Brief Communication

Molecular and Phenotypic Mapping of Genes Controlling Seed Coat Pattern and Color in Common Bean (*Phaseolus vulgaris* L.)

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Common bean (*Phaseolus vulgaris* L.) exhibits a wide variety of seed coat patterns and colors. From a historical perspective, extensive genetic analyses have identified specific genes that control seed coat pattern (*T, Z, L, J, Bip,* and *Ana*) and color (*P, C, R, J, D, G, B, V,* and *Rk*). Many of these genes exhibit epistatic interactions with other genes, interactions that define the many seed coat patterns and colors observed within the species. To better understand these complex interactions, we began a molecular marker discovery program that previously identified random amplified polymorphic DNA (RAPD) markers linked to many of these genes. We report here the discovery of RAPD markers linked to three additional genes—*C, G,* and *V.* These markers, and five RAPD markers we previously discovered linked to other seed coat pattern and color genes, were converted into easily scorable sequence tagged site (STS) markers. We next placed these markers onto a common molecular map shared by the *Phaseolus* research community and demonstrated a generally wide distribution of the genes throughout the common bean genome. A few previously unrecognized linkages were discovered. The probable existence of multiple genes controlling growth habit in common bean is discussed. Our approach demonstrates the usefulness and feasibility of marker discovery in one population followed by accurate mapping of that marker onto a core, community-wide map.

The color and pattern of the seed coat and flower are basic phenotypic features often used to distinguish genotypes of higher plant species. In common bean (*Phaseolus vulgaris* L.), color is controlled by a group of well-defined genes that appear to regulate the flavonol and anthocyanin biosynthetic pathways. Color expression in either the seed coat or flower is completely dependent on the multiple alleles at the *P* locus (Emerson 1909). The dominant allele at *P* potentiates color in the seed coat and flower. These tissues are white in homozygous recessive *pp* individuals, whereas individuals with nearly white flowers and gray-white seed coats are homozygous at p^{gri}, another allele at *P* (Bassett 1994). In *P_* individuals, a multiallelic, dominance series at *V* controls flower color: *V_* (purple) > v^{lae}_ (pink) > *vv* (white) (Lamprecht 1935). In addition, the seed coats of *V_* genotypes contain anthocyanin pigments, whereas the seed coats of *vv* genotypes contain flavonol pigments (Beninger et al. 1999, 2000). Alleles at seven other genes—*Gy, C, R, J, G, B,* and *Rk*—interact with *V* and among each other to determine the many color hues found in the common bean seed coat (Prakken 1972; Bassett et al. 2001a).

The pattern of the common bean seed coat is controlled by the action of two independent genes, *C* and *T.* As with all of these genes, each requires a dominant *P* allele for phenotypic expression. *C* is a complex locus consisting of many closely linked genes responsible for a variety of patterns including striping, speckling, and mottling (Prakken 1974). These patterns are generally distinguished by a light background color (usually cartridge buff) overlain with a darker pattern color determined by the genotype of the color genes just described. Tightly linked within the *C* locus complex is *R,* a gene responsible for some of the red seed coat colors (Prakken 1974).

Common bean seed coats can also be partly colored, in which a white background is overlain by a colored pattern. The hue of the pattern is determined by the genotype at the color genes just described. Partly colored seed coat patterns require the homozygous recessive *tt* genotype at *T* (Emerson 1909) and epistatic interactions among three other genes: *Z, L,* and *Bip* (Bassett and McClean 2000). Depending on the genotype within the series, the many partly colored seed coat phenotypes can range from the nearly completely colored *expansa,* to the partially colored *virgarcus,* to the slightly colored *bipunctata,* to completely white. The array of phenotypes is illustrated in Bassett and McClean (2000).

To deepen our understanding of these complex interactions we began developing molecular markers linked to these genes (Brady et al. 1998; Bassett et al. 1999b, 2000). Several of these markers were then used as tools to reveal previously unrecognized allelic relationships (Bassett et al. 1999a,b, 2000). To better understand the distribution of genes in common bean and the phenotypic effects that are manifested by their distribution, we are now mapping these genes and their markers onto a common bean linkage map. This article reports the development of new markers for several genes, the conversion of all RAPD markers to STS markers (Olson et al. 1989), the phenotypic mapping of several genes on the BAT93 × Jalo EEP558 linkage map (Freyre et al. 1998; Nodari et al. 1992), and the mapping of the STS markers onto the same linkage map.

