ms-15, and msk unknown | Yang, 1981; Yang et al., 1994 |
| msc-2* | Genic male sterility; spontaneous mutant found in China from 'Ying Ge Bai Er'; allelism with ms-1 to ms-15, msk-1 and msk unknown | Fan and Guo, 1994; Fan et al., 2004 |
| msk | N | Root-knot nematode resistance; resistance to M. incognita in 'Santaka' | Shifriss, 1973 |
| nf | | Nonflowering; no flowering throughout the growing season | Hare, 1957 |
| nl* | Narrow leaf; narrow leaves; reduced branches, flowers, and fruits; induced from 'PC 1' | Pathak et al., 1985 |
| O | Oblate fruit shape; fruit shape independent of fruit size; modifiers also affect fruit shape A, O, and sw1 are linked, the linear order and the map distances are A-6.5-O-18.8-sw1 | Peterson, 1959 |
| P | D | Pointed fruit apex; incomplete dominant to blunt | Miller and Fineman, 1938; Lippert et al., 1965 |

(Continued on next page)
Table 1. (Continued) The genes of Capsicum.

| Preferred symbol | Synonym | Character | References |
|------------------|---------|-----------|------------|
| p−cT, p−c2, p−c3 | Polytomatos; cotyledon number is frequently three to four; the stem is fasciated; sometimes after three to four nodes a pseudo-dichotomous branching with unequally developing of shoots may occur; all three are nonallelic | Csillery, 1980a |
| p−c | Petaloid calyx; the distal part of the calyx tube is transformed to a white corollalike tube and abscised after anthesis | Csillery, 1983 |
| Peli** | PED | The acute shape of the fruit pedicle attachment; dominant to the bulged shape; reassignment for PED | Ishikawa et al., 1998 |
| Pf | Parthemocarpic fruit; fruit size reduced; ovules began to degenerate 2 d after flower opening, and completely degenerate after 5 d; pollen fertility highly reduced | Pathak et al., 1983c |
| Pfr* | Phytophthora capsici foliar resistance; resistance to foliar rot in 'CM 334' | Walker and Bosland, 1999; Sy et al., 2005 |
| Pfr* | Fr | Phytophthora capsici fruit resistance; resistance to fruit rot in 'Waxy Globe'; reassigment for Fr | Saini and Sharma, 1978 |
| Ph | Procumbent hypocotyls; seedlings have a constriction below the cotyledonary node marked with a bright-green ring; at two to three leaf stage the hypocotyl bends down, the stem touching the ground; only the shoot tip turning upward; found in an interspecific hybrid between C. chinense and C. annum | Csillery, 1980a |
| pl | v | Plastid instability; green and white leaf variegation | Lippert et al., 1965 |
| pl-1 | Pl | Pallid lutescens; pale-yellow leaf color | Csillery, 1985 |
| Pn1 | Pn | Potyvirus necrotic; confers the system lethal necrotic response for specific resistance to PVY (0) in 'CM 334' | Dogimont et al., 1996 |
| Ppr | Proliferous plant; stamens are transformed into petals followed by equally transformed carpels, the latter form a closed structure, almost as a pistil, but inside the laminar structures are continuously formed petaloids and carpelloids | Csillery, 1983 |
| Ps | Pod separates easily from calyx; incomplete dominant; gene expressivity can be modified by genes controlling fruit shape | Greenleaf, 1986; Smith, 1951 |
| Psr* | Phytophthora stem rot resistance; resistance to stem rot in 'CM 334' | Sy et al., 2005 |
| psu-1 | c | Nonpungency; absence of capsaicinoids in fruit | Deshpande, 1935; Daskalov and Poulos, 1994 |
| prv-1 | C, C', C, C', C, C' | Potyvirus resistance; locus for resistance to TEV and PepMoV in 'PI 152225' and 'PI 169236' | Boiteux et al., 1996; Greenleaf, 1956, 1986; Kyle and Palloix, 1997; Pasko et al., 1996; Zitter, 1972 |
| prv-1* | prv2, yr, vy | Potyvirus resistance; resistance to PVY (0) in 'Yolo RP10' and 'Yolo Y' | Cook, 1961a; Gebre Selassie et al., 1983; Kang et al., 2005; Kyle and Palloix, 1997 |
| prv-1** | prv2, yr, yr, vy, yp | Potyvirus resistance; resistance to PVY (1) and TEV from 'PI 264281' and 'SC46252', and bred into 'Florida VR2' | Cook and Anderson, 1959; Gebre Selassie et al., 1983; Kang et al., 2005; Kyle and Palloix, 1997 |
| prv-1† | prv2 | Potyvirus resistance; polygenic resistance to PVY from 'Perennia' is a combination of QTLs including a major-effect gene mapped to the prv-2 locus; Ayme et al. (2004) propose to name prv-2 'the major-effect gene at the prv-2 locus' | Ayme et al., 2004; Caranta et al., 1978a |
| prv-3 | vy | Potyvirus resistance; monogenic resistance to PepMoV in 'Avelar' | Kyle and Palloix, 1997; Zitter and Cook, 1973 |
| Prv-4 | cy2, yr1–2, Pr4 | Potyvirus resistance; resistance to PVY pathotypes 0, 1, and 1–2, and PepMoV in 'CM 334' | Boiteux et al., 1996; Dogimont et al., 1996; Kyle and Palloix, 1997 |
| prv-5 | pr3 | Potyvirus resistance; resistance to PVY (0) in 'CM 334' | Caranta et al., 1999; Dogimont et al., 1996 |
| prv-6 | | Potyvirus resistance; resistance to PVYM from 'Perennia'; complementary with prv-2; no detectable effect without prv-2 | Caranta et al., 1996 |
| Prv-7 | Potyvirus resistance; resistance to the PepMoV Florida (V1182) strain from C. chinense Jacq. 'PI139236–9093'; Prv-7 is tightly linked to Prv-4 with recombination frequencies of 0.012 to 0.016 | Grube et al., 2000 |
| prv-8 | Potyvirus resistance; resistance to PVY isolate P-62–81 (PVY-1) from 'CM 334' | Arnedo–Andrés et al., 2004 |
| R-1, R-2 | Purple flower color; needs comparison with A and MoA | Daskalov and Poulos, 1994 |
| Riv | Resistance to infection by virus; a dominate gene in 'Rama' controlling a quantitative effect (i.e., reduction of the probability of an effective multiplication of CMV in the leaves after mechanical inoculation); this gene also modifies the reaction to TMV in the presence of L gene | Pochard, 1982 |

(Continued on next page)
Table 1. (Continued) The genes of Capsicum.

| Preferred symbol | Synonym | Character | References |
|------------------|---------|-----------|------------|
| rf-T             | Roundleaf; reduces the length of the leaf, but not the width, changing the length-to-width ratio from 1.5 to 1.24; mutant from ‘Highhart’ | Csillery, 1983; Greenleaf and Hearn, 1976 |
| rf-2             | Roundleaf; leaf apex is markedly blunt | Csillery, 1980a, 1983 |
| rf-L*            | Round leaf; leaf tip rounded; fewer branches, flowers and fruits; seed set very poor; induced from ‘PC 1’ | Aniel Kumar et al., 2001 |
| Rsr-1*, Rsr-2*   | Ralstonia solanacearum resistance; bacterial wilt resistance in ‘Mie-Midor’ shows incomplete dominance; at least two genes involved in resistance | Matsunaga et al., 1998 |
| ru-1, ru-2       | Rugose; cotyledons fleshy and curved downward; mature leaves rugose or savoyed and appear darker green in the field | Csillery, 1983 |
| S                | Soft juicy fruit; distinct from Ps | Greenleaf, 1986 |
| sd               | Scabrous diminutive; plants similar to those of gd; foliage with a uniform, fine-textured, surface roughness; found in PI 172768 | Bergh and Lippert, 1964 |
| sel-1*           | Seedless; produces normal pollen, but ovaries are malformed and nonfunctional | Curtis and Scarchuk, 1948 |
| sel-2*           | Seedless; placenta deformed; pericarp thick and fleshy; flowering significantly delayed | Prolaram et al., 1990 |
| sl-1             | Styleless; lacking normal style or stigma; incomplete female sterility | Bergh and Lippert, 1965 |
| sl-2*            | Styleless; flowers devoid of style and stigma, ovary intact, no fruit set; female sterility; isolated from ‘Kalyanpur Red’ | Pathak et al., 1983a |
| Sm               | Smooth leaf surface; interacts with H | Shuh and Fontenot, 1990 |
| sm-1*            | Small leaf; leaves small; fewer flowers and fruits; seed set poor; partially sterile; induced from ‘PC 1’ | Aniel Kumar et al., 2001 |
| sp               | Spinach; limited stem development, numerous large leaves developing just above ground level to form a dense whorl mass; flower buds; mutant found in PI 159280 | Bergh and Lippert, 1964 |
| (S) rf-1, rf-2   | Cytoplasmic male sterility; cytoplasmic male sterility controlled by two nuclear genes and the mutant cytoplasm S; the occurrence of the dominant alleles at both loci is necessary to restore pollen fertility; the influence of the mutant cytoplasm S is expressed only in the homozygously recessive constitution of the nuclear genes | Novac et al., 1971; Peterson, 1958; Shifriss, 1997 |
| Su               | Suppressor of indeterminate growth; suppressing the epistatic action of Ct | McCammon and Honma, 1984 |
| sw1, sw2, sw3, etc | Sultry white immature fruit color; immature fruit color is conditioned by a series of genes; dominant alleles control various green shades; number of genes and gene action not clearly established | Daskalov and Poulos, 1994; Odland and Porter, 1938 |
| t                | High beta-carotene; complementary with B | Lippert et al., 1965 |
| tal*             | Tall; plant height, 65 to 80 cm; fewer branches, flowers and fruits; induced from ‘PC 1’ | Aniel Kumar et al., 2001 |
| tl               | Taphrina leaf; leaves are deformed and rugose like peach leaves infected with T. deformans; stems tend to prostrate | Csillery, 1983 |
| tru              | Transition of fruit color; fruit color transition phenotype in the jalapeño cultivar ‘NuMex Pinata’ | Votava et al., 2000 |
| Tsw              | Tomato spotted wilt virus resistance; resistance to TSWV in C. chinense PI 159253; highly effective against TSWV isolates | Boiteux and de Avila, 1994 |
| tu               | Tube; cotyledons and leaves are rolled up like a tube, only the abaxial surface is exposed; relationship to cu not determined | Csillery, 1980a; Greenleaf, 1986 |
| un               | Undulate; small dark-green leaves with undulated surface | Daskalov, 1973b |
| up-1, up-2       | Upright pedicle and fruit orientation; fruit and pedicle are upright | Lippert et al., 1965; Gopalakrishnan et al., 1990 |
| vg**             | Variegated mottled; true leaves have yellow to light-green mottling; young leaves are intensely mottled to entirely yellow, particularly near the petiole; the allelic series vg⁻ (nonvariegated) > vg+ > vg⁻ in descending order of dominance | Lippert et al., 1964 |
| vg’              | Variegated virenscent; small seedlings with yellow cotyledons; cotyledons rapidly develop green coloration, but true leaves uniformly yellow; the pattern of yellow new growth, turning nearly normal green with maturity, continued throughout the growth cycle; allelic recessive to vg** | Lippert et al., 1964 |
recessive, all letters of the gene symbol are in small letters. A gene symbol cannot be assigned to a character until supported by statistically valid segregating data (i.e., F₂ and BC populations). After validation by allelic tests, multiple alleles have the same symbol followed by a Roman letter or Arabic number superscript, and mimics may either have distinctive names and symbols, or the same gene symbol followed by a hyphen and a unique Arabic numeral or Roman letter. A modifying gene may have either a symbol for an appropriate name, such as intensifier, followed by a hyphen and the symbol of the allele affected, or a distinctive name. New gene symbols should not be assigned to preassigned symbols, nor should the same trait be described by more than one symbol. Priority in publication should be the primary criterion for establishing the preferred symbol when the same symbol has been inadver- tently assigned to different genes or more than one symbol has been designated for the same gene or genes.

