# CLAVATA signaling ensures reproductive development in plants across thermal environments

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## SUMMARY

The ability to thrive in diverse environments requires that species maintain development and reproduction despite dynamic conditions. Many developmental processes are stabilized through robust signaling pathways which cooperatively ensure proper development [1]. During reproduction, plants like *Arabidopsis thaliana* continuously generate flowers on growing indeterminate inflorescences [2]. Flower primordia initiation and outgrowth depends on the hormone auxin and is robust across diverse environments [3-6]. Here we show that reproductive development under different thermal conditions requires the integration of multiple pathways regulating auxin dependent flower production. In colder/ambient temperatures, the receptor complex CLAVATA2/CORYNE (CLV2/CRN) is necessary for continuous flower outgrowth during inflorescence development. CLV2/CRN signaling is independent of CLAVATA1 (CLV1)-related receptor signaling but involves the CLAVATA3 INSENSTIVE RECEPTOR KINASE (CIK) family co-receptors, with higher order *cik* mutant combinations phenocopying *clv2/crn* flower outgrowth defects. Developing *crn* inflorescences display reduced auxin signaling and restoration of auxin biosynthesis is sufficient to restore flower outgrowth in colder/ambient temperatures. In contrast, at higher temperatures both *clv2/crn* signaling and heat induced auxin biosynthesis via *YUCCA* family genes are synergistically required to maintain flower development. Our work reveals a

novel mechanism integrating peptide hormone and auxin signaling in the regulation of flower development across diverse thermal environments.

## RESULTS AND DISCUSSION

Plants continually develop new organs throughout their life and do so across varied environmental conditions [7]. This indeterminate growth requires balanced cell proliferation and differentiation in stem cell niches, called meristems, at growing apices [8, 9]. During the reproductive phase of *Arabidopsis thaliana*, flower primordia are continuously produced from inflorescence meristems (IM) dependent on the hormone auxin [3-6]. Primordia then proliferate, forming flowers from secondary floral meristems [10, 11]. Cell recruitment into flower primordia is balanced by proliferation in the IM center. The conserved CLAVATA3 (CLV3) peptide signaling pathway dampens stem cell proliferation in shoot and floral meristems [12, 13]. CLV3 signals through a suite of receptors which repress the expression of *WUSCHEL* (*WUS*) in the center of the IM [8, 14]. *WUS* encodes a homeobox transcription factor that positively regulates stem cell proliferation [15]. Among these receptors is the atypical receptor pair CLAVATA2/CORYNE (CLV2/CRN), a leucine rich repeat (LRR) receptor-like protein and a transmembrane pseudokinase, respectively [16-19]. CLV2/CRN negatively regulate IM stem cell proliferation independent of other CLV3 receptors [17, 20]. Here we define a new role for CLV2/CRN in promoting auxin-dependent flower primordia outgrowth and show that signaling through this receptor complex contributes to an environmental buffering mechanism which ensures reproductive developmental stability.

### The CLV2/CRN receptor complex promotes flower primordia outgrowth and development

After the production of 1-5 normal flowers in *crn* null mutants (*crn-10*; in the Col-0 ecotype), we noticed a novel phenotype in which flower primordia initiate but fail to develop further and inflorescence internode elongation stalls (Figures 1A-1B; the termination phase). After ~30-40 of these terminated primordia, flower development and inflorescence elongation resumes (recovery

phase), indicating that continuous flower production requires *CRN* (Figures 1B and S1A; Movie S1). *clv2* null mutants (*rlp10-1*; in Col-0) displayed a similar phenotype to *crn* (Figure 1C), which was previously observed in *clv2* in a survey of mutants in receptor-like protein genes, but not characterized [21]. To quantify primordia termination, we classified the first 30 attempts to make flowers along the primary inflorescence as normal (complete flowers; formation of all four flower organs [22]), terminated flower primordia (no flower organs develop), or terminated flowers (some flower organs develop, but no gynoecium). *crn* and *clv2* single mutants displayed equivalent defects in flower production (Figure 1E), also observed in *clv2 crn* double mutants, consistent with the documented co-function of CLV2/CRN (Figures 1B-E). *clv2/crn* flower outgrowth defects, and floral meristem size (measured in carpels made per flower), were complemented by expressing fusion proteins from their native promoters (Figure S1B-S1C and S1I-S1J). Using standardized flower primordia staging, we found that *crn* primordia outgrowth deviated from WT (wild type) at flower primordia stage three (FP3), with little proliferation occurring afterwards (Figures 1F-1G; staging as in [23], or stage 2 using Smyth *et. al.* stages [22]). In *crn,* terminated primordia fail to develop floral organs, but occasionally produce bract-like structures, likely due to de-repression of cryptic bract outgrowth (Figure 1H) [24].

Consistent with a role in flower primordia development, *CRN* expression was detected as early as incipient primordia (before primordia outgrowth) and remained throughout primordia formation (Figure 1I). Both CRN (CRN-GFP) and CLV2 (CLV2-Citrine) fusion proteins, expressed by native promoters, confirmed this expression pattern (Figures S1B and S1C). Supporting previous *in situ* results [15], *WUS* expression did not overlap with *CRN* spatially or temporally during primordia specification and outgrowth, when *crn* inflorescence phenotypes diverge from WT (Figures 1I and 1J). The *WUS* domain was expanded in *crn* IMs compared to WT but ectopic *WUS* expression was not detected in terminated *crn* primordia (Figures 1J and 1K) [17]. Additionally, other mutants

known to have expanded *WUS* IM expression domains do not display *clv2/crn* primordia outgrowth defects (see below).

CLV2/CRN functions have been studied extensively in the Landsberg-*erecta* (Ler*)* background [16, 17, 25]. We found no flower primordia outgrowth defects in null *clv2-1* mutants in Ler, explaining why this phenotype has not been described in this ecotype (Figures S1D-S1E and S1I-S1J). Flower primordia termination was also not observed in *crn-1* (Ler background [17]) or two CRISPR-derived null alleles of *clv2* in Ler (*clv2-10* and *clv2-11*; Figures S1F-S1H and S1I-S1K). F1 plants from an ecotype-hybrid *clv2* null cross of *rlp10-1* (Col-0) X *clv2-1* (Ler) displayed mild termination (Figure S1L). Flower primordia outgrowth defects segregated in a digenic semi-dominant manner in the F2 population (Figure S1M), indicating that dominant modifiers in Col-0 underlie ecotype differences in *clv2/crn* flower outgrowth. Additionally, ecotypic differences were not attributable to the *erecta* (*er*) allele in Ler plants [26], as *clv2 er* double mutants in the Col-0 background had equivalent flower outgrowth defects to *clv2* (Figures S1N-S1P). Double mutants between *CLV2* and the floral identity gene *LEAFY* (*LFY*; *clv2 lfy*) displayed primordia outgrowth defects and *lfy*-like floral organ conversions (Figures S1Q-S1R), indicating that *CLV2/CRN* promote primordia formation independent of *LFY*-floral meristem specification [10, 27]. Collectively, these data show that CLV2/CRN signaling represents a novel ecotype-dependent process regulating primordia outgrowth following reproductive transition.

**CLAVATA2/CRN-mediated flower outgrowth requires CIK co-receptors**

The CLV2/CRN receptor complex lacks signaling capacity alone, as CRN is a transmembrane pseudokinase [19], suggesting CLV2/CRN require associated functional kinase(s) to signal. CLAVATA candidates with active kinase domains include the CLAVATA3 INSENSTIVE RECEPTOR KINASE1/2/3/4 (CIK1/2/3/4) family co-receptors, CLV1 and the CLV1-related BARELY ANY MERISTEM1/2/3 receptors (BAM1/2/3), which all regulate IM stem cell proliferation

[28-31]. CLV1/BAM signals independent of CLV2/CRN in shoot and floral stem cell control [20]. Consistent with this, we found negligible amounts of flower primordia termination in *clv1, bam1/2,* or *bam1/2/3* null mutants (in Col-0) and *crn* was additive in each higher order mutant combination (*crn clv1, crn bam1/2,* and *crn bam1/2/3*; Figures S2A-S2J). CIK1/2/3/4 are leucine-rich repeat (LRR)-II-receptor-like-kinase subfamily co-receptors with overlapping functions with CLAVATA primary receptors, several of which physically interact with CRN [31]. In a previous report we noticed a *crn*-like phenotype in specific *cik* mutant combinations [31]. To confirm this observation, we generated higher order CRISPR null alleles of *CIK1/2/4* in Col-0 (Figure S2K). In contrast to *clv1/bam* mutants, *cik1/2/4* displayed flower primordia termination equivalent to *crn* (Figures 2A-2C). Additionally, *cik1/2/4* had enlarged floral meristems quantitatively similar to *crn* (Figure 2D). The protein phosphatase POLTERGEIST (POL), a downstream component of CLAVATA signaling, suppresses *clv2/crn* meristem size defects [17, 32]. *pol* restored flower outgrowth and internode elongation defects in *crn* (*crn pol*; Figures 2E-2H). Collectively, these data demonstrate that CLV2/CRN signal alongside CIK1/2/4 co-receptors to promote flower outgrowth through a POL-dependent pathway.

CLAVATA receptors respond to CLAVATA3(CLV3)/EMBRYO-SURROUNDING REGION (CLE) peptide ligand(s), and there are 32 *CLE*s in Arabidopsis [33]. CLV3 and a suite of redundant CLE peptides signal via CLV1 to repress IM stem cell proliferation parallel to CLV2/CRN [13, 17, 34, 35]. Flower primordia outgrowth is not impaired in *clv3* or *dodeca-cle* higher order mutants, which combine *clv3* with several redundant *cle* alleles [13], suggesting that additional unknown CLE peptides regulate flower outgrowth through CLV2/CRN/CIK (Figure S2L-S2M). Consistent with this, *crn clv3* double mutants are additive with a clear disruption in flower primordia outgrowth and an enlarged disc-like IM (Figure S2N). The enlarged shoot and fasciated stem of *crn clv3* made quantification of terminated primordia difficult; however, these data support previous work suggesting that CLV2/CRN can act independently of CLV3 [20].

**Temperature and *CLV2/CRN* modulate auxin-dependent flower primordia outgrowth**

Many developmental programs are robust, ensuring optimal morphology/function across varied conditions [13, 36]. Populations of *A. thaliana* can be found throughout the Northern Hemisphere thriving in diverse environments [7, 37]. Natural variation in traits like flowering time and freezing tolerance are influenced by and/or directly correlated with adaptations to local conditions [7]. While investigating *clv2*/*crn* we observed remarkable quantitative variability in flower primordia termination at different temperatures. Flower outgrowth defects in *clv2/crn* were suppressed when grown at higher temperatures (31°C) compared to colder/ambient temperatures (16°C/24°C; Figures 3A-3G). Previous work noted shoot defects in *crn-1* mutants (Ler background) at high temperatures; however, we did not observe this under our conditions (Figures 3H-3I) [17]. Thermomorphogenic pathways regulate high temperature seedling growth by enhancing auxin biosynthesis [38, 39]. At higher temperatures, PHYTOCHROME INTERACTING FACTOR (PIF) family transcriptional regulators activate *YUCCA* (*YUC*) genes, which encode rate-limiting enzymes in auxin biosynthesis [40, 41]. Under colder/ambient temperatures, thermomorphogenesis is negatively regulated by the transcriptional repressor EARLY FLOWERING 3 (ELF3) [42]. As such, *elf3* seedlings display constitutive thermomorphogenic responses and higher auxin production across temperatures. To test if the thermomorphogenesis pathway was sufficient to suppress *clv2/crn* flower outgrowth defects, we generated *crn elf3* double mutants and grew them at colder/ambient temperatures. Consistent with high temperature mediated suppression of *crn*, flower primordia termination was suppressed in *crn elf3* at colder temperatures (Figures 3J-3L). In contrast to the suppression of *crn* primordia outgrowth, *elf3* slightly enhanced carpel numbers compared to *crn* (Figure 3M). This finding supports that CLV2/CRN-mediated outgrowth and CLV2/CRN-mediated meristem size regulation are separable with primordia outgrowth being highly sensitive to thermal conditions. Our data shows that CLV2/CRN/CIK signaling is critical for continuous flower production at colder/ambient

