Morphological, Biochemical, and Molecular Markers in Onion

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Among vegetables harvested in the United States during 1997, onion (*Allium cepa* L.) ranked second in yield per hectare, third in total production, fourth in total value, and fifth in the number of hectares harvested (U.S. Dept. of Agriculture, 1998). Five public and 21 private breeding programs in the United States are dedicated to improving onions (Frey, 1996). In addition, numerous other countries support public and private onion breeding programs. The study of onion genetics has been limited, relative to that of other crops, by the biennial nature of the onion plant, which increases the time required to complete these studies. Onion also possesses one of the largest plant genomes in terms of total genomic DNA (Arumuganathan and Earle, 1991) and has been difficult to culture, transform, and regenerate in vitro. The number of genetic markers in onion has increased several-fold with the identification and mapping of molecular markers (Bradeen and Havey, 1995; Havey et al., 1996; King et al., 1998). Recent papers have reviewed the classical markers (Havey, 1993; Pike, 1986; Rabinowitch, 1988), the biochemical markers (Peffley, 1993; Rabinowitch, 1988), and the molecular markers of onion (Peffley, 1993). However, concise review of the classical, biochemical, and molecular markers of onion has not been published. Information regarding the availability of onion populations possessing the described genes currently does not exist. This paper reviews all three types of markers and provides sources for each.

MORPHOLOGICAL MARKERS

Seedling and leaf

Several loci controlling the color of seedcoat and foliage have been described (Table 1). Seedcoat color is governed by the *b* locus with *B_* resulting in a black and *bb* resulting in a brown seedcoat (Davis, 1966). Several heritable chlorophyll-deficient mutants have been observed (Table 1). Albinism (*a*) is recessive, and homozygous recessive plants die soon after germination (Jones et al., 1944; Rasmusson, 1920). The yellow lethal loci, y_1 and y_2, when homozygous recessive, result in seedlings with light-yellow leaves (Jones et al., 1944; Rasmusson, 1920). Such seedlings die shortly after germination. Jones et al. (1944) proposed two yellow lethal loci because some seedlings with a yellow lethal allele also exhibited glossy foliage, while others did not. A third lethal locus, pale green (*pg*), results in pale-green-colored seedlings that die shortly after germination when the locus is homozygous recessive (Jones et al., 1944). Two recessive alleles at a fourth locus, virescent (*v*), result in seedlings with pale-green foliage at low temperatures (Jones et al., 1944). Virescent seedlings can survive, and the foliage gradually becomes similar in color to nonvirescent foliage, but bulb yields are greatly reduced (Jones et al., 1944).

Loci governing glossy foliage have also been described (Table 1). Glossy foliage is caused by the absence of wax on the surface of the leaf (Jones et al., 1944; Molenaar, 1984). Glossiness is conditioned by a recessive allele at the *gy* locus, which is reportedly linked to the yellow lethal (y_1) (Jones et al., 1944) locus and the locus governing thrips (*Thrips tabaci* Lindeman) resistance (Jones et al., 1944; Molenaar, 1984). Separate loci for scape glossiness have been reported and glossiness was conditioned by recessive alleles at both loci, gls_1 and gls_2, with gls_1 being epistatic to gls_2 (Molenaar, 1984). Double glossy scapes were produced when both loci had two recessive alleles. A dominant allele at the gls_2 locus produced a phenotype that was intermediate between single and double glossy scapes, whereas a dominant allele at the gls_1 locus produced single glossy scapes, regardless of the alleles at the gls_2 locus. Molenaar (1984) also suggested that the three loci for glossiness were linked.

Variation has also been observed for scape height. The inheritance of the dwarf scape locus, dw_1, has been described (Rabinowitch et al., 1984). Several other loci are thought to be involved in scape height, based upon F_3 segregations of families from crosses between dwarf and normal cultivars (Horobin, 1986).

Bulb

Bulb scale (dried basal portions of outer leaves) color has been studied extensively (Table 2). Scale color is controlled by five major loci (*I, C, R, L, G*) (Clarke et al., 1944; El-Shafie and Davis, 1967; Rieman, 1931). A dominant allele at the *I* locus inhibits pigment production and the scales are white (Clarke et al., 1944; Rieman, 1931). According to Clarke et al. (1944), *I* is incompletely dominant, such that an intermediate (buff) color was obtained with a genotype of *IiCCR_*. They had difficulty distinguishing *IiCCRR* and *IiCCrr* genotypes and also distinguishing *IiCCrr* genotypes from pure white, *II*. The *I* locus must be homozygous recessive to produce colored scales. In addition to the *I* locus, the *C* locus also influences the color of the bulb scales. When a dominant allele is present at the *C* locus, scales are colored; when the *C* locus is homozygous recessive, scales are white regardless of alleles for color at other loci (El-Shafie and Davis, 1967).

