Plants are unable to move and thus must respond appropriately to their environment. The sun is a key component of a plant’s environment, providing visible light for photosynthesis but also generating damaging ultraviolet (UV) rays and heat. In this study, we examine the genetic interactions between these two genes. In dark-grown seedlings, uvh6 exhibits a weak de-etiolated phenotype but does not affect the stronger de-etiolated phenotype of det1. In the light, det1 is epistatic to uvh6 with regard to chlorophyll level, but their effect on all size parameters is additive and therefore independent. With regard to UV tolerance, det1 UV resistance is epistatic to uvh6 UV sensitivity. In heat stress experiments, det1 enhances heat-induced tissue damage in the uvh6 background but suppresses heat-induced growth inhibition. Thus, det1 acts epistatically to uvh6 with respect to de- etiolation, chlorophyll level, UV tolerance, and heat-induced growth inhibition, whereas det1 and uvh6 act additively to regulate plant size and heat-induced cell death. These data provide insight into interplay between light and heat signaling.

**ABSTRACT**

Plants must adapt to a variety of abiotic inputs, including visible light, ultraviolet (UV) light, and heat. In Arabidopsis thaliana, DE-ETIOLATED 1 (DET1) plays a role in visible light signaling, UV tolerance, and development. UV-HYPERSENSITIVE 6 (UVH6) mutants are UV and heat sensitive, as well as dwarf and pale, like det1. In this study, we examine the genetic interactions between these two genes. In dark-grown seedlings, uvh6 exhibits a weak de-etiolated phenotype but does not affect the stronger de-etiolated phenotype of det1. In the light, det1 is epistatic to uvh6 with regard to chlorophyll level, but their effect on all size parameters is additive and therefore independent. With regard to UV tolerance, det1 UV resistance is epistatic to uvh6 UV sensitivity. In heat stress experiments, det1 enhances heat-induced tissue damage in the uvh6 background but suppresses heat-induced growth inhibition. Thus, det1 acts epistatically to uvh6 with respect to de-etiolation, chlorophyll level, UV tolerance, and heat-induced growth inhibition, whereas det1 and uvh6 act additively to regulate plant size and heat-induced cell death. These data provide insight into interplay between light and heat signaling.

**KEYWORDS**

Arabidopsis

DET1

UVH6

light

heat

**INVESTIGATION**
CULLIN 4 (CUL4) and RING-BOX 1 (RBX1) (Bernhardt et al. 2006; Chen et al. 2006). DET1 also interacts with histone 2B (H2B), suggesting it may be involved in regulating chromatin structure or transcription factor access (Benvenuto et al. 2002). Recently, DET1 was found to interact with the transcription factors CCA1 and LHY1 to act as a transcriptional repressor (Lau et al. 2011).

In addition to its role in visible light response, DET1 was recently found to be involved in UV tolerance. det1 mutants were found to be UV resistant as the result of increased levels of anthocyanin sunscreens, as well as increased expression of photolyase genes (Castells et al. 2010). UV light induces thymine dimers in DNA that interrupt transcription and DNA replication. These dimers can be removed via light repair, where photolyase enzymes use energy from visible light to directly cleave the dimer, or via dark or nucleotide excision repair (NER), where the lesions are recognized, unwound, removed and repaired in a multi-step process (Ganpudi and Schroeder 2011).

A key component of the NER pathway is the XERODERMA PIGMENTOSA D (XPD) helicase, which unwinds the region of UV-damaged DNA, facilitating its removal. In humans, mutation of XPD results in xeroderma pigmentosa, a UV-sensitive condition with increased skin cancer risk. The XPD helicase is a component of the TFIIH multi-protein complex and thus is also involved in transcription. Mutations in human XPD can also result in Cockayne syndrome or trichothiodystrophy, which include developmental and neurologic symptoms (Fuss and Tainer 2011).

A mutation in the Arabidopsis homolog of XPD was identified in a screen for UV-sensitive mutants as UV hypersensitive 6 (uvh6) (Jenkins et al. 1995; Liu et al. 2003). Like human xpd mutants, the uvh6-1 partial loss of function allele exhibits pleiotropic defects, including dwarf stature, decreased chlorophyll, and heat sensitivity (Jenkins et al. 1997). As the result of the overlapping phenotypes of uvh6 and det1, we generated the double mutants to examine the interactions between these two genes with respect to light signaling, UV tolerance, and heat response.

