

Comprehensive overview of plant development: Morphogenesis above ground

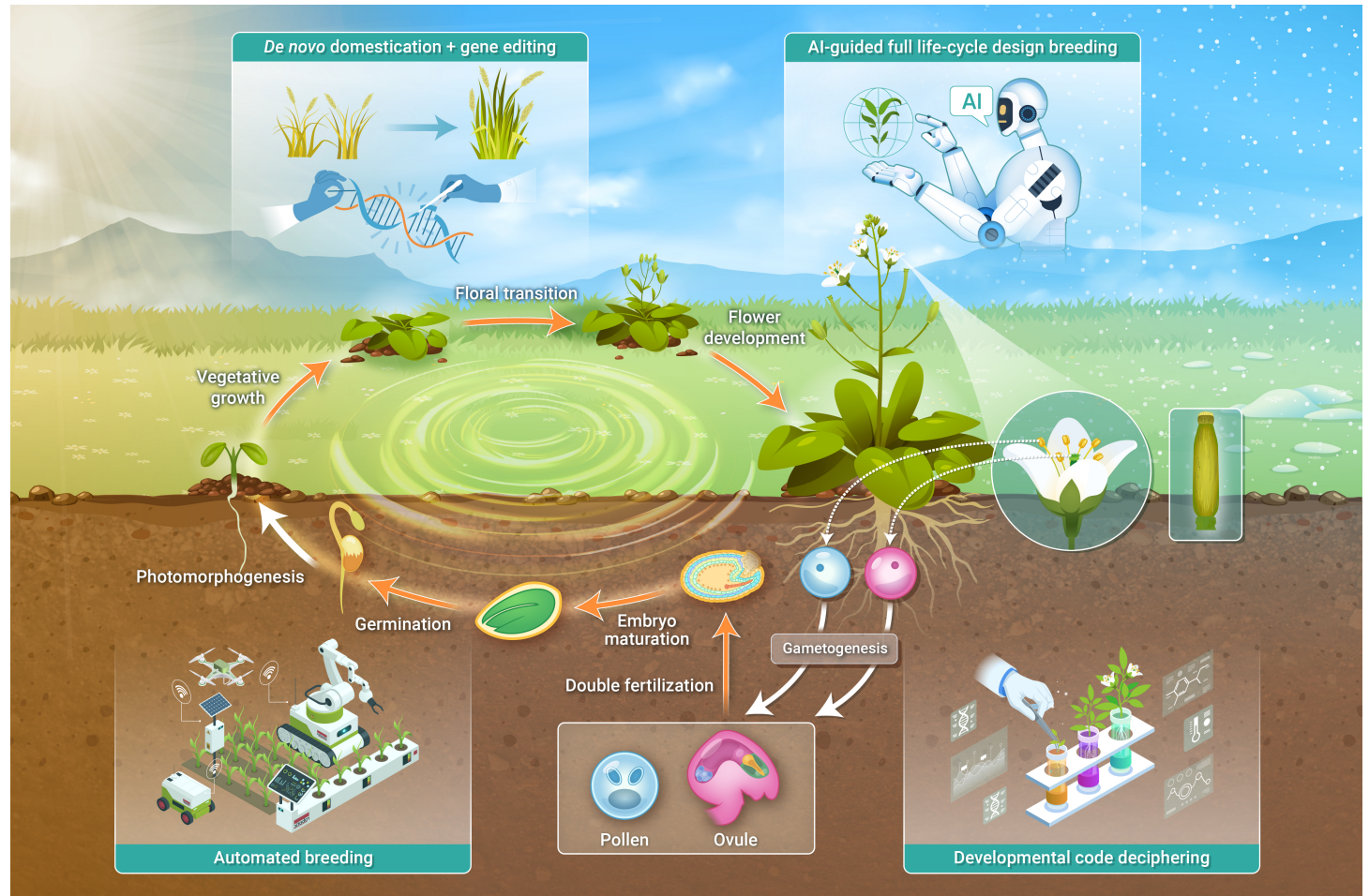
Xu Li,^{1,*} Ruoyan Liu,^{2,7,*} Shu-Yan Chen,^{2,*} Xiuchen Weng,^{3,*} Hongyan Shan,^{4,5,6,*} Hao-Ran Sun,^{4,5,6,*} Shuixian Li,^{4,5,6,*} Ming Luo,⁸ Steven M. Smith,⁹ Jiayang Li,^{2,10} Caihuan Tian,^{11,*} Hongtao Liu,^{1,*} Yang Zhu,^{3,*} Yang Dong,^{4,5,6,7,*} Hongzhi Kong,^{4,5,6,7,*} Hong-Ju Li,^{2,*} and Bing Wang^{2,*}

*Correspondence: tiancai@caas.cn (C.T.); htliu@szu.edu.cn (H.L.); zhuyang2020@zju.edu.cn (Y.Z.); yang.dong@ibcas.ac.cn (Y.D.); hzkong@ibcas.ac.cn (H.K.); hjli@genetics.ac.cn (H-J.L.); bingwang@genetics.ac.cn (B.W.)

Received: June 8, 2025; Accepted: November 12, 2025; Published Online: November 18, 2025; <https://doi.org/10.59717/j.xinn-life.2025.100172>

© 2025 The Author(s). This is an open access article under the CC BY license (<https://creativecommons.org/licenses/by/4.0/>).

GRAPHICAL ABSTRACT



PUBLIC SUMMARY

- Plant development determines agronomic traits.
- Plant development is the basis for domestication.
- Whole life-cycle plant development illustration facilitates smart breeding and intelligent cultivation.

Comprehensive overview of plant development: Morphogenesis above ground

Xu Li,^{1,*} Ruoyan Liu,^{2,7,*} Shu-Yan Chen,^{2,*} Xiuchen Weng,^{3,*} Hongyan Shan,^{4,5,6,*} Hao-Ran Sun,^{4,5,6,*} Shuixian Li,^{4,5,6,*} Ming Luo,⁸ Steven M. Smith,⁹ Jiayang Li,^{2,10} Caihuan Tian,^{11,*} Hongtao Liu,^{1,*} Yang Zhu,^{3,*} Yang Dong,^{4,5,6,7,*} Hongzhi Kong,^{4,5,6,7,*} Hong-Ju Li,^{2,*} and Bing Wang^{2,*}

¹College of Life Sciences and Oceanography, Shenzhen University, Shenzhen 518060, China

²State Key Laboratory of Seed Innovation, Institute of Genetics and Developmental Biology, Chinese Academy of Sciences, Beijing 100101, China

³Institute of Crop Science, Zhejiang University, Hangzhou 310058, China

⁴State Key Laboratory of Plant Diversity and Specialty Crops, Institute of Botany, Chinese Academy of Sciences, Beijing 100093, China

⁵Key Laboratory of Systematic and Evolutionary Botany, Institute of Botany, Chinese Academy of Sciences, Beijing 100093, China

⁶China National Botanical Garden, Beijing 100093, China

⁷University of Chinese Academy of Sciences, Beijing 100049, China

⁸South China Botanical Garden, Chinese Academy of Sciences, Guangzhou 510650, China

⁹School of Natural Sciences, University of Tasmania, Hobart 7001, Australia

¹⁰Yazhouwan National Laboratory, Sanya 572024, China

¹¹State Key Laboratory of Vegetable Biobreeding, Institute of Vegetables and Flowers, Chinese Academy of Agricultural Sciences, Beijing 100081, China

*These authors contributed equally

*Correspondence: tiancaihuan@caas.cn (C.T.); htliu@szu.edu.cn (H.L.); zhuyang2020@zju.edu.cn (Y.Z.); yang.dong@ibcas.ac.cn (Y.D.); hzkong@ibcas.ac.cn (H.K.);

hjl@genetics.ac.cn (H-J.L.); bingwang@genetics.ac.cn (B.W.)

Received: June 8, 2025; Accepted: November 12, 2025; Published Online: November 18, 2025; <https://doi.org/10.59717/j.xinn-life.2025.100172>

© 2025 The Author(s). This is an open access article under the CC BY license (<https://creativecommons.org/licenses/by/4.0/>).

Citation: Li X., Liu R., Chen S.-Y., et al. (2025). Comprehensive overview of plant development: Morphogenesis above ground. *The Innovation Life* 3:100172.

Plant development is a serial and dynamic process that encompasses various stages, from embryogenesis to senescence, influenced by both genetic and environmental factors. This review provides an in-depth exploration of the mechanisms underlying plant growth and development, highlighting key morphogenesis processes such as photomorphogenesis, plant growth, shoot branching, floral transition, flower development, fruit development and reproductivity specification. We delve into the molecular genetics of plant development, focusing on regulator and signaling pathways that govern critical developmental events. Furthermore, we discuss the role of phytohormones, including auxins, cytokinins, gibberellins, abscisic acid, and ethylene, in regulating developmental transitions. The interaction between plants and their environment, particularly light, temperature, and nutrient availability, is also examined, emphasizing how these external cues impact developmental pathways. Overall, this comprehensive overview offers insights into the intricate interplay between genetic programs and environmental stimuli in shaping plant architecture and life cycle, which will facilitate smart breeding and intelligent cultivation.

INTRODUCTION

Plant development embodies one of nature's most profound and intricate processes, where a minute seed unfurls into a magnificent organism capable of sustaining life on Earth. This extraordinary process, termed ontogeny, encompasses germination, growth, differentiation of tissues, organ formation, and ultimately reproduction. It reflects a harmonious interplay between genetics, hormonal signaling, and environmental responses, each playing a pivotal role in shaping the plant's form and function.¹

The tale begins with germination, a critical transition from dormancy to developmental activity. A seed equipped with stored nutrients and a protective coat awaits the optimal combination of moisture, temperature, and in some cases, light to break its slumber. Once triggered, enzymes degrade storage molecules, providing energy for the emerging radicle, the embryonic root. Concurrently, the plumule, which will become the shoot, perceives light signals and push upwards to complete photomorphogenesis. This initial phase sets the stage for subsequent developmental events, highlighting the plant's remarkable sensitivity to environmental stimuli.