**Genes Determining Morphological Traits**

**Plant height**

Nine genes have been identified that affect plant height. Daskalov (1974) found two dwarf mutants, dw-1 and dw-2, in 'Zlata Meda'. Csillery (1980b, 1983) reported two dwarf mutants from interspecific hybridizations—one from the interspecific hybridization between C. haccatum L. and C. annuum L., and the other between C. frutescens L. and C. annuum—and found that they were con- fered by dw-3 and dw-4, respectively. Re- staino (1991) identified a new dwarf mutant from the local Italian variety, 'Friarielli', that was controlled by dw-5, and was suitable for protected cultivation with high plant density. Yazawa and coworkers (1991) in Japan observed that some cultivars of C. annuum showed dwarfishness in their interspecific hybrids with C. chinense no. 3341. This dwarf- ness was incited by an interaction between nuclear genes dw-6 and dw-7 from C. annuum and the cytoplasm of C. chinense. The gene was linked 6 cM to the 610-bp RAPD marker amplified with SSU-2F (Inai et al., 1993; Yazawa et al., 1991). Aneil Kumar and colleagues (2001) found a recessive gene, dw-8, conferring the dwarf phenotype, and a recessive gene, tal, that was responsible for a tall mutant induced from C. annuum cv. PC 1.

The genetic conclusions of the plant height mutants were reached by using the mutants and the normal varieties from which these mutants were obtained. Interestingly, Todorov (1992) found that with the Romanian dwarf C. annuum variety Buketon, and the indeterminate, tall Bulgarian variety Gorogled 6, that plant height was controlled by two to three genes, with the environment having a stronger influence than genetic factors, suggesting additional genes may be involved in the genetic control of plant height.

**Flacid phenotypes**

Four kinds of flacid phenotypes have been described in Capsicum: diminished morphology (dm) (Csillery, 1983), scabrous diminutive (sd) (Bergh and Lippert, 1964), spinach (sp) (Bergh and Lippert, 1964), and flaccidity (fc) (Bosland, 2002).

**Branching habits**

The bl gene responsible for branching plants of PI 109113 was identified by Bergh and Lippert (1964). The “umbrella” branching in MSU 79-221 (C. annuum) was controlled by three major recessive genes—ct, dt, and fa—and modifiers (McCannom and Homma, 1984). Both dt and ct control plant habit; dt conditions determinate growth, whereas ct determines the number of axillary shoots. The dominant alleles, Dt and Ct, control indeterminate growth and are epistatic to one another. The fa gene conditions the fasciculate or clustered fruit-bearing habit. The dominant suppressor gene, Su, suppresses the epistatic action of Ct.

**Fasciculation**

Fasciculation in pepper is expressed as a shortening of internodes, resulting in compact, bushy plants, and expressed as flowers and fruits borne on bunched, compounded nodes conferred by the recessive fa gene (Lippert et al., 1965). Van der Beek and Litini (1990) observed the variation in fasciculation, inferring that minor genes could be involved in the expression of fasciculation, operating in the presence of the fa gene. The fa gene showed tight linkage with a distance of 3.5 cM to the dt gene (Yang and Park, 1999).

**Leaf Shape**

Leaves with an undulate surface were conferred by the recessive gene un (Daskalov,
Color of plant parts

So far, color mutants of cotyledons, leaves, and stems have been described by Kormos and Kormos (1955) and Pahlen (1966) (cited by Daskalov and Poulos, 1994). Zubrzycki and Pahlen (1974) reported a mutant, an, with golden cotyledons and leaves (cited by Daskalov and Poulos, 1994). Daskalov (1974) described a mutant, ya, with yellow anthers, but with purple spots on the stem and fruits. Later, Csillery (1980b, 1983) described color mutants of cotyledons, leaves, and stems, and showed that nine recessive genes (xa-2 to xa-10) controlled the xantha mutants; 18 genes (lut-1 to lut-18) controlled the lutecysens (yellow-green) mutants; three genes (yt-1, yt-2, and yt-3) determined yellow top mutants; 52 genes (mos-1 to mos-52) were responsible for the mosaic mutants; one gene (ism), a light-sensitive mosaic mutant; and eight recessive genes (al-1 to al-8), the anthocyaninless mutants. The lut genes are not allelic to each other, nor are the mosaic genes allelic among mos-1 to mos-9. Recently, Ma coworkers (2001) in China reported a lutecysens mutant conferred by gene lut-19. Theallelism between lut-19 and lut-1 to lut-18 was not investigated. Daskalov (1987) identified one Fvl gene determining yellow-green leaves (cited by Daskalov and Poulos, 1994). The relationship between lut and Fvl remains unknown.

An inteinitelyt complete genome, A, controls the anthocyanin color of stem, foliage, flowers, and immature fruits. The A gene is effective only in the presence of at least one A gene. In AA genotypes, the action of the A gene is intensified by a gene modifier, MoA (being ineffective alone). Additional genes for different anthocyanin accumulation in flower (R-1 and R-2), style (As), style and filament (Asf), and immature fruits (F) have been also reported. Odland (1960) found that the flower colors were conditioned by three genes (S, W, and A). The MoA locus was localized to chromosome 11 by sharing a linkage with L. confrons resistance to tobacco mosaic virus (TMV) (Ben Chaim et al., 2003). The A gene was assigned to chromosome 10 (Ben Chaim et al., 2003) and the gene Cc controlling the purple anther–filament color was assigned to chromosome 10 (Prince et al., 1993). The map position of A was identical to Cc, indicating that the two loci are allelic (Ben Chaim et al., 2003). Ben Chaim and colleagues (2003) also reported that the Cc locus was linked to a major quantitative trait locus, fsiT0.1, for fruit shape index (ratio of fruit length to fruit width).

Variegated seedlings

Six types of variegated seedlings have been reported. Hagiwara and Oomura (1947) reported the pi gene was responsible for green and white leaf variegation (cited by Lippert et al., 1965). Cook (1962) showed that the by gene was responsible for the bushy variegated mutant. Lippert and colleagues (1964) and Daskalov (1974, 1977) found four mutants with marbled leaves that were controlled by four genes: m-1, m-2, m-3, and m-4 (cited by Daskalov and Poulos, 1994). The variegated mottled mutant and variegated virencent mutant were controlled by the vg1 and vg2 genes respectively (Lippert et al., 1964). The greenish yellow variegation mutant was conditioned by the cy gene (cited by Lippert et al., 1965), and Zubrzycki and Pahlen (1974) determined that the deformed and undulated green virencent variegated leaves were conferred by the gene dvy (cited by Daskalov and Poulos, 1994).

Flowers

Multiple flowers are controlled by three major dominant genes: MF-1, MF-2, and MF-3 (Shuh and Fontenot, 1990). The expression of multiple flowers occurs when MF-1 is present and either MF-2 or MF-3 is present. Recessive homozygosity of the MF-1 locus reduces the multiple-flower nodes despite dominant genes present at both MF-2 and MF-3. Two genes affecting the time of flowering have been identified (Aniel Kumar et al., 2001): the gene ef for early flowering and the gene Pf for late flowering. Pathak and associates (1985) found a nonflowering mutant that was controlled by a monogenic recessive gene ef. Two closed-flower mutants are conditioned by the cf-1 and cf-2 genes (Pathak et al., 1983; Subramanya and Ozaki, 1984).

Fruit shapes

Six genes have been identified to affect the characteristics of fruit shape. Deshpande (1933) described the dominant gene P for pointed fruit apex, the recessive gene ph for fruit base nonbulging, and the gene ce for calyx enclosed around fruit base (Daskalov and Poulos, 1994). The round fruit shape is controlled by the major dominant gene O and other modifiers (Peterson, 1959), and recent molecular mapping studies confirm the existence of the major genes that control this trait (Ben Chaim et al., 2001, 2003; Rao and Parantho, 2003). Upright fruit orientation is controlled by two recessive genes—ap-1 and ap-2—that show specific dominant and recessive epistasis respectively (Gopalakrishnan et al., 1990; Lippert et al., 1965). Csillery (1983) identified the recessive gene, cy, responsible for the exocarp covered with transversely oriented, small suberized cracks at full fruit maturity. Ishikawa and coworkers (1998) reported the dominant Ap and Ped genes to condition the pointed shape of the fruit apex, and the acute shape of the fruit pedicel attachment respectively. The alleleism between Ap and P is unknown. Parthenocarpy is conferred by the pf gene from the variety ‘Kalynapurn Chaman’ (Pathak et al., 1983b).

Immature fruit color

Immature fruit color is conditioned by a series of alleles (Odland and Porter, 1938). The sulfur white, sw1, locus controls the various white and green shades of the fruit. The lettuce or yellowish green color, sw2, is dominant to the sulfur white, sw1, and recessive to the cedar green, sw3, whereas cedar green, sw3, is dominant to sulfur white.

Mature fruit color

Early studies found that the mature yellow color of Capsicum fruits is recessive to red and controlled by a single gene y (yellow) (Boswell, 1937). Smith (1950) found that the brown color of ripe pepper fruit was the result of the combination of the normal red pigment and the retention of chlorophyll. The brown color is governed by two genes: cl for retention of chlorophyll and y’ for red color. Hybridizations between red and white fruit varieties have shown that the mature fruit color is controlled by three genes: c-1, c-2, and y’. Smith and Hernandez (1985) reported the c-1 gene affects the amount, rather than the type, of carotenoids present, and the gene c-2 has a significant effect on the total level of carotenoid accumulation in fruit. The c-1 and c-2 genes reduced the pigmentation of y and y’ by the inhibition of beta-carotene. Popovsky and Parantho (2000) demonstrate the effect of the genetic background on fruit color segregation and provide additional evidence for two possible genotypes of the orange fruit color. In addition, the im gene was found to condition purple color at intermediate maturity in originally nonpurple immature fruit (Lippert et al., 1965).

