Inheritance and Epistasis of Loci Influencing Carotenoid Content in Petal and Pollen Color Variants of California Poppy (Eschscholzia californica Cham.)

PHILIPPA J. BARRELL, ANGELA M. WAKELIN, MICHELLE L. GATEHOUSE, CAROLYN E. LISTER, AND ANTHONY J. CONNER

From the New Zealand Institute for Plant & Food Research Limited, Christchurch 8140, New Zealand.

Address correspondence to Philippa J. Barrell at the address above, or e-mail: philippa.barrell@plantandfood.co.nz

Abstract

The model basal eudicot plant California poppy (Eschscholzia californica Cham.) typically has intense yellow to orange petals and orange pollen due to pigmentation by carotenoids. Flower color variants ranging from white to yellow and orange are common. We analyzed flower color inheritance in a diverse range of white and yellow color variants with reduced carotenoid content. The inheritance of the petal–pollen color of 24 variant flowers was investigated through complementation analysis by hybridization between different color variants and screening F1, F2, and BC1 populations for segregation of petal–pollen color. All white and yellow flower color variants exhibited the pleiotropic effect with each mutation influencing both petal and pollen color, with both petal and pollen color phenotypes co-inherited. A total of 5 complementation groups were identified with the color variants behaving as single recessive loci. Epistatic interactions among the loci were also identified. The white/yellow California poppy color variants described in this paper represent a unique genetic resource for analysis of carotenoid biosynthesis in this basal eudicot species.

Key words: California poppy, Eschscholzia californica, epistatic interactions, inheritance

Carotenoids are among the most widespread of all natural pigments (Garcia-Asua et al. 1998; DellaPenna and Pogson 2006; Maresca et al. 2008) and represent one of the largest classes of pigments in nature (van den Berg et al. 2000). The pigments are synthesized in algae, plants, and bacteria (Albrecht et al. 1995), where their colors vary through yellow and orange to red. Carotenoids have a diverse range of functions in plants. In algae and higher plants, carotenoids are essential for photosynthesis by contributing to light harvesting and maintaining the structure and function of photosynthetic complexes (Siefermann-Harms 1987; Green and Durnford 1996). Carotenoids quench chlorophyll triplet states, which is essential in preventing chlorophyll from bleaching in high-light conditions (Mozzo et al. 2008). Carotenoids are also required for thermal energy dissipation and scavenge reactive oxygen species (Garcia-Asua et al. 1998).

The biosynthesis of carotenoids has been extensively studied from a wide range of plant genera, and enzymes catalyzing the steps in the pathway have been identified and characterized from a number of species (Bartley et al. 1994; Cunningham and Gantt 1998; Cunningham 2002; Grotewold 2006). The first true carotenoid in the biosynthetic pathway is phytoene, a colorless compound formed via the condensation of 2 molecules of geranylgeranyl diphosphate. Subsequent desaturations of phytoene by phytoene desaturase produce the first colored carotenoids, neurosporene or lycopene, depending on the number of conjugated double bonds added. Addition of hydroxyl and methyl groups and further double bonds by other enzymes cause a multitude of different carotenoid structures to form (Garcia-Asua et al. 1998).

California poppy (Eschscholzia californica Cham.) typically has bright orange flowers. The principal carotenoid components of wild-type orange California poppy flowers are esters of a xanthophyll, eschscholtzxanthin (Strain 1938; Andrewes et al. 1979). Strain (1938) also reported the presence of zeaxanthin, lutein, and other xanthophylls, whereas Maoka et al. (2000) described 11 carotenoids, mostly xanthophylls, including a retro-carotenoid from the yellow petal tissue, 4’5-retro-β,β-carotene-3,5,3’-triol.

A number of California poppy flower color variants have been reported (Beatty 1936; Douwes 1943; Wakelin et al.
Previously, we have characterized a white-flowered variant that was deficient in carotenoid production in both petals and pollen (Wakelin et al. 2003). In this paper, we describe the genetic characterization of a diverse collection of additional 23 sources of white, pale yellow, and yellow flower color variants of California poppy. This defined 5 independent loci contributing to flower color. Total carotenoid levels assayed in petal tissue confirmed the flowers from each homozygous variant genotype to be deficient in carotenoid content, thereby confirming that the mutations causing the white and yellow flower colors are genes involved in carotenoid production.