A third locus, *R*, governs the amount of red pigment produced by the epidermal cells of the scale. The anthocyanins responsible are primarily glucosides of cyanidin with trace amounts of pelargonidin and peonidin (Fossen et al., 1996). Light-red or pink bulbs are conditioned by a dominant *R* allele (*iiC_R_*); yellow or brown bulbs have the recessive *r* allele (*iiC_rr*) (El-Shafie and Davis, 1967). To obtain dark-red bulbs, the *R* locus must be homozygous dominant and a fourth locus, *L*, also must be homozygous dominant (El-Shafie and Davis, 1967). Heterozygosity at either loci results in light-red or pink scales. Jones and Peterson (1952) described a complementary light-red bulb color that may be conditioned by the interaction of the *R* and *L* loci (El-Shafie and Davis, 1967). King et al. (1998) assigned the name *Crb-1* to one of these complementary loci, but the relationships among *Crb-1*, *R*, and *L* are not known. A dominant allele at the fifth locus, *G*, produces golden bulbs (*iiC_rrllG_*); when homozygous recessive (*iiC_rrllgg*), bulbs are chartreuse (El-Shafie and Davis, 1967).

Flower

Several loci governing floral morphology, particularly male sterility, have been described for onion (Table 1). In some plants, the perianth does not fully develop around the reproductive organs, such that the anthers appear to be protruding from the bud (Jones et al., 1944). This trait is referred to as *exposed anthers* (*ea*) and is conditioned by the recessive genotype at the locus. Both green and yellow anthers are observed. Yellow anthers, governed by the *ya* locus, are observed when two recessive alleles are present (Jones et al., 1944). In addition, white perianth has been reported to be reces-

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Table 1. The morphological markers of onion.

Locus	Character and comments	Reference[z]	Availability[y]
a	*Albino*. Chlorophyll-deficient seedlings soon die after germination.	Rasmusson, 1920; Jones et al., 1944	?
b	*Brown seed coat*. Wild-type is black.	Davis, 1966	H
C	*Colored* bulb scales. Must be dominant to get colored bulbs. All bulbs white when absent.	Clark et al., 1944	P
Crb-1	*Complementary red bulb*. May be either *R* or *L* (El-Shafie and Davis, 1967).	Jones and Peterson, 1952	H
dw_1	*Dwarf* seedstalk. Other genes may also be involved in seedstalk height.	Rabinowitch et al., 1984; Horobin, 1986	R
ea	*Exposed anther*. Perianth does not fully develop around anthers.	Jones et al., 1944	?
Foc1	*Fusarium basal plate rot resistance 1*. Partially dominant with *Foc2*.	Bacher, 1989; Bacher et al, 1989	P, H
Foc2	*Fusarium basal plate rot resistance 2*. Partially dominant with *Foc1*.	Bacher, 1989; Bacher et al, 1989	P, H
G	*Golden scale color*. Recessive allele gives chartreuse. Must have dominant allele for red bulb color.	El-Shafie and Davis, 1967	H
gl	*Glossy foliage*. Correlated with resistance to thrips.	Jones et al., 1944	H
gls_1	*Glossy scape 1*. Epistatic to gls_2. Correlated with resistance to thrips.	Molenaar, 1984	H
gls_2	*Glossy scape 2*. Correlated with resistance to thrips.	Molenaar, 1984	H
I	*Inhibitor* of scale color. Must be recessive to get colored scales.	Riemann, 1931	P, H
L	Results in red bulbs when dominant, particularly when the *R* locus is recessive.	El-Shafie and Davis, 1967	P
ms	*Male-sterile*. Dominant allele conditions male fertility for plants with S cytoplasm. All plants with N cytoplasm are male-fertile.	Jones and Clarke, 1943	H
pg	*Pale green foliage*.	Jones et al., 1944	P
Pd_1	*Downy mildew resistance 1*. Dominant gene from *A. roylei*.	Kofoet et al., 1990	CPRO
pr_1	*Pink root resistance*. Other genes may be involved in resistance.	Jones and Perry, 1956; Nichols et al., 1965	P, H
R	*Red* scale color. Recessive allele results in yellow or brown-colored bulbs.	Clark et al., 1944; El-Shafie and Davis, 1967	P
s_1	Downy mildew resistance 1. Found in 'Calred'.	Warid, 1952; Warid and Tims, 1952	P
s_2	Downy mildew resistance 2. Found in 'Calred'.	Warid, 1952; Warid and Tims, 1952	P
Ta	Nuclear restorer gene for CMS-T. Originally designated as *A*.	Schweisguth, 1973	P
Tb	Complementary nuclear restorer gene for CMS-T. *C* locus must also be dominant to restore male fertility. Originally designated as *B*.	Schweisguth, 1973	P
Tc	Complementary nuclear restorer gene for CMS-T. *B* locus must also be dominant to restore male fertility. Originally designated as *C*.	Schweisguth, 1973	P
v	*Virescent foliage*. Plants with this phenotype not as vigorous in growth as nonvirescent foliage plants.	Jones et al., 1944	P
y_1	*Yellow lethal*. Linked with glossy foliage.	Jones et al., 1944	?
y_2	*Yellow lethal*. Not linked with glossy foliage.	Jones et al., 1944	?
ya	*Yellow anther*.	Jones et al., 1944	P, H

[z]Associated references indicate the source of information for each loci.
[y]H = M.J. Havey; P = standard cultivars or accessions from the USDA Plant Germplasm Collection, Geneva, N.Y.; R = H. Rabinowitch, Hebrew Univ. of Jerusalem, Rehovot, Israel; CPRO = Centre for Plant Breeding Research, Wageningen, The Netherlands; ? = availability not known.

sive and controlled by a single locus (Davis, 1960); however, segregation data have not been presented.