Materials and Methods

Development and Mapping of RAPD and STS Markers

RAPD markers linked to a particular gene were discovered using the procedure described by Brady et al. (1998). Briefly, the steps are (1) an F_2 population ($n = 80$) segregating for an individual gene (Table 1)

Table 1. Genetic crosses used to map nine genes that control seed coat color and pattern in common bean (*Phaseolus vulgaris* L.)

Gene	Cross[a]	Recessive allele donor	Reference
Pattern genes			
T	*t* BC_2 5–593 × 5–593	Early Wax	Brady et al. (1998)
Z	*t z* virgarcus BC_3 5–593 × *t* All black BC_2 5–593	PI 527712	Brady et al. (1998)
D (= *Z*)[b]	*j d* BC_2 5–593 × *j v* BC_2 5–593	PI 527806	Bassett et al. (1999b)
J (= *L*)[c]	Thuringia × *t z* virgarcus BC_3 5–593	Thuringia	Bassett et al. (2001b)
Bip	*t z bip* bipunctata BC_3 5–593 × *t z* virgarcus BC_3 5–593	PI 527712	Bassett et al. (2000)
Ana (= *Bip*)	*t ana* BC_3 5–593 × *t z* virgarcus BC_3 5–593	PI 451802	Bassett et al. (2000)
Color genes			
c^v	c^v BC_2 5–593 × c^u BC_3 5–593	PI 527774	This article
G	*g b v* BC_2 5–593 × *b v* BC_3 5–593	Calima	This article
V	*g v* BC_2 5–593 × 5–593	PI 527830	This article
B	*g b v* BC_1 5–593 × *v* BC_3 5–593	L-64	This article
Gy	Wagenaar × *g b v* BC_3 5–593	Wagenaar	Bassett et al. (2001a)

[a] Breeding line 5–593 was the recurrent parent used to develop the genetic stocks. The 5–593 genotype is *T Bip P* [*C r*] *Z J G B V Rk Gy.* The genetic stocks, used to create the segregating populations, were developed by backcrossing a specific allele into 5–593. The nomenclature for the genetic stock includes the introduced allele and the level of backcrossing. Thus the designation *t* BC_2 5–593 means that *t* replaced the *T* allele by crossing through the second backcross generation.

[b] The hilum ring factor, *D,* was recently shown to be an allele of the zonal, *Z,* gene (Bassett et al. 1999b).

[c] The limiter gene, *L* (Schreiber 1940), was recently shown to be an allele of the joker, *J,* gene (Bassett et al. 2001b).

was scored phenotypically; (2) DNA bulks (Michelmore et al. 1991) were created from the two contrasting phenotypic classes; (3) RAPD polymorphisms were discovered; and (4) linkage between the RAPD fragment and the gene was determined by scoring the segregating population and calculating the Kosambi distance using MapMaker 3.0 (Lander et al. 1987). RAPD fragments were cloned, and the cloned fragment was hybridized to the original segregating population to ensure it represented the same polymorphism as the RAPD marker. The clone was sequenced using a Beckman CEQ2000XL capillary sequencer. STS primers were developed for each cloned fragment, and the optimum annealing temperature and Mg^{2+} concentration were determined. The genotypes BAT93 and Jalo EEP558 were scored for RAPD and STS polymorphisms (Table 2), and the BAT93 × Jalo EEP558 recombinant inbred (RI) population (n = 74; Freyre et al. 1998; Nodari et al. 1992) was scored for each polymorphic STS primer pair. If the STS pair was not polymorphic, the population was scored with the corresponding RAPD primers. Each clone corresponding to the STS polymorphism was hybridized to the BAT93 × Jalo EEP558 (hereafter referred to as BJ) RI population to confirm the segregating RAPD and STS fragment was homologous to the original RAPD fragment. The hybridization conditions are described in Rivkin et al. (1999). Each marker was placed on a linkage group relative to a set of core markers using MapMaker 3.0 (Lander et al. 1987) with the parameters described in Freyre et al. (1998) and Gepts (1999).