Materials and Methods

Plant Material

Seeds of 11 flower color variants of California poppy from naturalized populations in the South Island of New Zealand and from 12 commercially available cultivars were analyzed in this study (Supplementary Table A). All plants were grown in one greenhouse at the same time at Lincoln, Canterbury, under uniform conditions according to Wakelin et al. (2003) to allow direct comparison of petal–pollen color. Petal and pollen color of the color variants was compared visually with standardized color charts (Royal Horticultural Society 1966). For hybridization experiments, flowers were emasculated prior to hybridization and stigmas were covered for 48 h to exclude contamination with other pollen. For complementation analysis, the first filial generation (F1) represented the progeny resulting from hybridization of different sources of flower color phenotypes. The second filial generation (F2) was derived by hybridizing 2 F1 plants because most of the plants in this species were self-incompatible (Wright 1979). A total of 44 paired combinations were hybridized for complementation analyses (Supplementary Table B). Chi-square analysis was used to compare observed data with expected ratios for interpretation of inheritance.

Carotenoid Extraction

During all steps in the extraction process, samples were kept on ice and in the dark in order to minimize degradation of the carotenoids. Approximately, 50 mg of petal tissue was frozen in liquid nitrogen and ground for 10 s in 1.7-ml microfuge tubes with glass beads in a Silamat S6 dental amalgam mixer (Ivoclar Vivadent AG, Schaan, Principality of Liechtenstein). Four milliliters of acetone was added and the slurry transferred to a foil wrapped 15-ml tube on ice. Samples were rotated on a wheel in a working cool room in the dark for 1.5 h. The tubes were centrifuged at 3000 × g for 10 min at 4 °C. Aliquots of the supernatant were diluted into acetone, and 1 ml fractions were assayed in quartz cuvettes. Absorbance at 446 nm (β-carotene equivalents) and 550 nm (to correct for turbidity) was measured. Total carotenoid content was calculated by the following calculation:

\[
\text{total carotenoids (mg/g FW)} = \frac{D_{446} \times 10 \times V}{d \times E_{1\text{cm}} \times W}
\]

where, \(D_{446}\) = the absorbance at 446 nm minus the absorbance at 550 nm; \(V\) = original extraction volume (ml of acetone), \(d\) = path length (1 cm); \(E_{1\text{cm}}\) = extinction coefficient (2500; Goodwin 1955); \(W\) = weight of sample (g).

Results

Screen for Flower Color Variants

This study evaluated 23 color variants of California poppy of independent origin, including the previously reported white variant (Ben) (Wakelin et al. 2003). In addition, several yellow-flowered variants appeared unexpectedly among the progeny of a cross to determine the inheritance of the Incognitus white (Inc) variant, and were subsequently labeled Incognitus yellow (IncY), bringing the total number of flower color variants to 24. All variants were true breeding and did not display other defects in plant growth. Flower color variants ranged from white to yellow with intensity of color often varying with the source of the seed or the age of the flower. The petal and pollen colors of these plants are described in Supplementary Table A. Older petals generally were a slightly lighter shade than younger petals, as indicated by the codes matched to the Royal Horticultural Society (RHS) color charts. In all variants pollen color closely matched petal color. Although 24 color variants

Figure 1. Summary of the carotenoid biosynthetic pathway in plants. Carotenoids are in bold. Two molecules of geranylgeranyl diphosphate (GGPP) are condensed into phytoene, the first true carotenoid in the pathway by the enzyme phytoene synthase (PSY). Dashed arrows represent multiple enzymatic reactions. PDS: Phytoene desaturase; ZDS: Zeta-carotene desaturase.
were examined, only 15 separate color designations were assigned. For example, Benmore (Ben) found in a wild population in New Zealand, had the same color designation as Royal Fleur (RFL), and Vilmorin (VIL) commercial seed sources from France.

