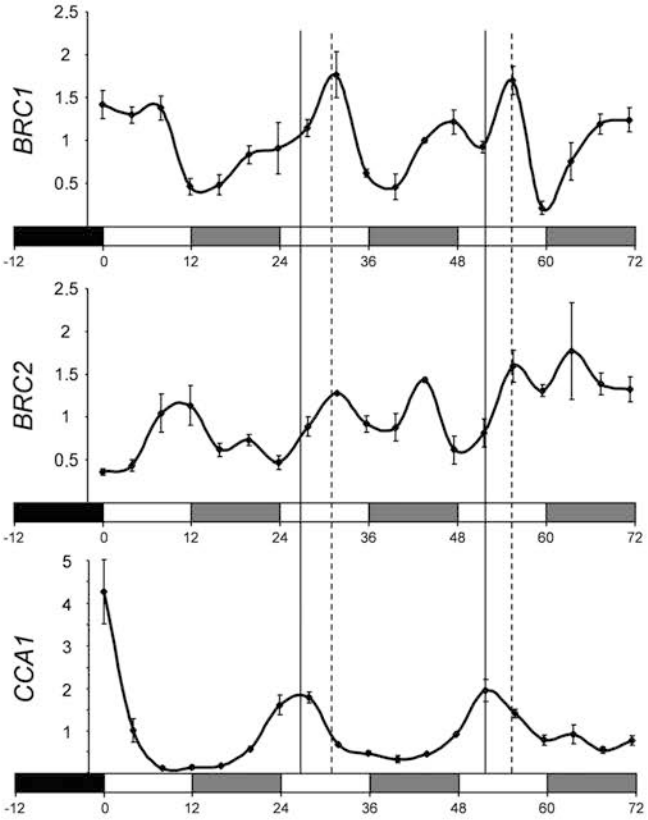
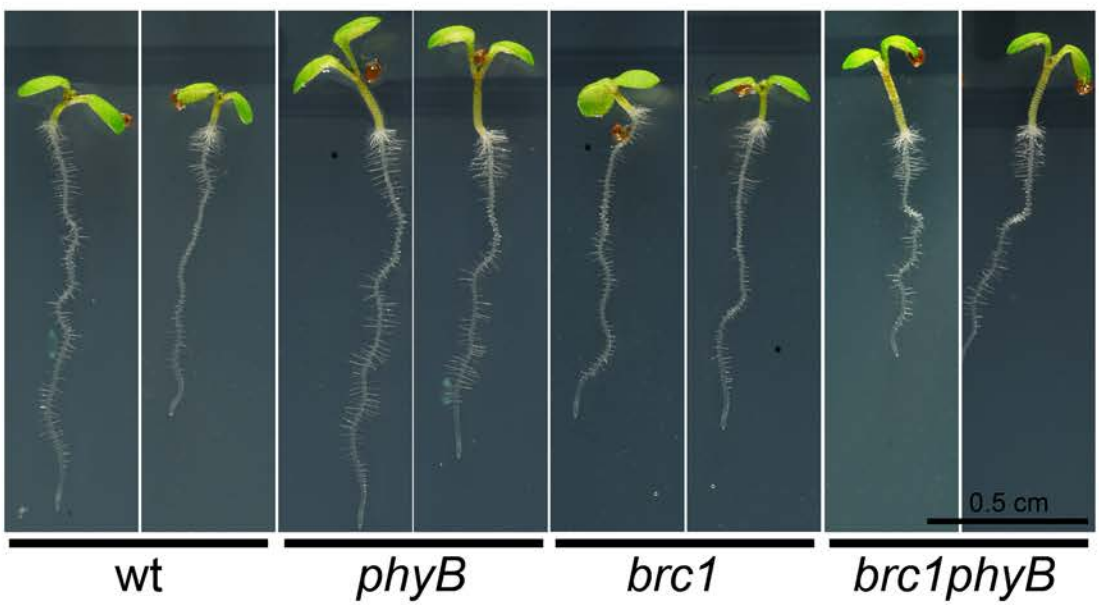
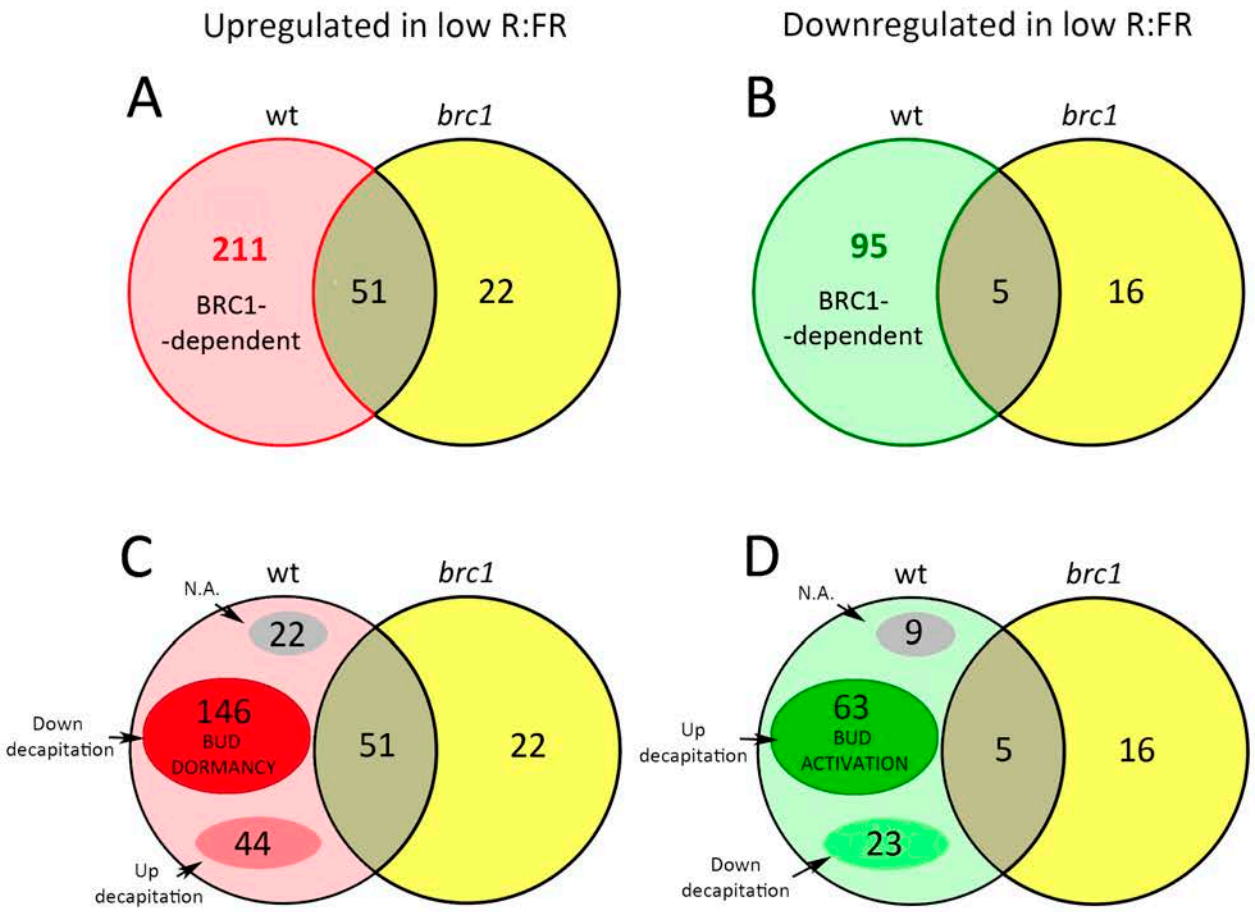


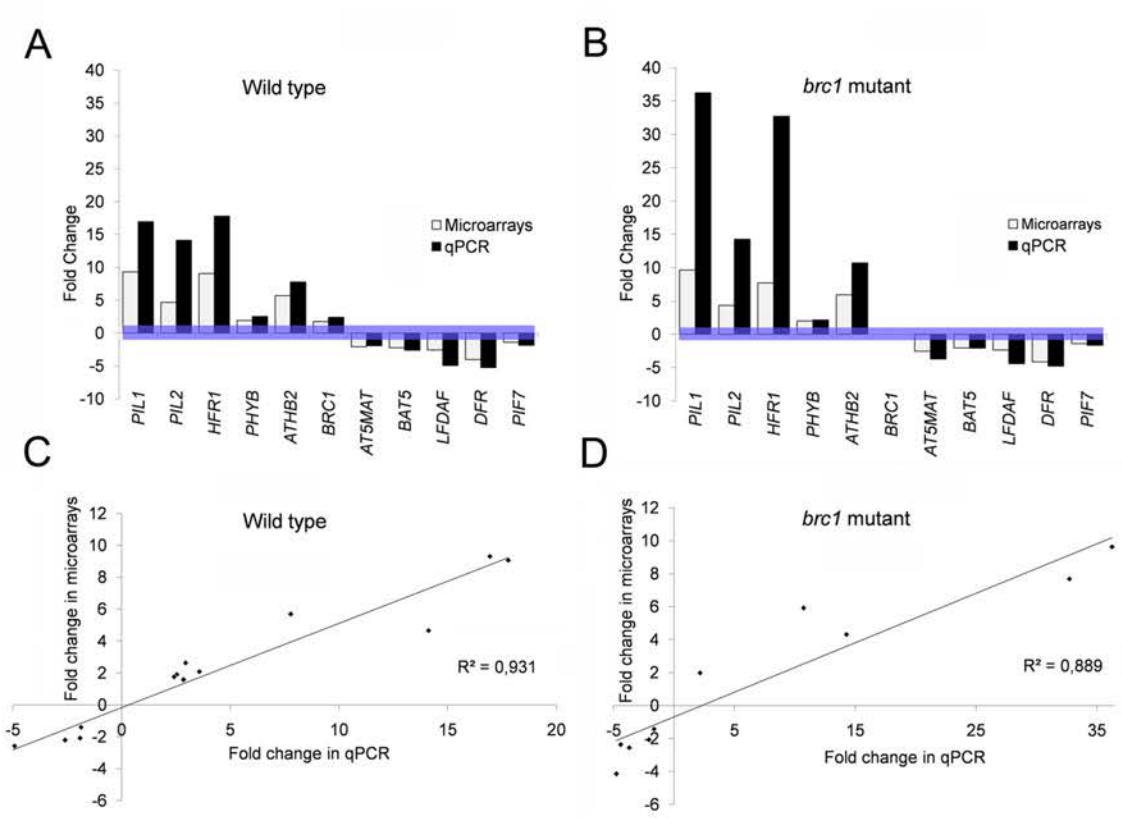
Supplemental Figure 1. Circadian expression of *BRC1* and *BRC2*. A, Expression of *BRC1*, *BRC2* and *CCA1* in continuous light quantified by qPCR. The time windows of highest expression of *CCA1* (black line) and *BRC1* (dotted line) are indicated. *BRC1* peak expression occurs 4 h after *CCA1* peak expression. Error bars represent SEM. Three biological samples were analyzed for each time point. The last night period before the experiment is indicated in black. White and grey areas correspond to 12 h periods during which the continuous light treatment was applied.