Following germination, plants enter postembryonic stages, in which all the organs and tissues iteratively occurs from shoot apical meristem (SAM), a reservoir of meristematic cells that nestled at the apex of growing stems and branches that drives vegetative growth and architectural determination.² The SAM orchestrates the continuous production of new organs, including leaves, flowers, fruits, and lateral branches, and the upward extension of the plant body, each meticulously positioned and timed according to the plant's

genetic blueprint and environmental cues, thereby shaping the plant's overall form and stature.^{3,4}

Notably, among the produced organs, flowers and fruits are unique to angiosperms (i.e., flowering plants) and represent key innovations in the history of plant evolution.^{5,6} From a developmental point of view, flowering marks the transition from vegetative growth to reproductive development.⁷ The remarkable diversity of flowers and fruits not only significantly improves the reproductive efficiency of flowering plants, but also produces plentiful food sources that human beings and animals rely on for survival. Moreover, the origin and diversification of flower and fruit enables by diverse pollination and dispersal strategies, fueled the rise and adaptive radiation of angiosperms, profoundly reshaping terrestrial ecosystems.

A critical turning point of flowering plant development is characterized by the production of germline cells, which is essential for sexual reproduction. Unlike animals that establish germline precursors during embryogenesis, angiosperms specify germinal lineages late in development from floral somatic tissues. A mature sporophyte, derived from embryonic development of the seed, consists almost entirely of somatic cells. Within floral organs, specific somatic cells undergo epigenetic reprogramming to initiate germline fate.⁸⁻¹¹ Crucially, meiotic recombination during sporogenesis and random fertilization ensure genetic diversity across life cycles. This process regenerates the diploid sporophyte through seed formation, completing the heteromorphic alternation of generations characteristic of angiosperms.¹² In the anthers, pollen mother cells undergo meiosis to produce pollen grains (male gametophytes),¹³ while in the ovules, megaspore mother cells undergo meiosis to yield megaspores that develop into embryo sacs (female gametophytes).⁹⁻¹¹ These gametophytes house reproductive cells, sperm, and egg cells. The process of fertilization, involving the union of sperm and egg cells within the embryo sac, initiates the development of a new seed, setting the stage for the next life cycle.^{12,14} This cycle ensures genetic diversity through the production of gametes for fertilization.

During the establishment of plant architecture, auxin, cytokinin (CK), gibberellin (GA), abscisic acid (ABA), ethylene, brassinolide (BR) and strigolactone (SL) act interactively to create a dynamic balance, ensuring that the plants' growth is adaptive and efficient.¹⁵ Genetic programs, such as genes encoding transcription factors and signaling proteins, are activated in precise spatial and temporal patterns.^{4,16-21} The ABC model of flower development, for instance, illustrates how combinations of three classes of genes direct the formation of sepals, petals, stamens, and carpels. Epigenetic modifications, such as DNA methylation and histone tail alterations, further refine gene expression, enabling plants to respond to environmental changes without altering their genetic blueprint.^{16,18} Environmental factors, such as light, temperature, and nutrient availability, act as vital conductors in this

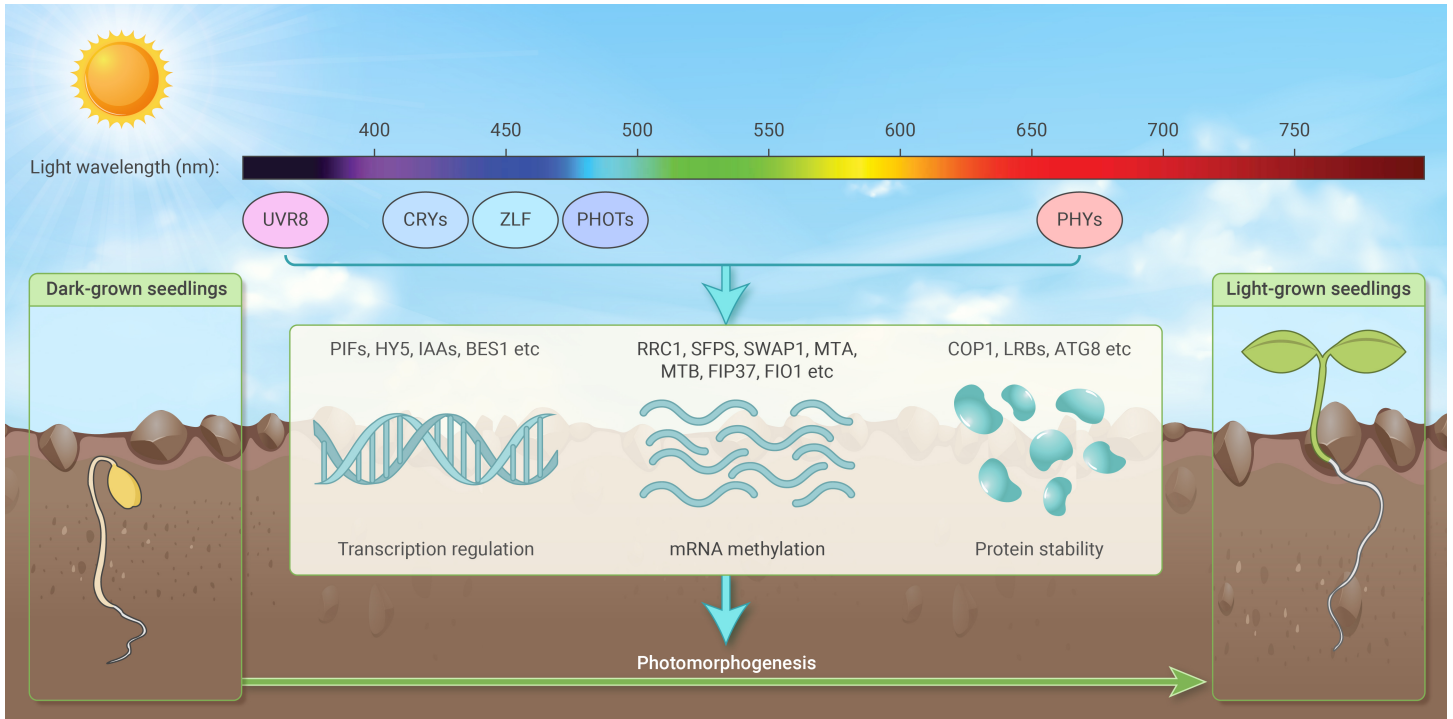


Figure 1. Light signaling pathways regulating photomorphogenesis in Arabidopsis The diagram depicts all identified photoreceptors in Arabidopsis, including the UV-B receptor UVR8, blue/UV-A light receptors cryptochromes (CRYs), the LOV-domain/F-box proteins ZTL/FKF1/LKP2 (collectively referred to as ZLF), PHOTs, and red/far-red light receptors phytochromes (PHYs). Each of these photoreceptors has a specific absorption spectrum, indicated by their respective positions under the light wavelength in nanometers (nm). Upon light perception, these photoreceptors activate downstream signaling cascades that involve at least three core regulatory layers: transcription regulation, mRNA methylation, and protein stability, thereby promoting the transition from skotomorphogenesis (dark-grown seedlings) to photomorphogenesis (light-grown seedlings). The diagram also highlights key interacting proteins involved in each regulatory pathway, underscoring the complexity and coordination of light-mediated developmental responses in plants.

symphony, fine-tuning the pace and pattern of development to ensure survival and adaptation to the surrounding conditions.²²⁻²⁴ Understanding the molecular mechanisms underlying plant development not only deepens our knowledge of biological complexity but also holds the key to enhancing crop yields, manipulating plant architecture, and mitigating the impacts of climate change on global food security. In this review, we summarize the regulatory mechanisms involved in different morphogenesis stages to provide a whole perspective on plant aboveground development, which will provide clues for further manipulations of crop development using genetic approaches.

PHOTOMORPHOGENESIS: TO LIVE OR TO DIE?

Light is important for our life. In addition to its well-known role as an energy source for plant photosynthesis, it is also an important environmental signal that regulates plant growth. In response to changing light conditions, plants perform appropriate physiological reactions and developmental programs, including seed germination, seedling de-etiolation (the transition from skotomorphogenesis to photomorphogenesis), vegetative growth, organ orientation, and the transition to reproductive development.^{25,26} The transition from full darkness beneath the soil to white light initiates de-etiolation. Etiolated seedlings exhibit slender, elongated stem, a hook-like curvature at the apex, small yellow-white leaves, with undifferentiated chloroplasts and no chlorophyll biosynthesis, as well as inhibited root elongation and reduced lateral root production. In contrast, light-grown seedlings develop short hypocotyls as well as open, expanded, and green cotyledons to increase photosynthesis. This developmental switch presents a life-or-death choice: exhaust finite resources, or deploy them to achieve photosynthetic autonomy (Figure 1).