**Complementation Analysis**

Multiple crosses were made between the various sources of white- and yellow-flowered variants and the progeny screened for flower color (Supplementary Table B). In all cases, the crosses between different sources of white variants resulted in all white-flowered progeny, indicating that they all had a mutation at the same locus which we assigned the notation “b.” In all cases, the crosses between white- and yellow-flowered variants resulted in complementation to restore the wild-type orange flowers in all progeny. Some crosses between different sources of yellow-flowered plants also resulted in complementation (all orange-flowered progeny), whereas other crosses produced all yellow-flowered progeny (no complementation). Overall, the 24 progeny), whereas other crosses produced all yellow-flowered progeny (no complementation). Although, 6% compared with wild type, the s locus was more easily distinguished from the other yellow loci, and observed segregation in all combinations did not significantly differ from the expected 9:3:4 ratios in the F2 generations.

Total carotenoid analyses of petal tissue from each of the 5 loci showed highly reduced carotenoid levels. b locus petals showed approximately 2% carotenoid levels compared with wild type, the x locus contained approximately 6% compared with wild type, s and f loci showed approximately 10%, and the i locus 19%, respectively (Table 6).

**Discussion**

In order to more fully understand the synthesis of carotenoid pigments in flowers, it is desirable to have a number of variants with mutations in different genes along biosynthetic pathways toward pigment accumulation. We present here a comprehensive genetic analysis of a collection of California poppy (E. californica Cham.) flower color variants with decreased carotenoid content in petals. A range of California poppy flower color variants were assembled from natural populations in New Zealand and commercially available cultivars. Their pollen and petal colors were described (Supplementary Table A), and the results of complementation analyses, inheritance, and/or epistatic interactions between loci determined.

Based on the results of the complementation crosses, the yellow variants were assigned to 4 independent loci (Table 1), each most likely representing a mutation in a different gene in the carotenoid pathway. These 4 yellow-flowered loci (i, j, k, and l) resulted from mutations in different genes to the previously described white b locus (Wakelin et al. 2003). Complementation crosses between these yellow variants and the original white b locus produced progeny with wild-type orange flowers (Tables 2–5; Supplementary Table B) confirmed that these variants represent mutations at different loci.

| Table 1 California poppy mutants grouped according to complementation analysis |
|-----------------|-----------------|-----------------|-----------------|-----------------|
| **b locus (white)** | **i locus (yellow)** | **f locus (yellow)** | **s locus (yellow)** | **x locus (pale yellow)** |
| Alb, Aur, Ben, Eyr, Inc, Ivc, Maks, Mnd, Ren, Rif, Rs1, TayD, TayR, Tck, and Vil | IncY | B1f3, B1f5, and Rs1Y | SelIII and HurP | Sell, Hur1, and BenY |

For interpretation of variant names and abbreviations, see Supplementary Table A.
The Ben plants used in sources 2, 3, and 4 represent plants following full-sib mating for 1, 2, and 5 generations, respectively, with selection for fertility

The Ben plant used was derived from full-sib mating with selection for fertility for 5 generations; ns, not significant (Source 4 (Ben

Source 3 (Ben

Source 2 (Ben

Table 3

Complementation and subsequent segregation of the yellow i locus (IncY), the yellow s locus (SelIII), and the pale yellow x locus (HurI), with the white b locus (Alba) and (Ben) alleles

Table 2

Complementation and subsequent segregation of the yellow i locus (IncY), the yellow s locus (SelIII), and the pale yellow x locus (HurI), with the white b locus (Alba) and (Ben) alleles

Descriptions of flower color variants in California poppy variants are limited (Beatty 1936; Douwes 1943; Wakelin et al. 2003). Beatty (1936) described the inheritance of 2 flower color loci, yellow (CC, Ce or white (cc) ground petal color and red (RR, Rr) or non-red (rr) additional petal color. Douwes (1943) carried out more comprehensive inheritance studies on several different orange and yellow petal color variants, including the presence and color of a “basal foot.” Although Beatty (1936) did not mention any pigment deficiencies associated with petal color, Douwes (1943) analyzed the carotenoid, anthocyanin, and anthoxanthin (flavonol) composition of various flower colors. Carotenoids were reported as absent in white flower petals. The petal colors described by Douwes (1943) used the horticultural color chart and so can be cross-referenced to the RHS standard colors used to characterize the color variants reported in this study. Of the 7 different colors described, 3 closely matched some of the variants reported. These are white (b locus), lemon yellow (i locus), and sulfur yellow with saffron yellow foot (x locus). We adopted similar locus notations based on the color similarities, and added the f locus for the Blf3 and Blf5 and the x locus for SelI, HurI, and BenY variants that did not clearly match the descriptors of Douwes (1943).