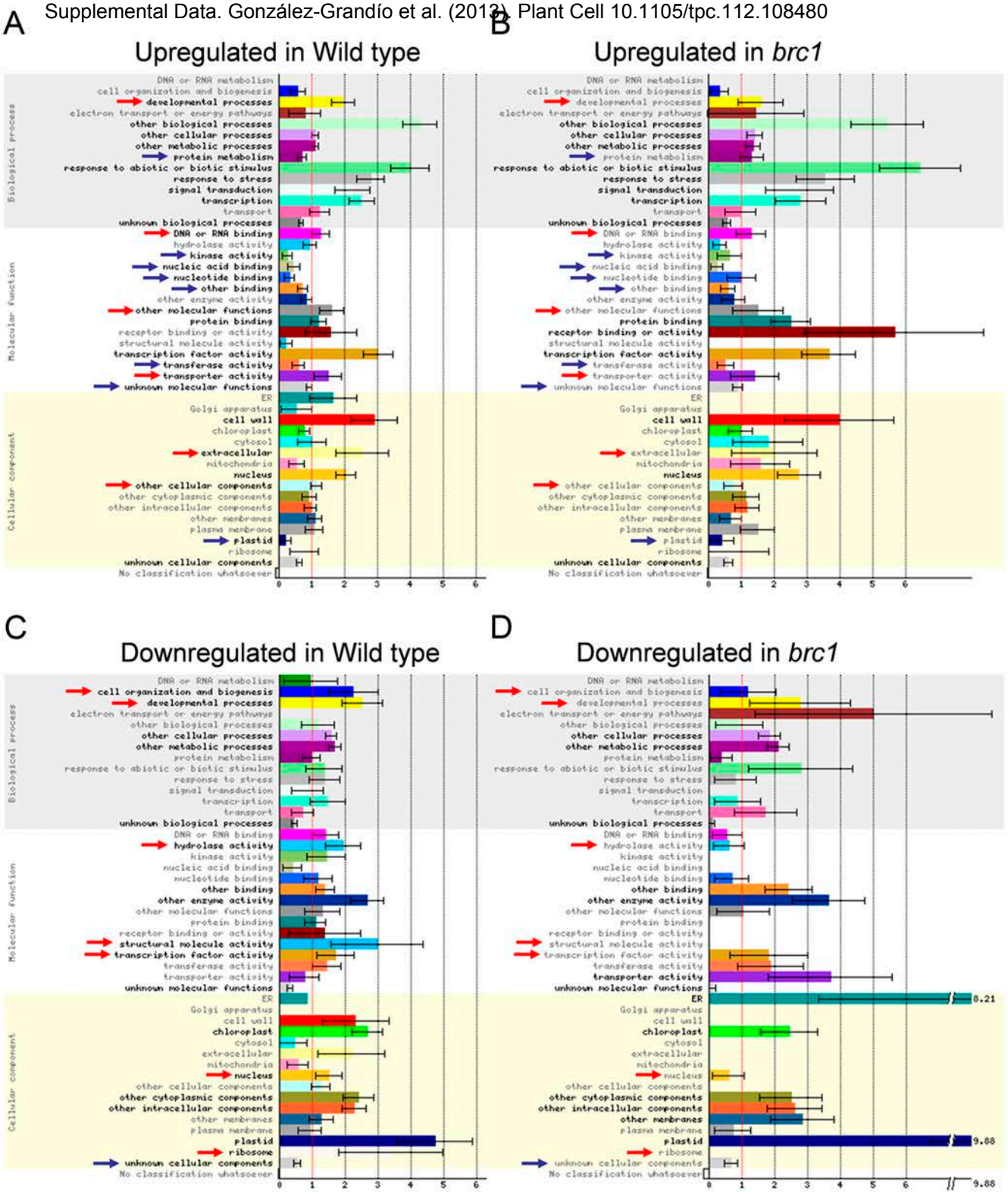
Supplemental Figure 2. *brc1* mutations do not suppress the long hypocotyl phenotype of *phyB* mutants. Five-day-old plants grown in continuous W, showing differences in hypocotyl length according to their genotype.