Photoreceptors mediate light-regulated photomorphogenesis

Although plants lack a dedicated “eye” organ, they utilize a series of photoreceptors to perceive external light signals, which act as their “eyes” to orchestrate growth and development across the entire life cycle.²⁷ Among the various regulatory processes controlled by these photoreceptors, photomorphogenesis is the most extensively studied. Taking the model plant

Arabidopsis thaliana as an example, in the 1970s, only phytochrome was known as photoreceptor.²⁸ Phytochrome is a bilin-binding protein that helps plants respond to all red/far-red reversible reactions. Due to scientists' obsession with “how plants see light”, this field has made rapid progress (Table S1). During nearly half a century of research, fourteen photoreceptors have been identified, including five phytochromes (phyA-phyE), eight blue light photoreceptors (two phototropins, three cryptochromes, and three F-box family members), and the ultraviolet-B (UV-B) light receptor of UV RESISTANCE LOCUS8 (UVR8).^{27,29}

As red/far-red light photoreceptors, phy exist as dimers and occur in two photo switchable forms: the biologically inactive Pr form and the biologically active Pfr form.³⁰ After light irradiation, phy translocate from the cytoplasm to the nucleus to transduce light signal by interacting with downstream factors, thus promoting photomorphogenesis.^{31,32} Blue light receptors consist of three different types of proteins. The plasma membrane-associated proteins phototropin1 (PHOT1) and PHOT2 contain two LOV domains that sense blue light and a serine-threonine kinase domain to modify downstream effectors.^{33,34} PHOT1 and PHOT2 mediate a series of light responses—including phototropism, stomatal opening, chloroplast relocation, leaf development and suppression of hypocotyl elongation—that collectively promote photomorphogenesis, ensuring proper light adaptation. Cryptochrome1 (CRY1) and CRY2 undergo photoresponsive photoreduction and homo-oligomerization, leading to their physiological activation. This enables them to mediate blue light-inhibited hypocotyl elongation, regulate the shade avoidance response, and modulate the circadian clock, thereby playing central roles in photomorphogenesis.³⁵⁻³⁸ The latest research has found that blue light not only activates the function of CRY2 under blue light but also turns off the function of CRY2 in darkness.³⁹ Non-photoactivated CRY2 at the root tips suppresses further root elongation, whereas in aerial tissues, it participates in the diurnal cycle to regulate photomorphogenesis, thereby contributing to the rational allocation of growth energy during early developmental stages. It updates our understanding of photoreceptors and revealing their dual identity in morphology regulation. CRY3 is localized at chloroplasts and mitochondria,⁴⁰ but its physiological function remains to be discovered.

The third type of blue light receptor, ZTL family members, primarily influence the circadian clock and photoperiodic flowering, although they also modulate light-regulated development.^{41,42} In 2012, a UV-B photoreceptor was unexpectedly discovered and even had a crystal structure, making it the latest known photoreceptor to be discovered but the earliest to have its full-length structure elucidated.^{29,43} The dimeric structure of the UVR8 protein dissociates into signaling-active monomers upon UV-B exposure, and these monomers rapidly interact with downstream signaling components to regulate gene expression.^{29,43} Structural biology information provides a basis for understanding how photoreceptors perceive and respond to different wavelengths of light. In 2016, scientists identified that the Blue-light Inhibitors of Cryptochromes (BICs) protein could negatively regulate the activity of CRYs in Arabidopsis.⁴⁴ Later, structural biologists discovered that blue light promotes the oligomerization of CRY2, and the constitutively active CRY2 mutant exists in both dimeric and tetrameric forms.⁴⁵ BIC2 acts as a rope, binding to the “antenna” CRY2, preventing it from interacting with other proteins and thus inhibiting signal transduction.⁴⁶ The cryo-EM structure of phyB in the inactive Pr state reveals a complex topology, with the PhyB dimer adopting a head-to-tail parallel arrangement of N-terminal photosensory module (PSM) and a head-to-head arrangement of the histidine-kinase-related domain (HKRD).⁴⁷ Red light triggers a conformational shift in the phytochrome-specific domain which consequently disrupts the head-to-tail dimerization of PSM.⁴⁸ This alteration enables the recognition and binding to downstream factor PIF6. The cryo-EM structure of phyA in the Pr state shows differences from that of phyB in the PSM domain, HKRD domain, and dimerization interface, which confer high photosensitivity to phyA.⁴⁹ These plant “eyes” regulate various photoresponses redundantly, coordinately, or antagonistically, partially through physical interactions between photoreceptors and the common downstream signal transduction components (Figure 1).

Light signal transduction pathways and transducers

Light-induced conformational changes enable photoreceptors to interact with signaling partners. Most studies have shown that photoreceptors utilize common transcription factors to integrate different wavelengths. All of the photoactivated phys, CRYs and UVR8 interact with the basic helix-loop-helix (bHLH) transcription factors PHYTOCHROME INTERACTING FACTOR4 (PIF4) and PIF5, which are major negative regulators of photomorphogenesis, to inhibit their DNA binding activity and disturb transcription of downstream targets.^{50–52} PIFs occupy a central hub position in the light signal transcription network and tightly integrate light signals with endogenous hormones to modify plant growth.⁵³ PIF4 binds to promoters of *YUCCA8* (*YUC8*) to regulate auxin biosynthesis.⁵⁴ PIF4 interacts with transcription factors BRASSINAZOLE RESISTANT1 (BZR1) and BRI1-EMS-SUPPRESSOR1 (BES1) to elevate BR levels and promote cell elongation.^{55,56} Protein levels of PIFs are negatively regulated by DELLAs, the key repressors of GA and mediators of photomorphogenesis suppression in darkness.⁵⁷ PIF4 interacts with the ETHYLENEINSENSITIVE3 (EIN3)/EIL1, the master transcription factor in the ethylene signaling pathway, to promote hook formation. Jasmonic acid (JA) inhibits hook development through MYC2-PIF4 interaction.⁵⁸ Photoreceptors not only regulate photomorphogenesis through PIFs but can also directly interact with hormone-related transcription factors to control growth. For example, photoexcited CRY1 and phyB directly interact with the AUXIN/INDOLE-3-ACETIC ACID-INDUCIBLE (AUX/IAA), ARF6 and ARF8 transcriptional regulators to either protect them from degradation or regulate their DNA-binding activity, thus modulating auxin signaling and cell elongation.^{59,60} CRYs and UVR8 interact with the transcription factors BES1 and BES1-INTERACTING MYC-LIKE1 (BIM1), which mediate BR-promoted plant growth, to inhibit their DNA binding activity and thus repress hypocotyl elongation.^{61,62} These transcription factors establish a complex and precise regulatory network, enabling hormones to jointly regulate photomorphogenesis.

Posttranslational protein modifications are another central mechanism by which photoreceptors initiate downstream signaling cascades. Among these, the E3 ubiquitin ligase CONSTITUTIVE PHOTOMORPHOGENIC1 (COP1), a central repressor of photomorphogenesis, stands out as the most classic downstream effector molecule of photoreceptors. Light-activated phys, CRYs, and UVR8 repress the activity of the COP1, thus stabilizing the positive

regulators ELONGATED HYPOCOTYL5 (HY5)/ HY5-HOMOLOG (HYH) and LONG AFTER FAR-RED LIGHT 1 (LAF1) to promote photomorphogenesis-related gene expression.^{63–65} COP1 interacts with the VP domain, which is present in both photoreceptors and downstream target transcription factors. Competitive binding by photoreceptors sequestering COP1 from its interacting substrate proteins. COP1 controls the stability of phys and CRYs to form feedback regulation.^{66–70} COP1 positively regulates PIF3 and EIN3/EIL to maintain skotomorphogenesis by targeting EIN3-BINDING F-BOX PROTEIN1 (EBF1) and EBF2, the E3 ligases for PIF3 and EIN3/EIL, for ubiquitination and degradation.^{71,72} EIN3/EIL are inhibited by phyB, thus attenuating ethylene responses and promoting photomorphogenesis.⁷² Under UV-B light, COP1 subverts its canonical role as a photomorphogenesis repressor by degrading REPRESSOR OF UV-B PHOTOMORPHOGENESIS 1 (RUP1)/RUP2, which stabilizes HY5 and triggers a functional reversal. This mechanism underscores COP1's versatility in fine-tuning light responses across wavelengths.⁷³ In addition to its role as an E3 ubiquitin ligase, COP1 promotes the nuclear retention of UVR8 through their interaction within the nucleus, thereby enhancing UVR8-mediated photomorphogenesis.⁷⁴ Besides COP1, the E3 ubiquitin ligase LRB1 (Light-Response Bric-a-Brack/Tramtrack/Broad) and LRB2 are also involved in modulating the stability of photoreceptors and key light signaling proteins. LRBs promote concurrent polyubiquitination and degradation of both PIF3 and phyB.⁷⁵ COP1 and LRBs catalyze the ubiquitination and degradation of CRY2 protein under different light conditions (continuous or transient light).⁷⁶ Additionally, LRB promotes the rapid degradation of CRY2 under low-temperature conditions, thereby participating in the process of photomorphogenesis at low temperatures.⁷⁷ Recent studies have revealed that CRY regulates photomorphogenesis by modulating the autophagy pathway. In particular, CRY1-dependent inhibition of HY5 selective autophagy plays a key role in this process, underscoring the mechanistic diversity of post-translational control in light signaling.⁷⁸

In addition to the previously mentioned light regulation of transcription and protein stability, recent studies have revealed new progress in the light regulation of mRNA metabolism. Photoreceptors have been shown to transduce signals through alternative pre-mRNA splicing (AS). PhyB can directly interact with splicing factors, including REDUCED RED-LIGHT RESPONSES IN CRY1CRY2 BACKGROUND1 (RRC1), SPLICING FACTOR FOR PHYTOCHROME SIGNALING (SFPS), and SUPPRESSOR-OF-WHITE-APRICOT/SURP RNA-BINDING DOMAIN-CONTAINING PROTEIN1 (SWAP1), forming a ternary complex to promote photomorphogenesis.⁷⁹ The red light-dependent interaction between phyB and these splicing factors may confer specificity to AS in light signaling. Another splicing factor, SWELLMAP 2 (SMP2), also physically interacts with phyB, and SMP2 genetically acts downstream of phyB to negatively regulate seedling photomorphogenesis.⁸⁰ Photoactivated phyB also induces a specific intron retention in the 5' UTR of PIF3 mRNA, thereby inhibiting PIF3 protein synthesis to promote photomorphogenesis,⁸¹ although the splicing factors involved in this process have not yet been identified. It has been reported that CRY2 also interacts with splicing factors, among which CRY2 INTERACTING SPLICING FACTOR 1 (CIS1) is associated with CRY2 in a blue light-dependent manner to regulate the alternative splicing of FLOWERING LOCUS M (FLM), a process critical for temperature modulation of flowering.⁸² Whether CIS1 also mediates CRY2 signaling to regulate photomorphogenesis under different temperatures remains to be further investigated. Moreover, additional pathways and detailed mechanisms through which CRYs regulate alternative splicing during photomorphogenesis remain to be discovered. In parallel, emerging evidence highlights a novel role for CRYs in the regulation of mRNA methylation. Blue light positively regulates mRNA methylation in Arabidopsis, and CRYs mediate this response by promoting the photo-condensation of the N⁶-methyladenosine (m⁶A) writer complex, which includes MTA, MTB, FKBP12-interacting protein 37 (FIP37) and FIONA1(FIO1) writer complex.^{83–85} Blue light promotes the formation of the condensed nuclear CRY2/SPA1 photobody, which recruits and activates FIO1, resulting in a sustained increase of N⁶-methyladenosine (m⁶A) deposition at the RRACH-like sequences of mRNAs and increased translation of these mRNAs.⁸³ Future investigations into the regulation of mRNA metabolism by photoreceptors are expected to yield further novel insights, advancing our understanding of light-mediated gene expression control in photomorphogenesis (Figure 1).