Phenotypic Scoring and Mapping of *J, G, B, C,* and *V*

The F_1 of the crosses of BAT93 and Jalo-EEP558 to the *j* BC_2 5-593 tester produced a nonallelic interaction. Therefore the two parents were homozygous for the *J* allele. The *G* and *B* genotypes of the BAT93, Jalo EEP558, and BJ RI lines were determined

Table 2. RAPD and STS marker polymorphisms and STS primer sequences and PCR conditions for genes controlling color and pattern in common bean seed coats

Gene	RAPD marker[a]	Linkage distance (cM)	BAT/Jalo RAPD polymorphism (bp)		STS primers[b]	STS PCR conditions[c]		BAT93/Jalo STS polymorphism (bp)	
			BAT93	Jalo EEP558		Annealing temperature (°C)	$[Mg^{2+}]$ (mm)	BAT93	Jalo EEP558
Pattern genes									
T	$OM19_{400}$	1.4	Null	400	F: 5′-CCTTCAGGCAGAGGAGGTTCCATT-3′ R: 5′-CCTTCAGGCACAATCTCAGGCTGA-3′	64	1.5	350	350
Z	$OAM10_{560}$	1.4	560	Null	F: 5′-GGAGGAAGGTAACAGTTCTCTCAG-3′ R: 5′-CCCTTCACATAAGTGGAAGTTGT-3′	67	1.0	490	Null
L	$OL4_{525}$	1.2	Null	500	F: 5′-CACACAGTGAAAGTGCTTAATTCC-3′ R: 5′-GACTGCACACTATCCATAAAAAT-3′	60	1.5	Null	500
Bip	$OJ17_{700}$	6.0	700	Null	F: 5′-AATAACCAAGTCGACGGAGAAAG-3′ R: 5′-GTAGAGATCGCACTCCCAAGTTAG-3′	65	1.5	550	750
Ana	$OM9_{200}$	5.4	200	Null	F: 5′-GTCTTGCGGAGCGATCCCTGGCAT-3′ R: 5′-GTCTTGCGGATGAAAGAAGTGAAT-3′	60	1.5	200	200
Color genes									
C	$OAP2_{700}$	4.9	Null	700	F: 5′-TCCCTAGAAGCAAACACAACATAA-3′ R: 5′-TTGCATGGATGTGTAAGAGTTTCT-3′	58	1.5	600	650
G	$OAP3_{1400}$	0.0	1400	Null	F: 5′-GTAAGGCGCATACTTGGACAACTT-3′ R: 5′-GTAAGGCGCAGGTACAGAGGGAGG-3′	70	1.5	1400	Null
G	$OAP7_{850}$	0.0	850	Null	F: 5′-ACCACCCGCTGCCTTATCACCAAC-3′ R: 5′-ACCACCCGCTTACTAAGCTCACTC-3′	70	1.5	850	Null
G	$OU14_{900}$	0.0	900	Null	F: 5′-CGAAAACTTCTAGGGTCAGGACAT-3′ R: 5′-ATTCATACTTCAAGTGGCGTGTT-3′	60	1.5	850	Null
V	$OD12_{800}$	0.0	800	Null	F: 5′-GAGAAGCAGGAGGACGAGAAG-3′ R: 5′-CTAGCATCTAGGGAGGAGGCAT-3′	65	1.0	490	Null
Gy	$OW17_{600}$	1.6	600	Null	F: 5′-GTCCTGGGTTTTGGAGGTTGATGT-3′ R: 5′-GTCCTGGGTTCAATTCCATAGTCC-3′	73	2.0	600	Null

[a] The corresponding designation for the STS is Primer-S-Largest Amplification Product Size. For example, the STS that corresponds to $OM19_{400}$ is $OM19S_{350}$.

[b] F = forward primer; R = reverse primer; underlined sequences represent the original RAPD primer sequence. For those primer pairs not underlined, the primers were designed to sequences internal to the original RAPD primer sequence.