Jones and Clarke (1943) described male sterility in onions and its interaction with the genotype of the cytoplasm. Male sterility is present when the *ms* locus, often referred to as the nuclear restorer locus, is homozygous recessive and the sterile (S) cytoplasm is present. All plants with normal (N) cytoplasm are male-fertile regardless of the genotype at the nuclear restorer locus. A second source of cytoplasmic male sterility has been described in European onion cultivars (Berninger, 1965). Three independently segregating restorer loci have been associated with T cytoplasm (Schweisguth, 1973). Dominant alleles at the *A* locus or at both the *B* and *C* loci restore fertility. Because the *a*, *b*, and *C* loci were previously designated in onion, we suggest that loci restoring male fertility in T cytoplasm be renamed to *Ta*, *Tb*, *Tc*.

Biotic and abiotic stresses

Loci that condition resistance to pink root [*Phoma terrestris* (E.M. Hans.) Gorenz, J.C. Walker, & R.H. Larson), Fusarium basal rot [*Fusarium oxysporum* Schlechtend.: Fr f. sp. *cepae* (H.N. Hans.) W.C. Snyder & H.N. Hans.], downy mildew [*Peronospora destructor* (Berk.) Casp. l.c. in Burk.], and purple blotch [*Alternaria porri* (Ellis) Cif.] have been described for onion (Table 1). Resistance to pink root is governed by a recessive allele at a single locus (Jones and Perry, 1956; Nichols et al., 1965). In this paper, the single locus for resistance will be designated pr_1. Nichols et al. (1965) also speculated that, for some crosses, pink root resistance was multigenic and additional loci for resistance were present. One locus (Tsutsui, 1991) or two loci (Bacher, 1989; Bacher et al., 1989) may be involved in resistance to Fusarium basal rot. Bacher et al. (1989) proposed the locus designation, *Foc1* and *Foc2*, for two loci conditioning Fusarium basal rot resistance. They surmised that resistance was partially dominant for both loci and that the gene effects at each locus were additive.

In addition to pink root and Fusarium basal rot resistance, resistance to downy mildew has been observed. Warid and Tims (1952) and Warid (1952) proposed that recessive alleles at two loci conditioned downy mildew resistance and designated those two loci as s_1 and s_2. They observed the greatest resistance to downy mildew when both loci were homozygous recessive. In addition, Kofoet et al. (1990) proposed the transfer of a single dominant locus (Pd_1) for downy mildew resistance from *A. roylei* Stearn to *A. cepa*. Resistance to purple blotch is controlled by a single gene when resistance is recessive (Ekanayake and Ewart, 1997). Resistance to onion smudge [*Colletotrichum circinans* (Berk.) Voglino] has been associated with another trait, bulb pigmentation (Walker, 1923). Pigmented bulbs are resistant to onion smudge, while nonpigmented bulbs are susceptible (Walker, 1923). Diplodia stain [*Lasiodiplodia theobromae* (Pat.) Griffon & Maubl.] is also associated with bulb pigmentation (Sumner, 1995); white onions are susceptible to the disease while yellow and red onions are resistant.

With regard to abiotic stresses, Engle and

Table 2. Genotypes conditioning onion scale color.

Genotype	Bulb color
II	White
IiCc	Buff white
Iicc	White
Iicc	White
IiCC	Pale pink
iiC_rrllgg	Chartreuse
*iiCCrrllG_*	Golden yellow
*iiCcrrL_*	Pink to light red
*iiCCRrL_*	Pink to light red
iiCCRrll	Pink to light red
iiCCRRLL	Red

Table 3. Biochemical markers of onion.[z]