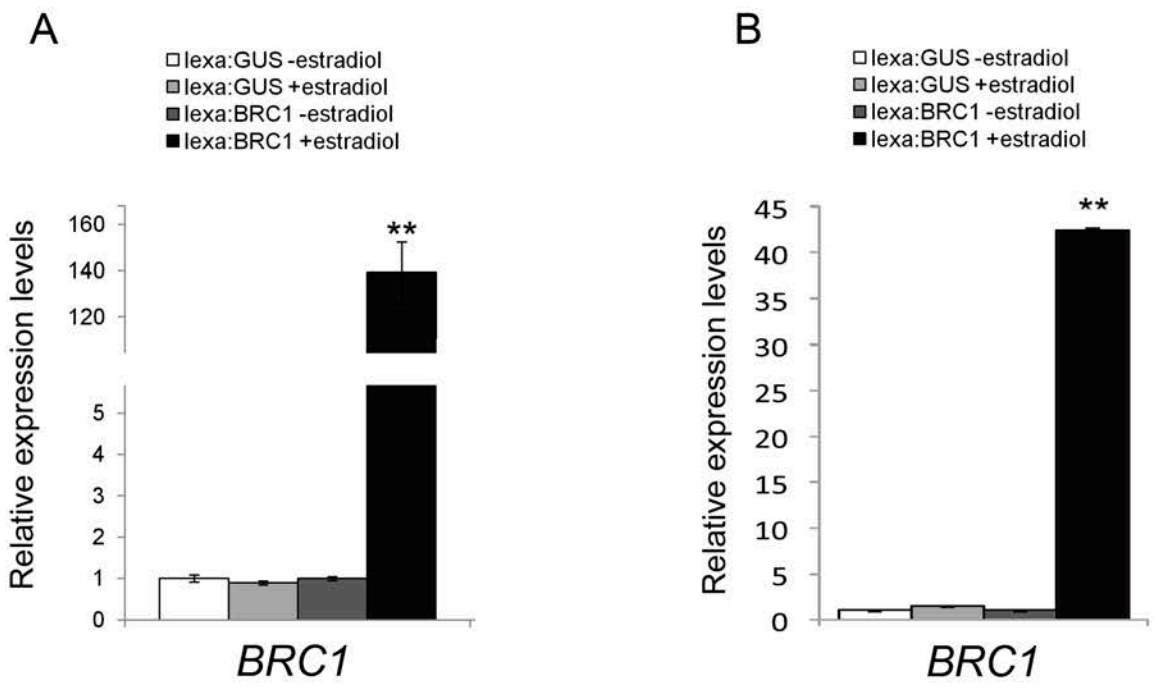
Supplemental Figure 3. Global responses in microarray hybridization experiment. Venn diagram (VENNY; Oliveros, 2007) showing the distribution of genes whose expression changes significantly (FDR<0.05) in our experiment in wild-type (A) and *brc1* mutant (B) plants. *BRC1*-dependent genes are genes significantly changing in wild type but not in *brc1* mutants. C and D, diagrams illustrating the distribution of Bud dormancy and Bud activation genes respectively, based on comparison with data from Tatematsu et