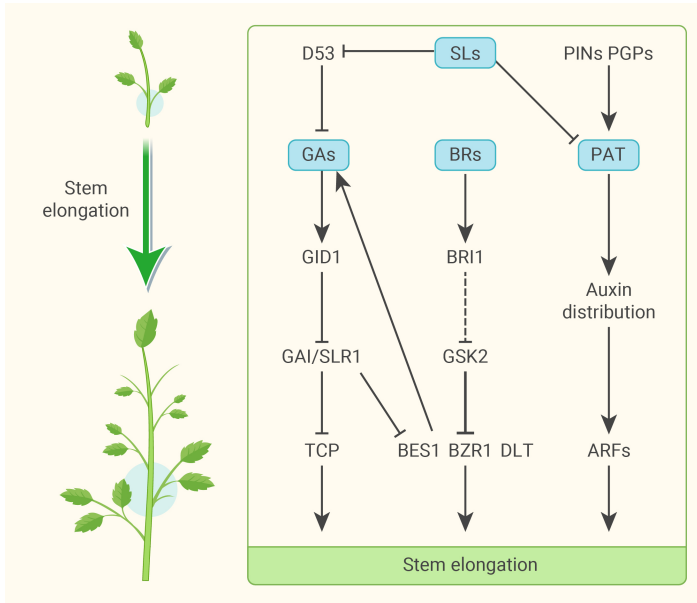


Figure 2. Genetic network regulating plant height Gibberellins (GAs) regulate plant height by controlling stem elongation DELLA proteins (e.g., GAI and SLR1) inhibit cell division by repressing transcription factors (e.g., TCP), but undergo GA-induced ubiquitination and degradation, releasing cell cycle suppression and promoting stem elongation. Brassinosteroids (BRs), regulate plant height by influencing cell expansion, apical meristem development, and microtubule dynamics through signaling components, such as the receptor BRI1, the kinase GSK2, and the transcription factors BES1, BZR1 and DLT. BES1, BZR1 and DLT increase GA levels, while DELLA proteins repress activities of BES1 and BZR1, thus mediating the coordinative regulation on stem elongation. Arrows indicate positive regulation, while lines ending with vertical bars represent repressive regulation. Dashed line indicates indirect mechanism.

When photomorphogenesis meets temperature

Photomorphogenesis is often accompanied by other environmental changes, such as temperature. There is crosstalk between photomorphogenesis and thermomorphogenesis.⁸⁶ Photoreceptors sense the difference between darkness and light, the level of irradiance, and also temperature. In particular, phyB senses temperature by changing the pool of light-activated Pfr (active form of phytochrome) and spontaneous phase separation.⁸⁷⁻⁸⁹ Plants perceive temperature to arrange chloroplasts for optimal photosynthesis while minimizing photodamage.⁹⁰ CRY1 that mediates blue light can repress high-temperature-promoted hypocotyl elongation.⁵¹ CRY2 proteins are degraded at low ambient temperatures by LRB proteins via the 26S proteasome.⁹¹ The loss of ZTL or UVR8 function results in defects in warm-temperature-promoted hypocotyl elongation, mainly by affecting PIF4 and auxin-related gene expression.^{92,93} The integration and adjustment of multiple factors in morphogenesis are important for plant survival, and they will be the focus of future research into plant morphogenesis.

PLANT HEIGHT REGULATION

Following the establishment of photomorphogenesis, which sets the foundation for early seedling development, plants transition into the vegetative growth phase, where key agronomic traits such as plant height regulation and shoot branching become critical. The formation of aboveground plant architecture is mainly determined by the activities of the meristems. Plant height is closely related to cell division and expansion of the shoot apical meristem and intercalary meristem.⁴ In the SAM, cells with high divisional activity continuously divide and proliferate to drive shoot growth, while the intercalary meristem supports continued cell division after maturation and promotes stem elongation. GAs and BRs play crucial roles in plant height by regulating cell division and expansion (Figure 2).

Gibberellin

GAs, a class of diterpenoid phytohormones, are pivotal in determining plant height through the regulation of stem elongation. Mutations in the biosynthesis and signaling pathways of GA lead to changes in plant height. The Green Revolution gene in rice, *Semidwarf 1* (*SD1*), encodes the rate-limiting enzyme

GA20ox in the GA biosynthetic pathway. Mutations in *SD1* produce a highly truncated enzyme, resulting in reduced endogenous GA synthesis and a semidwarf phenotype (Table S2).⁹⁴ The Arabidopsis *GA-INSENSITIVE* (*GAI*) gene encodes a DELLA protein which inhibits the transcriptional activity of the transcription factor TCP, thereby reducing the expression of genes essential for cell division.⁹⁵ In rice, the *GAI* homologue protein SLENDER RICE1 (*SLR1*) interacts with the GA receptor GIBBERELLIN INSENSITIVE DWARF1 (*GID1*) in the presence of GA. This interaction promotes the ubiquitination and subsequent proteasomal degradation of *SLR1*, relieving the suppression of cell division.⁹⁶ Consequently, rice *gid1* mutants exhibit a dwarf phenotype, whereas *slr1* mutants exhibit increased plant height. In wheat, the *Reduced height-1* (*Rht-1*) gene, a homologue of *GAI*, initiates tissue-specific translational reinitiation to generate an N-terminally truncated form of the DELLA protein, which is insensitive to GA-mediated degradation, leading to a semidwarf phenotype.⁹⁷ DELLA proteins further regulate plant height by modulating the stability or transcriptional activity of their interacting proteins. The GA-triggered degradation of *SLR1* not only promotes stem elongation but also triggers the degradation of *MONOCLUM1* (*MOC1*), leading to reduced tillering.⁹⁸ This discovery elucidates the molecular mechanism coordinating tillering and plant height regulation in rice.

Brassinosteroids

BRs are a class of steroidal hormones that regulate cell expansion and participate in cell wall remodeling activities.^{99,100} BRs impact plant height through their influence on the development of the apical meristem, cell division, microtubule formation, and directional control.¹⁰¹ In rice, the *DWARF2* (*D2*) and *DWARF11* (*D11*) genes encode proteins involved in BR biosynthesis, and the *d2* and *d11* mutants exhibit dwarf phenotypes due to abnormal BR synthesis (Table S2).¹⁰²⁻¹⁰⁴ The BR signal is perceived by the receptor BR INSENSITIVE1 (*BRI1*), which further represses GSK3/SHAGGY-like kinase 2 (*GSK2*) kinase to release its repression on the downstream transcription factors such as *BRI1 EMS SUPPRESSOR1* (*BES1*), *BRASSINAZOLE-RESISTANT 1* (*BZR1*), and *DWARF AND LOW-TILLERING* (*DLT*).¹⁰⁵⁻¹⁰⁷ *BES1* modulates plant height by regulating BR-responsive gene expression together with its interaction proteins.^{108,109} BRs may regulate microtubule formation and orientation to modulate cell elongation through *OsBZR1* and its downstream targets.¹¹⁰ *OsDLT* interacts with *ORYZA SATIVA HOMEBOX 15* (*OsOSH15*) to modulate rice internode elongation by orchestrating BR signaling and metabolism.^{109,111} BRs also work in concert with GAs and other phytohormones to regulate plant height. For instance, BRs enhance GA levels and cell elongation by activating the GA biosynthesis gene *OsGA3ox2* and inhibiting the GA inactivation gene *OsGA2ox3*.^{112,113} In addition, the *BZR1* and DELLA proteins interact with each other, mediating the crosstalk between BR and GA signaling pathways.¹¹⁴ The negative regulator DELLA proteins in the GA signaling pathway interact with transcription factor *BZR1* to inhibit the activity of *BZR1* (Figure 2).¹¹⁵

Regulation of other factors

In addition to GAs and BRs, auxin and SLs have also been recognized as critical regulators of plant height (Figure 2). Auxin regulates plant height through its polar transport. Transporters such as *OsPIN2* and P-glycoproteins (PGPs) establish auxin distribution patterns in the stem of rice, maize and sorghum. These patterns then modulate cell elongation and internode development, consequently regulating plant height.^{116,117} The auxin signaling component *TaARF12* promotes stem elongation and shows interaction with DELLA proteins in wheat.¹¹⁸ Furthermore, the auxin and BR signaling pathways cooperatively regulate plant height through the *SMOS1-SMOS2/DLT* complex in rice.¹¹⁹ SLs promote plant height in different plant species as indicated by the dwarf phenotypes of SL deficient and signaling mutants. But the precise mechanism through which SLs regulate plant height remains to be elucidated. Recent research identified the regulatory module of *GHD53-GhGARF-GhPH1/PAV^{PH1}* by which SLs promote plant height via influencing the endogenous GA levels in upland cotton.¹²⁰ SLs may also regulate plant height by affecting polar auxin transport (PAT), as indicated by the enhanced PAT in SL deficient mutants.^{121,122} Moreover, numerous genes regulate plant height by modulating cell wall development, cytoplasmic glutamine synthesis, RNA editing, cell division, the ubiquitin-proteasome pathway, and fatty acid