Isozyme	Reference
Alcohol dehydrogenase (*Adh*-1)	Hadacova et al., 1981; Peffley et al., 1985
Acid phosphatase (*Aps*-1,2)	Peffley et al., 1985
Alkaline phosphatase	Hadacova et al., 1981
Catalase	Hadacova et al., 1981
Cholinesterase	Hadacova et al., 1981
Esterase	Hadacova et al., 1981; Peffley et al., 1985; Cooke, 1985; Cooke et al., 1986
Glucose-6-phosphate dehydrogenase	Hadacova et al., 1981
Glutamate dehydrogenase	Hadacova et al., 1981
Glycerate dehydrogenase (*Gdh*-1)	Peffley et al., 1985
Glutamate oxaloacetate transaminase (*Got*-1,2,3)	Peffley et al., 1985
Isocitrate dehydrogenase (*Idh*-1)	Peffley et al., 1985; Peffley and Orozco-Castillo, 1987
Lactate dehydrogenase	Nakamura and Tahara, 1977
Malate dehydrogenase (*Mdh*-1)	Peffley et al., 1985; Ulloa-Godinez et al., 1995
NAD^+-glyceraldehyde-3-phosphate dehydrogenase	Hadacova et al., 1981
$NADP^+$-glyceraldehyde-3-phosphate dehydrogenase	Hadacova et al., 1981
NAD^+-malate dehydrogenase	Hadacova et al., 1981
$NADP^+$-malate dehydrogenase	Hadacova et al., 1981
$NADH_2$-tetrazolium reductase	Hadacova et al., 1981
$NADPH_2$-tetrazolium reductase	Hadacova et al., 1981
Peroxidase (*Prx*-1)	Peffley et al., 1985
6-phosphogluconate dehydrogenase (6*Pgdh*-1,2)	Peffley et al., 1985
Phosphoglucoisomerase (*Pgi*-1)	Peffley et al., 1985; Peffley and Orozco-Castillo, 1987
Phosphoglucomutase (*Pgm*-1)	Peffley et al., 1985; Peffley and Orozco-Castillo, 1987
Superoxide dismutase (*Sod*)	Hadacova et al., 1981

[z]Table adapted from Rabinowitch, 1988.

Gabelman (1966) reported resistance to ozone in several onion inbreds. For the inbreds tested, resistance was dominant and the mechanism of resistance was closure of leaky stomata that prevented destruction of inner tissues by ozone. Engle and Gabelman (1966) failed to designate this locus and we propose that this locus be designated *Oz*.

BIOCHEMICAL AND MOLECULAR MARKERS

Isozymes

Isozymes were identified as biochemical markers for onion during the 1980s. Currently, 24 different isozymes have been characterized in onion seeds and roots (Table 3) (Rabinowitch, 1988). Isozymes have been used primarily to compare other *Allium* species to *A. cepa*, common onion, and to identify the origins of chromosomal regions in interspecific hybrids (Havey et al., 1996; Peffley et al., 1985; Ulloa-Godinez et al., 1994, 1995). Two alleles for *Adh*-1 were characterized in *A. cepa*, while third and fourth putative alleles were identified in *A. fistulosum* L. (Peffley and Orozco-Castillo, 1987; Peffley et al., 1985). When 188 *A. cepa* breeding accessions and plant introductions were evaluated for four isozyme systems (*Adh*, *Idh*, *Pgi*, *Pgm*), only *Adh*-1 was variable (Peffley and Orozco-Castillo, 1987). Ulloa-Godinez et al. (1995) observed a fast band of *Mdh*-1 in *A. cepa* and a slow band in *A. fistulosum*. Work done by Cryder et al. (1991) suggested that *Idh*-1 and *Pgi*-1 were linked in backcross progeny between *A. cepa* and *A. fistulosum*. Genetic linkages between *Idh-1* and *Adh-1* and between *Pgi-1* and *Adh-1* were inconclusive. Except for linkage studies, few segregation analyses have been conducted to determine the number of genes at each isozyme locus. Segregation analyses for *A. fistulosum* suggest that 6-PGDH is governed by two loci, *6-Pgdh-1* and *6-Pgdh-2*, that have two alleles (1 and 2) each (Magnum and Peffley, 1994). In addition, both PGM and SKDH are governed by a single locus each with two alleles at each locus. Using alien chromosome addition breeding lines, Peffley and Currah (1988) assigned the location of *Adh*-1 to the sub-telocentric chromosome 5 and *Pgm*-1 to the sub-metacentric chromosome 4 in *A. fistulosum.*

RESTRICTION FRAGMENT LENGTH POLYMORPHISMS (RFLPS) AND RANDOMLY AMPLIFIED POLYMORPHIC DNAS (RAPDS)

Havey et al. (1996) described the challenges of identifying and mapping molecular markers in a plant, such as onion with a huge nuclear genome. Bradeen and Havey (1995) evaluated 580 decamer primers in replicated experiments and were able to establish the genetic bases of only 14 repeatable RAPDs (Table 4). The RFLPs were technically more difficult, but hybridization of cDNAs revealed adequate levels of polymorphism to develop a genetic map (Bark and Havey, 1995). King et al. (1998) constructed a low-density genetic map using 58 F_3 families generated from a cross between inbreds 'Brigham Yellow Globe (BYG) 15-23' and 'Alisa Craig (AC) 43'. They identified 128 segregating loci (112 RFLPs, 14 RAPDs, 2 morphological) and constructed a map consisting of 114 loci distributed over 11 linkage groups, one linked pair, and 12 unlinked markers (Table 4). The map covered 1064 centiMorgans (cM) with an average distance of 9.2 cM between loci. The RFLP loci were named with the probe–enzyme combination, revealing the polymorphisms with the sizes of the fragments, e.g., AOB272/*Eco*RI (10.0/12.0 kb). King et al. (1998) described an additional 69 cDNA clones that revealed RFLPs among elite inbreds of onion, but these RFLPs did not segregate in the BYG15-23 x AC43 family and the genetic bases of these polymorphisms were not established.