The gene Ccs, coding for capsanthin–capsorubin synthase that synthesizes the red carotenoid pigments, corresponds to the morphological locus y (Lefebvre et al., 1998; Popovsky and Parantho, 2000). Ccs, a single-copy gene in pepper, was assigned to pepper chromosome 6, 1 cM away from CT109. A QTL affecting the intensity of mature red color was also detected in this region (Thorup
A 500-bp fragment amplified by primer OPN-7 is linked with Ccs’Y at a distance of 7 cM (Thorup et al., 2000). In addition, one QTL affecting red chroma and another affecting red lightness were identified on chromosome 3 in an intraspecific C. annuum population (Thorup et al., 2000). In the locus cosegregated with phytoene synthase, Psy (Thorup et al., 2000), which is the locus responsible for the development of fruit color. Psy/C2 may be a major gene acting as a rate-limiting factor in carotenoid production. Psy is the candidate for C2 (Huh et al., 2001). A 700-bp fragment amplified by the primer OPO-12 and a 900-bp fragment amplified by the primer UBC-183 were mapped to chromosome 4, and were found to be 1 cM apart from each other and 36 cM away from Psy/C2 (Thorup et al., 2000). A QTL affecting the red hue of fruit was also mapped to this region (Ben Chaim et al., 2001), implicating Psy as a source of both qualitative and quantitative variability in carotenoid biosynthesis.

**Transition of fruit colors**

Votava and associates (2000) noticed that the jalepeno cultivar, NuMex Piñata, had a unique transition of colors as the fruits matured, from light green in immature fruits, maturing to yellow, then orange, and finally to red. It was determined that the inheritance of luteous foliage color and fruit color transition phenotype of ‘NuMex Piñata’ is the result of a single homozygous recessive gene (transition).

**Genes Determining Physiological Traits**

**Pungency**

Pungency, or the sensation of heat when eating pepper fruit, comes from the presence of the capsaicinoid alkaloids in the fruit. Early genetic studies reported that a single dominant gene C controlled the expression of heat (Deshpande 1935, Greenleaf, 1952), which can be modified by other genetic and environmental factors. This gene was later redesignated Pun (Daskalov and Poulos, 1994), and probably functions as an acyltransferase to complete the capsaicinoid synthesis (Stewart et al., 2005). Loaiza–Figureo and Tanksley (1988) reported a second non-pungency locus, pun2, in a wild, nonpungent pepper accession BG 3547; however, further studies cannot support the existence of this locus—most likely the nonpungent accession was expressing the low gene (Votava and Bosland, 2002). Votava and Bosland (2002) described two novel nonpungent C. chinense accessions ‘NMCA 30036’ and ‘PI 5431900’ in which loss of the vesicles (lov) on the placental walls is responsible for nonpungency. The lov gene is epistatic to pun1 and results in pungent progenies. Blum and associates (2002) mapped the Pun locus to chromosome 2 in a hybridization between a pungent C. frutescens accession and a non-pungent C. annuum bell pepper, and developed a polymerase chain reaction-based cleaved amplified polymorphic sequence (CAPS) marker linked to Pun using the sequence of the Capsicum fibrillum gene located 0.4 cM from Pun. Recently, the Pun-1 gene was cloned and characterized based on the candidate gene approach (Stewart et al., 2005). The full-length genomic sequence of Pun-1 is 1897 bp, containing two exons of 738 bp and 585 bp respectively, and one intron of 348 bp. The Pun-1 allele results from a large deletion at this locus.

**Beta-carotene contents**

Three genes—B, b, and bc—have been identified that confer high beta-carotene contents of ripe fruits. Chalukova and colleagues (1993) and Daskalov and coworkers (1995) described the mutant variety Orangeva Kapia obtained from ‘Pasardinzhiska Kapia’. This mutant variety produces immature green fruits and mature orange fruits containing 2 to 2.5-fold higher contents of beta-carotene than their red counterparts. High beta-carotene content in this mutant variety is conferred by a recessive gene, bc, which prevents hydroxylation of beta-carotene to beta-cryptoxanthin.

**Soft flesh and deciduous fruits**

Smith (1951) found the deciduous ripe fruit character to be controlled by a single dominant gene, S. Later, Jeswani and associates (1956) obtained similar findings for this trait (cited by Greenleaf, 1986). Kornos and Kornos (1957) reported that the soft-fruited trait was controlled by the dominant gene Ps (cited by Greenleaf, 1986). The genetic reports of these two traits were independent, so the two traits were considered as distinct from each other (Daskalov and Poulos, 1994; Greenleaf, 1986). In the gene lists of Daskalov and Poulos (1994), and Greenleaf (1986), the gene symbol S given by Smith (1951) to the deciduous fruit trait was assigned to the soft flesh trait, and the gene symbol Ps was reassigned to the pod separation trait and is considered distinct from S. However, the fruits of the wild C. frutescens accession ‘BG2816’ were both deciduous and had soft flesh, and these two traits cosegregated in the F2 progeny of ‘BG2816’ and the cultivar Maor (C. annuum). Therefore, it is likely that both characteristics in C. frutescens BG2816 are controlled by the same gene with pleiotropic effects (Rao and Parand, 2003). S is mapped to chromosome 10, and the polygalacturonase (PG) gene PG is a candidate gene for S (Rao and Parand, 2003).

**Genes Determining Sterility Traits**

**Genic male sterility**

Nearly 20 genes for genic male sterility have been reported. Shifriss and Frankel (1969) named the first male sterile gene ms-1 in a spontaneous stable male sterile mutant from the C. annuum cv. All Big. A second male sterile gene, ms-2, was identified in a spontaneous stable male sterile mutant from ‘California Wonder’ (Shifriss and Rylish, 1972). Daskalov (1973b) identified five genes (ms-3, ms-4, ms-6, ms-7, and ms-8) conferring male sterility in the irradiation-induced mutants. These seven nonallelic genes are mainly expressed as pollen sterility. Pochard found three genes (ms-9, ms-10, and ms-11) in three male sterile mutants obtained after treatment of monoploid materials with ethyl methanesulphonate (EMS) (cited in Daskalov and Poulos, 1994). Shifriss (1973) reported a recessive gene, ms-12, governing the male sterility in a spontaneous mutant from C. annuum cv. Gambo. Meshram and Narkhede (1982) identified the ms-13 gene that conditions male sterility in a mutant from C. annuum cv. Ca452–1. Pathak et al., (1983) isolated a male sterile mutant from cv. ‘Kalyanpur Selection’, and proved that male sterility was governed by the single recessive gene ms-14. In India, Deshpande and coworkers (1983) presented more than 20 male sterile mutants in India, whereas in China two genic male sterile genes (ms-1 and ms-2) have been used to produce F1 sweet pepper hybrids (Yang, 1981; Yang et al., 1994). Daskalov (1987) also reported a dominant genetic male sterility gene: Dms (cited in Daskalov and Poulos, 1994).

According to morphological changes exhibited in androecium, the male sterile mutants can be grouped into six categories (Deshpande et al., 1983): 1) androecium transformed into petaloid structure (ms-13); 2) shriveled anthers devoid of pollen grains, including ms-1, ms-3, ms-6, and ms-8 (Daskalov, 1974); 3) anthers that are not reduced severely and contain a small amount of fertile and sterile pollen grains in some flowers (ms-4 and ms-7) (Daskalov, 1974); 4) shrunken anthers that release numerous aborted pollen grains (ms-2) (Shifriss and Rylish, 1972); 5) anthers that appear to be normal, but pollen grains are sterile; and 6) yellow anther lobes that are flattened laterally to give an appearance of a fan blade and are devoid of pollen grains (Deshpande et al., 1983).

**Cytoplasmic male sterility (CMS)**

The inheritance of CMS in C. annuum was first studied by Peterson (1958), who showed that sterility was controlled by a major gene, ms, interacting with a specific S plasma type to generate (S) msms CMS plants. He also suggested that the second allelic pair was probably in another linkage group. Novac and colleagues (1971) worked with Peterson’s male sterile material and found that the male sterility was conditioned on two pairs of nonallelic nuclear genes. Both the nuclear genes ms-1 and ms-2 have complementogenic genetic interaction. The presence of at least one dominant allele at both loci is necessary to restore the pollen fertility in the plants with the S cytoplasm. Daskalov (1974) identified a CMS mutant from the variety ‘Kalinkov 800/7’ that is not identical with Peterson’s (1958) CMS line, and found that this male sterility was probably conferred by more nuclear genes, including the gene originally designated ms-5, and the S factor in the cytoplasm. In China and Korea, some CMS lines have been used to produce commercial F1 hybrids (Wang et al., 2003), but the
relationship between these CMS lines and Peterson’s (1958) CMS line has not been evaluated.

Shiffriss (1997) suggested the nuclear gene conferring CMS be redesignated \( R_f \) and the restorer of fertility allele as \( R_f \) to differentiate between the \( ms \) genes in genic male sterility lines and the male sterility genes interacting with \( N \) and \( S \) cytoplasm that were originally designated as \( ms \) genes (Daskalov, 1974; Peterson, 1958). The \( ms \) genes are nonallelic to the \( rf \) ones. Two RAPD markers tightly linked to a major fertility restorer gene were detected (Zhang et al., 2000): OP13\(_{Rf} \) with a genetic distance of 0.37 cM, and OW19\(_{Rf} \) on the opposite side with a distance of 8.12 cM. Recently, Kim and Kim (2005) developed two CMS-specific sequence-characterized amplified region (SCAR) markers for early identification of CMS genotype based on the restriction-length polymorphisms between male fertile and CMS cytoplasmic DNA at the \( msd \) and \( apet6 \) loci of the mitochondrial DNA of \( C. annuum \).

### Functional male sterility

Yuan and Li (2000) reported a spontaneous functional male sterile mutant from the cultivar Fudijian (\( C. annuum \)) and determined that this functional male sterility was controlled by a recessive gene: \( fms \).

### Female sterility

Six types of female sterile mutants have been reported: 1) the female sterility mutant conferred by the recessive \( f_s \) gene (Bergh and Lippert, 1964); 2) two female and male sterility mutants conferred by recessive \( fms \) and \( fM \) genes respectively (Aniel Kumar et al., 2001; Martin and Crawford, 1951); 3) a female sterile mutant with 27% to 55% pollen fertility conditioned by the recessive \( f_l \) gene (Pahlen, 1967; cited by Daskalov and Poulos, 1994); 4) two styleless mutants controlled by recessive genes \( sl-I \) and \( sl-2 \) (Bergh and Lippert, 1965; Pathak et al., 1983b); 5) two seedless mutants conditioned by recessive genes \( sel-1 \) and \( sel-2 \) (Prolaram et al., 1990); and 6) ‘complex’ mutants. The genes \( sp, bl, ft-1, prp, \) and \( pec \) have marked effects on both vegetative organs and female fertility (Bergh and Lippert, 1964; Caillery, 1983). Allelisms between \( sl-I \) and \( sl-2 \) are unknown. A conditional female sterility gene, \( cfs \), was identified by Daskalov and Mihailov (1988).