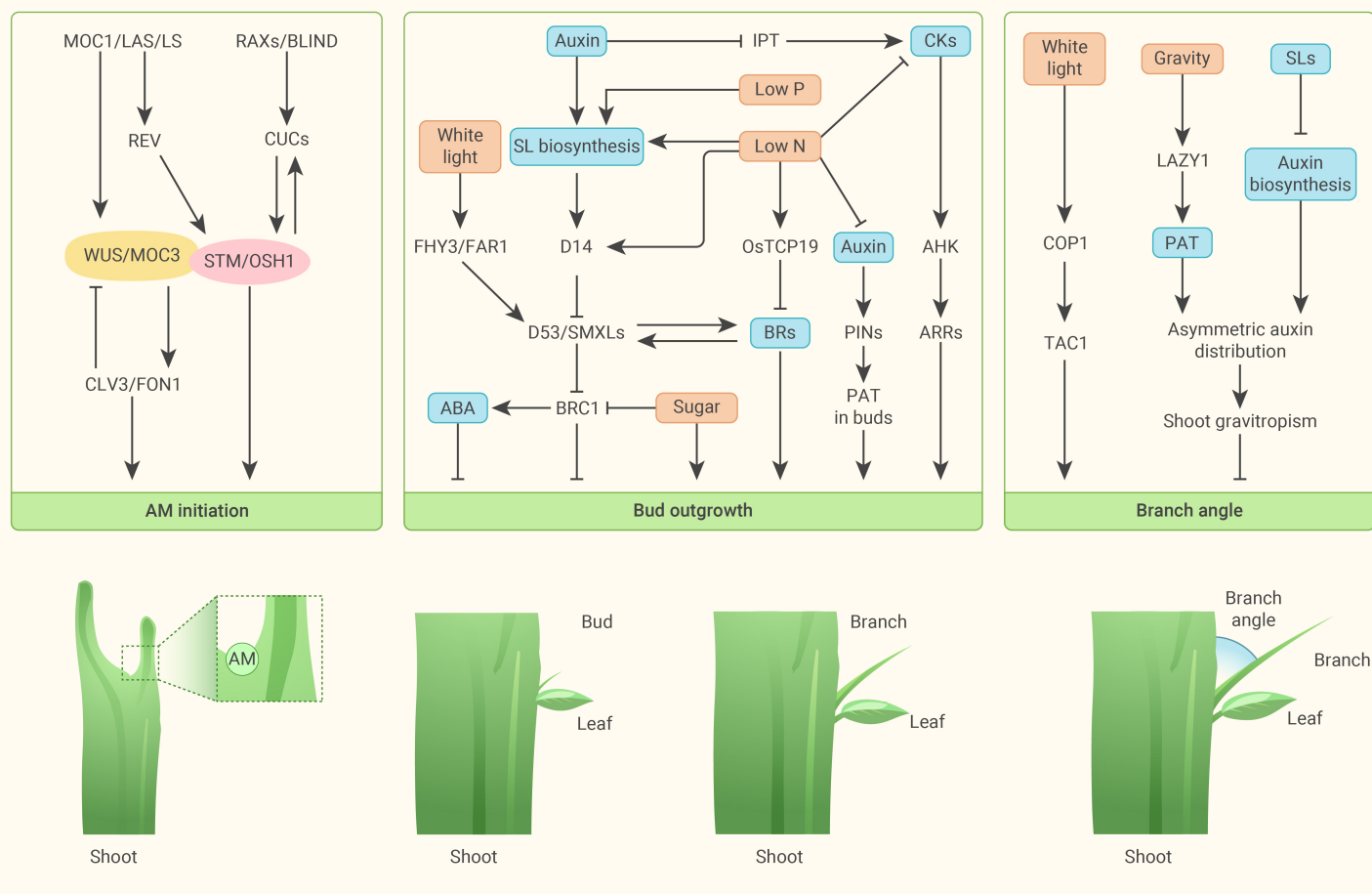


Figure 3. Genetic networks regulating shoot branching Branch architecture mainly includes the branch number and branch angle. The developmental stages of AM initiation and bud outgrowth determine branch number and are mainly regulated by key transcription factors, transcription regulators, receptors and kinases, which mediate endogenous hormone signals and environmental stimuli. Branch angle plasticity depends predominantly on shoot gravitropism and is also regulated by light conditions and plant hormones. The yellow and pink ovals indicate the WUS-STM complex, while the blue and orange rectangles represent plant hormones and environmental cues, respectively. Arrows indicate positive regulation, while lines ending with vertical bars represent repressive regulation.

metabolism.¹²³ The comprehensive mechanisms underlying plant height regulation and crosstalk between plant height and other architecture features need further investigation.

SHOOT BRANCHING

With plant growth, shoot branching begins to originate from the axillary meristem (AM). After AM specialization and bud formation, the lateral buds at the axils develop into lateral branches. Shoot branching is predominantly regulated by genetic factors, plant hormones, and external environmental signals. The branch number is determined by the activities of AMs and lateral buds. Plant hormones such as SLs, auxin and CKs affect shoot branching by regulating the elongation of lateral buds. Environmental signals affect branching architecture mainly by altering physiological and metabolic processes and endogenous hormone levels. Shoot branching is closely related to panicle number, plant density, photosynthesis, water and nutrient absorption, thus having profound effects on crop yield. Understanding the principles of shoot branching will facilitate crop design for ideal plant architecture, increased grain yield, and sustainable agricultural mechanization (Figure 3 & Table S3).

Genes controlling axillary meristem formation

Axillary meristem formation involves meristem initiation and development of meristem, which is regulated by a group of transcription factors. The 'lateral suppressor' (*ls*) mutant in tomato exhibited defects in meristem initiation.¹²⁴ The *monoclonum1* (*moc1*) mutant in rice has only a single primary stem with no tillers,¹²⁵ and the *lateral suppressor* (*las*) mutant in Arabidopsis shows a defect in lateral shoot formation. *LS*, *MOC1*, and *LAS* are homologous genes encoding GRAS family proteins that are critical for AM initiation during

the vegetative phase (Table S3). The *LAS* gene is required for the establishment and maintenance of *SHOOT MERISTEMLESS* (*STM*) expression in the AM.¹²⁶ The transcription factor *REVOLETA* (*REV*), which is downstream of *LAS*, directly upregulates *STM* expression to promote AM initiation.¹²⁷ *WUSCHEL* (*WUS*) is essential for the maintenance and differentiation of SAM by activating the expression of the secreted peptide *CLAVATA3* (*CLV3*), which in turn represses the expression of *WUS*.^{128,129} *MOC1* also functions as a co-factor of *MOC3*, a homologue of *WUS* in rice,¹³⁰ to promote the expression of *FLORAL ORGAN NUMBER1* (*FON1*).¹²⁹ In addition, *WUS* elevates its binding ability to *CLV3* by forming a heterodimer with *STM*.¹³¹

REGULATOR OF AXILLARY MERISTEM (*RAX*) encodes an R2R3-type MYB transcription factor that is essential for the early development of AMs by regulating the expression of *CUP-SHAPED COTYLEDON* (*CUC*) in the central axillary domain. The *rax1* mutant shows reduced number of rosette leaf branches and the *rax1 las* double mutant exhibits an additive phenotype, indicating that the *RAX* gene functions in a parallel pathway with *LAS* during AM initiation.^{124,132,133} *Blind* (*Bl*) is the ortholog of the *RAX* genes in tomato and pepper and regulate AM initiation,^{134,135} indicating a conserved role of *RAX* genes in AM formation. The *REGULATOR OF AXILLARY MERISTEM FORMATION* (*ROX*), which encodes a protein with a basic helix-loop-helix (bHLH) domain, functions downstream of *RAX* to regulate AM formation.^{136,137}

Genes controlling axillary bud elongation

After AM formation, axillary buds develop and form lateral branches under appropriate conditions. Genes that promote bud arrest have been described in both monocotyledons and dicotyledons (Table S3). The maize *TEOSINTE BRANCHED 1* (*TB1*) gene encodes a member of the TCP transcription factor family that is specifically expressed in axillary buds and prevents bud

outgrowth.^{138,139} Subsequently, its homologous genes were found in Arabidopsis (*BRC1*),¹⁴⁰ rice (*OsTB1*),¹⁴¹ pea (*PsBRC1*) and other plant species.¹⁴² Loss of function of *BRC1*, *OsTB1*, and *PsBRC1* results in increased number of branches and tillers,¹⁴⁰⁻¹⁴² proving that *TB1* homologues have conserved functions in repressing bud elongation. Light and SLs coordinately regulate *BRANCHED1* (*BRC1*) expression and shoot branching by converging at the *BRC1* promoter.¹⁴³ Sugar signaling regulates shoot branching partially through Sugar Transporter Protein 1 (STP1), which acts upstream of *BRC1* to repress *BRC1* expression.¹⁴⁴ CK and SLs regulate branch development by inhibiting and promoting the local accumulation of *BRC1*, respectively.^{145,146} SL and BR signaling coordinately regulate *BRC1* expression and shoot branching through repressors of SL signaling (SMXL6,7,8 and D53) and transcription factors involved in BR signaling (BES1 and OsBZR1).¹⁴⁷ The downstream signaling event of *BRC1* is also interesting. *BRC1* directly regulates the expression of *HOMEODOMAIN PROTEIN 21* (*HB21*), *HB40* and *HB53* to activate the expression of ABA biosynthetic gene *NINE-CIS-EPOXICAROTENOID DIOXYGENASE 3* (*NCED3*).¹⁴⁸ During the process of axillary bud dormancy, the expression of ABA responsive and biosynthetic genes is upregulated, and the application of ABA or overexpression of ABA biosynthetic genes represses the growth of axillary buds and rice tillers,^{149,150} indicating that ABA in turn inhibits bud growth.