Linkage was detected between RFLPs and loci affecting traits of economic importance. The *ms* locus was flanked by RFLP markers on linkage group B. One of the loci conditioning complimentary red bulb color (named *Crb*-1 by King et al., 1998) was linked to RFLP markers on group H. The RFLPs revealed with clones of alliinase [King et al., 1998 (AOB249); van Damme et al., 1992] showed this enzyme, important in the development of onion flavor, to be conditioned by loci on linkage groups A and I, as well as by one unlinked locus. In addition, rDNA sequences have been located to the telomeric regions of the short arms of chromosomes 6 and 8 using a probe from *Helianthus argophyllus* (pHAR1) (Ricroch et al., 1992).

Of the 112 RFLPs segregating for the map, 44 were dominant and 68 were codominant (King et al., 1998). Twenty percent of the cDNA clones revealed duplicated loci. Of those loci, 42% were tightly linked (<10 cM), 5% were loosely linked (10–30 cM), and 53% unlinked (>30 cM). Previous biochemical and cytogenetic research (Jones and Rees, 1968; Ranjekar et al., 1978) suggested that duplicated regions may exist in the genome. The predominance of dominant and duplicated RFLP loci observed in the low-density genetic map indicated that tandem, intrachromosomal duplications may have contributed to the large size of the onion genome (King et al., 1998).

CONCLUSIONS

Currently 28 morphological, 24 biochemical, and 126 DNA-length markers exist in onion. Four of the morphological markers are not currently available. Also, polymorphic phenotypes have been described in onion but

Table 4. Randomly amplified polymorphic DNAs (RAPDs) and restriction fragment length polymorphisms (RFLPs) in onion.

Marker[z]	Enzyme	Fragment Size (kb)[y]	Fragment Linkage group[x]
	RAPD		
AB14		0.7	G
AB16		1.2	D
AB20		0.8	U
AD19		0.9	D
AE09		0.7	D
AF07		0.6	L
AF12		0.8	F
AK20		0.9	I
AP12		0.7	F
C15		0.8	F
D03		0.7	A
D10		0.7	G
D12		0.5	A
AG19		0.8	U
	RFLP		
Alliinase	*Bam*HI	14.5/13.0	A
Alliinase	*Eco*RV	3.5	A
AJB006	*Eco*RI	9.5/22.0	C
AJB019	*Eco*RI	4.0/4.3	K
AJB032	*Eco*RI	20.0/9.5	F
AJB037	*Eco*RI	9.5/9.0	B
AJB037	*Eco*RI	5.0	U
AJB045	*Hin*dIII	8.0/10.0	K
AJB057	*Hin*dIII	17.0	C
AJB064	*Eco*RI	9.0/9.5	J
AJB064	*Eco*RI	10.9	J
AJB072	*Eco*RI	4.0	F
AJK028	*Hin*dIII	5.5	I
AJK084	*Eco*RI	15.0/13.0	D
AJK084	*Eco*RI	5.5/5.0	D
AJK085	*Eco*RV	22.0/20.0	J
AJK085	*Eco*RV	10.0	U
AJK242	*Eco*RV	9.0/3.5	A
AJK248	*Eco*RV	5.0	U
AJK252	*Eco*RV	20.0/8.0	D
AJK265	*Eco*RV	9.5	C
AJK265	*Eco*RV	5.0	C
AJK267	*Eco*RV	6.7	L
AJK295	*Hin*dIII	20.0/3.0	B
AOB041	*Hin*dIII	4.0/10.0	B
AOB046	*Eco*RV	20.0/9.5	E
AOB050	*Eco*RV	6.7	E
AOB074	*Eco*RV	6.7/15.0	K
AOB077	*Eco*RV	8.0	A
AOB087	*Eco*RV	4.0/3.5	C
AOB105	*Eco*RV	3.0/5.0	C
AOB107	*Eco*RV	10.0	U
AOB114	*Eco*RV	7.0	U
AOB115	*Eco*RV	3.0/4.3	G
AOB116	*Eco*RI	3.0/3.5	B
AOB117	*Eco*RV	6.7	U
AOB120	*Eco*RI	10.0	E
AOB150	*Hin*dIII	12.0	D
AOB151	*Eco*RV	20.0	A
AOB152	*Eco*RI	3.0	B
AOB152	*Eco*RI	0.7	H
AOB155	*Eco*RV	13.0/15.0	I
AOB156	*Eco*RI	4.3	G
AOB156	*Eco*RI	6.0/6.7	C
AOB162	*Eco*RI	15.0/18.0	A
AOB167	*Hin*dIII	5.0/4.0	D
AOB168	*Eco*RV	24.0	C
AOB168	*Eco*RV	10.0	F
AOB186	*Eco*RV	6.7/2.5	B
AOB187	*Eco*RI	7.6/9.0	C
AOB191	*Hin*dIII	3.0/5.5	D
AOB200	*Eco*RV	7.0/4.0	U
AOB210	*Hin*dIII	7.7	A
AOB210	*Hin*dIII	6.5/5.5	B

continued next column

Table 4. Continued.