**MATERIALS AND METHODS**

**Plant material and growth conditions**

Throughout this study, the Arabidopsis (Arabidopsis thaliana) ecotype Col-0 was used as the wild-type plant. The det1-1 partial loss of function mutation was previously described (Chory et al. 1989), and the uvh6-1 mutant line (Jenkins et al. 1995) (TAIR no. CS6375) was obtained from the Arabidopsis Stock Centre (http://www.arabidopsis.org/).

Unless otherwise indicated, plants were grown at 20° and 50% relative humidity. Light was supplied by cool white fluorescent bulbs with a photoperiod of 16-hr light (100 μmol photons m⁻² s⁻¹). Adult plants were grown in Sunshine mix number 1 (SunGro, Bellevue, WA).

**Growth analysis**

Seedlings: Seeds were plated on Linsmaier and Skoog (LS) media (Caisson) [1× LS salts, 0.8% phytoblend (Caisson), 2% sucrose], stratified at 4° for 2 days, transferred to either long-day conditions (light) or 6 hr of light then wrapped in foil (dark). After 7 days, seedlings were scanned and hypocotyl length and apical hook angle measured for dark-grown seedlings, or hypocotyl length and cotyledon width measured for light-grown seedlings, using NIH Image. Anthocyanin and chlorophyll analysis were done as previously described (Schroeder et al. 2002; Fankhauser and Casal 2004) using three replicates per genotype of 20 seedlings each. For gravitropism analysis, seedlings were grown in the dark on vertical plates for 7 days, then scanned and the angle between the hypocotyl and the vertical measured using NIH Image.

**UV tolerance**

Seedlings were grown on vertical plates [1× LS salts, 0.8% phytoblend (Caisson), 0.6% sucrose] in long-day conditions for 3 days, then irradiated with 600 J m⁻² of UV-C using a Model XX-15S UV lamp (UV Products). Plates were rotated by 90°, grown in long-day for an additional 2 days, and then scanned. NIH image was used to measure new root growth beyond the bend and data expressed as relative to unirradiated controls.

**Heat tolerance**

Tolerance of adult plants to heat stress was based on assays used in Jenkins et al. (1997). In brief, seedlings were grown on plates for 2 weeks, transferred to soil, 1 week later transferred to 37° for 5–7 days, then returned to 20°. One week after the start of heat treatment, rosette diameters were measured and leaf damage scored as damaged leaves/total leaves for each plant. Tolerance of dark-grown seedlings to heat stress was determined by assays used in Larkindale et al. (2005). In brief, seedlings were grown in the dark on small plates with 35 mL of media [1× LS salts, 0.8% phytoblend (Caisson), 0.6% sucrose] per plate for 3 days; transferred to 45° for 0, 2, or 4 hr; then returned to 20°. After an additional 4 days of dark growth, plates were scanned and hypocotyl length measured using NIH Image.

**Heat-induced gene expression**

Fifty seeds per genotype per treatment were plated on plates with 35 mL of media as described previously, stratified at 4° for 2 days, grown at 20° in long day conditions for 14 days, placed in a 45° incubator for 3 hr, allowed to recover at 20° for 1 hr, and then samples were collected. RNA was extracted using a QIAGEN RNeasy Plant Mini kit according to manufacturer instructions, including a DNase step, and quantified using a Nano-drop spectrophotometer (Thermo Scientific). cDNA was synthesized from 1 μg of total RNA using a Maxima First Strand cDNA synthesis kit (Fermentas) and diluted 40-fold for analysis. Real-time polymerase chain reaction (PCR) was performed in a 96-well plate on a iCycler equipped with iQ5 detection system (Bio-Rad) using iQ SYBR Green Supermix (Bio-Rad) in 20 μL of reaction volume. The following primers were used: At5g12030 HSP17.6 (CCGCCCTGAAGAACACCGAAG, TCCCTCTGTCTTTT GCCACTC), At4g27670 HSP21 (CGCTTAACCATGGACGTCTCTC, TCCCTCTGTCTTTT GCCACTC), At4g27670 HSP21 (CGCTTAACCATGGACGTCTCTC, TCCCTCTGTCTTTT GCCACTC), and At5g60390 EF1α (CTGGAG GTTTTGGAGGCTTGAT, CCAAGGGTAGAAGCAGAAGAGA). For a single experiment (four genotypes ± heat treatment), samples were assayed in triplicate (technical) and values normalized relative to the reference gene EF1α (Jain et al. 2006; Hossain et al. 2012) then expressed as relative to the untreated wild-type control. The entire experiment was repeated three times.