160 temperatures but can be bypassed by thermomorphogenic responses to higher temperatures. As
161 such, while Arabidopsis flower development is robust under various environmental conditions,
162 distinct mechanisms maintain this stability across different temperatures.
163
164 To define the mechanisms underlying *crn* primordia termination, we used RNA-seq to identify
165 differentially expressed genes (DEGs) in terminating *crn* IMs compared to WT. Using a strict cutoff
166 (p-value < 0.001), we found 460 DEGs between *crn* and WT IMs, with 236 upregulated and 224
167 downregulated in *crn* (Figure 4A; Table S1). Enriched Gene Ontology (GO) terms among DEGs
168 included meristem maintenance, flower development, and auxin function (Figure 4B; Table S2)
169 [43-45]. The first two GO term groups are consistent with CRN's role in meristem maintenance
170 [17, 20], and flower development, documented in this study (Tables S1 and S2). The
171 overrepresentation of auxin-associated genes in *crn* IM DEGs (Table S1) is complementary to the
172 thermomorphogenic suppression of *crn*'s primordia termination (Figure 3), suggesting CLV2/CRN
173 regulate auxin function during early flower primordia outgrowth. Therefore, we asked if *clv2/crn*
174 were defective in auxin outputs at lower temperatures and if auxin biosynthesis was required for
175 the high temperature suppression of *clv2/crn*. We visualized the auxin signaling reporter
176 *DR5::GFP* (where GFP positively correlates with increased auxin signaling output) and the auxin
177 perception reporter *DII-Venus* (where Venus negatively correlates with increased auxin
178 perception) in terminating *crn* IMs [46, 47]. There was a significant reduction in DR5::GFP signal
179 in *crn* IMs during termination, specifically in the L1 layer of incipient primordia (Figures 4C-4D and
180 S3A-S3C). Consistently, *DII::Venus* accumulated in the L1 layer of terminated *crn* IMs, a pattern
181 never observed in WT (Figures 4E-4F and S3D). DR5::GFP was restored to WT levels during
182 *crn*'s recovery phase (Figures S3E-S3F). During flower development, the PIN-FORMED1 (PIN1)
183 auxin efflux transporter concentrates auxin to the IM periphery, creating local maxima which
184 trigger flower primordia initiation and subsequent outgrowth [4, 5]. PIN1 reporter levels (PIN1-
185 GFP) [5] were decreased in terminating *crn* IMs (Figures S3G-S3H); consistent with *PIN1*

186 expression from our RNAseq DEG data. (Table S1). PIN1-GFP levels increased during *crn*'s
187 recovery phase, but not to WT levels (Figure S3I). These data demonstrate an overall reduction
188 in auxin signaling/perception within the IM and developing primordia of *crn* during the termination
189 phase. This decrease is transient and corresponds with flower outgrowth defects, indicating that
190 CLV2/CRN positively regulate auxin dependent flower primordia outgrowth in the IM.
191
192 Several auxin biosynthetic genes had decreased expression in *crn* compared to WT, including
193 *YUC* genes that regulate flower development (Figure S3J and Table S3) [48]. To test if low auxin
194 levels contributes to *crn*'s flower outgrowth defects, we expressed *YUC1* in developing primordia
195 of *crn* using the *AINTEGUMENTA* promoter (*ANTp::YUC1*) [6, 49] and grew plants in colder (16-
196 18°C) and ambient (22-24°C) temperatures. At 22-24°C, 9/13 T1 plants suppressed *crn* while only
197 2/18 plants partially suppressed *crn* at 16-18°C (Figures 4G-4H). This demonstrates that at
198 ambient temperatures (where *crn* terminates), ectopic *YUC1* can suppress flower outgrowth
199 defects; however, the degree of suppression correlates with temperature. Higher order mutant
200 combinations in IM-expressed *YUC1/2/4/6* severely impair floral and vasculature development;
201 however, *yuc1/4* double mutants produce more typical inflorescences with identifiable flowers
202 [48]. We generated *clv2 yuc1/4* triple mutants to reduce YUC-dependent auxin in *clv2* and test
203 whether high temperature suppression of *clv2*/*crn* flower termination was dependent on *YUC*-
204 mediated auxin biosynthesis. At 16-18°C, *clv2 yuc1/4* triple mutants displayed rates of flower
205 primordia termination comparable to *clv2* (Figure 4I). As such, CLV2/CRN promote auxin
206 mediated primordia outgrowth independent of YUC1/4 in colder temperatures. At 28-31°C, some
207 *clv2 yuc1/4* triple mutant plants displayed *clv2* flower primordia termination, indicating that at high
208 temperatures YUC1/4 contribute to heat induced suppression (Figure 4J). Surprisingly though,
209 the majority (~60%) of *clv2 yuc1/4* plants had a synergistic response to high temperatures
210 resulting in *pin*-like inflorescences completely lacking flower primordia (Figure 4J). These data

211 suggest that high temperature suppression of *clv2* is dependent on *YUC1/4*-mediated auxin
212 biosynthesis and that under high temperatures, *CLV2/CRN* and *YUC1/4* are synergistically
213 required to maintain flower primordia initiation and outgrowth.
214
215 Ensuring robust development and reproduction across environments is a challenge all organisms
216 face. Here we demonstrate that robust flower production in diverse thermal environments is
217 achieved through the synergistic deployment of CLV2/CRN-signaling and ELF3-regulated auxin
218 production via the thermomorphogenesis pathway in Arabidopsis. The relative contribution of
219 each to flower development varies across thermal clines, with CLV2/CRN signaling being critical
220 at colder/ambient temperatures and synergistic with heat-induced auxin production at higher
221 temperatures (Figure S4). Arabidopsis seedlings respond to high temperatures by promoting
222 auxin-dependent hypocotyl elongation, a process negatively regulated by ELF3 [38, 42]. Our work
223 demonstrates that high temperatures and *ELF3* also regulate auxin-dependent primordia
224 production. Interestingly, *clv2 yuc1/4* primordia outgrowth defects were strongly enhanced in
225 warmer conditions. Higher temperatures also enhance penetrance of seedling defects in loss-of-
226 function mutants in the TRANSPORT INHIBITOR RESPONSE1 (TIR)-family auxin receptors [50].
227 As such, heat might have an unappreciated negative impact on auxin function, with
228 thermomorphogenesis-induced auxin playing a protective role rather than simply directing growth.
229 How CLV2/CRN stimulate auxin-dependent flower initiation is unknown. Ectopic *YUC1*
230 expression or heat-induced auxin production is sufficient to restore primordia outgrowth to
231 *clv2/crn*. This suggests that CLV2/CRN are not critically required for TIR-dependent auxin
232 perception, ARF5/MONOPEROS dependent transcriptional activity, or the TRYPTOPHAN
233 AMINOTRANSFERASE OF ARABIDOPSIS (TAA1)-mediated conversion of tryptophan to indole-
234 3-pyruvate (IPA) step upstream of YUCCA in auxin biosynthesis [6, 51-53].
235

*clv2/crn* defects manifest early in inflorescence development and are transient. Recovery of primordia production is not linked to flower meristem identity or seed/fruit derived auxin production (*clv2 lfy* plants). This suggests that the transition from vegetative to reproductive meristem fate may be sensitized to CLV2/CRN signaling. Nevertheless, *clv2 yuc1/4* plants reveals that CLV2/CRN signaling is required at later steps in inflorescence development as well. Heat stress is known to damage crops in ways that negatively impact yield, including the loss of flower production [54, 55]. As climate change increases global temperatures it will be necessary to mitigate heat impacts on crop yield. If the environmental buffering capacity of CLV2/CRN-signaling is conserved in crop species perhaps it could be deployed to help improve plant responses to climate change.

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**AUTHOR CONTRIBUTIONS**

D.S.J. designed/performed experiments, analyzed data, acquired funding for support, and wrote the manuscript. A.J. designed/performed experiments and analyzed data. K.R.V performed Col-

0 x Ler population experiments. Z.L.N. conceptualized the project and experiments, analyzed data, acquired funding, and wrote the manuscript.

## DECLARATION OF INTERESTS

The authors declare no competing interests.

## MAIN TEXT FIGURE LEGENDS

**Figure 1. Flower primordia outgrowth is disrupted in *clv2*/*crn***

(A-D) Inflorescences of Col-0, *crn* (*crn-10*), *clv2 (rlp10-1)*, and *crn clv2* double mutants. (A'-D') Close up showing flower primordia termination in *crn, clv2* and *crn clv2*.

(E) Quantification of flower termination, classifying the first 30 attempts to make a flower as: normal (grey), terminated primordia (blue) or terminated flowers (yellow) in Col-0 (n=28), *crn* (n=27), *clv2* (n=25), and *crn clv2* (n=25).

(F and G) 3-D reconstruction of inflorescence meristems of (F) Col-0 (n=4) and (G) crn (n=6). Axial view of the (F', G') third (FP3) and (F", G") fourth (FP4) flower primordia (labeled 3 and 4 in (F) and (G)) revealing developmental differences. FP3 and FP4 were determined by identifying the 3$^{rd}$ and 4$^{th}$ earliest detectable primordia along the IM, respectively. Staging similar to [23].

(H) Side view of a young inflorescence meristem of *crn* during the termination phase (n=5).

(I-K) Expression patterns of *YPET-N7* reporter lines in the IM with XY view of L5 layer (I-K) and axial view (Z-axis) of same IM stack (I'-K') shown for each: (I) *CRNpro* in Col-0 (n=6), (J) *WUSpro* in Col-0 (n=6), (K) *WUSpro* in *crn* (n=6). Tissue stained with propidium iodide (PI; magenta).

Statistical groupings based on significant differences found using Kruskal-Wallis and Dunn's multiple comparison test correction (E). Scale bars, 50μm in (H), 20μm in (F-G) and (I-K).

See also Figure S1.

**Figure 2. CLV2/CRN-mediated flower outgrowth requires CIK1/2/4 co-receptors and a downstream *POL*-dependent pathway**

(A-B) Inflorescence of *crn* and *cik1/2/4* mutant plants reveal similar flower termination defects. Quantification of (C) flower termination and (D) carpel number across Col-0 (n=16), *crn* (n=17), and *cik1/2/4* (n=13).

(E-F) Inflorescence of *crn* and *crn pol* showing that flower termination is suppressed by *pol* (*pol-6*). Quantification of (G) flower termination and (H) carpel number across Col-0 (n=15), *crn* (n=15), *pol* (n=15), and *crn pol* (n=14).

Box and whisker plots show full range of data (min to max) with mean marked as "+".

Statistical groupings based on significant differences found using Kruskal-Wallis and Dunn's multiple comparison test correction (C-D and G-H).

See also Figure S2.

**Figure 3. High temperature responses modulate *clv2*/*crn* flower primordia outgrowth**

(A-F) Temperature-dependent inflorescence phenotypes of *crn* compared to Col-0. Flower primordia termination in *crn* is prevalent at (D) 16°C and (E) 24°C, but is suppressed at (F) 31°C.

(G) Quantification of flower primordia termination at 16°C: Col-0 (n=8), *crn* (n=9), *clv2* (n=9), *crn clv2* (n=7), 24°C: Col-0 (n=9), *crn* (n=9), *clv2* (n=9), *crn clv2* (n=9), and 31°C: Col-0 (n=8), *crn* (n=9), *clv2* (n=9), *crn clv2* (n=6).

(H-I) Inflorescences of Ler and *crn-1* grown at 31°C.

(J-K) Inflorescences of *elf3 (elf3-1)* and *crn elf3* grown at 16-18°C

(L-M) Quantification of (L) flower primordia termination and (M) carpel number in Col-0 (n=24), *crn* (n=18), *elf3* (n=27), *crn elf3* (n=26) grown at 16-18°C.

Box and whisker plots show full range of data (min to max) with mean marked as "+".

Statistical groupings based on significant differences found using Kruskal-Wallis and Dunn's multiple comparison test correction (L-M).

**Figure 4. Flower primordia outgrowth is maintained by separable auxin-dependent processes in different thermal conditions**

(A) Differentially expressed genes (DEG, p-value < 0.001) between *crn* and WT IMs; upregulated genes (red), downregulated genes (blue).

(B) Top GO terms enriched in *crn* DEGs, highlighting terms associated with flower development (blue), meristem maintenance (green) and auxin (red).

(C-D) Maximum intensity projection (MIP) of *DR5::GFP* (teal) in the IM of (C) Col-0 (n=14) and (D) *crn* (n=12). Stained with PI (magenta). Arrows point to primordia used for DR5 quantification/comparison.

(E-F) Axial view showing DII-Venus (green) expression pattern in (E) Col-0 (n=6) and (F) *crn* (n=8) IMs. Stained with PI (magenta). (E'-F') L1 layer of IMs from (E') Col-0 and (F') *crn* used to quantify percent of L1 cells with DII marker. Arrows point to organ boundaries where DII reporter can be detected in the L1 of Col-0 and *crn.*

(G-H) Flower primordia termination across independent T1 lines of *ANTpro::YUC1* in *crn* compared to Col-0 and *crn* when grown at (G) 16-18°C or (H) 22-24°C.

(I) *clv2 yuc1/4* inflorescence grown at colder temperatures (16-18°C) and quantification of flower outgrowth defects in Col-0 (n=9), *clv2* (n=9), *yuc1/4* (n=8), *clv2 yuc1/4* (n=10).

(J) *clv2 yuc1/4* pin-like inflorescence grown at hot temperatures (28-31°C) and quantification of flower outgrowth defects, including instances of pins (pink bar; right y-axis) in Col-0 (n=29), *clv2* (n=20), *yuc1/4* (n=20), *clv2 yuc1/4* (n=12).