Marker[z]	Enzyme	Fragment Size (kb)[y]	Fragment Linkage group[x]
	RFLP contd.		
AOB210	*Hin*dIII	10.0	I
AOB212	*Eco*RI	9.0/20.0	H
AOB213	*Hin*dIII	9.0/9.5	G
AOB213	*Hin*dIII	3.3	G
AOB232	*Eco*RV	24.0/30.0	B
AOB234	*Hin*dIII	8.0/7.0	C
AOB236	*Eco*RI	12.0/18.0	E
AOB237	*Eco*RI	20.0/9.0	J
AOB249	*Hin*dIII	9.5	I
AOB249	*Hin*dIII	4.3	U
AOB260	*Eco*RV	9.5	E
AOB262	*Hin*dIII	8.0/9.5	B
AOB262	*Hin*dIII	5.0	B
AOB271	*Eco*RI	15.0/9.5	E
AOB272	*Eco*RI	10.0/12.0	B
AOB290	*Eco*RV	9.5/7.0	E
AOB292	*Eco*RI	4.3/6.5	D
AOB302	*Eco*RI	10.0/9.5	A
AOB302	*Eco*RI	4.3	G
API10	*Hin*dIII	7.0/8.0	I
API14	*Hin*dIII	4.0/4.5	F
API14	*Hin*dIII	6.7/3.0	F
API15	*Eco*RI	3.0	E
API16	*Eco*RV	20.0	F
API18	*Eco*RV	9.0/6.0	A
API20	*Eco*RV	4.3/3.0	J
API21	*Eco*RV	4.5/4.1	B
API23	*Hin*dIII	12.0/6.5	E
API27	*Eco*RV	20.0/7.0	B
API29	*Hin*dIII	5.0/4.0	G
API29	*Hin*dIII	9.0/9.3	U
API31	*Eco*RV	8.0/5.0	J
API32	*Eco*RV	3.0/4.5	H
API40	*Eco*RV	7.0/4.3	D
API43	*Eco*RV	7.0/9.0	D
API43	*Eco*RV	2.5/4.0	D
API46	*Eco*RV	6.7	E
API47	*Eco*RI	15.0/20.0	E
API51	*Eco*RI	6.7/5.0	G
API51	*Eco*RI	0.6	G
API53	*Eco*RV	3.0	A
API53	*Eco*RV	6.5	G
API54	*Eco*RI	15.0/9.0	F
API55	*Eco*RV	9.0/15.0	A
API59	*Hin*dIII	15.0/9.5	E
API61	*Eco*RI	3.0	A
API61	*Eco*RI	8.0	J
API63	*Eco*RV	4.5/7.5	B
API65	*Eco*RV	10.0	B
API66	*Eco*RV	6.7/9.5	E
API73	*Eco*RV	9.5/20.0	F
API76	*Eco*RV	12.0/10.0	H
API81	*Hin*dIII	6.7	U
API82	*Eco*RI	9.0/9.5	F
API86	*Eco*RV	15.0/12.0	I
API89	*Eco*RI	5.0/4.3	D
API92	*Eco*RI	11.0/12.0	E
API94	*Eco*RI	20.0/10.0	H

[z]cDNA clones revealing RFLP markers in onion are designated as API, AJB, AJK, or AOB with a number corresponding to the order isolated (Bark and Havey, 1995; King et al., 1998). Clones are available from M.J. Havey. The others are decamer primers revealing randomly amplified polymorphic DNA markers (Bradeen and Havey, 1996).

[y]Approximate fragment sizes of RFLP and RAPD markers estimated relative to *Hin*dIII-digested l DNA. Only one fragment listed as dominant markers.

[x]Linkage groups as described by King et al. (1998). U = unlinked.

are not genetically characterized, e.g., white perianth. The designations of some morphological loci are redundant and we propose new names. Separate designations may describe the same locus, e.g., the *R* or *L* loci and *Crb-1*. Further work is required in onion to clarify these problems and to advance the knowledge of onion genetics.

LIterature Cited

Arumuganathan, K. and E. Earle. 1991. Nuclear DNA content of some important plant species. Plant Mol. Biol. Rpt. 9:208–218.

Bacher, J.W. 1989. Inheritance of resistance to *Fusarium oxysporum* f.sp. *cepae* in cultivated onions. MS Thesis, Dept. Hort., Michigan State Univ., East Lansing.

Bacher, J.W., S. Pan, and L. Ewart. 1989. Inheritance of resistance to *Fusarium oxysporum* f. sp. *cepae* in cultivated onions, p. 85–91. In: L. Jensen (ed.). Proc. 1989 Natl. Onion Res. Conf., Boise, Idaho.

Bark, O.H. and M.J. Havey. 1995. Similarities and relationships among open-pollinated populations of the bulb onion as estimated by nuclear RFLPs. Theor. Appl. Genet. 90:607–614.