**Statistical analysis**

Data were compared by Student’s t-test, and P values of 0.05 or less were considered to be statistically significant. All experiments were repeated at least three times.
RESULTS

Dark-grown seedlings

det1 mutants were originally identified via their light-grown phenotype when grown in the dark (Chory et al. 1989). To assess the genetic relationship between DET1 and UVH6 with respect to de-etiolation response, det1, uvh1, and the double det1 uvh6 mutant were grown in the dark and their phenotypes examined (Figure 1A). uvh6 single mutants exhibited a small-but-significant decrease in hypocotyl length as well as an increase in apical hook angle (Figure 1, B and C), suggesting a weak de-etiolated phenotype. det1 appears to be epistatic to uvh6, however, because the uvh6 det1 double mutant does not differ from det1 with regard to hypocotyl length (Figure 1B) or cotyledon opening angle (data not shown). In dark-grown seedlings, although uvh6 does not exhibit any difference in anthocyanin levels from the wild type, it enhances anthocyanin content in the det1 background (Figure 1D). The det1 single and uvh6 det1 double mutant exhibit curled hypocotyls in the dark (Figure 1A). This phenotype has previously been observed in cop/det/fus mutants (Hou et al. 1993) and indicates defects in gravitropism. Normally, in wild-type seedlings, light inhibits gravitropism (Fankhauser and Casal 2004); thus, another feature of the cop/det/fus phenotype is the constitutive inhibition of gravitropism in the dark. We quantified this phenotype by growing seedlings on vertical plates in the dark and measuring the angle by which hypocotyls deviated from the vertical (Figure 1E). In det1 mutants, hypocotyl orientation was basically random. uvh6 did not affect this phenotype in either the wild-type or det1 background. In contrast to the shoot gravitrophic response, root gravitropism in the dark was normal in all genotypes (data not shown). In summary, in dark-grown seedlings uvh6 single mutants exhibit slightly reduced hypocotyl length and increased apical hook opening but no change in anthocyanin content or shoot gravitropism. In the det1 background, uvh6 does not affect hypocotyl length or shoot gravitropism but slightly enhances anthocyanin content.

Light-grown seedlings

In light-grown seedlings, uvh6 had no detectable effect on hypocotyl length in either the wild-type or det1 background (data not shown). However, when cotyledon width was measured, uvh6 was found to result in decreased size in both the wild-type and det1 backgrounds (Figure 2, A and B), indicating this effect is independent of det1. Both uvh6 and det1 mutants have been reported to be pale in color with decreased levels of chlorophyll (Chory et al. 1989; Jenkins et al. 1997). In our assay, we could not detect a significant effect of uvh6 on chlorophyll level in either the wild-type or det1 background (Figure 2C). Interestingly, we did detect decreased levels of anthocyanin in the uvh6 single mutant (Figure 2D), perhaps contributing to its pale appearance. In contrast to dark-grown seedlings, anthocyanin levels did not differ between det1 and uvh6 det1 in light-grown seedlings.

Adults

Adult plants were grown and various growth parameters examined (Figure 3A). uvh6 did not affect flowering time as measured in either

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**Figure 1** Dark-grown seedlings. (A) From left: Col-0, uvh6, det1, uvh6 det1. (B) Hypocotyl length (n = 10). (C) Apical hook angle (n = 10). (D) Anthocyanin content (n = 3). (E) Angle of hypocotyl deviation from vertical on vertical plates (n = 20). Error bars indicate 95% confidence interval (95% CI), and *P ≤ 0.05 of single mutants relative to Col-0 or of double mutant relative to det1.
days or leaves in either the wild-type or det1 background (data not shown). With respect to size parameters, such as rosette diameter, height, and silique length, uvh6 resulted in decreased size in both the wild-type and det1 backgrounds (Figure 3, A2D), indicating that the dwarf phenotypes of uvh6 and det1 are independent and additive. uvh6 did not significantly affect apical dominance (Figure 3E).

**UV tolerance**

The UVH6/XPD helicase is a key component of the nucleotide excision repair pathway (Fuss and Tainer 2011). The uvh6-1 point mutant exhibits mild UV sensitivity (Jenkins et al. 1995) (Figure 4). det1 mutants have recently been reported to be UV resistant as the result of photolyase overexpression (Castells et al. 2010). As expected, the det1 UV-resistant phenotype is epistatic to the uvh6-sensitive phenotype (Figure 4) because in light conditions excess photolyase activity would compensate for defects in nucleotide excision (dark) repair.