Scale bars, 20μm in (C-D) and (E-F), 1mm in (I-J).

Bar plots show mean with SEM.

See also Figure S3, Tables S1-S3.

**STAR METHODS**

## RESOURCE AVAILABILITY

### Lead Contact

Information and resource/reagent requests should be directed to and will be fulfilled by the Lead Contact, Zachary Nimchuk (zackn@email.unc.edu).

### Materials Availability

Plasmids and Arabidopsis lines made during this study are freely available to academic researchers through the Lead Contact.

### Data and Code Availability

Raw RNAseq data described in this study has been deposited into the NCBI Short Read Archive (SRA) database under the BioProject PRJNA661065. Code used to analyze gene expression data can be found on the Nimchuk Lab GitHub page (https://github.com/NimchukLab). All other source data obtained throughout the course of this work have not been deposited to any public repository but are available upon request from the Lead Contact.

## EXPERIMENTAL MODEL AND SUBJECT DETAILS

*Arabidopsis thaliana* accession Columbia (Col-0) was used as our primary model system throughout this work. Some phenotypic comparisons were also made with the accession Landsberg-*erecta* (Ler), as noted.

### Plant growth conditions

Seeds were sterilized and plated on half-strength MS (Murashige-Skoog) media buffered with MES, pH 5.7. Plates were stratified in the dark at 4°C for 2 days and then moved to constant light in a custom-built grow room with environmental control (temperature maintained between 21-25°C), or in a Percival growth chamber (AR-75L3) when growing at a specified temperature. After

7-10 days, seedlings were transplanted to soil (Metro-Mix 360/sand/perlite supplemented with Marathon pesticide and Peter's 20:20:20 [N:P:K] at recommended levels) and then placed back into the chamber they were germinated in and grown until flowering for phenotypic analysis.

## METHOD DETAILS

### Plant materials

Mutant alleles used in this study are all in the Col-0 background, unless otherwise noted, and information for each is as follows: *crn* (*crn-10*) [20], *crn-1* in Ler [17], *clv2* in Col-0 (*rlp10-1*) [21], *clv2-1* in Ler [16], *erecta* (*er-105*) [56], *lfy* (*lfy-1*; ABRC CS6228) [27], *clv1* (*clv1-101*) [57], *bam1* (*bam1-4*) [30], *bam2* (*bam2-4*) [30], *bam3* (*bam3-2*) [30], *pol* (*pol-6*) [58], *clv3* (*clv3-9*) [30], *dodeca-cle* (*clv3-9;* CRISPR alleles of: *cle9, cle10, cle11, cle12, cle13, cle18, cle19, cle20, cle21, cle22, cle45)* [13], *elf3* (*elf3-1*; ABRC CS3787) [59], *yuc1* (SALK_106293) [48], and *yuc4* (SM_3_16128) [48]. Information for previously published transgenic complementation and reporter lines used are as follows: *CRNpro::CRN-GFP* in *crn* [20]*, CLV2pro::CLV2-CITRINE* in *clv2* [60], *WUSpro::Ypet-N7* in Col-0 (gift from Paul Tarr – Caltech), *DR5pro::GFP* in Col-0 [61, 62], *DII-Venus* in Col-0 [63], *PIN1pro::PIN1-GFP* in Col-0 [64]. The *DR5pro::GFP, DII-Venus,* and *PIN1pro::PIN1-GFP* lines were all crossed into *crn* (*crn-10*) for analysis of auxin signaling levels in *crn* shoots compared to WT.

### CRISPR mutagenesis of *CIK1/2/4* and *CLV2*

The *pCUT* vector system was used to simultaneously create *cik1*, *cik2* and *cik4* mutations in the Col-0 background, making the *cik1/2/4* higher order mutant [65]. Similarly, the *pCUT* system was used to make multiple unique mutations in *clv2* in the Ler background. A *pENTR-D/TOPO* entry vector was modified for golden gate cloning by TOPO cloning in PmeI and BsaI cut sites (*pENTR-GG)*. *pENTR-GG* was used to clone tandem cassettes (*cik1/2/4*) or a single cassette (*clv2*) of the

U6 promoter, 20 bp guide sequence, and the gRNA scaffold. These guide constructs were then gateway cloned into *pCUT4GTW* vector that expresses Cas9 from the *UBIQUITIN10* promoter. Hygromycin resistant plants were selected in the T1 generation and sequenced to detect editing of the target genes; *CIK1* (AT1G60800), *CIK2* (AT2G23950), and *CIK4* (AT5G45780) or *CLV2* (AT1G65380). To make stable *cik1/2/4*, T2 seed from editing *cik1/2/4* lines were grown for 2 weeks under standard conditions, heat shocked for 12-24 hours at 35°C [66], and screened for stable edits using dCAPS primers designed by the indCAPS webtool (http://indcaps.kieber.cloudapps.unc.edu/) [67]. Cas9 was segregated out of plants that had stable homozygous mutations in *CIK1/2/4.* To make stable *clv2* alleles in Ler, T2 seed, collected from single branches of T1 plants that had the *clv2* carpel phenotype, were grown in a Percival growth chamber at 16°C (see above), screened for stable mutations while segregating Cas9 out prior to phenotypic analysis.

**Columbia-0 X Landsberg-*erecta* hybrid *clv2* population**

Null *clv2* lines in the Col-0 (*rlp10-1*) and Ler (*clv2-1*) backgrounds were crossed to generate a hybrid population with fixed *clv2* mutations. Segregation of flower termination traits was assessed in the F2 generation. Phenotypic ratios were compared to expected values of a single causative locus (1:2:1) and digenic semi-dominant modifiers (7:6:3) using Chi-squared analysis (Microsoft Excel v.16.40) to determine the underlying genetic complexity of the ecotypic variability of *clv2* termination.

**Generation of binary vectors and transgenic lines**

New transgenic lines were generated using floral-dip transformation of binary vectors into specified backgrounds [68]. *pWUSpro::Ypet-N7* (gift from Paul Tarr – Caltech; cloning methods as in [69] but with *Ypet-N7*) was transformed directly into *crn* and the transgene was selected on

½ MS plates with Kanamycin. Four independent lines were selected for downstream analysis with the *WUS* expression domain being equivalent across all lines imaged (see imaging methods for data acquisition details). For *pCRNpro::Ypet-N7*, a pENTR-D *Ypet-N7* (2xYpet-N7 fusion) entry vector was recombined by LR reaction into the *pCRNpro::Gateway* binary vector in the *pMOA33* background [20]. For *pANTpro::YUC1*, the 4 kb 5' *ANT* promoter was amplified from genomic DNA, and cloned up stream of a gateway::OCS terminator cassette in the *pCR2.1* shuttle vector, and sequence verified. The resulting *ANTpro::Gateway::OCS* cassette was then mobilized as a Not1 fragment in to the *pMOA33* binary vector backbone as previously described to create *pANTpro::GTW* [30]. The *YUC1* CDS was amplified from Arabidopsis Col-0 cDNA and cloned into the *pENTR-D* topo vector and sequence verified. This vector was then recombined in an LR reaction into *pANTpro::GTW* to create the *pANTpro::YUC1*.

**Photography and time-lapse imaging**

Unless specified differently below, young inflorescences were staged at similar developmental timepoints and photographed using a Canon EOS Rebel T5 equipped with a Tokina 100mm f/2.8 AT-X M100 AF Pro D macro lens. Images were edited for brightness and contrast using Gimp v2.10.4 (https://www.gimp.org/). Young inflorescences from Col-0*, crn*, *clv2, crn clv2, cik1/2/4, yuc1/4,* and *clv2 yuc1/4* (Figures 1A'-D', 2B, 4E-F, and 4H-I) were imaged using a Zeiss Stemi 2000-C stereo microscope equipped with a Zeiss Axiocam 105-color digital camera and acquired using Zeiss ZEN software. Time-lapse imaging was done using the Lapse-it (http://www.lapseit.com/) app on an iPhone 6 operating iOS 12.3.1. Imaging began with ~3-week-old Col-0 (WT) and *crn* plants growing in our custom-built grow room. A single image was taken every 30 minutes over the course of 10-12 days. The final movie was compiled at 30 frames per sec and exported into iMovie where it was cropped to show only two plants for comparison of early flowering phenotypes.

**Confocal microscopy**

Live imaging of inflorescence meristems (IMs) was performed as previously reported [20, 70]. We used either an inverted Zeiss 710 (for: propidium iodide (PI) stained Col-0, *crn, CRNpro:Ypet-N7, WUSpro::Ypet-N7, DR5pro::GFP, DII-Venus, and PIN1pro::PIN1-GFP*) or a Zeiss 880 (for *CRNpro::CRN-GFP* and *CLV2pro::CLV2-CITRINE*) confocal laser scanning microscope equipped with an inverter (setup described in [71]). Young IMs were dissected immediately following floral transition in order to analyze expression patterns and reporter levels at the same developmental stage as *clv2/crn* flower primordia termination. When analyzing reporter status in recovered IMs of *crn*, shoots were dissected at a later timepoint after flower buds were visibly developing again. All IMs were briefly (~5 mins for WT and ~15 mins for *crn*) stained with PI (final concentration of 50μg/mL for WT shoots and 150μg/mL for *crn*) on ice and placed into a petri dish with 2% agarose (w/v) and immersed in cold water, ensuring no bubbles formed around the IM [70]. IMs were imaged using a W Plan-APOCHROMAT 40X (NA = 1.0) water dipping objective. Laser excitation and detected emission ranges were as follows: PI only – laser 561nm diode, PI channel 600-750nm; Ypet/Venus markers with PI – 514nm argon laser, Ypet channel 520-581nm, PI channel 655-758nm; GFP markers (on Zeiss 710) with PI – 488nm argon laser, GFP channel 493-556nm, PI channel 598-642nm; GFP on Zeiss 880 with PI – 488nm argon laser, GFP channel (GaAsP detector) 490-550nm, PI channel (GaAsP detector) 565-610nm; Citrine on Zeiss 880 with PI – 514nm argon laser, Citrine channel (GaAsP detector) 519-550nm, PI channel (GaAsP detector) 565-610nm. Whole IMs were imaged as a z-stack series with a step size optimized for three-dimensional reconstruction of data. All images comparing reporter levels in different backgrounds were obtained with identical specifications: *DR5pro::GFP, DII-Venus,* and *PIN1pro::PIN1-GFP* in both WT and *crn* shoots. Live inflorescence micrographs were all post-processed using ZEN (Zeiss) for three-dimensional reconstructions of IMs and Fiji/ImageJ v.2.0.0-rc-69/1.52u (National Institutes of Health) [72] for single scan images as well as axial views of

IMs. Channels corresponding to PI staining in *crn* IMs were almost always gamma corrected (0.8) as penetrance of this fluorescent dye in enlarged *crn* shoots was sometimes limited. *DR5pro::GFP* fluorescence quantification comparing WT and *crn* IMs was done as follows using Fiji/ImageJ v.2.0.0-rc-69/1.52u. Z-stacks were rendered as maximum intensity projections of the GFP channel only using data from the entire IM. A region of interest (ROI) was drawn around each of the first 3 developing primordia (identified as the 3 early primordia with the highest GFP intensities). GFP levels were quantified for each ROI, normalized to the final area of each ROI, and then averaged together to obtain a single value for WT and *crn* IMs. DII-Venus quantification was done using Fiji/ImageJ v.2.0.0-rc-69/1.52u. Due to rapid photobleaching of the Venus fluorescent reporter, single scan images were taken in the L1 layer of WT and *crn* IMs prior to z-stack scans and used for direct comparison. The percentage of L1 cells with the Venus reporter were determined across all imaged IMs, with WT IMs never having reporter in this layer.

Whole shoot reconstruction of *crn* during termination (Figure 1H) was done using fixed and cleared tissue, imaging structural autofluorescence (as in [13]). Young *crn* IM were fixed in FAA (2% formaldehyde, 5% acetic acid, 60% ethanol (w/v)) at 4°C overnight and then dehydrated in a graded ethanol series (70%, 80%, 95% and 100%) for 30 minutes each at room temperature. Tissue was then cleared overnight in methyl salicylate (catalog no. M6752; Sigma Aldrich) and placed in a small glass-bottom petri dish (catalog no. P35G-1.5-10-C; MatTek Corporation) and imaged on a Zeiss 710 CLSM using a Plan-APOCHROMAT 10X (NA = 0.45). Autofluorescence was detected using a 488nm argon laser for excitation and combining two channels for emission detection; Channel 1 – 504-597nm and channel 2 – 629-731nm. Data was gathered as a z-stack and three-dimensional reconstruction was done in Nikon NIS-Elements as a shaded render of both channels combined in grayscale.