Douwes (1943) designated the white mutation bb and found it was inherited as a single recessive allele through F2 and backcross data. These conclusions were supported by our previous data for the Ben variant (Wakelin et al. 2003). In an earlier study, Beatty (1936) also attributed white flower color to a single recessive allele (cc), with a yellow phenotype dominant. Unfortunately, Beatty does not include a description of the yellow color that would allow comparison with the yellow variants described in this paper. Neither Beatty (1936) nor Douwes (1943) mention pollen color reported by Wakelin et al. (2003). This study establishes that this pleiotropic effect occurs in 5 independent loci influencing flower color in California poppy (loci b, f, i, s, and x).

Table 3

Complementation and subsequent segregation of the f locus (Blf3 and Blf5) and the b locus (Alba and Ben)
The white variants reported in this paper (Supplementary Table A) originated from 7 natural New Zealand populations and 8 commercial sources from North America and Europe. When these variants were hybridized with the white-flowered Ben variant (Wakelin et al. 2003), only white progeny were recovered. This lack of complementation indicates that all the white-flowered variants reported in this study resulted from a mutation at the same locus, equivalent to the white-flowered locus reported by Beatty (1936) and Douwes (1943). Despite this, the white-flowered variants were independently assigned slightly different colors from the RHS color chart (Supplementary Table A). These slight differences in white flower color reflect the origin from geographically distinct populations and may be a consequence of different alleles at the \( b \) locus or the different genetic backgrounds of the plants. Similar minor differences were also apparent among the sources of yellow variants assigned to the \( f \), \( i \), and \( s \) loci.

Progeny segregation among the \( F_2 \) and BC generations were initially found to be significantly different to the expected ratios for some sources of the \( b \) locus (Table 3). This distorted segregation may result from a recessive lethal gene linked to the color loci, which is circumvented when using the \( b \) locus in a different genetic background. For example, further crosses made with a white mutant from a commercial source (Alba) produced progeny that were not significantly different from expected ratios for inheritance as a single recessive allele (Table 3). Beatty (1936) described an inbreeding effect in California poppy that resulted in lowered seed germination and longevity of plants from closely related parents. Low seed germination and seedling survival were noted in many of the crosses performed to determine the inheritance of various flower colors in California poppy in this study, causing low total progeny numbers. It is possible that inbreeding depression and accumulation of recessive lethal alleles caused the progeny

**Table 4** Complementation and subsequent segregation among progeny of crosses between the \( f \) (Blf3 and Blf5), \( i \) (IncY), and \( s \) loci (SelIII and HurP)

<table>
<thead>
<tr>
<th>Cross</th>
<th>Number of progeny</th>
<th>Expected ratio</th>
<th>Chi-square</th>
</tr>
</thead>
<tbody>
<tr>
<td>( f ) locus (Blf3) \times ( i ) locus (IncY)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( F_1: ) iiFF (yellow) \times iIFF (yellow)</td>
<td>4</td>
<td>0</td>
<td>---</td>
</tr>
<tr>
<td>( F_2: ) iiiFF (orange) \times iiFF (orange)</td>
<td>20</td>
<td>17</td>
<td>9:7</td>
</tr>
<tr>
<td><strong>BC:</strong> iIFF (yellow) \times iIFF (orange)</td>
<td>57</td>
<td>60</td>
<td>1:1</td>
</tr>
<tr>
<td><strong>BC:</strong> IIFF (yellow) \times iIFF (orange)</td>
<td>28</td>
<td>30</td>
<td>1:1</td>
</tr>
<tr>
<td>( f ) locus (Blf3) \times ( i ) locus (IncY)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( F_1: ) iiFF (yellow) \times iIFF (yellow)</td>
<td>54</td>
<td>0</td>
<td>---</td>
</tr>
<tr>
<td>( F_2: ) iiiFF (orange) \times iIFF (orange)</td>
<td>116</td>
<td>76</td>
<td>9:7</td>
</tr>
<tr>
<td><strong>i</strong> locus (IncY) \times ( s ) locus (SelIII)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( F_1: ) iiSSW (yellow) \times IIss (yellow)</td>
<td>41</td>
<td>0</td>
<td>---</td>
</tr>
<tr>
<td>( F_2: ) iisS (orange) \times IisS (orange)</td>
<td>57</td>
<td>38</td>
<td>9:7</td>
</tr>
<tr>
<td><strong>s</strong> locus (HurP) \times ( f ) locus (Blf5)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( F_1: ) ssFF (yellow) \times SSff (yellow)</td>
<td>48</td>
<td>0</td>
<td>---</td>
</tr>
<tr>
<td>( F_2: ) SsFf (orange) \times SsFf (orange)</td>
<td>45</td>
<td>42</td>
<td>9:7</td>
</tr>
</tbody>
</table>