**Heat tolerance**

uvh6 mutants show increased heat sensitivity (Jenkins et al. 1997). We examined heat tolerance in uvh6, det1, and the double mutant. In adult plants, 2 and 3 days of heat treatment killed the uvh6 single mutant but did not result in significant leaf damage in wild type (Figure 5, A and B). det1 mutants exhibited low levels of leaf damage even in control conditions; however, this was not significantly increased by heat treatment. In the uvh6 det1 double mutant, 1 day of heat treatment resulted in dead plants, indicating that det1 enhanced heat-induced tissue damage in uvh6. Another effect of heat is inhibition of growth. To quantify this effect, we measured rosette diameters in all genotypes and treatments and calculated relative rosette diameter (Figure 5C). Heat treatment resulted in a significant reduction in rosette diameter in uvh6 relative to the wild type. In det1, heat inhibition of growth was similar to that observed in wild type. In uvh6 det1, relative rosette diameter was intermediate between the two single mutants. It was not significantly different from det1 in any condition and was significantly greater than that of uvh6 after 2 and 3 days of heat treatment. Thus, in adult plants, det1 suppressed heat inhibition of growth in uvh6 while enhancing heat-induced tissue damage. To investigate heat tolerance at other stages of development, we examined the effect of heat on hypocotyl length in dark-grown seedlings (Figure 5D). Again, uvh6 exhibited increased growth inhibition relative to the wild type. det1 mutants exhibited slightly decreased inhibition relative to the wild type at intermediate treatment duration. As in the adult assay, the uvh6 det1 double mutants were not significantly different from det1 in any condition but exhibited significantly less inhibition than uvh6 in both heat treatments. Therefore, in both adults and dark-grown seedlings, det1 suppressed heat inhibition of growth in uvh6.

**Heat regulation of gene expression**

As a component of TFIH, XPD/UVH6 plays an important role in transcription. uvh6 mutants have been reported to exhibit aberrant levels of several RNAs and proteins (Jenkins et al. 1997; Liu et al. 2008;
With respect to its role in heat tolerance, *uvh6* was reported to contain increased levels of HSP21 (Jenkins et al. 1997) but normal levels of HSP101 and sHSPs (Larkindale et al. 2005). *det1* mutants also misexpress hundreds of genes. Interestingly, many heat shock protein genes are overexpressed in *det1* mutants in light conditions (supporting information, Table S1) (Maxwell 2001; Schroeder et al. 1990; Schroeder et al. 2002). We examined expression levels of several heat shock protein genes in light-grown seedlings with or without heat treatment using real-time reverse-transcription PCR. HSP21 protein was previously found to be present at increased levels in *uvh6* mutants (Jenkins et al. 1997). An increase in At4g27670 HSP21 transcript levels was detected in untreated *uvh6* seedlings (Figure 6A). In contrast, HSP21 levels were lower in untreated *det1* and *uvh6 det1* than in the wild type. After heat treatment, however, HSP21 levels were greater in *det1*. *uvh6* did not appear to affect HSP21 levels after heat treatment in either the wild type or *det1* background. At5g12030 HSP17.6 encodes a class 1 small HSP, which had previously been shown to be unchanged in *uvh6* mutants (Larkindale et al. 2005). We observe a decrease in induced HSP17.6 levels in both *uvh6* relative to wild type and in the double mutant relative to *det1* (Figure 6B).

**DISCUSSION**

In this study we examined the genetic interactions between the pleiotropic *det1* and *uvh6* mutations. In dark-grown seedlings, *uvh6* exhibited a mild de-etiolated phenotype, consisting of a slight decrease in hypocotyl length and an increase in apical hook angle. *det1* appears to be epistatic to this phenotype because the *uvh6 det1* double mutants do not differ from *det1* with respect to hypocotyl length or agravitropism. The double mutants do, however, exhibit an increase in anthocyanin in the dark relative to *det1*, indicating a mild enhancement of this phenotype. In contrast, in light-grown seedlings, *uvh6* single mutants exhibit decreased anthocyanin levels relative to the wild type, perhaps contributing to their pale appearance. *det1* is epistatic to this phenotype because *uvh6* does not affect *det1* anthocyanin levels in the light. The basis of this differential effect of light on *uvh6* anthocyanin regulation is unknown.