### Genes Determining Resistance to Diseases, Nematodes, and Herbicides

#### Resistance to tobacco mosaic virus (TMV)

Holmes (1937) found that resistance to TMV was controlled by a series of multiple alleles: \( L \) (localization of TMV), \( L' \) (imperfect localization of TMV), and \( L'' \) (motiling), with \( L > L' > L'' \). Boukema et al. (1980a) determined that the resistance of 10 \( C. chilense \) accessions (\( PI 152225, PI 159236, PI 315008, PI 315023, PI 315024, PI 159223, PI 213917, PI 257117, PI 257284, \) and \( PI 224424 \)) was inherited monogenically and was partially dominant, and that the resistant genes in these accessions appeared to be allelic and were allelic with the alleles \( L \) and \( L' \). She designated the symbol \( L' \) for the new allele, and redesignated Holmes’s allelic series \( L' > L'' > L' > L'' > L' > L'' \). Later, Boukema (1984) reported that the \( L' \) allele originated from \( C. chacoense \) \( PI 260429 \) and \( SA185 \). Recently, Salomon and associates (2001) revealed the resistant allele \( L'^{2} \) in ‘Gregyo’, which behaved like the allele \( L' \) with characteristics of \( C. frutescens \) cv. Tabasco. The identity of the allele \( L'^{2} \) relative to \( L' \) is not known.

However, the conferred resistance of \( L \) locus lessens when the air temperature surpasses 30 °C (Palloix, 1992). Daubeze and colleagues (1990) revealed the presence of secondary genes that stabilize the expression of \( L' \) under high temperature (32 °C). Such modifier genes were found in Chinese accesses (‘Zao Feng’ and ‘Ben Xi’ with \( L' \), but also in tropical varieties susceptible to TMV (‘Perennial’ and ‘PI 322719’). The Mexican variety Criollo de Morelos 334 (CM 334) also bears a particular allele at the \( L \) locus that confers resistance to TMV (0) at high temperatures, and secondary genes that control the rapid production of smaller local lesions in the inoculated organs (Palloix, 1992).

Zatýko and Moor (1998) confirmed a close linkage between the \( al \) and \( l' \) genes in the pepper line ‘TL 791’. SCAR marker WA31–15005 was linked to the \( L' \) gene within a distance of 1.5 cM (Matsunaga et al., 2003).

### Resistance to cucumber mosaic virus (CMV)

Resistance to CMV has showed the characteristics of genetic diversity (for a review see Wang et al., 1996). Some accessions have the recessive resistance gene (Cook, 1982; Herison et al., 2004; Singh and Thakur, 1977), some have oligoigenes (Herison et al., 2004; Saito et al., 2004), and others carry polygenic resistance (Greenleaf, 1986; Pochard et al., 1983). Singh and Thakur (1977) confirmed the presence of resistance to CMV in the line ‘Perennial’, and designated the gene symbol \( cm \). Resistance in ‘Perennial’ was under polygenic control (Gil-Ortega and Arteaga, 1988), and four QTLs were significantly associated with resistance to CMV in this line, among which the QTL-controlling percentage (16%–33%) of the observed phenotypic variation was linked to the \( L \) locus that confers resistance to TMV (Ben Chaim et al., 2001). Pochard (1982) designated three resistant components of partial resistance to CMV, (i.e., tendency to escape infection, restriction of viral multiplication, and slowing of viral migration in the resistant reaction to CMV in different cultivars). Later, researchers added four more resistance components—namely, the ability to recover from systemic infection (Pochard and Daubeze, 1991), resistance of virus installation in host cells (Caranta et al., 1997b), restriction of virus multiplication in the whole plant (Nono–Wondim et al., 1993), and restriction of long-distance virus movement (Caranta et al., 2002). Three QTLs significantly affecting restriction of CMV installation in host cells were detected, and each QTL from ‘Perennial’ was associated with an increased resistance (Caranta et al., 1997a). Partial restriction of CMV long-distance movement in ‘Vanita’ is inherited as a dominant trait, and several QTLs, including one major-effect and several minor-effect QTLs, were associated with this resistance (Caranta et al., 2002). The genes for resistance to CMV are not linked with those genes conferring fruit weight and size (Shiffriss and Cohen, 1987).

### Resistance to potyvirus

Pepper may be infected by six major potyviruses: potato virus Y (PVY), tobacco etch virus (TEV), pepper mottle virus (PepMoV), pepper veinal mottle virus (PVMV), chile veinal mottle virus (CVMV), and potyvirus E (PVE) (Green and Kim, 1991). PVY isolates have been traditionally classified into three pathotypes (\(-1, -1\), and \(-1.2\)) (Cook, 1963; Gobre Selassie et al., 1983). Recently, a new pathotype has been described and provisionally named as PVY-PRW (Luis Artega et al., 1997). The gene designations for resistance in \( C. annuum \) to three potyviruses (PVY, TEV, and PepMoV) have not followed regular convention, so confusion has occurred. Kyle and Palloix (1997) proposed a nomenclature that clarifies the genetic and biological relationships among potyvirus resistance genes in \( C. annuum \). They proposed the symbol \( prv \) for the potyvirus resistance locus. Until now, eight major resistance genes (\( prv \) genes) and several QTLs showing phenotypically distinct types of responses to potyvirus. The \( prv-1 \) gene is the locus with alleles for recessive resistance to TEV and PepMoV in ‘\( PI 152225 \)’ (Greenleaf, 1956) (originally \( er' \) and \( er'' \) and ‘\( PI 169236 \)’ (Greenleaf, 1986) (originally \( er' \) and \( er'' \) ). These \( PI \) accessions are allelic for PepMoV resistance and for TEV resistance (Murphy et al., 1998). An allelic series at the \( prv-2 \) locus controls the recessive resistance to PVY, Tabasco, and Palloix (1997). For resistance to PVY (0), the resistance allele \( prv-2 \) replaces \( cr \) and \( vv \) from ‘Yolo RP10’ and ‘Yolo Y’ respectively, (Cook, 1960, 1961a; Gobre Selassie et al., 1983). For resistance to PVY (1) and TEV derived from ‘\( SC46252 \)’ and introduced to ‘Florida VR2’, \( prv-2 \) replaces \( cr' \), \( cr'' \), and \( vv' \) (Cook and Anderson, 1959; Gobre Selassie et al., 1983).

In addition, a polygenic resistance to PVY from ‘Perennial’ was dissected into a combination of QTLs, including a major-effect gene that was mapped to the \( prv-2 \) locus (Caranta et al., 1997a). Ayne and colleagues (2004) proposed the name \( prv-2 \) for this major-effect gene at the \( prv-2 \) locus. Both \( prv-2 \) and \( prv-2 \) alleles are shown to correspond to the eukaryotic translation initiation factor 4E (eIF4E) (Rufleff et al., 2002). Recently, genetic complementation analysis demonstrated the interaction of \( prv-2 \), \( prv-2 \), and \( prv-2 \) (Kang et al., 2005; Rufleff et al., 2004). Hence, \( prv-2 \) and \( prv-2 \) have been redesignated \( prv-1 \) and \( prv-1 \) respectively.
(Kang et al., 2005). The third locus, pvr-3, was proposed for an allele for monogenic recessive potyvirus resistance to PepMoV in *C. annuum* 'Avalera' (Zitter and Cook, 1973).

Several resistance genes against potyviruses have been described in CM 334; the *Pvr-4* locus was named for a gene that gives resistance to a known potyvirus isolate of PVY and to PepMV in *C. annuum* CM 334 (Boiteux et al., 1996; Dogimont et al., 1996; Palloix, 1992; Pasko et al., 1992). Pasko and coworkers (1992) described several levels of resistance in CM 334 against different PVY pathotypes. CM 334-derived materials show different responses, indicating that, besides the *Pvr-4* allele, other genes conferring resistance at different levels are also present (Arnedo-Andres et al., 1998). The gene conferring only the resistance to PVY (0) in CM 334 was designated *pvr-3* (Dogimont et al., 1996).

The gene *Pvr-1* confers the necrotic response to PVY (0, 1, 2) isolates in 'Mat'. The genetic control for the appearance of systemic necrotic symptoms in 'CM 334 (11)' after inoculation of PVY (1–2) isolate P-22–88 is the result of a codominant gene expressed only when *Pvr-4* is not present and with a maximum expression that is observed in the homozygous condition (Arnedo-Andres et al., 2004).

A recessive gene for resistance to PVVMV from 'Perennial' is designated *pvr-6* (Caranta et al., 1996). The gene *pvr-6* is complementary to the *pvr-1* locus for resistance to PVVMV. The dominant gene, *Pvr-7*, from *C. chinense* Jacq. 'PI 159263', confers resistance to PepMoV Florida (V1182) strain (Grube et al., 2000). This gene is tightly linked to the dominant potyvirus resistance gene, *Pvr-4*, with observed recombination frequencies of 0.012 to 0.016. Resistance to PVY in the A97011 line, derived by intraselection from CM 334, is controlled by a recessive and independent gene: *pvr-8* (Arnedo-Andres et al., 2004).

Caranta and Palloix (1996) found the polygenic resistance of 'Perennial' to PVY and PVE consists of a combination of isolate-specific and broad-spectrum QTLs. In their studies, 11 chromosomal regions were determined to be associated with quantitative resistance in 'Perennial' to PVY and PVE. QTLs for potyvirus resistance, in some cases, coincide with positions for *pvr* loci. QTLs for PVY and PVE resistance are detected in the vicinity of the *pvr-2* and *pvr-6* loci. These results suggest a possible allelism between major genes and QTLs.

The modes of action of these major resistance genes have been determined. *pvr-2* and *pvr-2* both control a complete inhibition of virus accumulation in infected cells (Deom et al., 1997); *pvr-2* impairs cell-to-cell movement (Ponz et al., 1994; Arroyo et al., 1996) whereas *pvr-3* slows long-distance movement (Murphy and Kyle, 1995). Interaction between the potyvirus genome-linked protein (VPg) and eIF4E are important for PVY virus infectivity, suggesting that the *pvr-2* recessive resistance could be the result of incompatibility between the VPg and eIF4E in the resistant genotype (Ruffel et al., 2002). Mouri and associates (2004) point out that 15 nucleotide changes corresponding to five putative amino acid differences in the same region of the VPg of PVY affected virulence toward the *pvr-2* and *pvr-2* resistance. Both the *Pvr-4* and *pvr-3* control a complete inhibition of potyvirus replication or accumulation. This mechanism is pathotype specific when controlled by *pvr-5*, whereas it is effective against three different pathotypes and against PepMoV as well when controlled by *Pvr-4* (Caranta et al., 1998).