al. (2005). N.A. Genes not analyzed due to lack of data in the Affymetrix arrays.



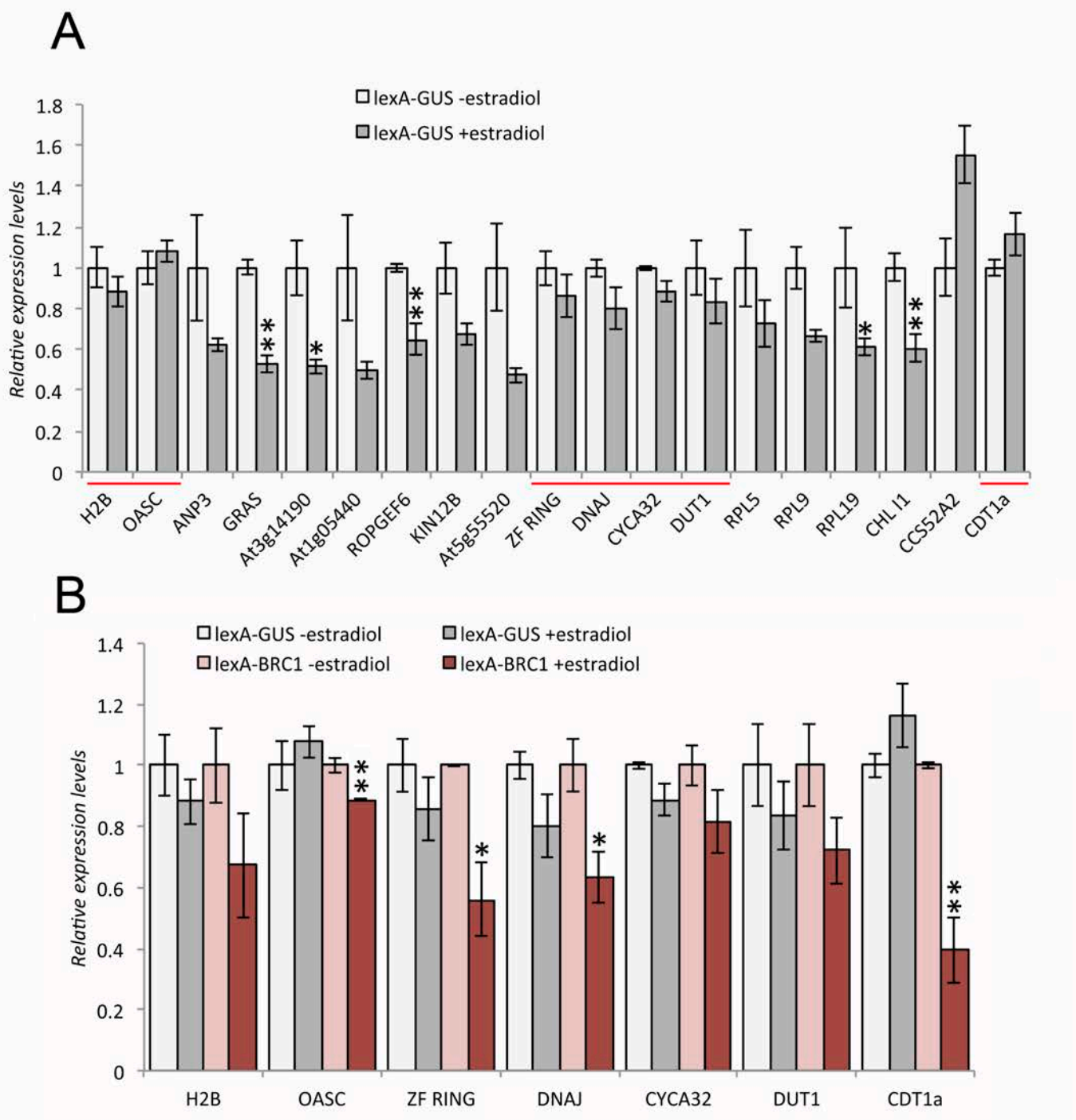
Supplemental Figure 4. Validation of microarray data by QPCR. A and B, mRNA levels of a subset of genes identified as responding to the W+FR treatment both in wild-type and *brc1* samples. C and D, Pearson's correlations between gene expression levels determined by q-PCR and microarray expression profiling for the same genes. Although the correlations of both datasets are high, microarray data underestimate the degree of change.