\( \text{ns, not significant } (P > 0.05) \)

**Table 5** Complementation and subsequent segregation among progeny of crosses between the \( x \) locus (HurL and BenY) and the \( b \) (Ben), \( i \) (IncY), \( f \) (Blf5), or the \( s \) (HurP) loci

<table>
<thead>
<tr>
<th>Cross</th>
<th>Number of progeny</th>
<th>Expected ratio</th>
<th>Chi-square</th>
</tr>
</thead>
<tbody>
<tr>
<td>( b ) locus (Ben) \times ( x ) locus (BenY)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( F_1: ) bbXX (white) \times BBXX (pale yellow)</td>
<td>34</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>( F_2: ) BbXx (orange) \times BbXx (orange)</td>
<td>35</td>
<td>---</td>
<td>17</td>
</tr>
<tr>
<td>( x ) locus (HurL) \times ( i ) locus (IncY)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( F_1: ) xxII (pale yellow) \times XXii (yellow)</td>
<td>23</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>( F_2: ) XxIi (orange) \times XxIi (orange)</td>
<td>39</td>
<td>21</td>
<td>17</td>
</tr>
<tr>
<td>( f ) locus (Blf5) \times ( x ) locus (HurL)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( F_1: ) ffXX (yellow) \times FfXX (pale yellow)</td>
<td>38</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>( F_2: ) FfXx (orange) \times FfXx (orange)</td>
<td>43</td>
<td>19</td>
<td>20</td>
</tr>
<tr>
<td>( x ) locus (HurL) \times ( s ) locus (HurP)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( F_1: ) xxSS (pale yellow) \times XXss (yellow)</td>
<td>18</td>
<td></td>
<td></td>
</tr>
<tr>
<td>( F_2: ) XxSs (orange) \times XxSs (orange)</td>
<td>22</td>
<td>12</td>
<td>10</td>
</tr>
</tbody>
</table>

\( ^a \) The Ben plants used were derived from full-sib mating with selection for fertility for 5 generations; ns, not significant \( (P > 0.05) \).

754
observed segregation was consistent with modified F2 
they represent mutations in genes at different loci. The 
loci produced all orange progeny (Table 4), showing that 
recessively, the recessive, effectively blocking the entire pathway. Alterna-
carotenoid pathway that is nonfunctional when homozygous 
that is critical for the expression of all the genes in the 
ss, or 
the expression of the homozygous yellow phenotypes (ii, ff, 
the white 
locus is not homozygous recessive. Therefore, the white 
locus exhibits dominant epistasis over the i 
locus, that is, homozygous recessive white (bb) suppresses the expression of the homozygous yellow phenotypes (ii, ff, 
loci are only expressed when they are homozygous recessive and 
when the b locus is not homozygous recessive. Therefore, 
the white b locus exhibits dominant epistasis over the i 
locus, which is likely to have reduced the effect of any recessive lethal alleles.

Table 6  Quantification of total petal carotenoids in California 

<table>
<thead>
<tr>
<th>Locus</th>
<th>Carotenoid content (mg/gFW)</th>
</tr>
</thead>
<tbody>
<tr>
<td>b</td>
<td>0.034 ± 0.002</td>
</tr>
<tr>
<td>i</td>
<td>0.317 ± 0.016</td>
</tr>
<tr>
<td>s</td>
<td>0.165 ± 0.027</td>
</tr>
<tr>
<td>f</td>
<td>0.175 ± 0.019</td>
</tr>
<tr>
<td>x</td>
<td>0.110 ± 0.023</td>
</tr>
<tr>
<td>Wild type</td>
<td>1.715 ± 0.043</td>
</tr>
</tbody>
</table>

The mean carotenoid content ± standard error of the mean (n = 3) are shown as milligrams per gram of fresh weight.

ratios from the Ben × Blf3 F2 and BC plants to be skewed. 
This is supported by the absence of distorted segregation 
following 5 generations of full-sib mating and selection for 
fertility (Table 3), which is likely to have reduced the effect of 
any recessive lethal alleles.