The *pvr-1* locus, linked with tomato marker TG56, has been genetically mapped to a small linkage group with synteny to the short arm of tomato chromosome 3 (Murphy et al., 1998). Yeam and colleagues (2005) developed the allele-specific CAPS markers based on point mutations in resistance alleles at the *pvr-1* locus encoding eIF4E in *Capsicum*. Arnedo-Andres and associates (2002) identified one RAPD marker (UBC191425) linked in repulsion phase to *Pvr-4* and converted it into a dominant SCAR marker (SCUB191425). Caranta and associates (1999) mapped eight amplified fragment length polymorphism (AFLP) markers in an interval from 2.1 ± 0.8 to 13.8 ± 2.9 cM around the *Pvr-4* locus, and converted the closest codominant AFLP marker into a dominant CAPS marker.

**Resistance to tomato spotted wilt tospovirus (TSWV)**

The hypersensitive resistance to TSWV was determined by a single gene, *Tsw*, in *C. chinense* Jacq. accessions (PI 152225, PI 143219, PI 163192, PI 163193, PI 163195, and PI 235047) (Cook and Guevara, 1982, 1984; Cook and Stall, 1963; Hibberd et al., 1987; Kim and Hartmann, 1985; Sahin and Miller, 1997). The *Bs-2* gene from *C. chacoense* specifically recognizes and confers resistance to strains of Xcv that contain a virulence gene *avrB2* (Minsavage et al., 1990). The *Bs-2* gene is a member of the nucleotide binding site leucine-rich repeat class of plant disease resistance genes (Tai et al., 1999a), and is predicted to reside in the plant cytoplasm.

Recently Jones and colleagues (2002) found a nonhypersensitive resistance to race 6 in three pepper genotype ECW-12346 that was developed with bacterial spot resistance derived from 'Pep13', PI 271322, and ECW123 (Early CalWonder containing *Bs-1*, *Bs-2*, and *Bs-3* genes), and identified that two recessive genes—*bs-5* and *bs-6* derived from PI 271322 and ‘Pep13’ respectively—determined this resistance. ECW12346 inhibits the population buildup of this Xcv strain without inducing the typical hypersensitive reaction. Hungarian scientists described another type of nonhypersensitive resistance against Xcv in *C. annuum* PI 163192, which used a general defense system of the host plant (Caillère et al., 2004; Szarka and Caillère, 1995). This resistance is regulated by a recessive gene: *gds* (general defense system). This gene localizes the pathogen with an entirely different strategy than the hypersensitive reaction. The resistance conditioned by *gds* promotes cell growth and cell wall thickening. The instability of avirulence genes in Xcv has stimulated the interest for quantitative resistance. Accession CNPH703 was identified with a race nonspecific and nonhypersensitive resistance to Xcv, and low narrow-sense heritability for three components of resistance: lesion number, lesion diameter, and total lesion area (Poulos et al., 1991). Poulos and colleagues (1992) determined that dominance, additive, and interacting gene effects of at least two genes were involved in the inheritance of quantitative resistance to Xcv in *C. annuum*.

Tai and associates (1999b) identified one AFLP marker, A2, co segregated with the *Bs-2* locus, and two other markers, F1 and B3, flanking the *Bs-2* locus and being within 0.6 cM. Also, two RAPD markers, OPD05 and OPF10, were identified to link with the *Bs-2* gene (Kim et al., 2001). OPD05 was located 5.3 cM from *Bs-2* gene whereas OPF10 was located 4.9 cM from the *Bs-2* on the opposite
Resistance to phytophthora

Phytophthora capsici Leon. causes several disease syndromes (i.e., phytophthora root rot, stem rot, foliar rot, and fruit rot) (Sy et al., 2005; Walker and Bosland, 1999). Resistance to Phytophthora root rot exhibited the characteristics of genetic diversity (for a review see Wang et al., 1995). A single dominance gene was present in some accessions (Choi et al., 1990; Kim and Hur, 1992), oligogenes in some accessions (Cristinzio et al., 1992; Gil Ortega et al., 1991, 1992; Reifschneider et al., 1992), and quantitative genes in others (Thabuis et al., 2003). One dominant gene was necessary for resistance in CM 334 when the susceptible parent was ‘Early Jalapeno’ (Sy et al., 2005; Walker and Bosland, 1999).

A single dominant gene, Psr, conferred stem resistance in ‘Crillo de Morelos-334’ when choosing ‘Early Jalapeno’ as a susceptible parent (Sy et al., 2005). A dominant gene, Plo, was required for foliar resistance in CM 334 when the susceptible parent was ‘Early Jalapeno’ (Sy et al., 2005; Walker and Bosland, 1999). Although when the susceptible parent was ‘Keystone Resistant Giant no. 3’, at least one dominant gene must be expressed for both root rot and foliar blight resistance besides a dominant gene required for foliar resistance and a different dominant gene required for root resistance (Walker and Bosland, 1999). However, the allelism tests were not determined. Resistance to Phytophthora fruit rot was controlled by the dominant gene Pfr in ‘Waxy Globe’ (Saini and Sharma, 1978).

Recently, six chromosomal regions (Phyto4.1, Phyto5.1, Phyto5.2, Phyto6.1, Phyto11.1, and Phyto12.1) were identified to be involved in one or more components of resistance to P. capsici (Thabuis et al., 2003). The Phyto5.2 QTL may be widely distributed in highly resistant accessions. SCAR marker Di04.717 located in chromosome 5 was tightly linked with Phyto5.2 (Quarin et al., 2005).

Resistance to anthracnose

Resistance of C. annuum cv. Chirongyung to Colletotrichum dematium was conferred by a dominant allele, Anr1, when the resistance was measured as a lesion with a diameter less than 18.1 mm (Park et al., 1992). The dominant genes were present for resistance to C. gloeosporioides: a single gene, Anr-2, in the accession BGH3077; and two genes, Anr-3 and Anr-4, in BGH2850 and BGH15085 (Fernandes and Ribeiro, 1998). One dominant gene, Anr-5, was responsible for the resistance to anthracnose incited by C. capsici in the line ‘83–168’ at four days after inoculation (Lin et al., 2002). Allelism among Anr gene series is unknown.

Voorrips and colleagues (2004) scored three resistance-related traits for anthracnose in an F2 population derived from the susceptible C. annuum cv. Jatilaba and the resistance C. chinense accession PR195030. When fruits are inoculated with C. gloeosporioides, one main QTL, B1, was identified on all three traits, and three other QTLs were detected for overall lesion diameter and true lesion diameter, of which two also had an affect on infection frequency. When fruits are inoculated with C. capsici, the marker B1 was found for overall lesion diameter when marker G1 was used as cofactor in multiple QTL analysis. No significant QTLs were detected for two other traits.

Resistance to ralstonia solanacearum

The bacterial wilt resistance in the sweet pepper variety ‘Mie-Midori’ showed incomplete dominance, and at least two genes were involved in resistance (Matsunaga et al., 1998).

Resistance to powdery mildew

Leveillula taurica causes powdery mildew of pepper. The line H-V-12 immune to L. taurica races from Israel depends on three pairs of genes: lmr-1, lmr-2, and lmr-3 (Shifriss et al., 1992). Daubeze and associates (1991) determined that at least three genes appeared to control resistance in the Ethiopian variety H3 to L. taurica. Later, Lefebvre and coworkers (2003) detected seven genomic regions, including additive QTLs and epistatic interactions contributing to the resistance of the variety H3.

Resistance to root knot nematodes (Meloidogyne spp.)

There are four genetically important root-knot nematode species: Meloidogyne incognita (Kofoid and White) Chitwood, M. arenaria (Neal) Chitwood, M. javanica (Treub) Chitwood, and M. hapla Chitwood. As early as 1957, Hare (Fery and Harrison, 1990) identified a dominant gene, N, for resistance to M. incognita acrita in the C. annuum ‘Sanatka XS’ line. Expression of the N gene in bell pepper is modified and decreased at temperatures more than 28°C (Thies and Fery, 1998, 2002). Cytoplasmic factors are not involved in expression of N-type resistance. The resistance of ‘Carolina Hot’ to M. incognita is conditioned by two genes: one dominant allele to the dominant resistance gene N, and one recessive (Fery and Dukes, 1996). The resistance to M. incognita in C. chinense ‘PA-426’ is conditioned by a single dominant gene that is allelic to the dominant gene N in the C. annuum ‘Carolina Cayenne’ (Fery and Thies, 1998). Resistance to M. arenaria race 1 in C. chinense lines PA-353 and PA-426 was conditioned by a single dominant gene that is allelic to a resistance gene in C. annuum ‘Carolina Cayenne’ (Fery and Thies, 2000). However, the allelism tests did not demonstrate conclusively that the M. arenaria race 1 resistance gene in C. chinense is the N gene in C. annuum. Five main resistance dominant genes, Me-1 to Me-5, in C. annuum line PM217 (PI 201234) and PM687 (PI 322719) were identified by Hendy et al. (1985) to confer resistance to Meloidogyne spp. (cited by Dijian–Caporalino et al., 1999, and Souza–Sobrinho et al., 2002), all acting individually in a gene-for-gene interaction. The Me-6 gene specifically controlled resistance to M. arenaria and M. javanica (French population in ‘Yolo Wonder’ (Dijian–Caporalino, personal communication). Me-7 was found in CM 334, conferring a high level of resistance to M. arenaria, M. incognita, and M. javanica (Pegard et al., 2005). Two other loci, Mech-1 (in ‘PM217’) and Mech-2 (in ‘CM334’), were identified to control the resistance to M. chitwoodi (Dijian–Caporalino et al., 2004). Among them, three dominant and thermostable loci with broad-spectrum resistance (Me-3, Me-1, and Me-7), Mech-1, and Mech-2 suppressed nematode reproduction (Dijian–Caporalino et al., 1998, 1999, 2004). It appears that the N gene and the Me-3 gene both confer higher resistance than the Me-1 gene (Thies, 2004).

Fine mapping with AFLP markers flanked the resistance genes in coupling, and the nearest marker was located less than 2 cM from Mech-1, 3 cM from Me-1, and 1 cM from Mech-2 (Dijian–Caporalino et al., 2004). The Me-3 nearest AFLP marker was 10.1 cM from a RAPD marker Q4Q_0.3 and 2.7 cM from RFLP marker CT135. Me-4 was linked 10 cM to Me-3 (Dijian–Caporalino et al., 2001). The genes Me-3, Me-1, Me-7, Mech-1, and Mech-2 are effectivly different, but linked, and were all assigned to chromosome P9 (Dijian–Caporalino et al., 2004).