Berninger, E. 1965. Contribution à l'étude de la stérilité mâle de l'oignon (*Allium cepa* L.) Ann. Amelior. Plant. 15:183–199.

Bradeen, J.M. and M.J. Havey. 1995. Randomly amplified polymorphic DNA in bulb onion and its use to assess inbred integrity. J. Amer. Soc. Hort. Sci. 120:752–758.

Clarke, A.E., H.A. Jones, and T.M. Little. 1944. Inheritance of bulb color in onion. Genetics 29:569–575.

Cooke, R.J. 1985. Characterization of cultivars of *Allium cepa* L. (onions) by ultrathin-layer isoelectric focusing of seed esterases. Electrophoresis 6:572–573.

Cooke, R.J., T.M., Smith, and A.G. Morgan. 1986. The use of isoelectric focusing of seed esterases to classify cultivars of *Allium cepa* L. J. Natl. Inst. Agr. Bot. 17:209–218.

Cryder, C.M., J.N. Corgan, N.S. Urquhart, and D. Clason. 1991. Isozyme analysis of progeny derived from (*Allium fistulosum* x *A. cepa*) x *A. cepa*. Theor. Appl. Genet. 82:337–345.

Davis, E. 1966. An improved method for producing hybrid onion seed. J. Hered. 57:55–57.

Davis, E.W. 1960. Marker genes to facilitate roguing onion-seed fields. Seed World 87:4, 6.

Ekanayake, N. and L.C. Ewart. 1997. Inheritance of resistance to *Alternaria porri* (Ellis) Cife in cultivated onion (*Allium cepa* L.). Allium Improv. Nswl. 7:20–22.

El-Shafie, M. and G. Davis. 1967. Inheritance of bulb color in *Allium cepa*. Hilgardia 9:607–622.

Engle, R.L. and W.H. Gabelman. 1966. Inheritance and mechanism for resistance to ozone damage in onion, *Allium cepa* L. J. Amer. Soc. Hort. Sci. 89:423–430.

Fossen, T., O.M. Andersen, D.O. Ovstedal, A.T. Pedersen, and A. Raknes. 1996. Characteristic anthocyanin pattern from onions and other *Allium* spp. J. Food Sci. 61:703–706.

Frey, K. J. 1996. National plant breeding study—I. Human and financial resources devoted to plant breeding research and development in the United States in 1994. Spec. Rpt. 98. Iowa Agr. Home Econ. Expt. Sta., Iowa State Univ., Ames.

Hadacova, V., E. Klozova, K. Pitterova, and V. Turkova. 1981. The screening of the enzyme and isoenzyme patterns in seeds of *Allium cepa* cultivar Vsetatska. Biol. Plant. (Prague) 23:442–448.

Havey, M.J. 1993. Onion. *Allium cepa* L., p. 35–49.

In: G. Kalloo and B.O. Bergh (eds.). Genetic improvement of vegetable crops. Pergamon Press, Tarrytown, N.Y.

Havey, M.J., J.J. King, J.M. Bradeen, and O.H. Bark. 1996. Molecular markers and mapping in bulb onion, a forgotten monocot. HortScience 31:1116–1118.

Horobin, J.F. 1986. Inheritance of a dwarf seed stalk character in onion. Ann. Appl. Biol. 108:199–204.

Jones, H.A. and A.E. Clarke. 1943. Inheritance of male sterility in the onion and the production of hybrid seed. Proc. Amer. Soc. Hort. Sci. 43:189–194.

Jones, H.A., A.E. Clarke, and F.J. Stevenson. 1944. Studies in the genetics of the onion (*Allium cepa* L.). Proc. Amer. Soc. Hort. Sci. 44:479–484.

Jones, H.A. and B.A. Perry. 1956. Inheritance of resistance to pink root in the onion. J. Hered. 47:33–34.

Jones, H.A. and C.E. Peterson. 1952. Complementary factors for light-red bulb color in onions. Proc. Amer. Soc. Hort. Soc. 59:457.

Jones, R., and H. Rees. 1968. Nuclear DNA variation in *Allium*. Heredity 23:591–605.

King, J.J., J.M. Bradeen, O. Bark, J.A. McCallum, and M.J. Havey. 1998. A low-density genetic map of onion reveals a role for tandem duplication in the evolution of an extremely large diploid genome. Theor. Appl. Genet. 96:52–62.

King, J.J., J.M. Bradeen, and M.J. Havey. 1998. Variability for restriction fragment-length polymorphisms (RFLPs) and relationships among elite commercial inbred and virtual hybrid onion populations. J. Amer. Soc. Hort. Sci. 123:1034–1037.

Kofoet, A., C. Kik, W.A. Wietsma, and J.N. DeVries. 1990. Inheritance of resistance to downy mildew [*Peronospora destructor* (Berk.) Casp.] from *Allium roylei* Stearn in the backcross *Allium cepa* L. x (*A. roylei* x *A. cepa*). Plant Breeding 105:144–149.

Magnum, P.D. and E.B. Peffley. 1994. Inheritance of ADH, 6-PGDH, PGM, and SKDH in *Allium fistulosum* L. J. Amer. Soc. Hort. Sci. 119:335–338.