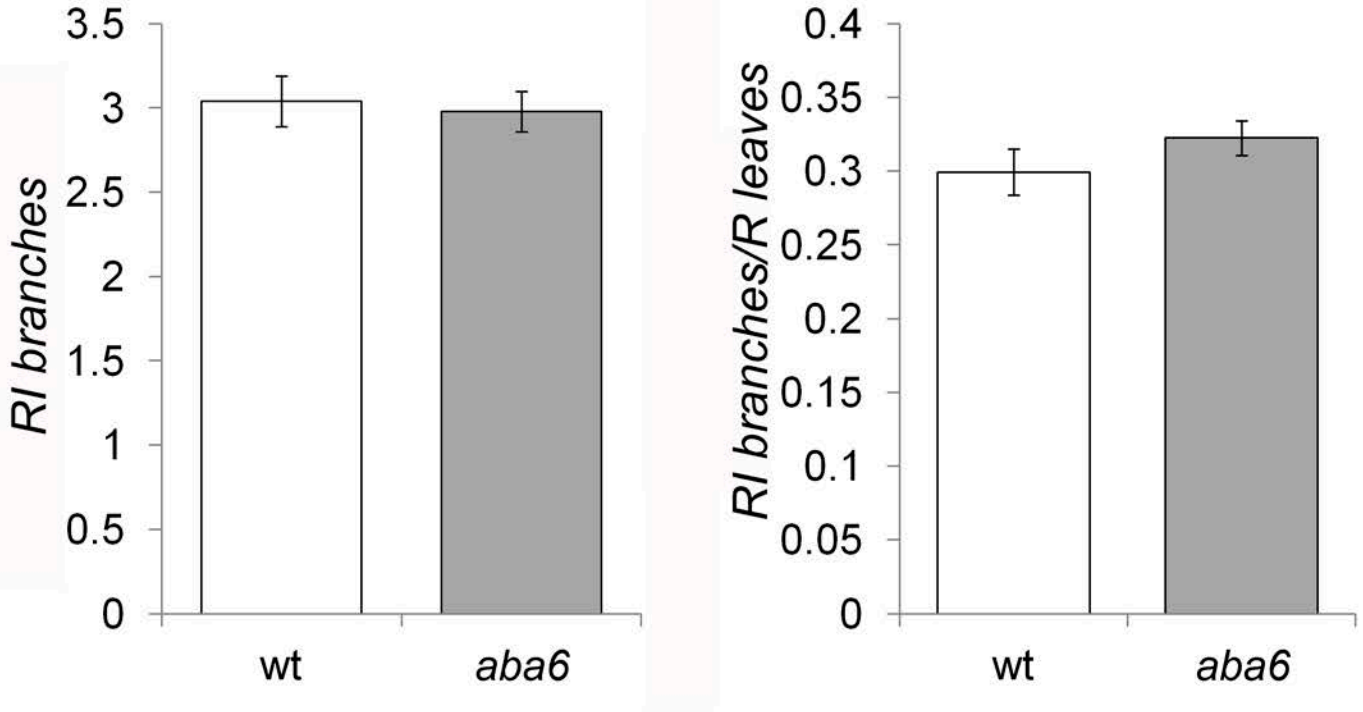
Supplemental Figure 5. Functional classification of genes changing in wild type and *brc1* after a W+FR treatment. Normalized gene category overrepresentation in our datasets calculated with the Classification SuperViewer Tool (bar.utoronto.ca). Categories in bold correspond to those over or underrepresented in the dataset (to the right or the left of the red dotted line, respectively) relative to the total number of genes of this category in the whole Arabidopsis genome. Red arrows indicate categories overrepresented in wild type but not in *brc1* mutants and blue arrows, categories underrepresented in wild type but not in *brc1* mutants. Normalized class scores and standard deviation for the scores were calculated by the Classification SuperViewer Tool as described in http://bbc.botany.utoronto.ca/ntools/cgi-bin/ntools_classification_superviewer.cgi.



Supplemental Figure 6. *BRC1* mRNA levels in estradiol-inducible lines 7 h after estradiol induction, quantified by qPCR. A, Seven-day old seedlings (10 μM estradiol). B, axillary buds (20 μM estradiol). Error bars are SEM. Three biological samples were analyzed for each treatment and genotype. Asterisks are significant mRNA level differences between treatments and genotypes (T-student, **=P<0.05).



Supplemental Figure 7. *BRC1* negatively regulates bud activation genes and cell cycle genes in axillary buds. A, mRNA levels, quantified by qPCR, of bud activation genes in axillary buds of plants carrying an estradiol inducible control construct lexa:GUS, 7 h after beginning of treatment (mock or 20 µM estradiol). Red underlining indicates genes that do not respond to the estradiol treatment in the control lines and which were tested in the *BRC1* inducible lines. B, mRNA levels, quantified by qPCR, of bud activation genes in axillary buds of plants carrying lexa:BRC1, 7 h after beginning of treatment (mock or 20 µM estradiol). Error bars are SEM. Three biological samples were analyzed for each treatment and genotype. Asterisks are significant differences between mock and estradiol treated plants (T-student, *=P<0.1; **=P<0.05).