Bentazon herbicide tolerance

The dominant gene Bzt is responsible for a high level of tolerance to the herbicide, bentazon, in C. annuum (Fery and Harrison, 1990; Wolff et al., 1992). A possible cytoplasmic involvement was present in the expression of the Bzt gene in the Santaka cultivar, and modifying genes affected the major gene, Bzt, controlling tolerance in the Bohemian Chilli cultivar.

Conclusions and Perspectives

Capsicum genetics have been extensively studied for nearly a century, and Capsicum breeding has benefited greatly from this knowledge. Some characteristics/trait appearances in the Capsicum genetic literature are not included in this list because complete inheritance data were provided. Many gene mutants or lines are not available. Hence, a need to have genetic stocks deposited and maintained in a repository for further research is warranted. Unfortunately, resources for a Capsicum Gene Stock Center are not available. Nevertheless, the Chilli Pepper Institute at New Mexico State University maintains some of the mutants and makes them available to bonified Capsicum
Scientists. Future genetic exploration of *Capsicum* genes will include studying the allelic relationship among the similar genes, obtaining more gene mutants by the application of mutagens, developing a set of chromosome location lines using information from the tomato genome sequencing project, establishing linked molecular markers to genes, and characterizing gene function.

**Literature Cited**

Aniel Kumar, O., V. Anitha, K. Roseline Subbathini, and K.G. Raja Rao. 2001. Induced morphological mutations in *Capsicum annuum* L. Capsicum Eggplant Nswl. 20:72–75.

Arnedo-Andres, M.L., M.L. Arteaga, and R.G. Ortega. 1998. Response of ‘Serrano Criollo de Morelos-334’ to PVY pathotypes. Proc. 10th Eucarpia Meeting on Genetics and Breeding of *Capsicum* and Eggplant, 7–11, Sept. 1998, Avignon, France, 105–109.

Arnedo-Andres, M.L., M.L. Arteaga, and R.G. Ortega. 2004. New genes related to PVY resistance in *C. annuum* L. Capsicum Eggplant Nswl. 20:149–153.

Bosland, P.W. 1992. Chilies: A diverse crop. Hortotechnology. 2:6–10.

Bosland, P.W. 2002. Inheritance of a novel flaccid mutant in *Capsicum annuum*. J. Hered. 93:380–382.

Bosland, P.W. and E.J. Votava. 2000. Peppers: Vegetable and spice Capsicums. CAB International Publishing, New York.

Boswell, V.R. 1937. Improvement and genetics of tomatoes. Amer. Nat. 71:266–270.

Cook, A.A. 1960. Genetics of resistance in *Capsicum annuum* to two virus diseases. Phytopathology. 50:364–367.

Cook, A.A. 1961a. A mutation for resistance to potato virus Y in pepper. Phytopathology. 51:550–552.

Cook, A.A. 1961b. Inheritance of mutant-1 phenotype in the pepper. J. Hered. 52:118–120.

Cook, A.A. 1962. Studies of a foliage variegation in pepper. Proc. Amer. Soc. Hort. Sci. 81:390–395.

Cook, A.A. 1968. Introduction of resistance genes to pepper to three strains of potato virus Y. Phytopathology. 53:720–722.

Cook, A.A. 1982. Disease resistance studies and new releases from Florida. Capsicum Eggplant Nswl. 1:43–44.

Cook, A.A. and C.W. Anderson. 1959. Multiple virus disease resistance in a strain of *Capsicum annuum*. Phytopathology. 49:198–201.

Cook, A.A. and Y.G. Guevara. 1984. Hypersensitivity in *Capsicum annuum* to race 1 of pepper. Plant disease 68:329–330.

Cook, A.A. and R.E. Stall. 1963. Inheritance of resistance in pepper to bacterial spot. Phytopathology. 53:1060–1062.

Csillery, G. 1980b. Self-eliminating genes suitable for the purpose of hybrid seed production. Proc. 4th Eucarpia Meeting of Capsicum Working Group, 17–19 May 1980, Wageningen, the Netherlands. 135–146.

Csillery, G. 1984. Linkage between an ‘anthocyaninless’ and a ‘mosaic’ gene. Capsicum Eggplant Nswl. 3:52–53.

Csillery, G. 1985. Abnormal segregation ratio in a ‘lutescens’ hybrid in *Capsicum baccatum*. Capsicum Eggplant Nswl. 4:43.

Csillery, G., E. Szarka, E. Sardi, J. Mityko, J. Kapitany, B. Nagy, and J. Szarka. 2004. The unity of plant defense: Genetics, breeding and physiology. Proc. 12th Eucarpia Meeting on Genetics and Breeding of *Capsicum* and Eggplant, 17–19 May 2004, Noordwijkhout, the Netherlands. 145–146.

Csillery, G. 1983. New *Capsicum* mutants found on seedling growth types, leaf, flower and fruit. 5th Eucarpia Meeting of Capsicum and Eggplant Working Group, 4–7 July 1983, Plovdiv. 127–130.

Chalukova, M., S. Daskolov, E. Lukarska, and D. Baraliieva. 1993. Beta-orange mutant in pepper (*Capsicum annuum* L.). Capsicum Eggplant Nswl. 12:57–58.

Choi, K.S., D.H. Pae, Y.H. Om, and C.H. Lee. 1998. Studies on the breeding of disease resistance in red pepper (*Capsicum annuum* L.). 4. Breeding of Jangsusogochu, a multi-disease resistant variety and the mode of inheritance of resistance to Phytophthora capsici and TMV. Research Reports of the Rural Development Administration, Horticulture. 30:1–8.

Cook, A.A. 1966. Genetics of resistance in *Capsicum annuum* to two virus diseases. Phytopathology. 50:364–367.

Daskalov, S. 1968. A male sterile (*Capsicum annuum* L.) mutant. Theor. Appl. Genet. 38:370–372.

Daskalov, S. 1971. Two new male sterile mutants by pepper (*C. annuum*). C.R. Acad. Sci. Agr. Bulg. 4:291–294.
Daskalov, S. 1973a. Gene list for the pepper. Genet. Plant Breeding 6:401–408.
Daskalov, S. 1973b. Investigation of induced mutants in Capsicum annuum L. III. Mutants in the variety Zlaten Medal. Genet. Plant Breeding 6:419–420.
Daskalov, S. 1974. Investigation on induced mutants in sweet pepper (Capsicum annuum L.). Proc. 1st Meeting of the Capsicum Breeding and Genetics, Budapest, Hungary. 81–90. 1–4 July 1974.
Daskalov, S. 1987. Investigations on mutagenesis and heterosis in pepper (Capsicum annuum L.). D.Sc. Thesis [in Bulgarian].
Daskalov, S., M. Chalulova, D. Baraleva, and E. Lukarska. 1995. Biochemical investigations of an induced beta-orange mutant in sweet pepper (Capsicum annuum L.) and developing varieties with increased beta-carotene content. Proc. 9th Eucarpia Meeting on Genetics and Breeding of Capsicum and Eggplant, 21–25 Aug. 1995. Budapest, Hungary. 24–27.
Daskalov, S. and L. J. Biol. 120(8). A new method for hybrid seed production based on cytoplasmic male sterility combined with a lethal gene and a female sterility pollinator in Capsicum annuum L. Theor. Appl. Genet. 76:530–537.
Daskalov, S. and J.M. Polous. 1994. Updated Capsicum gene list. Capsicum Eggplant Nswl. 13:16–26.
Daubeze, A.M., A. Palloix, and E. Pochard. 1990. Resistance of androgenic autotriploids of pepper to Phytophthora capsici and tobacco mosaic virus under high temperature. Capsicum Nswl. 8:47–48.
Daubeze, A.M., E. Pochard, and A. Pochard. 1989. Inheritance of resistance to Leveillula taurica and relation to other phenotypic characters in the haplodiploid progeny issued from an African pepper line. Proc. 7th Eucarpia Meeting on Genetics and Breeding of Capsicum and Eggplant, 27–30 June 1989, Palanka, Yugoslavia. p. 229–232.
Deom, C.M., J.F. Murphy, and O.R. Pugou. 1997. Resistance to tobacco etch virus in Capsicum annuum L. Inhibition of virus RNA accumulation. Mol. Plant Microbe Interact. 10:917–921.
Deshpande, R.B. 1933. Studies in Indian chillies. 3. The inheritance of some characters in Capsicum annuum L. Indian Jour. Sci. 3:219–300.
Deshpande, R.B. 1935. Studies in Indian chillies. 4. Inheritance of pungency in Capsicum annuum L. Indian Jour. Sci. 5:513–516.
Deshpande, A.A., C.S. Pathak, and D.P. Singh. 1983. Types of male sterility in chilli pepper (Capsicum spp.). Capsicum Nswl. 2:97–98.
Dijan-Caporalino, C., F. Bertilou, A. Fazari, V. Lefebvre, A. Palloix, A. Pegard, and L. Pijarowski. 2004. Genetic, cytological and molecular bases of the resistance to root-knot nematodes (Meloidogyne spp.) in pepper (Capsicum annuum L.) using AFLPs. Proc. 10th Eucarpia Meeting on Genetics and Breeding of Capsicum and Eggplant, 7–11 Sept. 1998. Avignon, France. 125–128.
Dijan-Caporalino, C., L. Pijarowski, A. Januel, V. Lefebvre, A. Daubeze, A. Palloix, A. Dalmasso, and P. Abad. 1999. Spectrum of resistance to root-knot nematodes and inheritance of heat-stable resistance to pepper (Capsicum L.). Theor. Appl. Genet. 99:496–502.
Dogmott, C., A. Palloix, A.M. Daubeze, G. Marchoux, K. Gehrke Selassie, and E. Pochard. 1996. Genes of broad spectrum resistance to potyviruses in haplodiploid progenies of pepper (Capsicum annuum). Euphytica. 88:231–239.
Fan, Y. and J. Guo. 1994. Breeding and application aspect of male sterile lines in sweet pepper, p. 256–259. In: G. Dong and L. Meng (eds.). Advances in horticultural science. China Agriculture Press, Beijing, China. [in Chinese].
Fan, Y., Y. Liu, and L. Yan. 2004. Breeding and application of male sterile lines in sweet pepper. J. Hebei Agr. Sci. 8:26–29 [in Chinese].
FAO. 2005. Agricultural statistics for 2005. Food Agr. Org. United Nations, Rome.
Fernandes, and R. de L. D. Ribeiro. 1998. Mode of inheritance of resistance in Capsicum annuum accessions to Gomphus gloeosporioides. Proc. 10th Eucarpia Meeting on Genetics and Breeding of Capsicum and Eggplant, 7–11 Sept. 1998. Avignon, France. 170.
Ferry, R.L. and P.D. Dukes. 1996. The inheritance of resistance to the southern root-knot nematode in 'Carolina Hot' cayenne pepper. J. Amer. Soc. Hort. Sci. 121:1024–1027.
Ferry, R.L. and H.F. Harrison Jr. 1990. Inheritance and assessment of bentazon herbicide tolerance in 'Santaka' pepper. J. Amer. Soc. Hort. Sci. 115:854–857.
Ferry, R.L. and J.A. Thies. 1998. Genetic analysis of resistance to the southern root-knot nematode in Capsicum chinense Jacq. J. Amer. Soc. Hort. Sci. 123:1008–1011.
Ferry, R.L. and J.A. Thies. 2000. Inheritance of resistance to the peanut root-knot nematode in Capsicum frutescens Jacq. J. Amer. Soc. Hort. Sci. 125:615–618.
Gehrke Selassie, K.E., G. Marchoux, and E. Pochard. 1983. Biological and serological characterization of tomato virus Y strains affecting peppers and other related strains. Capsicum Nswl. 2:134–136.
Gil Ortega, R. and M.L. Arteaga. 1988. Response of pepper to two Spanish isolates of CMV. Plant Disease. 72:65–66.
Gil Ortega, R., C. Palazon Espana, and J. Cuartero. 1993. Genetic analysis of stunted growth of pepper due to Phytophthora capsici. Plant Breed. 108:118–125.
Gil Ortega, R., C. Palazon Espana, and J. Cuartero. 1992. Genetic relationships among four pepper genotypes resistant to Phytophthora capsici. Plant Breed. 100:118–125.
Gopalakrishnan, T.R., P.K. Gopalakrishnan, and K. Raghavacharyulu. 1983. Types of male sterility in chilli pepper (Capsicum spp.). J. Amer. Soc. Hort. Sci. 108:262–267.
Gopalakrishnan, T.R., P.K. Gopalakrishnan, and A. Raghavacharyulu. 1985. Inheritance of root-knot nematode resistance in Capsicum annuum L. J. Hered. 76:211–213.
Herron, C., Rustikawati, and Sudarsono. 2004. Gene analysis of resistance of cucumber mosaic virus (CMV) in hot pepper (Capsicum annuum L.). Capsicum Eggplant Nswl. 23:113–116.
Hibberd, A.M., M.J. Bassett, and R.E. Stahl. 1987. Allelism tests of three dominant genes for hypersensitive resistance to bacterial spot of pepper. Phytopathology. 77:1304–1307.
Holmes, F.O. 1934. Inheritance of ability to localize tobacco mosaic virus. Phytopathology. 24:984–1002.
Holmes, F.O. 1937. Inheritance of resistance to tobacco mosaic disease in the pepper. Phytopathology. 27:637–642.
Huth, J.H., B.C. Kang, S.H. Nahm, S. Kim, K.S. Ha, M.M. Lee, and B.D. Kim. 2001. A candidate gene approach identified phytoene synthase as the locus for mature fruit color in red pepper (Capsicum spp.). Theor. Appl. Genet. 102:524–530.
Hurtado-Hernandez, H. and P.G. Smith. 1985. Inheritance of mature fruit color in Capsicum annuum L. J. Hered. 76:211–213.
Inai, S., K. Ishikawa, O. Nunifu, and H. Ikeshi. 1993. Genetic analysis of stunted growth by nuclear-cytoplasmic interaction in interspecific hybrids of Capsicum by using RAPD markers. Theor. Appl. Genet. 87:416–422.
Ishikawa, K., T. Janos, and O. Nunifu. 1998. Inheritance of the fruit shape at the apex and the peduncle attachment of pepper. Capsicum Eggplant Nswl. 17:30–33.
Jahn, M., I. Parin, K. Hoffmann, E.R. Radwanski, K.D. Livingstone, R.C. Grube, E. Aftergoot, M. Lapidoth, and J. Moyer. 2000. Genetic mapping of the Tsw locus for resistance to the tospovirus spotted wilt virus in Capsicum spp. and its relationship to the Sw-S gene for resistance to the same pathogen in tomato. Mol. Plant Microbe Interact. 13:673–682.
Jones, J.B., G.V. Minsavage, P.D. Roberts, R.R. Johnson, C.S. Kousik, S. Subramanian, and...
R.E. Stall. 2002. A non-hypersensitive resistance in pepper to the bacterial spot pathogen is associated with two recessive genes. Phytopathology. 92:273–277.