**RNA Sequencing and Data Analysis**

Total RNA was isolated using the EZNA Plant RNA kit (Omega Bio-tek) from 45-50 inflorescence meristems for three biological replicates of both Col-0 and *crn* (*crn-10*) plants. RNA was treated with RNase-free DNase (Omega Bio-tek). Approximately 1.5 ug RNA was used as input material for library preparation, using the Stranded mRNA-Seq kit (Kapa Biosystems) at the High-throughput Sequencing Facility at UNC Chapel hill. 50bp paired-end reads were generated on the NovaSeq 6000 sequencer (illumina) with a read depth of 23-35 million reads per biological replicate. Trimmed raw data was aligned to the *A. thaliana* genome (TAIR10.1) using HISAT2 version 2.2.0 [73] and reads were counted using Subread version 1.5.1 [74]. Subsequent analysis was performed on RStudio with reads normalized using EDASeq version 2.22.0 [75] and RUVseq version 1.22.0 (upper quartile normalization) [76] and differentially expressed genes were identified with a p-value <0.001 using EdgeR version 3.33.0 [77]. These top 460 DEGs were used for GO term analysis from Panther [78]. To obtain the average TPM counts of auxin biosynthetic genes, reads were pseudoaligned to the *Arabidopsis* transcriptome (TAIR10 from plants.ensembl.org) using Kallisto version 0.44.0 [79] and quantified with Sleuth version 0.33.0 [80].

**QUANTIFICATION AND STATISTICAL ANALYSIS**

Quantitative data from all experiments was compiled and analyzed in GraphPad Prism v.8.3.2. We performed at least two biological replicates for each experiment ensuring consistent results (sample sizes indicated in figure legends). For comparisons of flower termination across conditions, genotypes and/or transgenic lines (Figures 1E, 2C, 2G, 3G, 3L, 4G-H, S1I, S2C-D, S2I-J), only % flower primordia termination (as defined in the paper) was compare across samples using a non-parametric Kruskal-Wallis and a Dunn's multiple comparison test correction where significance was defined as p-value < 0.05. For comparisons of carpel number across genotypes (Figures 2D, 2H, 3M, and S1J), 10 consecutive flowers on the primary inflorescence were

counted, starting after the recovery phase in *crn*, *clv2*, and *cik1/2/4* while starting at the 11$^{th}$ flower in genotypes that had no flower primordia termination or partially suppressed flower primordia termination (Col-0, Ler, *clv2-1, crn-1, pol, crn pol, elf3,* and *crn elf3*). Carpel number was compared statistically using a non-parametric Kruskal-Wallis and a Dunn's multiple comparison test correction where significance was defined as p-value < 0.05. For comparison of *DR5pro::GFP* levels, WT and *crn* IM values were compared statistically using an unpaired t-test, where the p-value = 0.0007. Sample size (n) for all analyses can be found with each figure in the legend and refers to the number of individual plants analyzed.

## SUPPLEMENTAL INFORMATION

**Table S1. Normalized differentially expressed genes (DEG) list in *crn* IMs. Related to Figure 4 and Table S2**

Genes with p-value < 0.001 showing their log2FC, p-value and false discovery rate (FDR). Red log2FC values indicate upregulation and blue log2FC values indicate downregulation. Red, green and blue highlights refer to GO processes indicating auxin, meristem and floral related genes, respectively, while purple highlights indicate the gene belongs to multiple of these groups.

**Movie S1 – Time-lapse of flower termination and recovery in *crn.* Related to Figure 1**

Time-lapse of Col-0 (left) and *crn* (right) during early stages of flowering revealing both *crn*'s termination and recovery phases. Entire movie spans ~10 days. Movie rendered at 30 frames per second while each frame was captured in 30-minute intervals (see methods).

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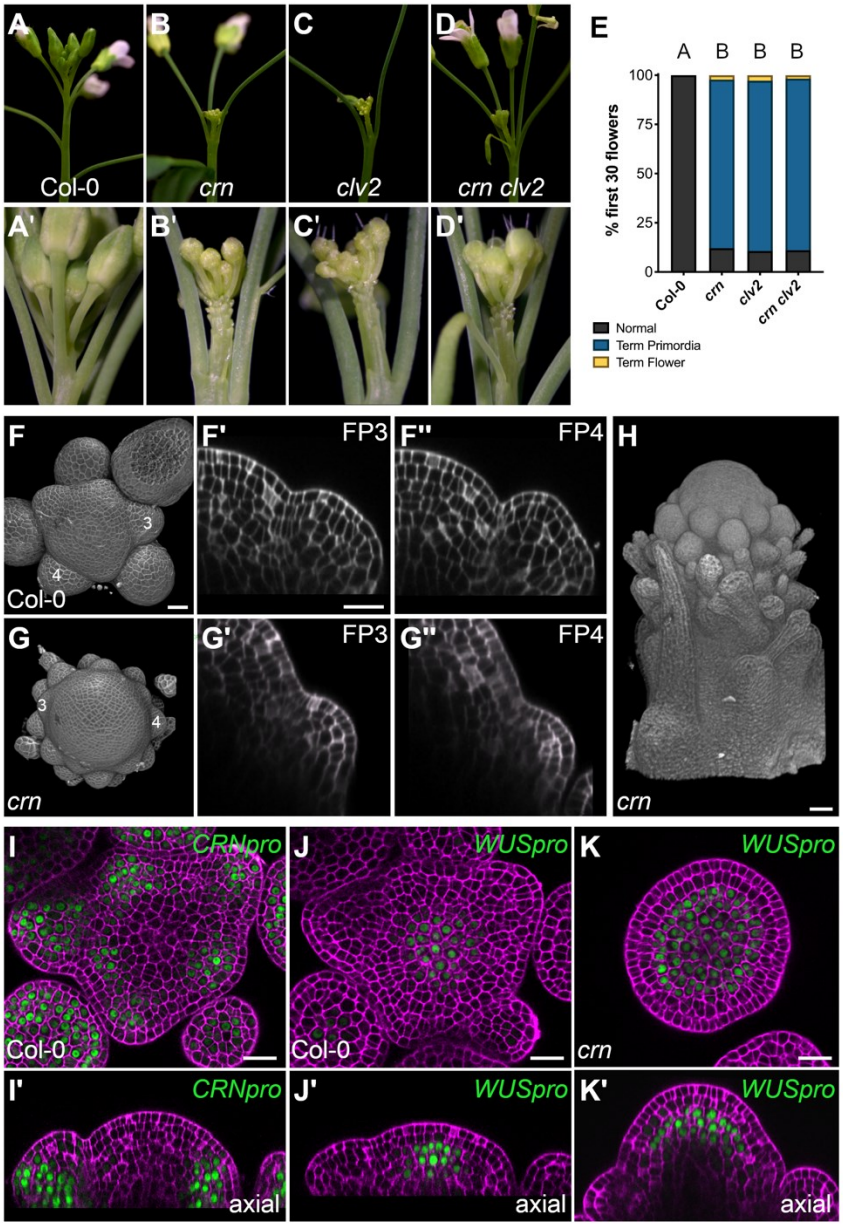
**Figure 1**


A
B
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Col-0
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crn clv2
A'
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C'
D'
E
A B B B
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25
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% first 30 flowers
Col-0
crn
clv2
crn clv2
Normal
Term Primordia
Term Flower
F
F'
FP3
F''
FP4
H
3
4
Col-0
G
G'
FP3
G''
FP4
3
4
crn
crn
I
CRNpro
J
WUSpro
K
WUSpro
Col-0
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WUSpro
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axial
axial

763 **Figure 2**

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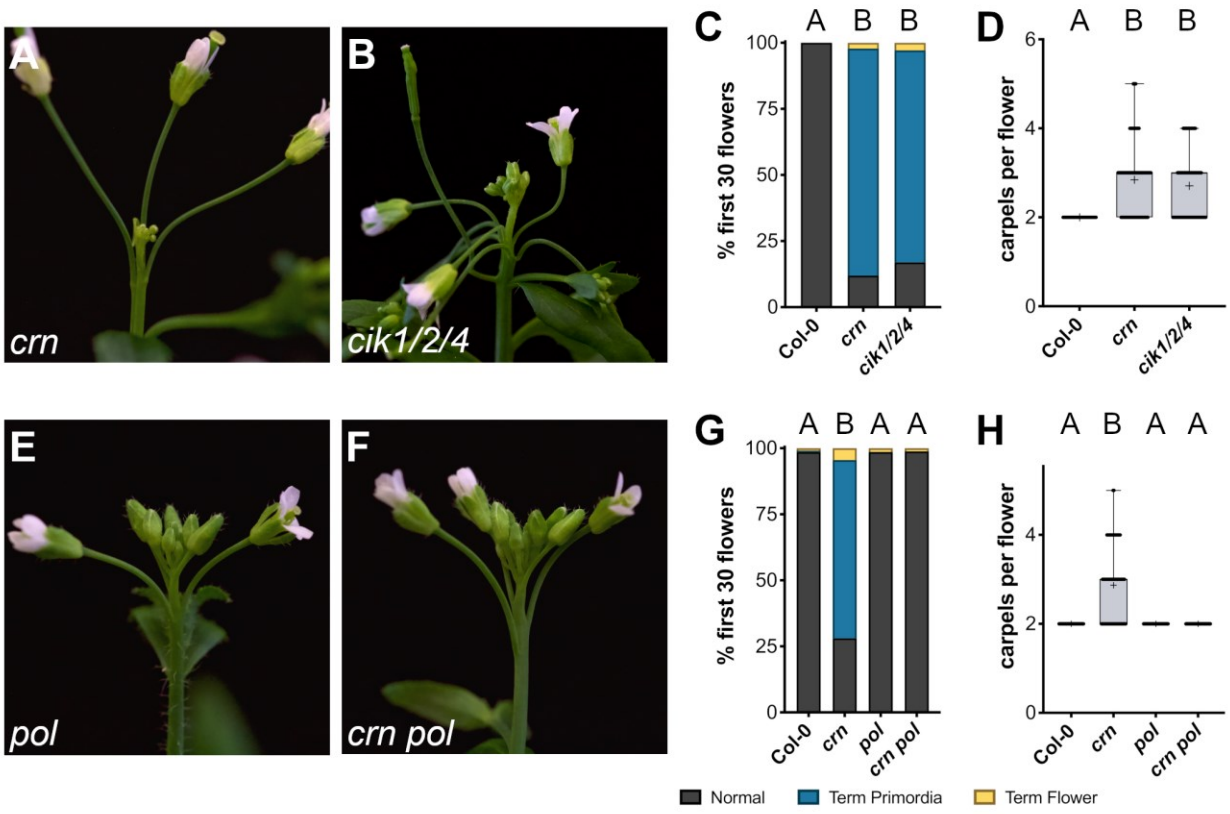