Supplemental Figure 8. Branching phenotype of sextuple ABA receptor mutants.
Sextuple mutants impaired in six PYR/PYL receptors (*aba6*), namely PYR1, PYL1, PYL2, PYL4, PYL5, and PYL8 have a wild-type branching phenotype. Error bars are SEM. Student´s *t*-test were performed and no significant differences were found.
N_{wt}= 52, N_{aba6} =50.

Supplemental Methods

Statistical analysis of response in gene lists

To assess whether specific hormone pathways or gene categories were significantly activated or repressed after the W+FR treatment, the response of all the Arabidopsis genes represented in the arrays belonging to this category was studied. Gene lists containing hormone-specific markers or genes responding to SL (Nemhauser et al., 2006, Mashiguchi et al. 2009) and lists elaborated by (Lopez-Juez et al., 2008) complemented manually were used (Supplemental Table 2). The global proportion of genes upregulated and downregulated in the arrays was analyzed, considering upregulation a fold change of >1.2 and downregulation a fold change of <-1.2. This proportion was compared to the proportion of genes up- or downregulated within our gene lists. If both proportions were significantly different, this indicated that the gene category was differentially up- or downregulated with respect to the overall behavior of the genes in the array. The genes responding to hormone pathways were analyzed by studying whether, in our experiment, the hormone marker genes followed the behavior expected in response of its corresponding hormone (up- or downregulation) and whether the proportion of genes responding as expected was significantly different from the proportion of genes changing accordingly in the array. These analyses were performed by a Wilson approximation to the hypothesis test of equality of two proportions defining binomial distributions (Wilson, 1927) (R function: prop.test). To analyze whether the overall hormone response was significantly different in the wild type (WT) and *brc1* mutant (MUT) considering at the same time the genes that followed the expected behavior and those that were against expectations, the following analysis was performed. We called p^{+}_{WT} and p^{+}_{MUT} =proportion of genes up-regulated in wild type and *brc1* respectively; p^{-}_{WT} and p^{-}_{MUT} = proportion of genes down-regulated in wild type and *brc1* respectively; N_H =number of genes related to each hormone and analyzed in the microarray; n^{+}_{WT} and n^{-}_{WT} = number of genes (within N_H) that behave as expected or against expectations, respectively. n^{+}_{WT} was treated as a random variable and its probability density function (PDF) was computed as the Binomial distribution of parameters $\left(N_H, \frac{n^{+}_{WT}}{N_H}\right)$.

Similarly, the number of genes behaving as expected, $n_{WT}^{+,\exp}$, has a PDF given by the Binomial distribution of parameters $\left(N_H, p_{WT}^{+} + p_{WT}^{-}\right)$. Using these two PDFs, the PDF of the random variable $d_{WT}^{+} = n_{WT}^{+} - n_{WT}^{+,\exp}$ was numerically computed. This procedure was repeated for the genes changing against expectations and the PDF of the random variable $d_{WT}^{-} = n_{WT}^{-,\exp} - n_{WT}^{-}$ was computed. Finally, these two differences were combined in a single variable $d_{WT} = d_{WT}^{+} + d_{WT}^{-}$. The whole procedure was performed in wild-type and *brc1* data and the random variable $d = d_{WT} - d_{MUT}$ that compared the differences between the wild type and *brc1* was constructed. Using the PDF of d, the hypothesis that there was a difference between d_{wt} and d_{MUT} was tested. For that, the p-values of this inference test were calculated. The analysis for the different gene categories (Figure 4B) and the genes coregulated with cluster I and II (Supplemental Figure 5 online) was carried out as above except that n_{WT}^{+} and n_{WT}^{-} =number of upregulated and downregulated genes related to the pathway, respectively. The expected number of up and downregulated genes followed a Binomial distribution with parameters $\left(N_H, p_{WT}^{+}\right)$ and $\left(N_H, p_{WT}^{-}\right)$, respectively.