Molenaar, N.D. 1984. Genetics, thrips (*Thrips tabaci* L.) resistance and epicuticular wax characteristics of nonglossy and glossy onions (*Allium cepa* L.). PhD Diss., Dept. of Horticulture, Univ. of Wisconsin, Madison.

Nakamura, S. and M. Tahara. 1977. Studies on the determination of species and cultivars on the basis of electrophoretic patterns of seed protein and seed enzymes. II. *Allium* spp., cucumber and melon, pea, and garden bean. J. Jpn. Soc. Hort. Sci. 46:233–244.

Nichols, C. G., W.H. Gabelman, R.H. Larson, and J.C. Walker. 1965. The expression and inheritance of resistance to pink root in onion seedlings. Phytopathology 55:752–756.

Peffley, E.B. 1993. Molecular markers in *Allium*: A review. Proc. 1993 Natl. Onion Res. Conf. p. 115–120.

Peffley, E. B. , J.N. Corgan, K.E. Horak, and S.D. Tanksley. 1985. Electrophoretic analysis of *Allium* alien addition lines. Theor. Appl. Genet. 71:176–184.

Peffley, E.B. and L. Currah. 1988. The chromosomal locations of enzyme-coding genes in *Adh*-1 and *Pgm*-1 in *Allium fistulosum*. Theor. Appl. Genet. 75:945–949.

Peffley, E. B. and C. Orozco-Castillo. 1987. Polymorphism of isozymes within plant introductions of *Allium cepa* L. and *A. fistulosum* L. HortScience 22:956–957.

Pike, L.M. 1986. Onion breeding, p. 357–394. In: M. Bassett (ed.). Breeding vegetable crops. AVI Press, Westport, Conn.

Rabinowitch, H.D. 1988. Genetics and breeding: state of the art or too slow but not too late, p. 57–70. In: Proc. EUCARPIA 4th *Allium* Symp., Inst. Hort. Res., Wellesbourne, Warwick, U.K.

Rabinowitch, H.D., B. Friedlander, and R.J. Peters. 1984. Inheritance and characterisation of dwarf scape in onions: A progress report, p. 83–89. In: Proc. EUCARPIA 3rd *Allium* Symp., Inst. Hort. Plant Breeding, Wageningen, The Netherlands.

Ranjekar, P.K., D. Pallotta, and J.G. LaFontaine. 1978. Analysis of plant genomes. V. Comparative study of molecular properties of DNAs of seven *Allium* species. Biochem. Genet. 16:957–970.

Rasmusson, J. 1920. Mendelnde chlorophyllfaktoren bei *Allium cepa*. Hereditas 1:128–134.

Ricroch, A., E.B. Peffley, and R.J. Baker. 1992. Chromosomal location of rDNA in Allium: in situ hybridization using biotin- and fluorescein-labelled probe. Theor. Appl. Genet. 83:413–418.

Rieman, G.H. 1931. Genetic factors for pigmentation in the onion and their relation to disease resistance. J. Agr. Res. 42:251–278.

Schweisguth, B. 1973. Etude d'un nouveau type de stérilité mâle chez l'oignon, *Allium cepa* L. Ann. Amelior. Plant. 23:221–233.

Sumner, D. R. 1995. Diplodia stain, p. 28–29. In: H.F. Schwartz and S.K. Mohan (eds.). Compendium of onion and garlic diseases. APS Press, St. Paul, Minn.

Tsutsui, K. 1991. Inheritance of resistance to *Fusarium oxysporum* in onion. MS Thesis, Dept. Hort., Univ. Wisconsin, Madison.

Ulloa-Godinez, M., J.N. Corgan, and M. Dunford. 1994. Chromosome characteristics and behavior differences in *Allium fistulosum* L., *A. cepa* L., their F_1 hybrid, and selected backcross progeny. Theor. Appl. Genet. 89:567–571.

Ulloa-Godinez, M., J.N. Corgan, and M. Dunford. 1995. Evidence for nuclear-cytoplasmic incompatibility between *Allium fistulosum* and *A. cepa*. Theor. Appl. Genet. 90:746–754.

U.S. Dept. of Agriculture. 1998. Vegetables, Annual Summary for 1997. U.S. Dept. Agr., Washington, D.C.

van Damme, E.J.M., K. Smeets, S. Torrekens, F. van Leuven, and W.J. Peumans. 1992. Isolation and characterization of alliinase cDNA clones from garlic (*Allium sativum* L.) and related species. Eur. J. Biochem. 209:751–757.

Walker, J.C. 1923. Disease resistance to onion smudge. J. Agr. Res. 24:1019–1040.

Warid, W.A. 1952. Inheritance studies in the onion, *Allium cepa* L. PhD Diss., Dept. of Bot., Bact., and Plant Path., Louisiana State Univ., Baton Rouge.

Warid, W.A. and E.C. Tims. 1952. Studies on the inheritance of resistance to downy mildew studies in onion incited by *Peronospora destructor*. Phytopathology 42:22.