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767 **Figure 3**

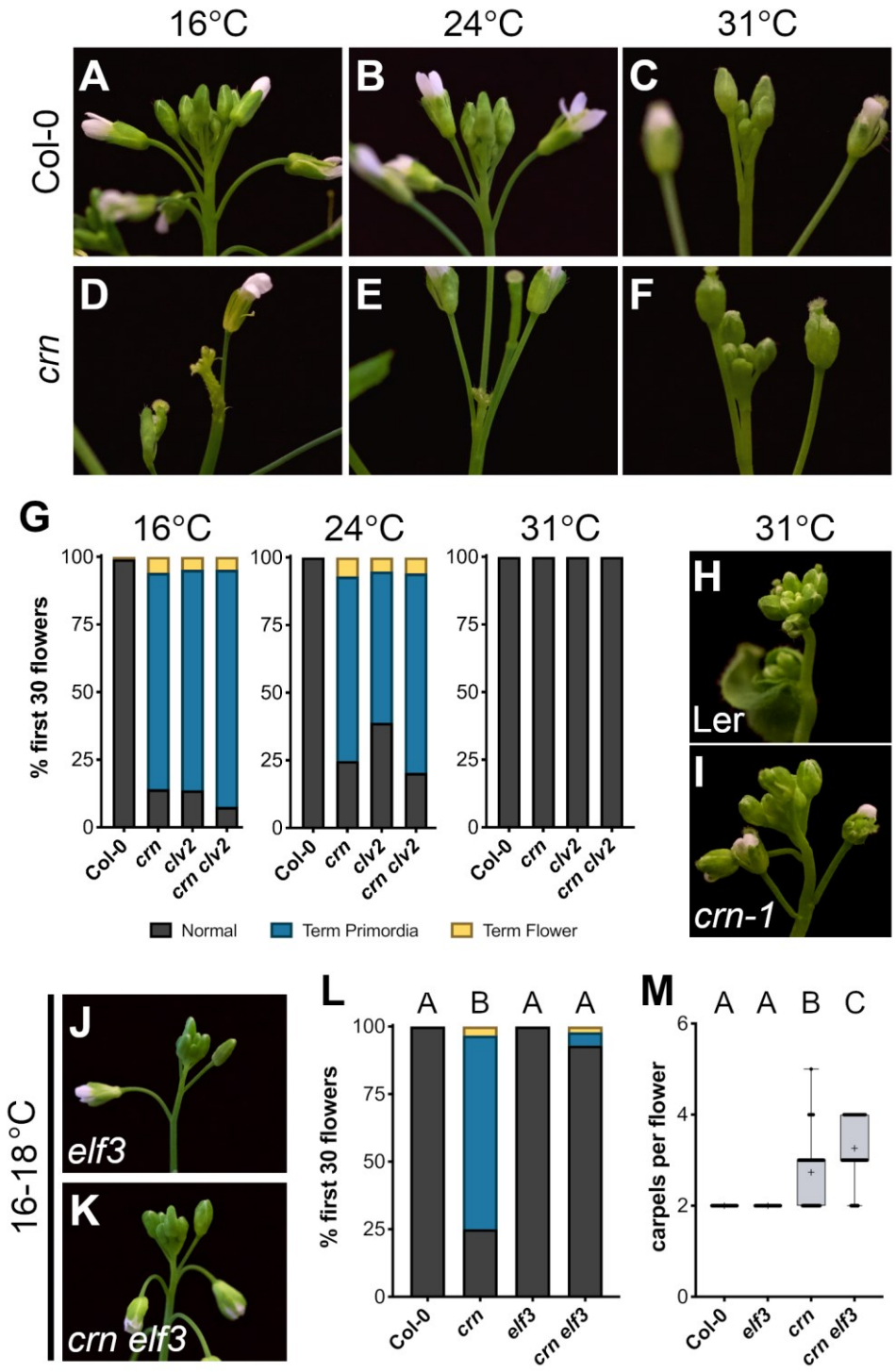


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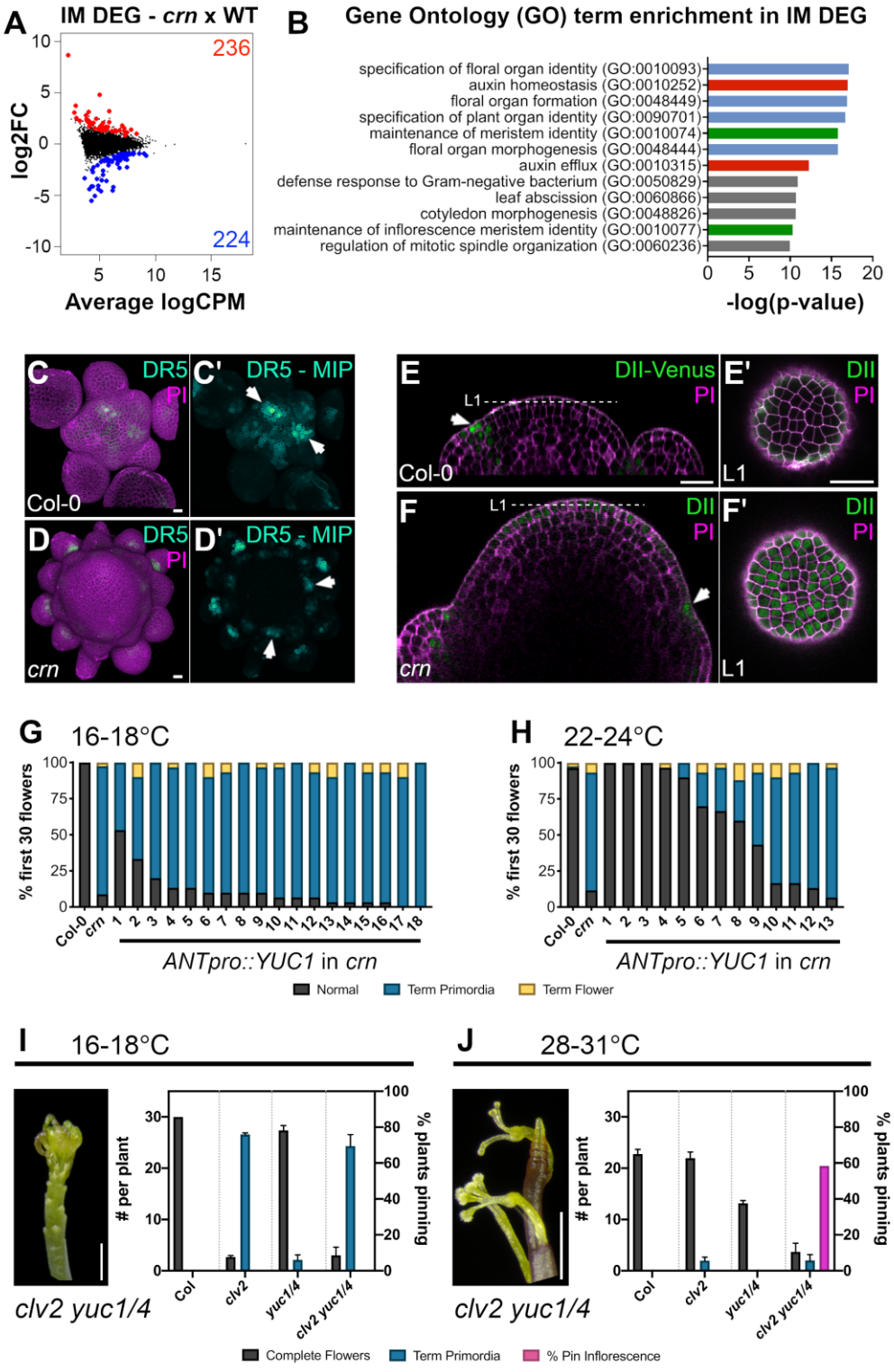
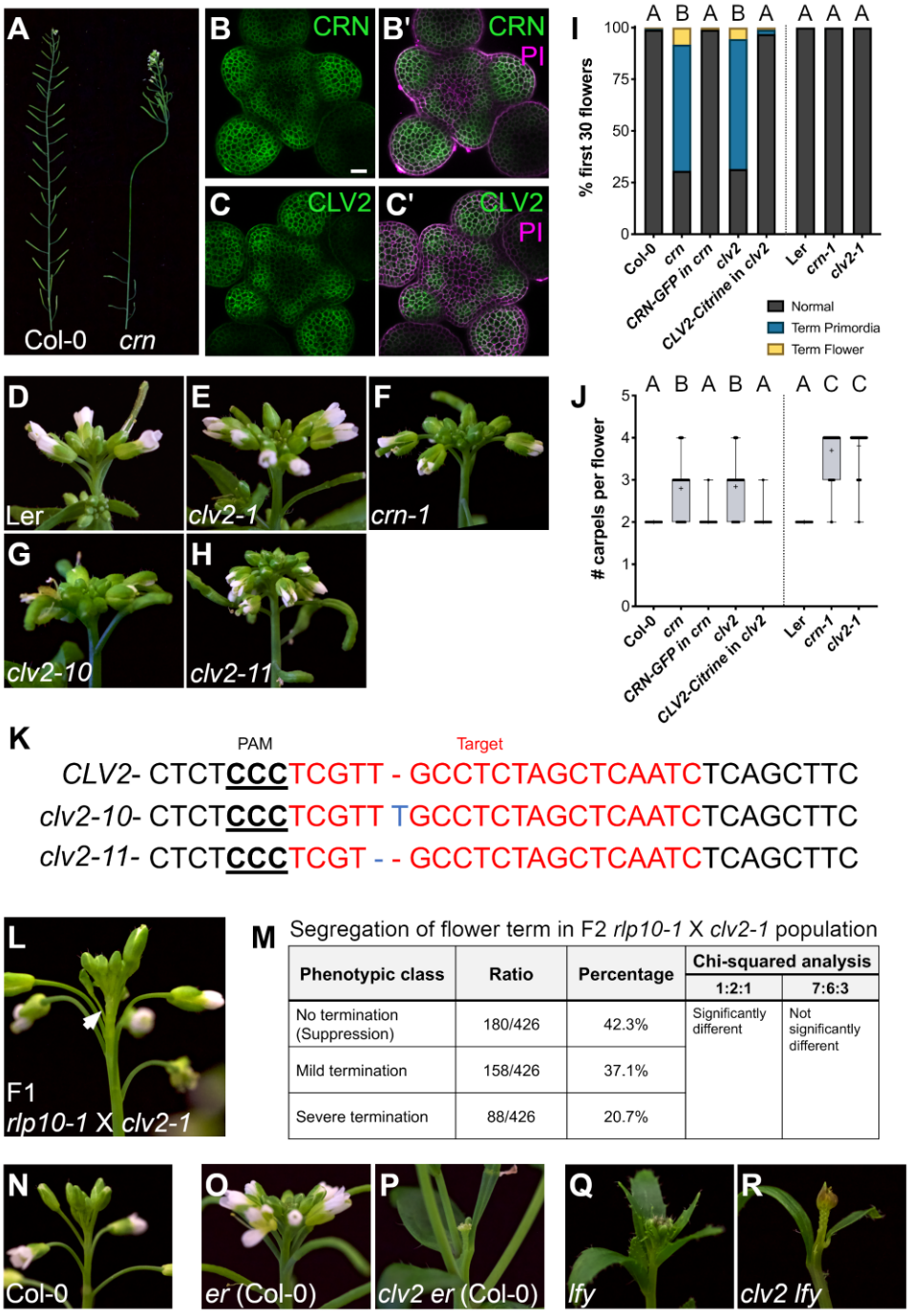
**Figure 4**


A
IM DEG - crn x WT
236
224
log2FC
Average logCPM
B
Gene Ontology (GO) term enrichment in IM DEG
specification of floral organ identity (GO:0010093)
auxin homeostasis (GO:0010252)
floral organ formation (GO:0048449)
specification of plant organ identity (GO:0090701)
maintenance of meristem identity (GO:0010074)
floral organ morphogenesis (GO:0048444)
auxin efflux (GO:0010315)
defense response to Gram-negative bacterium (GO:0050829)
leaf abscission (GO:0060866)
cotyledon morphogenesis (GO:0048826)
maintenance of inflorescence meristem identity (GO:0010077)
regulation of mitotic spindle organization (GO:0060236)
-log(p-value)
C
DR5
PI
C'
DR5 - MIP
Col-0
D
D'
crn
E
DII-Venus
L1
E'
DII
F
F'
G
16-18°C
% first 30 flowers
ANTpro::YUC1 in crn
H
22-24°C
Normal
Term Primordia
Term Flower
I
J
clv2 yuc1/4
# per plant
% plants pinning
Col
clv2
yuc1/4
Complete Flowers
% Pin Inflorescence

**Supplemental**




| Phenotypic class | Ratio | Percentage | Chi-squared analysis | |
|---|---|---|---|---|
| | | | 1:2:1 | 7:6:3 |
| No termination (Suppression) | 180/426 | 42.3% | Significantly different | Not significantly different |
| Mild termination | 158/426 | 37.1% | | |
| Severe termination | 88/426 | 20.7% | | |

**Figure S1. CLV2/CRN regulate primordia outgrowth in an ecotype-dependent manner. Related to Figure 1**

(A) Inflorescence architecture of Col-0 and *crn,* showing bare stem (flower termination) and recovery of *crn* compared to continuous flower production in Col-0. (B-C) *CRNpro::CRN-GFP* in

*crn* and *CLV2pro::CLV2-GFP* in *clv2* (*rlp10-1*) are expressed in developing flower primordia. PI (magenta) stained IM's of (B') *CRN-GFP* (green; n=6) and (C') *CLV2-Citrine* (green; n=5). (D-H) Inflorescences of *crn-1* and multiple *clv2* mutant alleles in the Ler background.(I-J) Quantification of (I) flower termination and (J) carpel number in Col-0 (n=16), *crn* (n=15), *CRNpro:CRN-GFP* in *crn* (n=16), *clv2* (*rlp10-1*) (n=15), *CLV2pro:CLV2-Citrine* in *clv2* (*rlp10-1,* n=16), Ler (n=18), *crn-1* (n=16) and *clv2-1* (n=14).(K) Novel CRISPR alleles of *clv2* in the Ler background. Target sequences are in red, edits (insertions and deletions) are shown in blue, and protospacer adjacent motif (PAM) is underlined. (L) Inflorescence of F1 hybrid from *clv2* (*rlp10-1* X *clv2-1*). Terminated primordia shown by arrow. (M) Segregation of flower termination defects in F2 mapping population of *rlp10-1* X *clv2-1*. (N-R) Inflorescences of (N) Col-0, (O) *erecta* (*er-105*), (P) c*lv2 er* (in the Col-0 background), (Q) *leafy* (*lfy-1*), and (R) *clv2 lfy* (in the Col-0 background). Scale bars, 20μm (B-C). Box and whisker plots show full range of data (min to max) with mean marked with "+". Statistical groupings based on significant differences found using Kruskal-Wallis and Dunn's multiple comparison test correction, significance defined as p-value < 0.05 (I-J).