Classical Mendelian 9:3:4 dihybrid progeny ratios in the 
F2 generations following complementation of the i, f, s, and 
x loci with the b locus indicated that an epistatic interaction 
occurred between these yellow loci and the white b locus 
(Table 2). The yellow phenotypes of the i, f, s, and x loci are 
only expressed when they are homozygous recessive and 
when the b locus is not homozygous recessive. Therefore, 
the white b locus exhibits dominant epistasis over the i 
locus, that is, homozygous recessive white (bb) suppresses the expression of the homozygous yellow phenotypes (ii, ff, 
loci are required to determine the individual carotenoids and/or 
carotenoid intermediates present in the flowers of each of 
the 4 yellow loci.

Pairwise complementation crosses between the i, f, and s 
loci produced all orange progeny (Table 4), showing that 
they represent mutations in genes at different loci. The observed segregation was consistent with modified F2 
dihybrid segregation expected for complementary gene 
action for epistatic loci (9:7) with the mutant yellow form 
(IncY), f (Blf5), and the s (HurP) loci did not significantly differ from the 
expected 9:3:4 ratios in the F2 generations (Table 5).

These results indicate that these yellow phenotypes are 
due to 4 independently segregating gene pairs, all of which 
produce a yellow phenotype when homozygous recessive. 
The complementary epistatic effect of 2 dominant alleles in 
pairwise combinations of these loci produces the orange-
flowered phenotype. An interpretation of these results on 
a functional basis could be that the i, f, s, and x loci code for 
sequential enzymes in the carotenoid pathway. When the 
earlier locus is homozygous recessive, yellow carotenoids 
accumulate. However, if the earlier locus has a functional 
dominant allele, the pathway progresses to the next step, 
which, if homozygous recessive, also results in an accumulation 
of yellow carotenoids. When all enzymes are functional, 
that is, have a dominant allele present, the pathway progresses 
to where orange-colored carotenoids predominate. Alterna-
tively, these variants may contain the same carotenoids as the 
white type, but at a reduced concentration, thus affecting their 
color. In this case, the loci for these variants could be 
regulatory, and when homozygous recessive cause a down-
regulation of the carotenoid pathway in general.

The reduced total carotenoid content found for each of 
the 5 loci petals (Table 6) confirmed that the mutations 
causing the yellow flower phenotypes described in this paper 
are due to mutations in the carotenoid biosynthetic pathway 
in floral tissues. The much reduced carotenoid content 
observed for the b locus petals is in agreement with our 
previous observations (Wakelin et al. 2003). Further analyses 
are required to determine the individual carotenoids and/or 
carotenoid intermediates present in the flowers of each of 
the 4 yellow loci.

Several genes have been cloned and characterized from 
the carotenoid biosynthetic pathway in plants. Very little 
direct connection has been made between the genes and 
their phenotypic expression (Huh et al. 2001). The IncY, 
Blf3, Hirl, and SelIII yellow mutants are inherited as single 
loci (Tables 2 and 4), but functional assignment of the mutation 
will require definitive identification and quantitation 
of the carotenoids, as well as the cloning, sequencing, 
and molecular complementation and characterization anal-
ysis of the relevant genes (Huh et al. 2001). California poppy 
gains attention in recent times as a model basal eudicot 
species especially in evolution and developmental genetic 
studies (Becker et al. 2005; Zahn et al. 2006; Orashakova 
et al. 2009). As part of the floral genome sequencing 
consortium, (www.floralgenome.org) more genetic resour-
ces for California poppy are becoming available. These 
resources will aid in the isolation and sequencing of genes 
volved in carotenoid biosynthesis in California poppy.

Supplementary Material

Supplementary material can be found at http://www.jhered .oxfordjournals.org/.

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