Kang, B.C., L. Yeam, J.D. Lewans, J.F. Murphy, and M.M. Jahn. 2005. The prel locus in pepper encodes a translation initiation factor eIF4E that interacts with tobacco etch virus RNAV. Plant J. 42:392–405.

Kim, K.T., Y. Song, H.J. Kim, D.H. Pae, J.Y. Yoon, and B.D. Kim. 2001. Development of DNA markers linked to bacterial leaf spot resistance in chilli. Acta Hort. 546:597–601.

Kim, B.S. and R.W. Hartmann. 1985. Inheritance of a gene (Bs3) conferring hypersensitive resistance to Xanthomonas campestris pv. vesicatoria in pepper (Capsicum annuum L.). Mol. Cells. 20:416–422.

Kyle, M.M. and A. Palloix. 1997. Proposed revision of information on cytoplasmic male sterility gene in Capsicum. Euphytica. 97:183–188.

Lefebvre, V., A.M. Daubeze, J. Kouppe van der Voort, J. Peleman, M. Bardin, and A. Palloix. 2003. QTL for resistance to powdery mildew in pepper under natural and artificial infections. Theor. Appl. Genet. 107:661–666.

Lefebvre, V., M. Kuntz, B. Camara, and A. Palloix. 1998. The capsanthin capsorubin synthase gene: A candidate gene for the y locus controlling the red fruit color in pepper. Plant Mol. Biol. 36:785–789.

Lefebvre, V., A. Palloix, C. Caranta, and E. Palanka. 1995. Functional male sterility in chilli (Capsicum annuum L.). Mol. Plant Microbe Interact. 17:322–329.

Moury, B., M. Noiraude, J. Poirot, and B. Tanyolac. 1997. Allelation of restricted systemic spread of pepper mottle potyvirus in Capsicum annuum cv. Avelar by coinfection with a cucumovirus. Phytopathology. 87:561–566.

Matsunaga, H., T. Saito, M. Hirai, T. Nunome, and T. Yoshida. 2003. DNA markers linked to pepper mild mottle virus (PMoMV) resistance locus (L4) in Capsicum. Jpn. Jpn. Soc. Hort. Sci. 72:218–222.

Matsunaga, H., T. Sato, and S. Monna. 1998. Inheritance of bacterial wilt resistance in the sweet pepper cv. Mie-Midori. Proc. 10th Eucarpia Meeting on Genetics and Breeding of Capsicum and Eggplant, Rome, Italy. 7–10 Sept. 1998, Avignon, France. 172–178.

McCannom, K.R. and S. Homma. 1984. Genetics of the ‘umbrella’ branching habit in Capsicum annuum L. Theor. Appl. Genet. 68:541–545.

Mesihram, J.L. and M.N. Narkhede. 1982. Natural male sterile mutant in hot chilli (Capsicum annuum L.). Euphytica. 31:1003–1005.

Hou, B.S., R.V. Choudhuri, B.Y. Kakade, and M.W. Marawar. 1992. Functional male sterility in chilli (Capsicum annuum L.). Proc. 8th Eucarpia Meeting on Genetics and Breeding on Capsicum and Eggplant, Rome, Italy. 7–10 Sept. 1992, Avignon, France. 172–178.

Miller, J.C. and Z.M. Fineman. 1938. A genetic study of some qualitative and quantitative characters of the genus Capsicum. Proc. Amer. Soc. Hort. Sci. 36:647–651.

Mourey, B., C. Morel, E. Johansen, L. Guilbaud, S. Souque, V. Ayme, C. Caranta, A. Palloix, and M. Jacquemond. 2004. Mutations in potato virus Y genome-linked protein determine virulence toward recessive resistances in Capsicum annuum and Lycopersicon hirsutum. Mol. Plant Microbe Interact. 17:322–329.

Moury, B., A. Palloix, K.G. Selassie, and G. Marchoux. 1997. Hypersensitive resistance to tomato spotted wilt virus in three Capsicum chinense accessions is controlled by a single gene and is overcome by virulent strains. Euphytica. 94:45–52.

Panda, R.C., O.A. Kumar, and K.G. Raja Rao. 1987. Desynaptic mutant in chilli pepper. J. Hered. 78:101–104.

Paran, I., J.K. van der Voort, V. Lefebvre, M. Jahn, L. Peleman, M. Bardin, B. Tanyolac, C. Caranta, A. Ben Chaim, K. Livingstone, A. Palloix, and J. Peleman. 2004. An integrated genetic linkage map of pepper (Capsicum spp.). Mol. Breed. 13:251–261.

Park, H.K., B.S. Kim, and W.S. Lee. 1990. Inheritance of resistance to anthracnose (Colletotrichum sp.) in pepper (Capsicum annuum L.). II. Genetics analysis of resistance to Colletotrichum dematium. J. Korean Society for Horticultural Science 31:207–212.

Pasko, P., M. Luis Arteaga, and R.G. Ortega. 1992. Different kinds of reactions to PYY1-2 in Capsicum annuum L. cv. ‘SCM-334’. Proc. 8th Eucarpia Meeting on Genetics and Breeding of Capsicum and Eggplant, Rome, Italy. 7–10 Sept. 1992, Avignon, France. 172–178.

Pasko, P., R.G. Ortega, and M.L. Arteaga. 1996. 2006. Molecular genetics of pepper and its wild relatives for genetic control. Proc. 8th Eucarpia Meeting on Genetics and Breeding of Capsicum and Eggplant, Rome, Italy. 7–10 Sept. 1992, Avignon, France. 172–178.

Pathak, C.S., D.P. Singh, and A.A. Deshpande. 1985a. Inheritance of male sterile character. Z. Pflanzenzucht. 65:129–140.

Pathak, C.S., D.P. Singh, and A.A. Deshpande. 1983b. Male and female sterility in chilli pepper (Capsicum annuum L.). J. Korean Society for Horticultural Science 29:99–100.

Pathak, C.S., D.P. Singh, and A.A. Deshpande. 1983c. Pathenocarpny in chillies (Capsicum annuum L.). Capsicum Nswl. 2:151–157.

Pathak, C.S., A.A. Deshpande, and D.P. Singh. 1985. Non-flowering mutant in chillies (Capsicum annuum L.). Capsicum Nswl. 4:41–42.

Pegard, A., G. Brizzard, A. Fazari, O. Soucaze, P. Abad, and C. Djian–Caporalino. 2005. Historical characterization of resistance to different root-knot nematode species related to phenolics accumulation in Capsicum annuum. Phytopathology. 95:158–165.

Peterson, P.A. 1958. Cytoplasmically inherited male sterility in Capsicum. Amer. Nat. 92:111–119.

Peterson, P.A. 1959. Linkage of fruit shape and color genes in Capsicum. Genetics. 48:407–419.