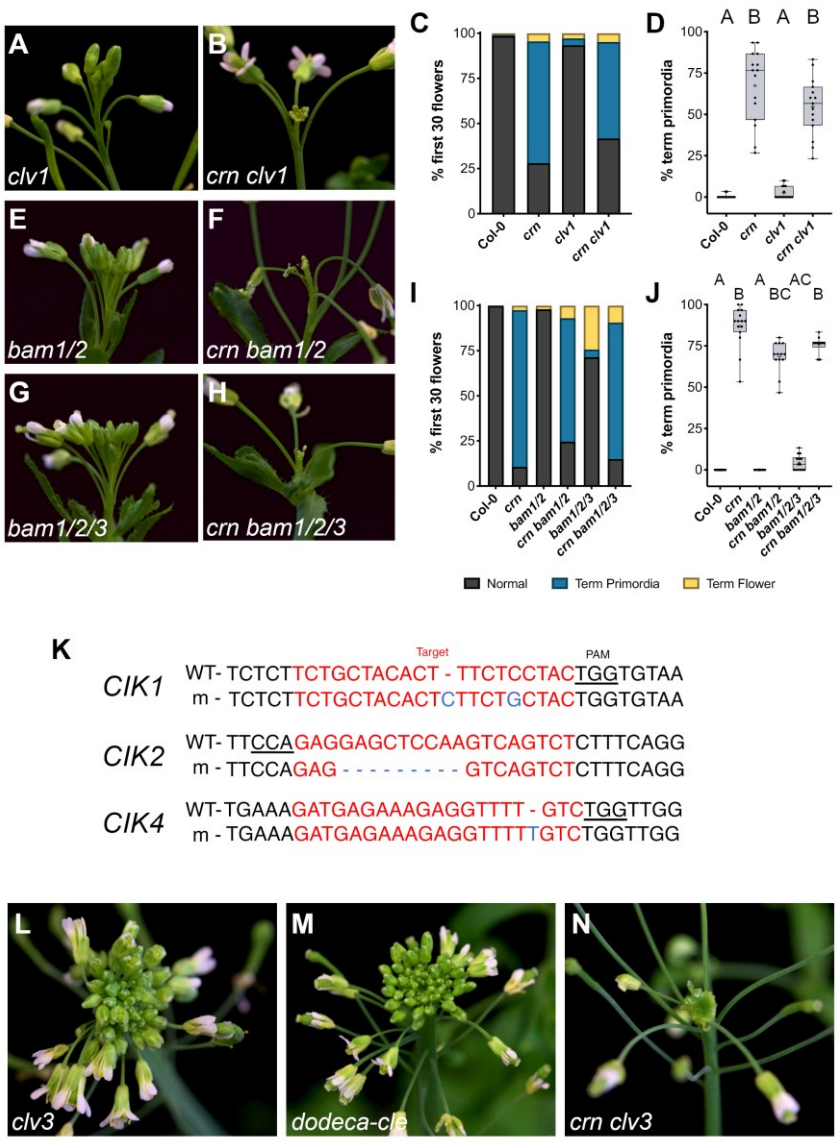



**Figure S2. CLV2/CRN-mediated flower primordia outgrowth is independent of CLV1/BAM receptors and CLV3 peptide signaling. Related to Figure 2**

(A-D) Inflorescences of (A) *clv1* (*clv1-101*) and (B) *crn clv1*. (C-D) Quantification of (C) flower primordia termination and (D) comparisons of termination percentages in Col-0 (n=15), *crn* (n=15), *clv1* (n=17), and *crn clv1* (n=15). (E-J) Inflorescences of (E) *bam1/2* (*bam1-4, bam2-4*), (F) *crn bam1/2*, (G) *bam1/2/3* (*bam1-4/bam2-4/bam3-2*) and (H) *crn bam1/2/3*. (I-J) Quantification of (I) flower primordia termination and (J) comparisons of primordia termination percentages in Col-0 (n=16), *crn* (n=15), *bam1/2* (n=10), *crn bam1/2* (n=13), *bam1/2/3* (n=14), *crn bam1/2/3* (n=10). (K) Novel CRISPR alleles of *cik1/2/4* (*cik1-3/cik2-3/cik4-3*). Sequences of wildtype (WT) and edited (m) genomic DNA of *CIK1, CIK2,* and *CIK3*. Target sequences are in red, edits (insertions and deletions) are shown in blue, and protospacer adjacent motif (PAM) is underlined. (L-N) Inflorescences of (L) *clv3* (*clv3-9*), (M) *dodeca-cle* (*clv3-9, cle9, cle10, cle11, cle12, cle13, cle18, cle19, cle20, cle21, cle22, cle45*) and (N) *crn clv3*. Box and whisker plots show full range of data (min to max) with mean marked with "+". Statistical groupings based on significant differences found using Kruskal-Wallis and Dunn's multiple comparison test correction, significance defined as p-value < 0.05 (D and J).

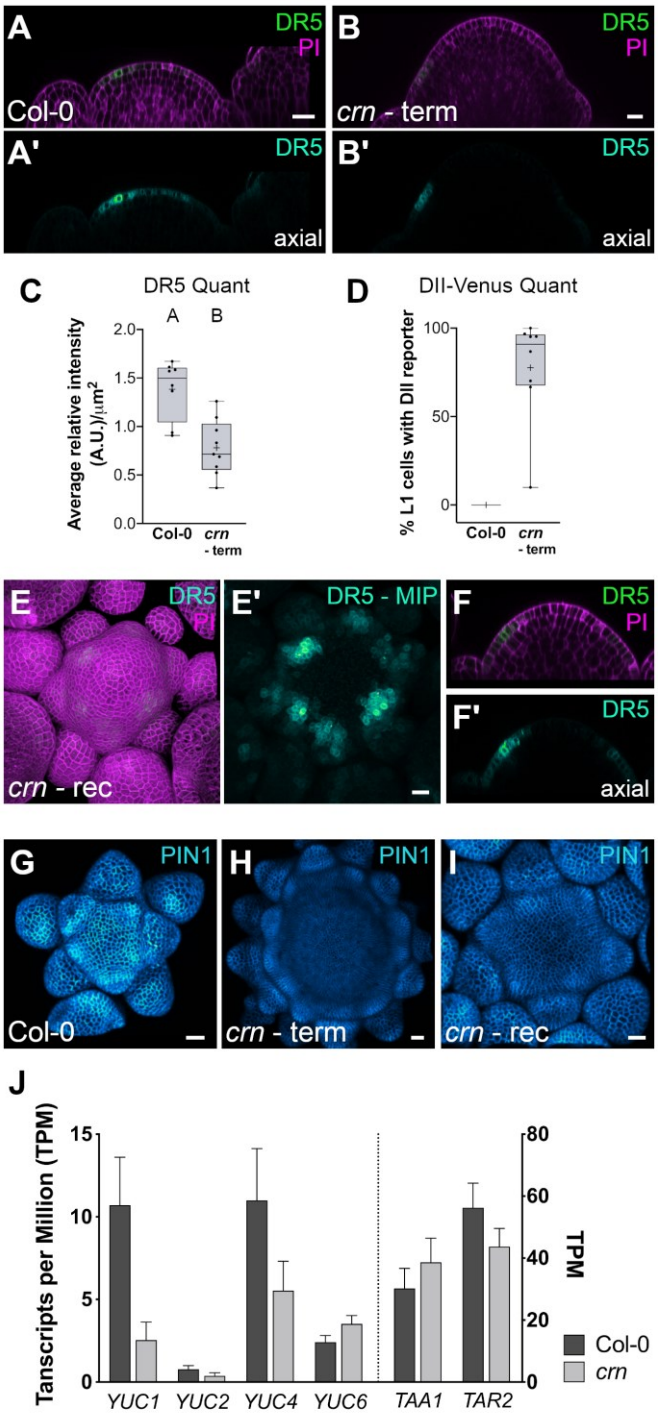



**Figure S3. Auxin signaling is reduced in the *crn* inflorescence meristem during the termination phase. Related to Figure 4**

(A) Axial view of Col-0 inflorescence meristem showing L1 expression of (A) *DR5::GFP* (green) co-labeled with PI (magenta) and (A') a heatmap of the *DR5::GFP* (teal) signal on its own. Section corresponds to the developing primordia that had the highest DR5 levels. (B) Axial view of *crn* inflorescence meristem with L1 expression of (B) *DR5::GFP* (green) co-labeled with PI (magenta)

and (B') a heatmap of the *DR5::GFP* (teal) signal on its own. Section corresponds to the developing primordia that had the highest DR5 levels. (C) Quantification of *DR5::GFP* levels in Col-0 (n=8) compared to terminated *crn* (n=9) IMs. (D) Quantification of *DII-Venus* in Col-0 (n=4) compared to terminated *crn* (n=8) IMs, using percentage of L1 layer cells with reporter for comparison. (E) Maximum intensity projection (MIP) of *DR5::GFP* (teal) in the inflorescence meristem of *crn* after floral recovery (n=4). Tissue stained with PI (magenta). (F) Axial view of recovered *crn* inflorescence meristem (*crn* - rec) with L1 expression of (F) *DR5::GFP* (green) co-labeled with PI (magenta) and (F') a heatmap of the *DR5::GFP* (teal) signal on its own. Section corresponds to the developing primordia that had the highest DR5 levels. (G-I) Maximum intensity projection (MIP) of *PIN1pro::PIN1-GFP* (blue heatmap) in the inflorescence meristem of: (G) Col-0 (n=16), (H) *crn* during termination (*crn* - term; n=19), (I) *crn* after floral recovery (*crn* - rec; n=5). (J) Expression levels of key auxin biosynthesis genes in Col-0 and *crn* IMs from RNAseq analysis. Genes included are *YUCCA* (*YUC*) *1/2/4/6*, *TRYPTOPHAN AMINOTRANSFERASE OF ARABIDOPSIS* (*TAA1*), and *TRYPTOPHAN AMINOTRANSFERASE RELATED 2* (*TAR2*). *YUC1/2/4/6* levels shown on left y-axis; *TAA1* and *TAR2* levels are shown on right y-axis. Box and whisker plots show full range of data (min to max) with mean marked with "+". Statistical grouping based on unpaired t-test with a p-value = 0.0007. Error bars = standard deviation. Scale bars, 20μm.

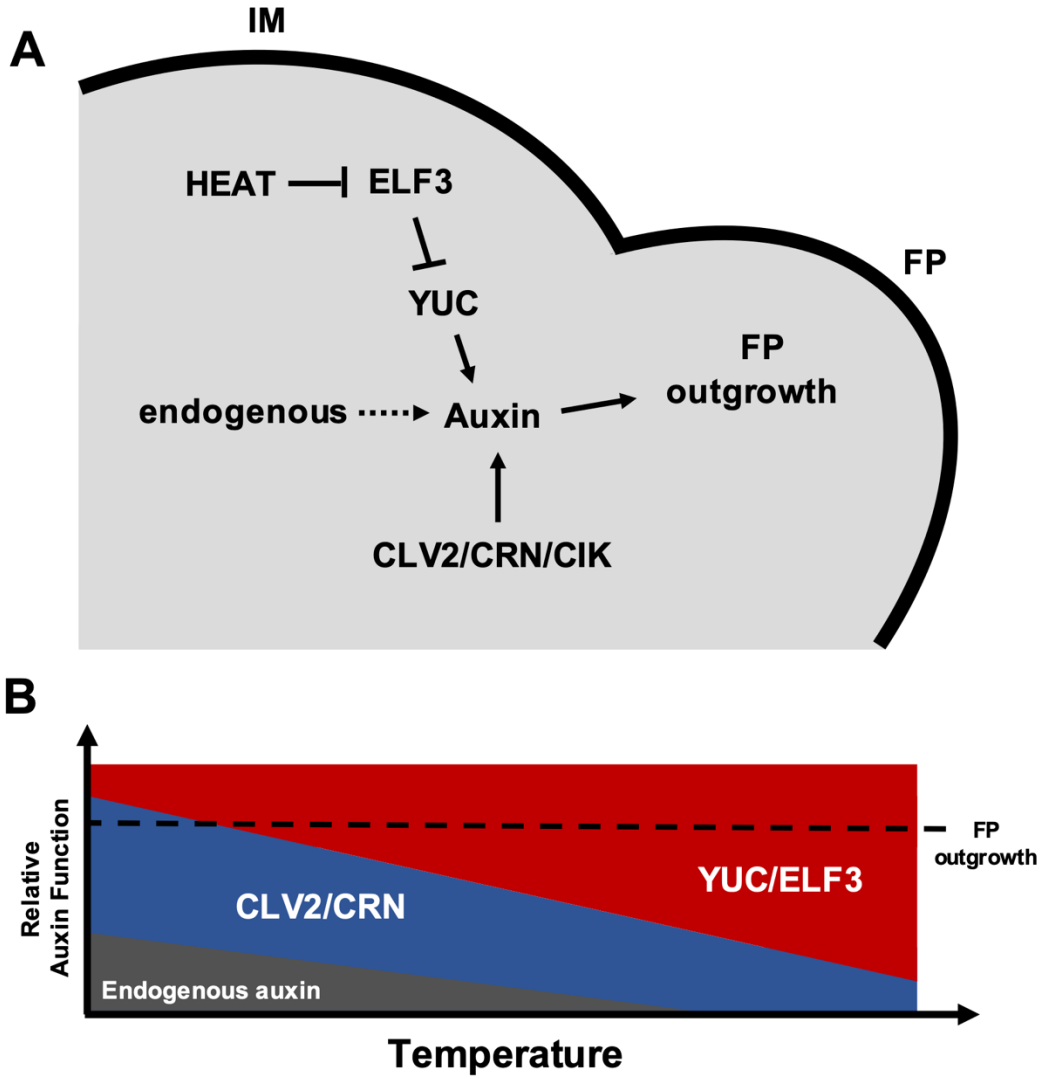



**Figure S4. Model of flower primordia outgrowth. Related to Figures 1, 3, and 4**
(A) Schematic of auxin-mediated flower primordia (FP) outgrowth where CLV2/CRN/CIK signaling and ELF3-regulated auxin production via YUC biosynthetic genes converge to regulate FP outgrowth. (B) Model depicting relative contributions of CLV2/CRN-signaling and ELF3/YUC towards auxin production in increasing temperatures. CLV2/CRN signaling is crucial at lower temperatures while ELF3/YUC is more important but functions synergistically with CLV2/CRN signaling at higher temperatures for auxin-dependent primordia development. Dotted line represents threshold required for floral primordia outgrowth.