[c] PCR conditions: 94°C, 1 min; annealing temperature, 1 min; 72°C, 2 min; 40 cycles.

by scoring F_1s following a cross to the *g b v* BC_2 5-593 tester (Bassett 1998). Those individuals who's F_1 expressed either buffy citrine (*gBv*) or chamois (*gbv*) seed coat color were scored as *gg,* whereas those expressing mineral brown (*GBv*) or yellow brown (*Gbv*) seed coat color were scored as *GG.* Those individuals who's F_1 expressed either shamois (*gbv*) or yellow brown (*Gbv*) seed coat color were scored as *bb,* whereas those expressing mineral brown (*GBv*) or buffy citrine (*gBv*) seed coat color were scored as *BB.* The *C* genotypes of the two parents and derived BJ RI lines were determined by scoring F_1s following a cross to the c^u BC_2 5-593 tester (Bassett 1995). Those individuals expressing strong marbling in the F_1 generation were scored as $C^{hm}C^{hm}$ (where C^{hm} is a tentative gene symbol for heterozygous marbling), whereas those expressing solid color in the F_1 generation were *CC.* The *V* genotypes of the two parents and derived BJ RI lines were determined by scoring flower color (v^{lae} = pink; *v* = white). Those genes segregating in the RI population were placed on a linkage group relative to a set of core markers (Freyre et al. 1998) by using the MapMaker software with the parameters described in Freyre et al. (1998) and Gepts (1999).

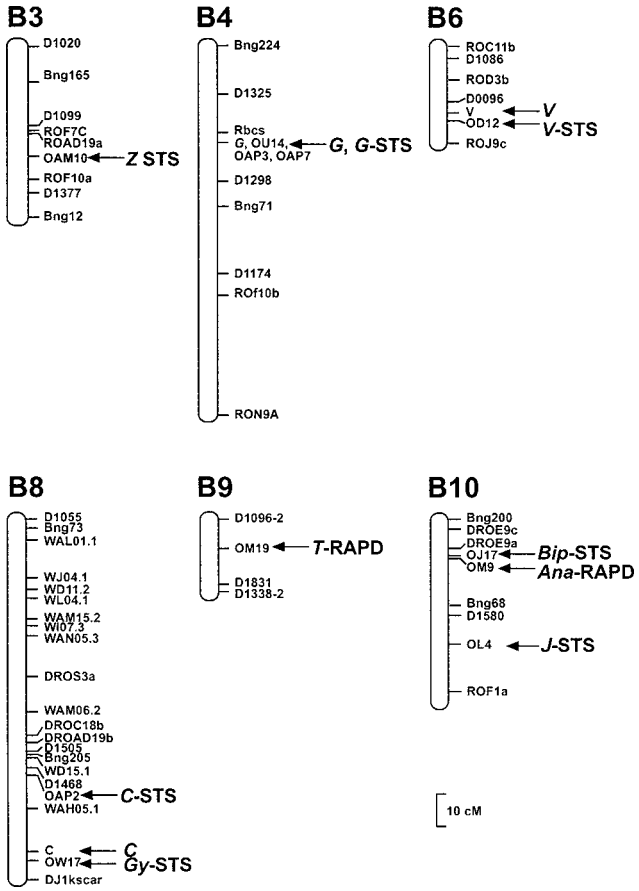

Figure 1. STS and RAPD marker distribution of eight loci controlling seed coat color and patterning in common bean (*Phaseolus vulgaris* L.). Each marker is noted by its primer name. See Table 2 for the fragment size of the marker. The location of each phenotypic gene and marker mapped on the population is highlighted by bold notation to the right of the linkage group.

Results and Discussion

We previously published the identification and application of a number of molecular markers linked to several genes that control seed coat patterning in common bean (Bassett et al. 1999a,b, 2000; Brady et al. 1998). Using an approach that combines the phenotypic and genotypic analysis of several F_2 segregating generations along with RAPD screening and scoring procedures (Brady et al. 1998), we have extended that research here and report the discovery of markers linked to three important seed coat color genes—*C, G,* and *V.* The population segregating for *C* (Table 1) fit the expected 3 gray brown:1 cartridge buff ratio (χ^2 = 0.7, *P* = .80). A RAPD marker ($OAP2_{700}$) linked in repulsion to the c^v allele was discovered. All cartridge buff plants contained the marker fragment, and all plants lacking the fragment were gray brown. The distance between $OAP2_{700}$ and *C* was estimated to be 4.9 cM (Table 2). The population segregating for *G* (Table 1) fit the expected 3 yellow brown:1 pale greenish yellow ratio (χ^2 = 1.64, *P* = .20). Three RAPD markers ($OAP3_{1400}$, $OAP7_{850}$, and $OU14_{900}$) were discovered to be in coupling phase with the *G* allele, and each cosegregated with the locus (Table 2). The population segregating for *V* (Table 1) fit the expected 12 black:3 mineral brown:1 buffy citrane ratio (χ^2 = 4.32, *P* = .12). RAPD marker $OD12_{800}$, linked in coupling phase with the dominant *V* allele, cosegregated with the locus (Table 2).