*det1* is best known for overexpressing light-regulated genes in the dark (Chory et al. 1989), but in the light it actually underexpresses light-regulated genes such as *CAB1*, *CAB2*, and *LHCB2.4* (Chory and Peto 1990; Schroeder et al. 2002). *DET1* regulation of the *CAB2* promoter in the light requires a HY5-binding element (Maxwell et al. 2003), and *hy5* mutants suppress the *det1* pale phenotype (Chory 1992). *uvh6* has been described as yellow-green with decreased chlorophyll level and poorly organized thylakoid membranes (Jenkins et al. 2009). With respect to its role in heat tolerance, *uvh6* was reported to contain increased levels of HSP21 (Jenkins et al. 1997) but normal levels of HSP101 and sHSPs (Larkindale et al. 2005).
Figure 5 Heat tolerance. (A) Adult plants after 0–3 days of heat treatment. (B) Fraction of damaged leaves after 0–3 days of heat treatment (n = 6). (C) Relative rosette diameter after 0–3 days of heat treatment (n = 6). (D) Relative hypocotyl length after 0–4 hr of heat treatment. For C and D, error bars indicate SE, and *P ≤ 0.05 of single mutants relative to Col-0 or of double mutant relative to det1 in the same conditions.

For all parameters that describe plant size in light conditions, such as light-grown seedling cotyledon width, as well as adult rosette diameter, height, and silique length, uvh6 and det1 act additively. Although the basis of the det1 dwarf phenotype is not entirely clear, it is partially suppressed by the ted mutants, including hy5 (ted5) and the peroxisomal protein gene ted3, suggesting that transcription and peroxisome function play a role (Chory 1992; Pepper and Chory 1997; Liu et al. 1999), as well as increased levels of genes associated with light stress such as photolyases (Hu et al. 2002; Castells et al. 2010). Light stress can induce cell death in plants, and the blue light receptor CRY1 is required for this response (Danon et al. 2006). det1 exhibits constitutive light signaling in a number of pathways, including CRY1. Combined heat and light treatments result in reduced plant survival (Larkindale and Knight 2002; Larkindale et al. 2005). Stress response with uvh6 heat sensitivity. In contrast to the enhanced heat-induced tissue damage in the uvh6 det1 double mutants, det1 suppresses heat-induced growth inhibition in the uvh6 background. These differential effects may be attributable to the nature of the det1 phenotype. det1 mutants are small, stressed plants. When combined with the heat sensitivity of the uvh6 mutants, the double mutants are hypersensitive to heat stress at the cellular level but do not exhibit additional heat-induced growth inhibition. These data suggest that basis for being small in det1 is epistatic to heat-induced inhibition of growth in uvh6.

With respect to heat regulation of gene expression, for HSP21 we detect increased levels in uvh6 mutants, consistent with previous studies showing increased HSP21 proteins levels (Jenkins et al. 1997). For HSP17.6, however, we detect reduced levels in uvh6 mutants, in contrast to the unchanged amounts of class 1 sHSP protein previously described (Larkindale et al. 2005). This difference could be the result of differential regulation of RNA vs. protein or differences in developmental stage or heat treatment. Although in some studies authors indicate enhanced response to heat treatment by uvh6 (e.g., Jenkins et al. 1997)), others observe reduced effects. For example, Liu et al. (2008) show that heat treatment reduced levels of AtKu70 and AtKu80 transcript in wild type, but this down-regulation did not occur in uvh6. In response to another stress, cold, Hall et al. (2009) found that uvh6 mutants failed to induce some cold stress genes but not all. Thus, the uvh6-1 mutant appears to exhibit abnormal regulation of a subset of genes rather than global defects in transcription (Hall et al. 2009). In det1 mutants, we also detect abnormal levels of HSP transcripts, consistent with previous studies implicating DET1 in regulation of gene expression (Benvenuto et al. 2002; Schroeder et al. 2002; Ma 1997; Liu et al. 2003). In seedlings, however, we did not detect a significant effect of uvh6 on chlorophyll levels in either the wild-type or det1 background.
the reference gene
real-time reverse-transcription PCR. Values were normalized relative to
same conditions.

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suggesting that the enhanced heat induced tissue damage in the dou-

of the genes examined exhibit enhanced levels in the double mutant,

direct regulation of transcription is still unclear. Nonetheless, neither
variation in transcription factor abundance, chromatin structure, or

In summary, we

Figure 6 Heat-induced gene expression. mRNA levels of HSP21 (At4g27670) (A) and HSP17.6 (At5g12030) (B) in the absence and
presence of heat treatment (3 hr 45° + 1 hr 20°) as determined by
real-time reverse-transcription PCR. Values were normalized relative to
the reference gene EF-1α then expressed as relative to the untreated
wild-type control. Error bars indicate SE (n = 3) and *P ≤ 0.05 of single
mutants relative to Col-0 or of double mutant relative to det1 in the
same conditions.

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types have been shown to require HY5 activity (Chory 1992; Pepper
and Chory 1997; Maxwell et al. 2003; Castells et al. 2010). Perhaps
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