GO Term Analysis

| GO Term | GO biological process | Annotation | Significant | Expected | over/under | fold Enrichment | P-value | FDR |
|---|---|---|---|---|---|---|---|---|
| GO:0010093 | specification of floral organ identity | 13 | 5 | 0.21 | + | 23.8 | 7.18E-06 | 9.14E-04 |
| GO:0060866 | leaf abscission | 8 | 3 | 0.13 | + | 23.21 | 5.99E-04 | 3.26E-02 |
| GO:0090701 | specification of plant organ identity | 14 | 5 | 0.23 | + | 22.1 | 9.62E-06 | 1.13E-03 |
| GO:0010077 | maintenance of inflorescence meristem identity | 9 | 3 | 0.15 | + | 20.63 | 7.90E-04 | 4.00E-02 |
| GO:0060236 | regulation of mitotic spindle organization | 10 | 3 | 0.16 | + | 18.57 | 1.01E-03 | 4.67E-02 |
| GO:0060237 | regulation of spindle organization | 10 | 3 | 0.16 | + | 18.57 | 1.01E-03 | 4.63E-02 |
| GO:0090224 | auxin efflux | 15 | 4 | 0.24 | + | 16.5 | 2.02E-04 | 1.41E-02 |
| GO:0048449 | floral organ formation | 25 | 6 | 0.4 | + | 14.85 | 8.21E-06 | 9.83E-04 |
| GO:0050829 | defense response to Gram-negative bacterium | 20 | 4 | 0.32 | + | 12.38 | 5.21E-04 | 2.94E-02 |
| GO:0048826 | cotyledon morphogenesis | 21 | 4 | 0.34 | + | 11.79 | 6.12E-04 | 3.30E-02 |
| GO:0010252 | auxin homeostasis | 39 | 7 | 0.63 | + | 11.11 | 7.68E-06 | 9.58E-04 |
| GO:0010074 | maintenance of meristem identity | 45 | 7 | 0.73 | + | 9.63 | 1.77E-05 | 1.86E-03 |
| GO:0048444 | floral organ morphogenesis | 45 | 7 | 0.73 | + | 9.63 | 1.77E-05 | 1.83E-03 |
| GO:0006342 | chromatin silencing | 35 | 5 | 0.57 | + | 8.84 | 4.14E-04 | 2.43E-02 |
| GO:0019827 | stem cell population maintenance | 55 | 7 | 0.89 | + | 7.88 | 5.67E-05 | 4.65E-03 |
| GO:0010374 | stomatal complex development | 55 | 7 | 0.89 | + | 7.88 | 5.67E-05 | 4.59E-03 |
| GO:0098727 | maintenance of cell number | 55 | 7 | 0.89 | + | 7.88 | 5.67E-05 | 4.53E-03 |
| GO:0045814 | negative regulation of gene expression, epigenetic | 41 | 5 | 0.66 | + | 7.55 | 7.97E-04 | 4.01E-02 |
| GO:0010928 | regulation of auxin mediated signaling pathway | 42 | 5 | 0.68 | + | 7.37 | 8.80E-04 | 4.15E-02 |
| GO:0048481 | plant ovule development | 53 | 6 | 0.86 | + | 7.01 | 3.45E-04 | 2.13E-02 |
| GO:0009914 | hormone transport | 90 | 10 | 1.45 | + | 6.88 | 4.52E-06 | 6.14E-04 |
| GO:0035670 | plant-type ovary development | 55 | 6 | 0.89 | + | 6.75 | 4.15E-04 | 2.41E-02 |
| GO:0048825 | cotyledon development | 55 | 6 | 0.89 | + | 6.75 | 4.15E-04 | 2.38E-02 |
| GO:0060918 | auxin transport | 87 | 9 | 1.41 | + | 6.4 | 2.29E-05 | 2.21E-03 |
| GO:0009926 | auxin polar transport | 69 | 7 | 1.11 | + | 6.28 | 2.08E-04 | 1.40E-02 |
| GO:0009965 | leaf morphogenesis | 93 | 9 | 1.5 | + | 5.99 | 3.72E-05 | 3.32E-03 |
| GO:0090698 | post-embryonic plant morphogenesis | 198 | 19 | 3.2 | + | 5.94 | 2.31E-09 | 1.15E-06 |
| GO:0048440 | carpel development | 76 | 7 | 1.23 | + | 5.7 | 3.60E-04 | 2.20E-02 |
| GO:0010016 | shoot system morphogenesis | 170 | 15 | 2.75 | + | 5.46 | 3.08E-07 | 8.00E-05 |
| GO:0009908 | flower development | 354 | 30 | 5.72 | + | 5.24 | 9.63E-13 | 1.44E-09 |
| GO:0090567 | reproductive shoot system development | 369 | 31 | 5.96 | + | 5.2 | 4.93E-13 | 9.83E-10 |
| GO:0003002 | regionalization | 155 | 13 | 2.5 | + | 5.19 | 3.18E-06 | 5.14E-04 |
| GO:0048467 | gynoecium development | 89 | 7 | 1.44 | + | 4.87 | 8.66E-04 | 4.15E-02 |
| GO:0048437 | floral organ development | 243 | 19 | 3.93 | + | 4.84 | 4.96E-08 | 1.41E-05 |
| GO:0006928 | movement of cell or subcellular component | 91 | 7 | 1.47 | + | 4.76 | 9.79E-04 | 4.54E-02 |
| GO:0010073 | meristem maintenance | 145 | 11 | 2.34 | + | 4.69 | 4.29E-05 | 3.72E-03 |
| GO:0007389 | pattern specification process | 188 | 14 | 3.04 | + | 4.61 | 4.87E-06 | 6.48E-04 |
| GO:0048438 | floral whorl development | 193 | 14 | 3.12 | + | 4.49 | 6.46E-06 | 8.40E-04 |
| GO:1905393 | plant organ formation | 111 | 8 | 1.79 | + | 4.46 | 6.47E-04 | 3.37E-02 |
| GO:0009909 | regulation of flower development | 167 | 12 | 2.7 | + | 4.45 | 3.20E-05 | 2.90E-03 |
| GO:0090697 | post-embryonic plant organ morphogenesis | 117 | 8 | 1.89 | + | 4.23 | 8.94E-04 | 4.18E-02 |
| GO:0048367 | shoot system development | 724 | 49 | 11.7 | + | 4.19 | 2.04E-16 | 1.22E-12 |
| GO:0048507 | meristem development | 217 | 14 | 3.51 | + | 3.99 | 2.23E-05 | 2.19E-03 |
| GO:0048646 | anatomical structure formation involved in morphogenesis | 204 | 13 | 3.3 | + | 3.94 | 4.90E-05 | 4.19E-03 |
| GO:0048831 | regulation of shoot system development | 268 | 17 | 4.33 | + | 3.93 | 3.73E-06 | 5.44E-04 |
| GO:0048827 | phyllome development | 492 | 31 | 7.95 | + | 3.9 | 4.46E-10 | 2.96E-07 |
| GO:0001666 | response to hypoxia | 261 | 16 | 4.22 | + | 3.79 | 1.07E-05 | 1.23E-03 |
| GO:0045944 | positive regulation of transcription by RNA polymerase II | 214 | 13 | 3.46 | + | 3.76 | 7.76E-05 | 6.03E-03 |
| GO:0036293 | response to decreased oxygen levels | 265 | 16 | 4.28 | + | 3.74 | 1.28E-05 | 1.42E-03 |
| GO:0070482 | response to oxygen levels | 266 | 16 | 4.3 | + | 3.72 | 1.34E-05 | 1.46E-03 |
| GO:0046777 | protein autophosphorylation | 188 | 11 | 3.04 | + | 3.62 | 3.68E-04 | 2.18E-02 |
| GO:0048366 | leaf development | 332 | 19 | 5.36 | + | 3.54 | 4.19E-06 | 5.83E-04 |
| GO:0090696 | post-embryonic plant organ development | 176 | 10 | 2.84 | + | 3.52 | 8.34E-04 | 4.12E-02 |
| GO:0010817 | regulation of hormone levels | 249 | 14 | 4.02 | + | 3.48 | 9.11E-05 | 6.99E-03 |
| GO:0071456 | cellular response to hypoxia | 235 | 13 | 3.8 | + | 3.42 | 1.87E-04 | 1.33E-02 |
| GO:0071453 | cellular response to oxygen levels | 237 | 13 | 3.83 | + | 3.39 | 2.03E-04 | 1.39E-02 |
| GO:0036294 | cellular response to decreased oxygen levels | 237 | 13 | 3.83 | + | 3.39 | 2.03E-04 | 1.38E-02 |
| GO:0009888 | tissue development | 605 | 31 | 9.78 | + | 3.17 | 4.35E-08 | 1.30E-05 |
| GO:1905392 | plant organ morphogenesis | 403 | 20 | 6.51 | + | 3.07 | 1.73E-05 | 1.85E-03 |
| GO:0014070 | response to organic cyclic compound | 287 | 14 | 4.64 | + | 3.02 | 3.66E-04 | 2.19E-02 |
| GO:0009733 | response to auxin | 299 | 14 | 4.83 | + | 2.9 | 5.40E-04 | 2.99E-02 |
| GO:0009653 | anatomical structure morphogenesis | 886 | 41 | 14.32 | + | 2.86 | 4.47E-09 | 1.91E-06 |
| GO:0099402 | plant organ development | 996 | 44 | 16.09 | + | 2.73 | 4.48E-09 | 1.79E-06 |
| GO:0048580 | regulation of post-embryonic development | 395 | 17 | 6.38 | + | 2.66 | 3.65E-04 | 2.21E-02 |
| GO:2000241 | regulation of reproductive process | 374 | 16 | 6.04 | + | 2.65 | 5.73E-04 | 3.15E-02 |
| GO:0006357 | regulation of transcription by RNA polymerase II | 430 | 18 | 6.95 | + | 2.59 | 3.41E-04 | 2.12E-02 |
| GO:1902680 | positive regulation of RNA biosynthetic process | 521 | 21 | 8.42 | + | 2.49 | 1.84E-04 | 1.35E-02 |
| GO:1903508 | positive regulation of nucleic acid-templated transcription | 521 | 21 | 8.42 | + | 2.49 | 1.84E-04 | 1.33E-02 |
| GO:0048608 | reproductive structure development | 1198 | 48 | 19.36 | + | 2.48 | 1.79E-08 | 6.69E-06 |
| GO:0061458 | reproductive system development | 1200 | 48 | 19.39 | + | 2.48 | 1.87E-08 | 6.58E-06 |
| GO:2000026 | regulation of multicellular organismal development | 568 | 22 | 9.18 | + | 2.4 | 2.94E-04 | 1.91E-02 |
| GO:0010557 | positive regulation of macromolecule biosynthetic process | 569 | 22 | 9.19 | + | 2.39 | 2.98E-04 | 1.89E-02 |
| GO:0045893 | positive regulation of transcription, DNA-templated | 519 | 20 | 8.39 | + | 2.38 | 6.15E-04 | 3.29E-02 |
| GO:0042592 | homeostatic process | 522 | 20 | 8.43 | + | 2.37 | 6.40E-04 | 3.39E-02 |
| GO:0051254 | positive regulation of RNA metabolic process | 550 | 21 | 8.89 | + | 2.36 | 4.66E-04 | 2.65E-02 |
| GO:0031328 | positive regulation of cellular biosynthetic process | 603 | 23 | 9.74 | + | 2.36 | 2.39E-04 | 1.59E-02 |
| GO:0009891 | positive regulation of biosynthetic process | 620 | 23 | 10.02 | + | 2.3 | 3.22E-04 | 2.03E-02 |
| GO:0045935 | positive regulation of nucleobase-containing compound metabolic p | 570 | 21 | 9.21 | + | 2.28 | 6.46E-04 | 3.39E-02 |
| GO:0009791 | post-embryonic development | 1472 | 54 | 23.79 | + | 2.27 | 3.87E-08 | 1.22E-05 |
| GO:0051173 | positive regulation of nitrogen compound metabolic process | 739 | 27 | 11.94 | + | 2.26 | 1.67E-04 | 1.23E-02 |
| GO:0048731 | system development | 1849 | 67 | 29.88 | + | 2.24 | 1.09E-09 | 5.92E-07 |
| GO:0003006 | developmental process involved in reproduction | 1427 | 51 | 23.06 | + | 2.21 | 2.66E-07 | 7.25E-05 |
| GO:0051239 | regulation of multicellular organismal process | 625 | 22 | 10.1 | + | 2.18 | 1.09E-03 | 4.88E-02 |
| GO:0050793 | regulation of developmental process | 781 | 27 | 12.62 | + | 2.14 | 2.94E-04 | 1.89E-02 |
| GO:0031325 | positive regulation of cellular metabolic process | 797 | 27 | 12.88 | + | 2.1 | 5.23E-04 | 2.93E-02 |
| GO:0010604 | positive regulation of macromolecule metabolic process | 771 | 26 | 12.46 | + | 2.09 | 7.31E-04 | 3.74E-02 |
| GO:0065008 | regulation of biological quality | 955 | 32 | 15.43 | + | 2.07 | 1.42E-04 | 1.08E-02 |
| GO:0009725 | response to hormone | 1237 | 41 | 19.99 | + | 2.05 | 2.66E-05 | 2.45E-03 |
| GO:0009719 | response to endogenous stimulus | 1274 | 42 | 20.59 | + | 2.04 | 2.12E-05 | 2.15E-03 |
| GO:0048856 | anatomical structure development | 2824 | 93 | 45.63 | + | 2.04 | 6.09E-11 | 5.21E-08 |
| GO:0007275 | multicellular organism development | 2436 | 80 | 39.36 | + | 2.03 | 2.53E-09 | 1.16E-06 |