If a molecular marker derived in one population is to be mapped to a linkage group in a second population, it is important to confirm the marker is actually mapping to its correct genetic location. One approach to address this issue is to phenotypically map several genes in the mapping population. If the marker and gene are also linked in the mapping population, confidence can be placed in the map location of the marker. We applied that approach by first determining the *C, G, B,* and *V* genotypes of BAT93 and Jalo EEP558 and then phenotypically mapping these four genes in the BJ core common bean mapping population. The markers linked to these genes were then mapped in the same population. In each case, the marker and its linked gene mapped to the same linkage group, and the genetic distance between the marker and the gene was similar to that observed in the original population used to discover the marker.

We determined the genotype of BAT93 to be $CJGBv^{lae}$ and the genotype of Jalo EEP558 to be $C^{hm}Jgbv$. *B* mapped to the end of linkage group B2, but the LOD value of the marker order did not meet our criteria. It was shown previously that the bean common mosaic virus resistance gene *I,* known to be tightly linked to *B* (Kyle and Dickson 1988), also maps to the end of B2 (Freyre et al. 1998). This observation suggests that the location of *B* on B2 is correct.

Phenotypic mapping placed *G* on linkage group B4 of the core *P. vulgaris* linkage map (Freyre et al. 1998) (Figure 1). As in the original mapping population, three independently derived STS markers ($OAP3S_{1400}$, $OAP7S_{850}$, $OU14S_{850}$) cosegregated with the *G* phenotype. *V* was mapped phenotypically to linkage group B6 (Figure 1), and the independently derived *V* marker ($OD12S_{490}$) mapped 2.8 cM from the *V* locus. This linkage distance

represented recombination events not observed in the population in which the marker was discovered. The mapping of *V* to B6 is consistent with the BJ F_2 mapping results, which placed FLCO (= *V*) on linkage group D6 (Nodari et al. 1992). Of interest to any future cloning experiments, the RFLP marker D0096 cosegregated with the gene (Figure 1).

With the phenotypic mapping approach, *C* was placed on linkage group B8 (Figure 1). The mapping of the *C* STS marker, $OAP2S_{650}$, to group B8 is consistent with this phenotypic mapping result. Both of these results are consistent with those of Miklas et al. (2000) who mapped *R,* a locus very tightly linked to *C* (Prakken 1974), to B8. $OAP2S_{650}$ was located 22.7 cM from the *C* locus in the BJ population. In the mapping population used to derive the marker, $OAP2S_{650}$ was located 4.9 cM from *C.* The original mapping population consists of two derivatives of the same parent, 5-593, whereas the BJ cross is more divergent. Therefore the linkage distance differences may reflect variable recombination rates in the two populations. However, this difference appears to be locus specific because the distances between *G* and *V* and their linked markers in both the BJ and original mapping populations were similar (Table 1 and Figure 1).

Gy, a recently described gene (Bassett et al. 2002a), was located to a linkage group based on its marker location. The recessive allele at *Gy* controls the strong greenish-yellow color expressed in the cultivar Wagenaar. Molecular mapping placed $OW17S_{600}$, an STS derivative of the *Gy*-linked RAPD marker (Bassett et al. 2002a), on linkage group B8. The closest RFLP marker, DJ1kscar, was located 5.7 cM from the *Gy* STS marker. The mapping analysis also showed that the *Gy* STS marker was linked to *C* at a distance of 2.7 cM (Figure 1).

Because the BJ population does not segregate for seed coat patterning genes (other than *C*), those genes were located to linkage groups based on the map location of linked markers. The RAPD marker for *T* ($OM19_{400}$; Brady et al. 1998) mapped to linkage group B9. The closest RFLP marker, D1096-2, was located 9.0 cM from the *T* marker (Figure 1).

