

Figure S1. Comparison of previously reported dark- or heat- responsive genes with the dataset in this study.

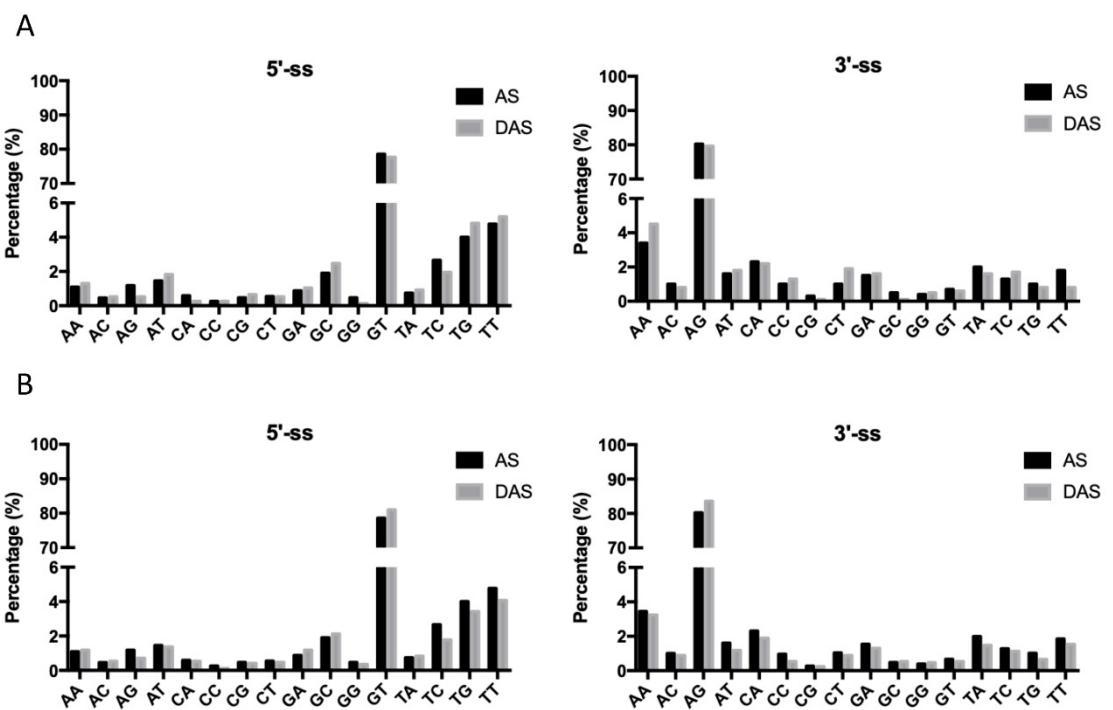


Figure S2. Analysis of the splice sites (ss) between total AS events and DAS events regulated by dark (A) and heat (B) treatments.



Figure S3. GO analysis of dark- or heat- regulated DEGs and DAS genes. Biological processes were sorted by fold enrichment and top ten pathways are shown.

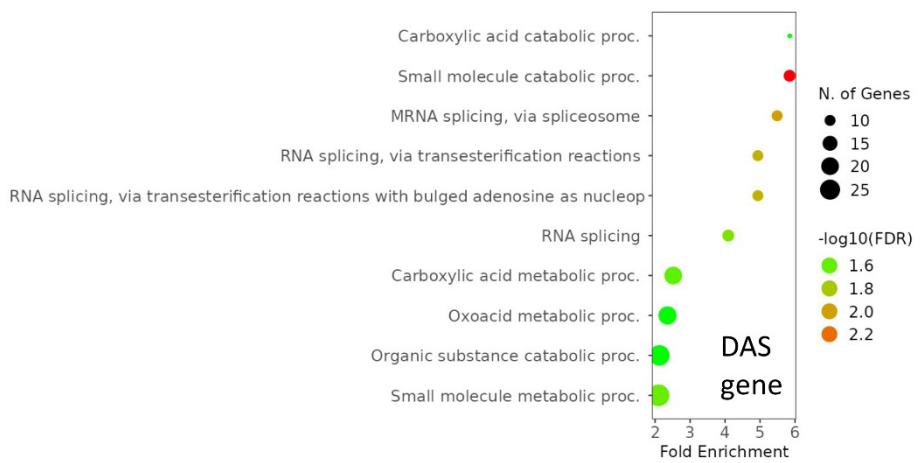
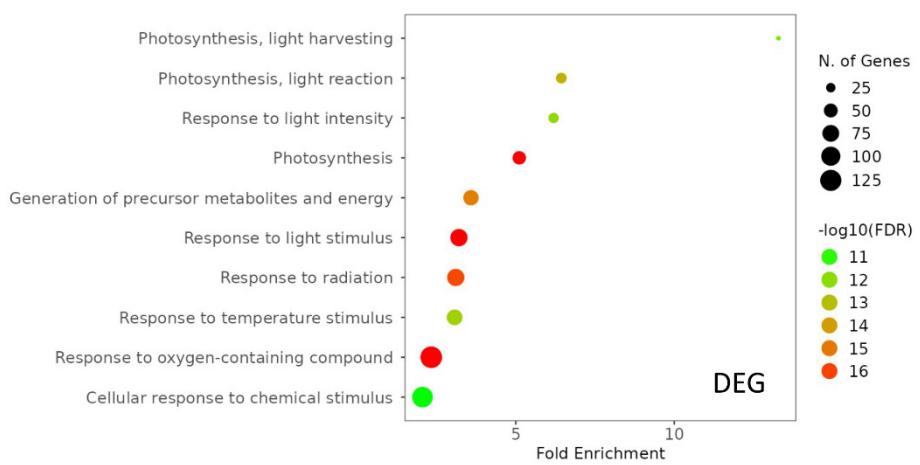


Figure S4. GO analysis of DEGs and DAS genes that were co-regulated by dark and heat treatments. Biological processes were sorted by fold enrichment and top ten pathways are shown.