Petty, S., M. L纳斯, T. Labaye, A. Ballwora, J. Veuakens, M. Canal, and U. Bonas. 2000. High-resolution genetic mapping of the pepper resistance locus Bs3 governing recognition of the Xanthomonas campestris pv. vesicatoria Avr33 protein. Theor. Appl. Genet. 101:255–263.

Pochard, E. 1982. A major gene with quantitative effect on two different viruses, CMV and TMV. Caracas Veg. 1:57–58.

Pochard, E. and A.M. Daubeze. 1989. Progressive construction of a polygenic resistance to cucumber mosaic virus in the pepper. Proc. 7th Eucarpia Meeting on Genetics and Breeding of Capsicum and Eggplant, Rome, Italy. 27–30 June 1989, Palanka, Yugoslavia. 187–192.

Pochard, E., R. Dumas de Vaulx, and A. Florent. 1983. Linkage between partial resistance to CMV and susceptibility to TMV in the line ‘Perennial’. Analysis on androgenetic homozygous lines. Capsicum Nswl. 2:33–33.

Pospivskiy, S. and I. Paran. 2000. Molecular genetics of the y locus in pepper: Its relation to capsanthin–capsorubin synthase and to fruit color. Theor. Appl. Genet. 101:86–89.
Poulos, I.M., F.I.B. Reischnieder, and W.R. Coff- 
man. 1991. Heterobility and gain from selection 
for quantitative resistance to Xanthomonas campestris pv. vesicatoria in Capsicum annuum L. Euphytica. 56:161–167. 
Poulos, J.M., F.I.B. Reischnieder, and W.R. Coff- 
man. 1992. Inheritance of quantitative compo- 
nents of resistance to Xanthomonas campestris pv. vesicatoria in pepper line ‘CPN91705’. Proc. 8th Eucarpia Meeting on Genetics and Breeding on 
Capsicum and Eggplant, Rome, Italy. 7–10 Sept., 1992. 166–171. 
Prince, J.P., E. Pochard, and S.D. Tankelsy. 1993. 
Construction of a molecular linkage map of pepper and a comparison of synteny with 
tomato. Genome. 36:404–417. 
Prolaram, B., T. Christoph, and Subhash. 1990. 
Seedless fruit mutant in Capsicum. Capsicum 
Nswl. 8/9:34–35. 
and Eggplant, Rome, Italy. 7–10 
Capsicum 
Xanthomonas campestris 
spp.) resistance in a cross 
Xanthomonas campest-
rius pv 

Salamon, P., G. Venczel, L. Zatykó, and Z. Sági. 
2001. Studies on the Tobamovirus resistance of 
the pepper (Capsicum annuum L.) cultivar 
Gregyo. Int. J. Hort. Sci. 7:71–75. 
Shifriss, C. 1973. Additional spontaneous male-
sterile mutant in Capsicum annuum L. Euphy- 
tica. 22:527–529. 
Shifriss, C. 1995. Male sterility in Capsicum. 
Capsicum and Eggplant Nswl. 14:11–25. 
Shifriss, C. 1997. Male sterility in pepper (Capsi-
cum annuum L.). Euphytica. 93:83–88. 
Shifriss, C. and M. Pilyosky. 1992. A progress 
in breeding for resistance to CMV in pepper. 
Capsicum Nswl. 6:60. 
Shifriss, C. and R. Frankel. 1969. A new male 
 sterility gene in Capsicum annuum L. J. Amer. 
Soc. Hort. Sci. 94:385–388. 
Shifriss, C. and Y. Hakim. 1977. Segregation for 
prebifurcation shooting, stem length and leaf 
number of main stem in two crosses of Capsi-
cum annuum L. Euphytica. 66:123–126. 
Shifriss, C. M. Pilyosky, and J. M. Zack. 1992. 
Resistance to Leveillula mildew (Olpidiopsis 
taurica) in pepper. Proc. 8th Eucarpia Meeting 
Genetics and Breeding on Capsicum and Eggplant, 
Rome, Italy. 7–10 Sept. 1992. 172–177. 
Shifriss, C. and J. Rybicki. 1972. A male sterile 
(mns-2) gene in ‘California Wonder’ pepper (C. annuum). HortScience. 7:36. 
Shih, D.M. and J.F. Fontenot. 1990. Gene transfer 
of multiple flowers and pubescence leaf from 
Capsicum chinense into Capsicum annuum 
backgrounds. J. Am. Soc. Hort. Sci. 115:499–502. 
Singh, J., and M.R. Thakur. 1977. Genetics of 
resistance to tobacco mosaic virus, cucumber 
mosaic virus and leaf curl virus in hot pepper 
(Capsicum annuum). Proc. 3rd Eucarica Meeting 
going on Capsicum Working Group, Montfavit, 
Avignon, France. 5–8 July 1977. 119–126. 
Smith, P.G. 1950. Inheritance of brown and 
green mature color in peppers. J. Hered. 41:138–140. 
Smith, P.G. 1951. Deciduous ripe fruit character in 
peppers. Proc. Amer. Soc. Hort. Sci. 57:343–344. 
Souza–Sobrinho, F., W.R. Maluf, L.A.A. Gomes, 
W.R. Maluf, I. Slamov, and M. Jahn. 2000. Candi-
dous for the 
bell pepper genotypes homozygous and hetero-
ygous for the N gene. J. Amer. Soc. Hort. Sci. 
127:371–375. 
Takai, T., D. Dahlbeck, R.E. Stall, J. Pelamans, and 
B.J. Staskawicz. 1999b. High-resolution genet- 
ic and physical mapping of the region 
containing the Bs2 resistance gene of pepper. 
Theor. Appl. Genet. 99:1201–1206. 
Toborek, J. 1993. Origin and inheritance of resistance to 
Phytophthora blight in pepper germplasm: Evidence for conserved 
loci across Solanaceae and for a 
large genetic diversity. Theor. Appl. Genet. 
106:1473–1485. 
Thies, J.A. 2004. IFAFS final report. 
Thies, J.A. and R.L. Fery. 1999. Modified expression of 
the N gene for southern root-knot nematode 
resistance in pepper at high soil temperatures. 
J. Amer. Soc. Hort. Sci. 123:1012–1015. 
Thies, J.A. and R.L. Fery. 2002. Heat stability of resistance 
to southern root-knot nematode in bell pepper genotypes homozgous and hetero-
ygous for the N gene. J. Amer. Soc. Hort. Sci. 
10:6–99. 
Uzo, J.O. 1984. Hybrid vigor and gene action 
for qualitative traits of flavor peppers in 
Nigeria. Scientia Horticulturae 22:321–326. 
van den Berkmortel, L.G. 1977. Breeding pepper 
for resistance to a strain of TMV. Proc. 3rd 
Eucarpia Meeting on Genetics and Breeding 
of Capsicum and Eggplant, 5–8 July 1977. 
Avignon-Montfavet, France. 89–92. 
Van der Beek, J.G. and A. Lilt. 1990. Variation in 
fasciculation in F2 populations of pepper. 
Capsicum Eggplant Nswl. 8/9:34–35. 
Voorrips, R.E., R. Finkers, L. Sanjaya, and 
R. Droenwold. 2004. QTL mapping of anthrac- 
ose (Colletotrichum spp.) resistance in a cross 
between Capsicum annuum and C. chinense. 
Theor. Appl. Genet. 109:1275–1282. 
Votava, E.J., C. Balok, D. Coon, and P.W. Bosland. 
2000. Inheritance of unique fruit and foliage 
colour mutation in NuMex Pinata. J. Hered. 
91:60–69. 
Votava, E.J. and P.W. Bosland. 2002. Novel 
 sources of non-pungency in Capsicum species. 
Capsicum Eggplant Nswl. 21:66–68. 
Walker, S.J. and P.W. Bosland. 1999. Inheritance of 
Phytophthora root rot and foliar blight 
resistance in pepper. J. Amer. Soc. Hort. Sci. 
124:14–18. 
Wang, H., D Wang, and Y. Li. 2003. Advances in 
the heterosis breeding using the CMS lines. J. Guangdong Agri. Sci. 5:16–18. [in Chinese] 
Wang, D., M. Wang, Y. Wang, and Y. Yan. 1995. 
Advances in the inheritance of and breeding for 
resistance to Phytophthora blight. China Veg. 
3:50–53. [in Chinese] 
Wang, D., M. Wang, Y. Wang, and Y. Yan. 1996. 
Advances in the inheritance of and breeding for 
resistance to cucumber mosaic virus (CMV). China Veg. 
1:51–55. [in Chinese] 
Wolff, D.W., W.W. Collins, and T.J. Monaco. 
1992. Inheritance of tolerance to the herbicide 
bentazon in peppers (Capsicum annuum L.). J. Amer. 
Soc. Hort. Sci. 117:985–990.
Yang, S. 1981. Breeding of male sterile lines in hot pepper. Acta Horticulturae Sinica. 8:49. [in Chinese].

Yang, T. and H. Park. 1998. The study on inheritance of several characters in Capsicum annuum L. RDA J. Hort. Sci. 40:1–8.

Yang, F., S. Yang, E. Jiang, Z. Wang, and J. Li. 1994. Breeding and application of male sterile line 'AB92' in bell pepper. J. Liaoning Agr. Sci. 6:15–18. [in Chinese].

Yazawa, S., T. Sato, and T. Namiki. 1991. Interspecific hybrid dwarfism and geographical distribution of the dwarfness gene in Capsicum. J. Jpn. Soc. Hort. Sci. 58:609–618.

Yeam, I., B.C. Kang, W. Lindeman, J.D. Frantz, N. Faber, and M.M. Jahn. 2005. Allele-specific CAPS markers based on point mutations in resistance alleles at the pvr1 locus encoding eIF4E in Capsicum. Theor. Appl. Genet. 112:178–186.

Yuan, J. and S. Li. 2000. Study of floral organ morphology and inheritance of a new functional male sterile pepper line. Hereditas (Beijing). 22:28–30. [in Chinese].

Zatyko, L., and A. Moor. 1998. Linkage between an al gene (anthocyanin less) and the L1 gene of TMV resistance in pepper. Proc. 10th Eucarpia Meeting on Genetics and Breeding of Capsicum and Eggplant, Avignon, France. 7–11 Sept. 1998. 249.

Zewdie, Y. and P.W. Bosland. 2001. Allelic test for closed flower trait in Capsicum. Capsicum Eggplant Nswl. 20:58–59.

Zhang, B., S. Huang, G. Yang, and J. Guo. 2000. Two RAPD markers linked to major fertility restorer gene in pepper (Capsicum annuum L.). Euphytica. 113:155–161.

Zitter, T.A. 1972. Naturally occurring pepper virus strains in south Florida. Plant Dis. Rept. 56:586–590.

Zitter, T.A. and A.A. Cook. 1973. Inheritance of tolerance to a pepper virus in Florida. Phytopathology. 63:1211–1212.