Regulatory mechanism of plant hormones

SLs play fundamental roles in repressing branch number through the inhibition of bud elongation (Figure 3). Based on the structure features, SL compounds are divided into canonical and noncanonical SLs. Canonical SLs probably act as signals to microbes and plants in the rhizosphere, while noncanonical SLs are proposed to primarily function as plant hormones.¹⁵¹ In Arabidopsis, the SL synthesis pathway mainly includes Arabidopsis DWARF27 (AtD27), MORE AXILLARY GROWTH 3 (MAX3), MAX4, MAX1, CLA methyltransferase (CLAMT), and Lateral Branching Oxidoreductase (LBO), which are responsible for sequential catalysis from all-*trans*- β -carotene to 9-*cis*- β -carotene, 9-*cis*- β -apo-10'-carotenal, carlactone, carlactonic acid (CLA), methyl carlactonoate (MeCLA) and an SL-like product. The corresponding mutants displayed multibranch phenotypes due to deficiency in SL biosynthesis.¹⁵²

In the SL signaling pathway, the receptor protein DWARF14 (D14), F-box protein DWARF3 (D3)/MAX2, and repressor protein D53/SUPPRESSOR OF MORE AXILLARY GROWTH2-LIKE6,7,8 (SMXL6,7,8) form a complex upon SL perception, triggering ubiquitination and degradation of D53/SMXL6,7,8 through the 26S proteasome.¹⁵³⁻¹⁵⁶ In the absence of SLs, D53/SMXL6,7,8 remain stable and recruit TPL/TPR proteins through their ethylene-responsive element binding factor-associated amphiphilic repression (EAR) motif to inhibit downstream signaling pathways. SMXL6, 7, and 8 function as transcriptional repressors through interaction with transcription factors and repressing the transcriptional regulatory effects of transcription factors on target genes. For instance, SLs repress shoot branching by activating *BRC1* transcription, which induces *HB40* expression and promotes ABA biosynthesis in lateral buds in Arabidopsis.¹⁴⁶ In rice, SLs promote ABA biosynthesis mainly through activating the expression of *HOMEODOMAIN 12* (*OsHOX12*) and ABA biosynthetic gene *9-CIS-EPOXYCAROTENOID DIOXYGENASES* (*OsNCEDs*).¹⁴⁹ Ideal Plant Architecture 1 (IPA1) is a transcription factor that directly binds and activates the *OsTB1* promoter. D53 interacts with IPA1 and represses its transcriptional activation. Moreover, IPA1 can directly bind to the *D53* promoter and promote *D53* expression, forming a negative feedback regulatory loop.¹⁵⁷ Interestingly, SMXL6,7,8 can directly bind to their own promoters and repress gene expression, demonstrating that SMXL6,7,8 function as transcription factors to regulate the homeostasis of SL signaling in Arabidopsis.¹⁴⁶ A recent study has demonstrated that SMXL5 also participates in the SL signaling pathway by inhibiting the SMXL7 degradation and attenuating the downstream transcriptional regulation, thus regulating shoot branching.¹⁵⁸ Notably, exciting research discovered that the microbial metabolite cyclo (Leu-Pro) can directly bind to the SL receptor OsD14 and activate the SL signaling pathway, thus repressing tiller development in rice.¹⁵⁹ This suggests that the rhizosphere microbial community is also involved in the regulation of shoot branching.

The endogenous levels and spatial-temporal distribution of auxin are

pivotal in the regulation of lateral branch development (Figure 3). Auxin is mainly produced in the shoot apex and is transported to other parts of plants through polar auxin transport (PAT). Auxin prevents lateral bud outgrowth and establish apical dominance by regulating the secondary messengers CK and SLs and by establishing an auxin gradient.¹⁶⁰ On the one hand, auxin can inhibit the growth of AMs by downregulating the activity of *isopentenyl transferase* (*IPT*), thereby suppressing CK biosynthesis and maintaining apical dominance.¹⁶¹ Auxin also upregulates the expression of *MAX3* and *MAX4* through their signaling pathway to promote SL synthesis and inhibit bud outgrowth.¹⁶² On the other hand, the growth of lateral buds depends on the efflux of auxin from the buds through PAT, which relies on the efflux carrier PIN proteins on the plasma membrane.¹⁶³ SLs also reduce the accumulation of PIN1 on cell membranes by removing PIN1 from the plasma membrane, slowing or inhibiting auxin transport from the bud to the stem.¹⁶⁴ In addition, auxin regulates lateral branch development via auxin synthesis, metabolism, and signaling pathways.

BR, CK, ABA and GA are also important in regulating lateral bud growth (Figure 3). In BR signaling, the key transcription factor BZR1 directly targets and suppresses the expression of *CUC3*, thereby affecting the expression of *LAS* and inhibiting axillary bud formation. BES1 and OsBZR1 also bind directly to the *BRC1* promoters, and SMXL6,7,8 and D53 repress *BRC1* expression partially through interaction with BES1 and OsBZR1, indicating crosstalk between BR and SL signaling in bud outgrowth.^{147,165-167} CKs activate WUS expression during AM initiation and promote lateral bud development by promoting cell division.¹⁶⁸ The STM protein in Arabidopsis regulates meristem proliferation by activating CK synthesis.^{169,170} Moreover, CKs inhibit the local accumulation of *BRC1* to promote lateral bud elongation.¹⁴⁵ The biosynthesis of ABA in buds is regulated by *BRC1* and plays an essential role in the switch between bud dormancy and outgrowth, as mentioned above. The increase in *OsTB1* mRNA levels in the GA catabolic mutant GA2ox correlated well with increased tillering, indicating that GA may inhibit tillering development in rice by repressing *OsTB1* expression.¹⁷¹ Moreover, GA promotes the sustained growth of pea buds after auxin treatment,¹⁷² suggesting that GA may also regulate shoot branching through auxin transport and apical dominance establishment.

Influence of environmental factors on shoot branching

The availability of nutrients in the soil, particularly nitrogen and phosphorus, serves as critical environmental factor regulating branch development (Figure 3). For instance, nitrate and ammonium ions in the soil can stimulate rice tillering.⁹⁴ High nitrogen levels enhance the transport of CKs from roots to shoots and promote shoot branching, whereas low nitrogen levels reduce CK synthesis, resulting in reduced branching.^{4,173} Furthermore, low nitrogen levels induce the expression of SL biosynthetic genes in roots, leading to increased SL content and decreased shoot branching.^{174,175} Importantly, low nitrogen also induces phosphorylation of the SL receptor D14 to stabilize D14 and enhance SL perception, thus repressing tiller development in rice.¹⁷⁶

Nitrogen-mediated Tiller Growth Response 5 (NGR5), a nitrogen-induced transcription factor, plays a pivotal role in nitrogen-mediated chromatin remodeling and regulates the expression of genes involved in tillering development. NGR5 can be recognized and degraded by the GA receptor GID1, mediating the inhibitory effect of GA on tillering development.¹⁷⁷ Nitrogen supply inhibits the expression of *OsSTP28*, which results in glucose accumulation in the apoplast of tiller buds and in turn suppresses the expression of a transcriptional inhibitor OSH15, thus activating gibberellin catabolism in shoot base and promoting the rice tiller number.¹⁷⁸ In addition, OsTCP19 is a critical transcription factor that regulates rice tillering in response to nitrogen and is capable of directly binding to the *Dwarf and Low-Tillering* (*DLT*) promoter in the BR signaling pathway, thereby regulating tiller number.¹⁷⁹ Furthermore, polar auxin transport and the expression of auxin transporters are responsive to N limitation and ammonium application, leading to changes in auxin levels and lateral bud elongation.^{180,181}

Phosphorus deficiency reduces tiller number, and PHOSPHATE STARVATION RESPONSE2 (*OsPHR2*) is the master transcription factor involved in tiller repression under Pi deficiency.¹⁸² Low-Pi stress also significantly enhances the synthesis of strigolactones. Pi deficiency results in a several hundred-fold increase in SL content in rice roots and activates SL signaling