AS5

ACACTGTGCTGTTCATCGTACGATGACGTACGTGAGAGACTCCACGAGCAGTAGTCAGACGACGAGCCCACCGCACCGCTCCCGTG
GTGCGATCATCGGGTGCAGATGTTAGATGAAGCGGCATGAGGCCACCAATATGTTGACTCATCATGGCACGCAGCTTCAATTATGC
TGCCTCGTATGTTGAAATTGGAGCGGATACCATTACACAGGAAACATCTATGACCATGATTACGCCAAGCTCAGATTACCCAC
TAAAGGTACTAGTCTGCAGGTTAACGAATTGCCCTCACCATGAGTAGCCGATGGAATCGTACGATCTACGTGGGATTGCTGG
AGATATTGCAAGTGTGAGGGTGAAGATCTCTACAAGTATGGACCAATTGAGCTTGAAGATTCCACCGAGACCTCTGG
TTATGCCCTTGTGAGTTGAAGATCCTCGTGCAGACGATGCAATTATGGACGTGATGGTTATGTTGATGGGTGACTTCG
GGTTAGTAAACGATGAAAGCTAGCTTAATTCTGTAATTCTGAAAGGTGTTATCTTGTGATGTTTAAAGTTGAGATTG
ACATGGTGGTGTAGATTTACCATCAGTGTAGGTACAGCAGCAGTACAGTGCAGGCCGTGACCTTCAAGACGCTCTGACTACC
GCGTGTGACCGGATTACGCCCTGCTCGAGGACCTAAGGATCACATGCGCAAAGCTGGAGATGTCGTTCTGAA
GTTTCCCTGACCGTAAAGGCATGTCGGGTTGGATTATAGCAACTATGATGATATGAAGTACGCAATAAGGAAACTTGATGCCAC
TGAATTGAAATGCTTCTAGTGTCTTATACGGGTGAGGGATATGAGTCGAGGAGTGTGAGTCAAGGCCAGATGATTCTAAA
GCTATAGAAGCAGGAGTCGGAGCCGGTCAAGCTGAGCTAGCTAGTAGCAAGAGCAGGAGTGTGACCTGCTAGATCCATTCCCC
GCGTTACGGCCCTTAGTCGTTCTCGCTCGTACAGCTGCTCAAGGTCCTAACAGATCAAAATCAAGATCAAGATCAAGATCAAGATC
GAATTCTCAGTTCACCTGTGATATGGTGAAGGGCGAATTGCGCCGCTAACATGCCCTATAGAATGCC

AS6

GGGCATCTATAGGGGATTGATTAGCGGCCGGAATTGCCCTCACCATGAGTAGCCGATGGAATCGTACGATCTACGTTGGGAAATT
TGCCTGGAGATATTGCAAGTGTGAGGGTGAAGATCTTCTACAAGGTTGAAATTCTCTCGATAAAAATTGAATTCTTCTCG
TGACTTGTGGTTCTAAATTGCAATTCTGCTTGAGACAATTAAATGACTCTTATGTTGTTGAGTGGACCAATTG
TGGACATTGATTGAAGATCCACCGAGACCTCTGGTTATGCCTTGTGAGTTGAAGATCCTCGTGTGAGACGATGCAATTATG
GACGTGATGGTTGATTTGATGGGTGTCGACTCCTGGGTTGAGATTGACATGGTGGTGTAGATTCACCATCAGTTGATAGGTAC
AGCAGCAGCTACAGTGCAGCCGTGACCTTCAAGACGCTCTGACTACCGCTGCTGTGACCGGATTACCGCTTCTGCTCG
GGACCTTAAGGATCACATCCGAAAGCTGGAGATGTCGTTCTGAAAGTTCCCTGACCGTAAAGGCATGTCGGGTTGTGGATT
ATAGCAACTATGATGATATGAAGTACGCAATAAGGAAACTGATGCCACTGAATTGCAAATGCTTCTAGTGTCTTATACGGGTGA
GGGAATATGAGTCGAGGGAGTGTGAGTCGAAGGCCAGATGATTCTAAAAGCTATAGAAGCAGGAGTGTGAGCCGTGGTCAAGCTGTA
GCTATAGTAGCAAGAGCAGGAGTGTGTCACCTGCTAGATCATTCCCCCGCGTTACGGCCCTTAGTCGTTCTCGCTCGTACAGCT
CTGTCAGGTCCTAACAGATCAAAATCAAGATCAAGATCAAGATGAAATTCCAGTTCACCTGTGATATCTGGTTGAAAGGGCG
AATTGTTAACCTGCAGGACTAGTACCTTATTGAGGGTTAATTGAGCTGGCGTATCATGGCATAGCTGGCTTCTGTGAAATTG
ATCCGCTCACATTCCCCAATACGGCCGGAGAAAAAGTAAGCTGGGGCTATGAGGGCTACCATATGGTGGCTCTGCCCTTCA
CTCGAACCTACTGCCCGCTAACATGCACCCCGCAGACGAGTGCATTGGGTC

Figure S5. Sequences of SR30 isoforms identified by Sanger sequencing. Shaded sequences indicate those correspond to the sequences of SR30. Underlined sequences indicate the retained intron sequence.