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| GO Term | GO biological process | Annotation | Significant | Expected | over/under | fold Enrichment | P-value | FDR |
|---|---|---|---|---|---|---|---|---|
| GO:0006468 | protein phosphorylation | 1037 | 34 | 16.76 | + | 2.03 | 1.45E-04 | 1.09E-02 |
| GO:0048522 | positive regulation of cellular process | 1055 | 34 | 17.05 | + | 1.99 | 2.67E-04 | 1.75E-02 |
| GO:0070887 | cellular response to chemical stimulus | 1088 | 35 | 17.58 | + | 1.99 | 2.01E-04 | 1.41E-02 |
| GO:0010033 | response to organic substance | 1711 | 55 | 27.65 | + | 1.99 | 1.77E-06 | 3.91E-04 |
| GO:0022414 | reproductive process | 1714 | 55 | 27.7 | + | 1.99 | 1.83E-06 | 3.79E-04 |
| GO:0000003 | reproduction | 1724 | 55 | 27.86 | + | 1.97 | 2.86E-06 | 5.04E-04 |
| GO:0032502 | developmental process | 2979 | 93 | 48.14 | + | 1.93 | 8.62E-10 | 5.16E-07 |
| GO:0009605 | response to external stimulus | 1508 | 47 | 24.37 | + | 1.93 | 2.42E-05 | 2.30E-03 |
| GO:1901700 | response to oxygen-containing compound | 1511 | 47 | 24.42 | + | 1.93 | 2.50E-05 | 2.34E-03 |
| GO:0032501 | multicellular organismal process | 2711 | 83 | 43.81 | + | 1.89 | 2.37E-08 | 7.89E-06 |
| GO:0043207 | response to external biotic stimulus | 1092 | 33 | 17.65 | + | 1.87 | 8.62E-04 | 4.20E-02 |
| GO:0051707 | response to other organism | 1092 | 33 | 17.65 | + | 1.87 | 8.62E-04 | 4.16E-02 |
| GO:0009607 | response to biotic stimulus | 1093 | 33 | 17.66 | + | 1.87 | 8.68E-04 | 4.12E-02 |
| GO:0006355 | regulation of transcription, DNA-templated | 2158 | 64 | 34.87 | + | 1.84 | 3.42E-06 | 5.38E-04 |
| GO:1903506 | regulation of nucleic acid-templated transcription | 2158 | 64 | 34.87 | + | 1.84 | 3.42E-06 | 5.24E-04 |
| GO:2001141 | regulation of RNA biosynthetic process | 2160 | 64 | 34.9 | + | 1.83 | 3.48E-06 | 5.21E-04 |
| GO:0016310 | phosphorylation | 1216 | 36 | 19.65 | + | 1.83 | 6.87E-04 | 3.55E-02 |
| GO:2000112 | regulation of cellular macromolecule biosynthetic process | 2340 | 69 | 37.81 | + | 1.82 | 2.08E-06 | 4.02E-04 |
| GO:0044419 | interspecies interaction between organisms | 1120 | 33 | 18.1 | + | 1.82 | 1.09E-03 | 4.94E-02 |
| GO:0010556 | regulation of macromolecule biosynthetic process | 2353 | 69 | 38.02 | + | 1.81 | 2.22E-06 | 4.16E-04 |
| GO:0019219 | regulation of nucleobase-containing compound metabolic process | 2320 | 68 | 37.49 | + | 1.81 | 2.92E-06 | 4.85E-04 |
| GO:0031326 | regulation of cellular biosynthetic process | 2451 | 71 | 39.6 | + | 1.79 | 2.28E-06 | 4.13E-04 |
| GO:0009889 | regulation of biosynthetic process | 2488 | 72 | 40.2 | + | 1.79 | 1.81E-06 | 3.88E-04 |
| GO:0051252 | regulation of RNA metabolic process | 2260 | 65 | 36.52 | + | 1.78 | 8.13E-06 | 9.93E-04 |
| GO:0007165 | signal transduction | 1330 | 38 | 21.49 | + | 1.77 | 8.22E-04 | 4.10E-02 |
| GO:0031323 | regulation of cellular metabolic process | 2956 | 84 | 47.76 | + | 1.76 | 4.50E-07 | 1.08E-04 |
| GO:0060255 | regulation of macromolecule metabolic process | 3030 | 86 | 48.96 | + | 1.76 | 3.95E-07 | 9.86E-05 |
| GO:0050794 | regulation of cellular process | 4726 | 134 | 76.36 | + | 1.75 | 2.97E-11 | 2.96E-08 |
| GO:0051171 | regulation of nitrogen compound metabolic process | 2722 | 77 | 43.98 | + | 1.75 | 2.02E-06 | 4.03E-04 |
| GO:0042221 | response to chemical | 2682 | 75 | 43.34 | + | 1.73 | 3.92E-06 | 5.59E-04 |
| GO:0065007 | biological regulation | 5956 | 166 | 96.24 | + | 1.72 | 9.50E-14 | 2.84E-10 |
| GO:0080090 | regulation of primary metabolic process | 2806 | 78 | 45.34 | + | 1.72 | 2.87E-06 | 4.91E-04 |
| GO:0010468 | regulation of gene expression | 2648 | 73 | 42.79 | + | 1.71 | 1.09E-05 | 1.23E-03 |
| GO:0019222 | regulation of metabolic process | 3302 | 91 | 53.36 | + | 1.71 | 4.96E-07 | 1.14E-04 |
| GO:0050789 | regulation of biological process | 5374 | 148 | 86.84 | + | 1.7 | 1.46E-11 | 1.75E-08 |
| GO:0051716 | cellular response to stimulus | 2521 | 69 | 40.74 | + | 1.69 | 2.20E-05 | 2.19E-03 |
| GO:0007154 | cell communication | 1618 | 44 | 26.14 | + | 1.68 | 1.09E-03 | 4.91E-02 |
| GO:0050896 | response to stimulus | 5567 | 149 | 89.95 | + | 1.66 | 9.48E-11 | 7.09E-08 |
| GO:0006950 | response to stress | 3090 | 79 | 49.93 | + | 1.58 | 5.67E-05 | 4.71E-03 |
| GO:0009987 | cellular process | 11959 | 237 | 193.24 | + | 1.23 | 3.89E-05 | 3.42E-03 |
| GO:0016070 | RNA metabolic process | 1293 | 7 | 20.89 | - | 0.34 | 8.59E-04 | 4.21E-02 |
| GO:0010467 | gene expression | 1601 | 8 | 25.87 | - | 0.31 | 5.35E-05 | 4.51E-03 |
| GO:0006396 | RNA processing | 785 | 1 | 12.68 | - | 0.08 | 6.77E-05 | 5.33E-03 |

**Table S2. Full Gene Ontology terms list of top 460 DEGs in *crn* lMs. Related to Figure 4**
The number of significant genes in our DEG list (p-value < 0.001) out of the annotated A. thaliana reference list from GO panther was used to calculate fold-enrichment, p-value and FDR. Red, green and blue highlighted GO processes indicate auxin, meristem and floral related genes, respectively, detailed further in Table S1.

| Auxin Biosyntheric Genes TPM Count | | | | | |
|---|---|---|---|---|---|
| **Gene Name** | **ATG - isoform** | **Col-0 TPM** | ***crn* TPM** | **p-value** | **q-value** |
| YUC1 | AT4G32540.1 | 9.62163605 | 2.22777304 | 0.000150561 | 0.011150848 |
| YUC1 | AT4G32540.2 | 1.08021526 | 0.30754248 | 0.066447724 | 0.246747862 |
| YUC2 | AT4G13260.1 | 0.77819885 | 0.3726434 | 0.12364161 | 0.313064639 |
| YUC3 | AT1G04610.1 | 0.05658348 | 0.05274052 | 0.542276947 | 0.648222557 |
| YUC4 | AT5G11320.1 | 9.47973542 | 4.69456935 | 0.00471094 | 0.062519474 |
| YUC4 | AT5G11320.2 | 1.51754126 | 0.83461118 | 0.044222831 | 0.204729205 |
| YUC5 | AT5G43890.1 | 0.00390573 | 0 | - | - |
| YUC6 | AT5G25620.1 | 2.41963745 | 3.5137845 | 0.054387715 | 0.226726537 |
| YUC7 | AT2G33230.1 | 0.0058149 | 0 | - | - |
| YUC8 | AT4G28720.1 | 0.07420622 | 0.02084606 | - | - |
| YUC9 | AT1G04180.1 | 0.01755266 | 0.00867234 | - | - |
| YUC10 | AT1G48910.1 | 0.01773398 | 0 | - | - |
| YUC11 | AT1G21430.1 | 0 | 0 | - | - |
| TAA1 | AT1G70560.1 | 30.226102 | 38.6519243 | 0.213430557 | 0.388882748 |
| TAR2 | AT4G24670.1 | 9.30982625 | 5.82646658 | 0.065158626 | 0.244976736 |
| TAR2 | AT4G24670.2 | 46.8980927 | 37.8726915 | 0.020707299 | 0.14162109 |
| AMI1 | AT1G08980.1 | 38.6695782 | 61.3469622 | 0.00102772 | 0.027829535 |
| RHM3 | AT3G14790.1 | 32.7962069 | 25.4270094 | 0.000729834 | 0.023526653 |
| ASB1 | AT1G25220.1 | 36.6762453 | 34.5094303 | 0.096033747 | 0.286370868 |
| ASB1 | AT1G25220.2 | 0.02064712 | 0.05240687 | - | - |
| VAS1 | AT1G80360.1 | 20.8728462 | 13.2519822 | 0.006027497 | 0.07158872 |
| VAS1 | AT1G80360.2 | 9.6028417 | 4.83430674 | 0.008223945 | 0.085497555 |
| VAS1 | AT1G80360.3 | 2.59071657 | 2.7720062 | 0.388299567 | 0.529284096 |
| VAS1 | AT1G80360.4 | 10.1510957 | 9.71721332 | 0.566011827 | 0.666634099 |
| AAO1 | AT5G20960.1 | 0.85296647 | 0.92154705 | 1 | 1 |
| AAO1 | AT5G20960.2 | 3.28165823 | 2.49653366 | 0.181870638 | 0.363877752 |

**Table S3. Average TPM and statistics of auxin biosynthetic genes in Col-0 and *crn* IMs. Related to Figure 4**

Grey boxes show *YUCCA* biosynthetic genes, light grey boxes show *TAA* family genes and white for other auxin biosynthetic genes.