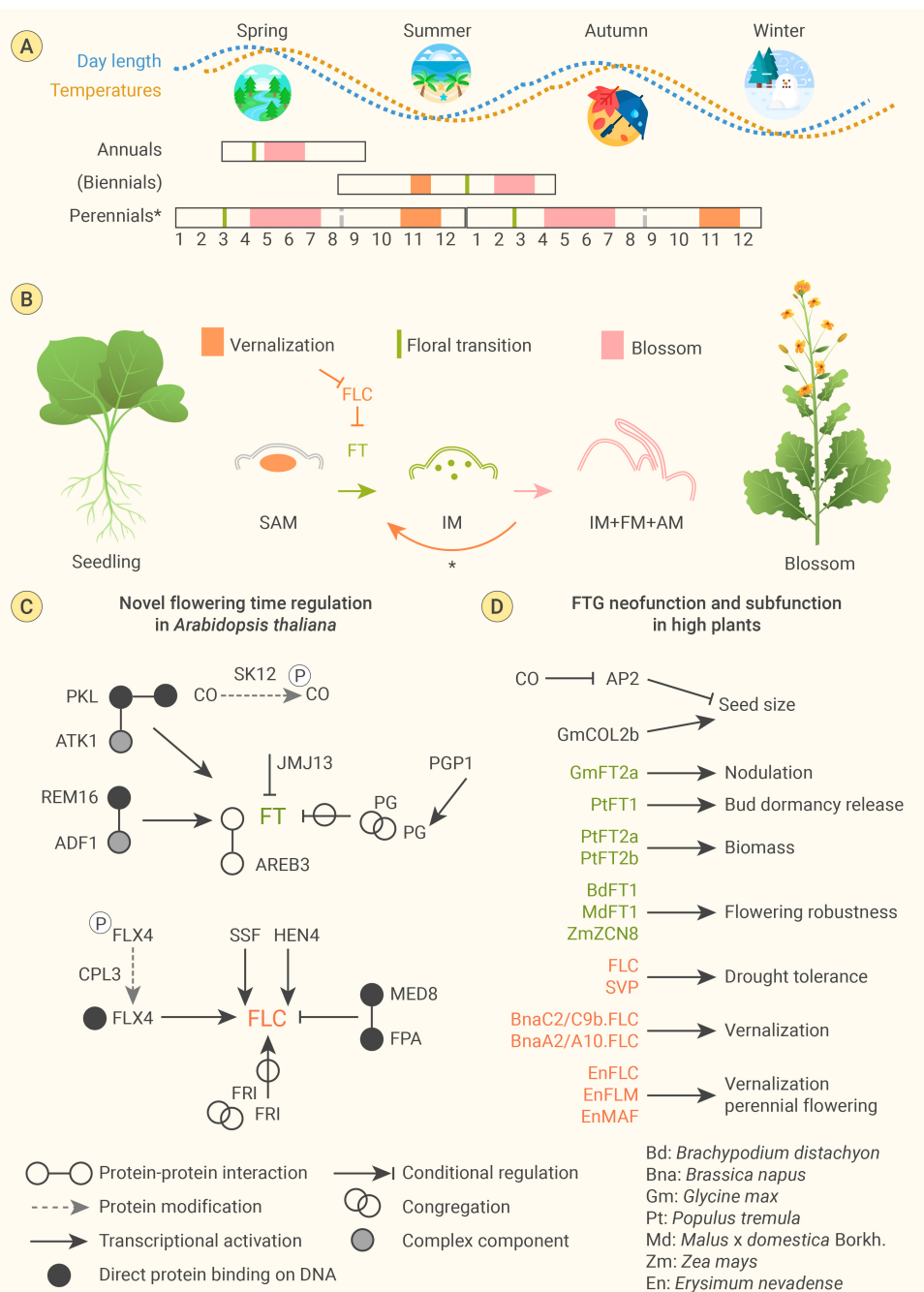


Figure 4. Regulated transition of flowering via new FTG Screening of flowering time genes (FTG) is never stopped. Traditionally, floral transition makes the shoot apical meristem (SAM) turn to inflorescence meristem (IM). During favorable conditions, IM keeps formations of flower meristems (FM) and axillary meristems (AM). New regulators involved floral transition exhibit many different regulatory ways. Transcription factors can work together with epigenetic regulators and perform concise regulation. Beyond gene transcriptional regulation, proteins can conjugate with different other molecules for an additional layer of control. In different plant species, FTG undergo evolution and domestication which show unexpected critical functions. Compared with annual plants, biennial and perennial plants experience winter cold temperatures. Low temperatures are not only required for flowering promotion but also needed for multiple rounds of flowering.

TILLER ANGLE CONTROL 1 (*TAC1*), thus reducing the branch angle when *Arabidopsis* is grown under continuous dark or far-red light conditions.¹⁸⁵ Understanding the molecular mechanisms of light-regulated shoot branching is essential for the breeding of crop varieties with reduced shade avoidance syndrome that facilitates increased cultivation densities and yields.

Gravity is the primary factor regulating the branch angle of plants via the PAT (Figure 3). Statocyte cells contain amyloplasts to sense gravitational stimuli in the plant stem endodermis. Amyloplast sedimentation may trigger signal transduction to change the direction of auxin transport and induce the asymmetric redistribution of auxin in gravity-responsive cells.¹⁸⁶ SLs inhibit auxin biosynthesis and weaken the asymmetric redistribution of auxin, thus regulating shoot branch angle.¹⁸⁷ In rice, the *LAZY1* (*LA1*) gene negatively regulates PAT to affect auxin distribution and consequently alter the branching angle (Table 7).¹⁸⁸ In *Arabidopsis*, gravity triggers the phosphorylation of LAZY proteins, which facilitates the enrichment of LAZY proteins on amyloplasts and the repolarization of LAZY proteins on membrane, thus resulting in asymmetrical auxin distribution and promoting differential root growth.¹⁸⁹ It is interesting to investigate whether LAZY

pathways to suppress tillering by elevating *OstB1* expression levels.¹⁸³ Dissecting the regulatory mechanisms underlying tillering regulation by soil nutrients, such as nitrogen and phosphorus, is crucial for minimizing fertilizer use in agriculture, enhancing crop yields, and achieving sustainable agricultural development.

Light influences plant growth and development, particularly by affecting the number and angle of shoot branches (Figure 3). Plants sense changes in light quality through light signal receptors such as phyA, phyB, UVR8, and CRY1/2. These receptors relay signals to downstream factors, subsequently modulating the expression of target genes such as *BRC1* to either inhibit or promote bud elongation.¹⁸⁴ Phytochrome and SL signaling synergistically regulate branch development through SQUAMOSA PROMOTER BINDING PROTEIN-LIKE9,15 (*SPL9,15*) and *SMXL6,7,8*, which form protein complexes to control *BRC1* expression. In addition, FAR-RED ELONGATED HYPOCOTYL3 (*FHY3*) and FAR1-RED IMPAIRED RESPONSE1 (*FAR1*) also directly upregulate the expression of *SMXL6* and *SMXL7* to promote branching.¹⁴³ *CONSTITUTIVE PHOTOMORPHOGENESIS 1* (*COP1*) is a core inhibitor of the light signaling pathway that suppresses the expression of the branch angle control gene

proteins sense gravity in shoot through a conserved mechanism and to evaluate its contribution to branch angle.

TRANSITION TO FLOWERING CAN OCCUR ONCE OR REPEATEDLY

With appropriate environmental signal, plants will transit from vegetative to reproductive development, which termed as floral transition. During the tightly regulated developmental transition, the SAM undergoes morphological changes toward the inflorescence meristem (IM).¹⁹⁰ The IM further gives rise to floral meristems (FMs, Figure 4) and axillary meristems (AMs, Figure 3) at the flanking regions, which further grow out in a relative order and number and ultimately become various types of plant inflorescence architectures.¹²⁶ Annual plants, such as the model organisms *A. thaliana* Columbia-0 and most crops *Oryza sativa* ssp. *indica*, undergo irreversible floral transition one time in the lifespan. The majority of perennial plants, such as the arctic-alpine *Arabis alpina*,¹⁹¹ *Populus* trees¹⁹² and newly interspecifically hybridized Asian rice *Oryza sativa* ssp. *indica* RD23,¹⁹³ repeatedly flower in consecutive seasonal growth periods and maintain vegetative growth even after flowering (Figure 4A).

This critical switch occurring in favorable conditions is one of the most important life-history strategies for plant adaption to growth conditions. Active SAMs of all annuals and a few unique perennials completely convert to inflorescence and flower meristems and thus produce a massive flowering episode and sufficient seeds immediately followed by the phase of whole-plant senescence and death. These plants are also known as monocarpic plants (Figure 4A). In modern agricultural cropping system, cereal crops, such as maize, rice and barley, and prominent oil crops like soybean and sunflower, can complete their life cycles in a calendar year despite being kept in optimal environments. In the plant kingdom, some extreme perennials, such as bamboo¹⁹⁴ and silversword, may live for many years before a single flowering and subsequent death. In contrast, most perennial species use a polycarpic life strategy, in which only converted IMs stop growing after seed production and more importantly maintain a supply of vegetative SAMs for permits of the next rounds of growth in future seasons. Moreover, some perennials can cycle back and forth between vegetative and reproductive growth modes because reproductive meristems are still able to change back to the vegetative meristems even after flowering. The in-between seasonal life expectancy is the character of biennial plants, which remain vegetative the first year, reproduce the second year, and then die. Biennials are relatively rare in the angiosperms, and the winter oil crop *Brassica napus* and winter cereal crop wheat (*Triticum aestivum*) are both considered as the important herbaceous biennial plants. Differences in flowering behavior between annual and perennial plant species contribute to different life adaptation strategies. Plants dynamically sense seasonal environment, mainly changing in day length, light quality, light strength, temperature, and nutrients (Figure 4). With comprehensive studies in the last century, hundreds or even thousands of flowering time genes (FTG) from up to eight hundred diverse plant species have been identified.¹⁹⁵ The identification of several well-characterized functional genes unveil regulatory networks, which allow floral transition to occur in a tightly controlled manner. To date, the model plant *A. thaliana* contains more than three hundreds of FTG, which play specific or multi-layer roles through photoperiod, vernalization, aging or phytohormone pathways.¹⁹⁶ In the allopolyploid plant *B. napus*, the FTG has largely increased to more than thirteen hundreds because of the whole genome triplication.¹⁹⁷ Most orthologous FTG get triplicated, but some remain at low copy numbers, which may result from domestication. In *B. napus*, the flowering time integrator *FLOWERING LOCUS C (FLC)* has even nine or ten paralogues in different accessions, each of which shows different expression dynamics resulting in a wide range of vernalization requirements and adaptive flowering time in response to different levels of cold conditions.¹⁹⁸

Here, we will pinpoint some recent interesting findings of novel important regulators and additional in-depth molecular mechanisms in flowering time regulation and then broaden the discussion to neofunction of classic FTG and subfunction of FTG orthologs (Figure 4 & Table S4).