Recent research demonstrates that *Ana* and *Bip* are allelic (Bassett et al. 2000), and *Ana* has subsequently been given the allelic designation bip^{ana}. That study also describes two markers, one linked to each allele, from independent segregating populations. Their linkage was confirmed by molecular analysis of the BJ population, where the STS markers for *Bip* ($OJ17S_{750}$) and bip^{ana} ($OM9S_{200}$) mapped to linkage group B10, 0.7 cM apart (Figure 1).

Recently a molecular marker ($OL4_{525}$) was discovered to be linked to *Limiter* (*L*), and the marker provides supporting evidence regarding the allelic relationship between *L* and *Joker* (*J*; Bassett et al. 2002b). The gene symbol *J* was given preference. The corresponding STS marker, $OL4S_{500}$, mapped to linkage group B10, 8.9 cM from the RFLP marker (D158) and 27.7 cM from the bip^{ana} STS marker (Figure 1).

$OAM10S_{490}$, developed from the *Z*-associated RAPD marker (Brady et al. 1998), was positioned on linkage group B3. The closest RFLP marker, D1096-2, is located 6.7 cM away. At first glance the placement of *Z* on B3 seems to disagree with the research of Koinange et al. (1996). That group positioned *fin,* a gene that controls determinate versus indeterminate growth habit and is tightly linked to *Z* (Bassett 1997; Surface 1916), within linkage group B1. Several technical difficulties might explain the result, but none seem plausible. It might be argued that the linkage detected between *Z* and the RAPD marker $OAM10_{490}$ is not correct. Yet the marker is also found to be linked to *D,* the hilum ring factor gene that was recently demonstrated to be an allele of *Z* (Bassett et al. 1999b). It is also possible that the fragment amplified in the BJ population was not related to the fragment originally shown to be linked to *Z.* This relationship, though, was confirmed by hybridization (data not shown). Finally, it is possible that a translocation event occurred between B1 and B3 during the development of the BJ population. Chromosome rearrangements appear unlikely since both B1 and B3 markers are colinear in three separate mapping populations (Freyre et al. 1998). Based on current data, we are led to conclude that multiple determinate growth habit genes exist in common bean. The following narrative explains our rationale.

The source of indeterminacy used by Bassett (1997) to discover the linkage between growth habit and gene *Z* was Steuben Yellow Eye. Based on its "T" phaseolin pattern and its elongated, cylindrical seed shape, this line belongs to the Nueva Granada race of the Andean gene pool (Singh et al. 1991). In contrast, G12873, used by Koinange et al. (1996) as the source of indeterminacy to map growth habit to B1, is a small-seeded wild bean with the "M" phaseolin pattern (Gepts et al. 1986) from the Middle American gene pool. In addition, the determinate genotypes used by Bassett (1997) and Koinange et al. (1996) are recessive at both loci, and the two indeterminate genotypes they used are homozygous dominant at only one growth habit locus. Finally, if growth habit evolved separately in each gene pool, as suggested by Singh et al. (1991), the two determinate growth habit loci mapped in these two distinctly different crosses would not necessarily, as we discovered, be located on the same linkage group.

Support for the existence of multiple growth habit genes in common bean is provided by the recent research of Ta'an et al. (2001). They mapped the growth habit gene in Sanilac to a linkage group colinear with the Florida linkage group K. Linkage group K is equivalent to B9 of the BJ linkage map (Freyre et al. 1998). By comparison, Koinanage et al. (1996) mapped the G12873 growth habit gene to B1. Since the Sanilac and G12873 growth habit genes map to different linkage groups, multiple growth habit genes must exist in common bean. The existence of multiple growth habit genes could be explicitly proven genetically by crossing two indeterminate (Steuben Yellow Eye and G12873) and two determinate (5-593 and Midas) genotypes and analyzing their segregating progenies.

This research provides the first global perspective of the genomic distribution of eight genes responsible for a number of seed coat patterns and colors in common bean. These genes are essential components that define each of the major market classes of dry bean with respect to seed coat color and pattern. The observation that, in general, these genes are not linked provides a clear genetic explanation for the wide variation in seed coat phenotype seen in common bean. It also explains why crosses among market classes generate a wide range of seed coat phenotypes.

The research also emphasizes the value of extensive genetic tester stocks (Bassett 1998) for marker discovery and the utility of a variable mapping population. Continued mapping in the BJ RI population will provide the *Phaseolus* research community a common mapping source upon which further comparative genetic and molecular research can be based.

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Received April 3, 2001
Accepted November 26, 2001

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