New flowering time genes and novel regulatory mechanisms

Regulation of flowering time has been well studied in the model dicot plant *A. thaliana*⁷ and the cereal monocot crop rice.^{199,200} Generally, plants sense seasonal cues, such as changes of photoperiod and temperature, to trigger phase transition (Figure 4). These processes are concisely regulated by hundreds of functional genes, in terms of responsive cues and regulatory networks that have been well summarized into several flowering time pathways. Plants receive changes of day length through the photoperiod pathway. In Arabidopsis, long days, normally referred to 16-hour light, promote flowering time, which is indicated by the number of rosette leaves formed from SAM before floral transition (Figure 4B). The day-length-dependent accumulation of *CONSTANS (CO)* transcript and protein is crucial for the rhythmic activation of *FLOWERING LOCUS T (FT)* expression at dusk under long days. *PICKLE (PKL)*, also designated as *EPPI* for *ENHANCED PHOTOMORPHOGENESIS*, shows a mild rhythmic expression pattern with one peak at zeitgeber time 4 (ZT4) and another at ZT16 under long days. PKL interacts with CO via its ATPase domain, and the PKL and CO complex is critical for the direct activation of *FT*.²⁰¹ Furthermore, PKL recruits ARABIDOPSIS HOMOLOG OF TRITHORAX1 (ATX1) that belongs to the Trithorax Group (TrxG) proteins to maintain the level of trimethylated lysine 27 of histone 3 (H3K27me3) at various regions across the *FT* locus.²⁰² One glycogen SYNTHASE KINASE 3

(GSK3) member, SHAGGY-LIKE KINASE 12 (SK12), interacts with CO for the phosphorylation at threonine 119, thus facilitating CO degradation (Figure 4C).²⁰³ As we all know, *FT* can be induced by FR. Still, the rhythmic activation peak of *FT* expression in the morning is influenced by the accumulation of R: FR-adjusted light (R: FR ratio was adjusted to 1 with FR supplement) from the previous afternoon to the next morning. The expression level of *FT* in *phyA-211* is lower than Col-0, especially at ZT4. PhyA-mediated high-irradiance response (HIR) also regulates the *FT* bimodal expression patterns.²⁰⁴

The mobile floral stimulus *FT*¹⁹⁰ belongs to the phosphatidylethanolamine-binding proteins (PEBPs) family, which includes structurally, but not functionally, lipid-binding proteins reported from bacteria to humans and plants.²⁰⁵ *FT* protein is synthesized in the vasculature and moves directly to its destination that could be shoot apex²⁰⁶ or root nodules.²⁰⁷ *C-TERMINALDOMAIN PHOSPHATASE-LIKE 2 (CPL2)* is highly expressed in the vasculature of leaves, loss-of-function of which is much earlier flowering (Figure 4C). By using RNA sequencing and *FT:GUS* lines, *FT* upregulation could be causal for flowering promotion in the *cpl2-2* mutant. Furthermore, protein-protein interaction assays together show that CPL2 directly interacts with and dephosphorylated EARLY FLOWERING MYB PROTEIN (EMF). The CPL2-EMF complex has the ability to directly bind to *FT* gene locus for inhibiting the transcriptional activity of *FT* and thereby suppressing flowering.²⁰⁸ In the shoot apex, *FT* works with the bZIP transcription factor FD as a transcriptional coregulator.²⁰⁹ The FD-related bZIP transcription factor *AREB3* is spatially and temporally co-expressed with *FD* at the SAM.²¹⁰ Tissue-specific overexpression of *FT* could not promote flowering time in the *areb3-1 fd-3* double mutant. Interestingly, *FT* also interacts with other transcription factors previously known in the ABA pathways (Figure 4C).²¹⁰ Different from long-day facultative Arabidopsis, soybean (*Glycine max*) is one of typical short-day crops, which is sensitive to photoperiod and long-day conditions strictly delay flowering time. Promoting flowering can enhance the adaptability of soybean to grow in high latitudes area in China. By using diverse accessions and GWAS analysis, *TIME OF FLOWERING 5 (Tof5)* in chromosome 5 is localized with a highly significant QTL locus. Although *Tof5* could be annotated to either *GmFUL2a* or *SEPAL-LATA4 (GmSEP4)*, correlation analysis between haplotypes and flowering-time phenotypes indicates that *GmFUL2a* is likely to be a flowering activator. Furthermore, null mutant and overexpression of *GmFUL2a* lines demonstrate that *GmFUL2a* can directly activate the expression of soybean *FT* homologs *GmFT2a* and *GmFT5a*, and thus promote flowering time.²¹¹ Plants sense the environmental temperatures and Arabidopsis flowering time changes in response to one degree Celsius. Generally, lower temperatures delay flowering and higher temperatures promote flowering. At short-day high temperatures, such as 28°C, plants with loss-of-function of *JUMONJI 13 (JM13)*, an H3K27me3 demethylase, still occurs very early flowering through activation of *FT*.²¹² At a lower 16°C ambient temperature, the anionic phospholipid phosphatidylglycerol (PG) directly binds to *FT* to sequester *FT* activity. In the mutant of *PHOSPHATIDYLGLYCEROLPHOSPHATE SYNTHASE 1 (PGP1)*, a key PG biosynthetic gene, flowering time is much earlier under low temperatures, because of the presence of more soluble and mobile *FT* proteins in companion cells of leaf vasculatures (Figure 4C). In addition, mutagenesis of other PG biosynthetic genes significantly changes flowering time under low temperatures.²¹³ In maize, PGs are important metabolites involved in plant responses to low temperatures. In Mexico, highland domesticated maize shows introgression with a teosinte subspecies *mexicana*. Adaptation to low temperatures and phosphorus deficiency in highland environments drove *mexicana* genetic divergence from the lowland *parviglumis*. By linkage mapping, *HIGH PHOSPHATIDYLCHOLINE 1 (HPC1)* encoding a phospholipase A1 enzyme has been identified as a major driver of flowering time and phospholipid variation in highland maize. *HPC1* exhibits similar expression patterns to *ZmZCN8*, an *FT*-like homolog.²¹⁴ Moreover, the small actin protein ACTIN DEPOLYMERIZING FACTOR 1 (ADF1), which is involved in cytoskeleton organization, physically interacts with a new flowering time activator REM16, an AP2/B3-like transcription factor, to promote *FT* expression.²¹⁵

In Arabidopsis, based on flowering time analysis of FTG mutants, the frequency of late-flowering phenotypes is much greater than that of early-flowering phenotypes, indicating that the number of flowering time activators is possibly much greater than the repressors. Negative floral transition information is mainly integrated by two MADS-box transcription factors, *FLC* and

SHORT VEGETATIVE PHASE (SVP). Recent studies have revealed additional *FLC* regulators. High expression of *FLC* in winter Arabidopsis annuals is maintained by the activator complex containing FRIGIDA (FRI), *FLC* EXPRESSOR (FLX), and FLX-LIKE 4 (FLX4). However, summer Arabidopsis annuals lose *FRI* but still have *FLX* and *FLX4* to establish baseline expression of *FLC*. An RNA polymerase II C-TERMINAL DOMAIN PHOSPHATASE-LIKE 3 (CPL3) protein interacts with and dephosphorylates FLX4 to prevent precocious flowering.²¹⁶ In addition, CPL3 is involved in GA-mediated plant development. CPL3 interacts with RGA and GAI, inhibiting their degradation by the 26S proteasome, enhancing the stability of DELLA proteins.²¹⁷ *HUA ENHANCER 4* (*HEN4*) that encodes a K-homology RNA-binding protein (RBP) positively activates *FLC* under 16 °C and executes critical functions, such as repression of the juvenile-to-adult vegetative phase and subsequent floral transition.²¹⁸ Mediator is a transcriptional cofactor complex widely existed in all eukaryotes. Arabidopsis MEDIATOR SUBUNIT 8 (MED8) has been newly identified as flowering time activator (Figure 4C). MED8 creates direction protein interaction with FPA and performs transcriptional repression of *FLC* via the bindings on both the first and sixth introns of *FLC*.²¹⁹ *SISTER of FCA* (*SSF*) encodes an FCA-like protein but plays antagonistic roles to FCA homologs in *FLC* expression. Interestingly, FCA and SSF appear to have arisen through gene duplication after dicot-monocot divergence.²²⁰ CPK32 interacts with the FCA protein, phosphorylating the WW domain of the FCA protein in a Ca²⁺-dependent manner, to maintain the interaction between FLOWERING LOCUS Y (FY) and FCA protein (Figure 4C). This interaction represses the expression of the core flowering repressor gene *FLC*.²²¹ Beyond genetic exploration, advances in biochemistry technologies decipher that FRI proteins condense in the nucleus upon cold treatment, result in quickly reducing FRI occupancy at the *FLC* regulation region and *FLC* repression.²²²

To explore how plants survive under arid climates, 335 Arabidopsis accessions from the Cape Verde Islands (CVI), which is more arid than the coastal Morocco, have been collected for whole-genome resequencing. The genetic information of CVI Arabidopsis is different from that of the Eurasia and Morocco. Due to short period of precipitation and arid climate, CVI Arabidopsis use less time to flower, which substantially increases their fitness. By accounting for the natural bolting dates and performing quantitative trait loci (QTL) GWAS analysis, data clearly show that fitness improvement is mediated by convergent de novo loss of function of FRI and *FLC*.²²³ Similarly, by using two Arabidopsis accessions widely collected from Sichuan and Tibet in China, respectively, which are naturally exposed to different growth conditions, such as UV light, seasonal precipitations and daily temperature fluctuations, genome-wide association studies (GWAS) of a recombinant inbred line (RIL) population demonstrate that the change from number 96 cytosine (C) in Tibet Arabidopsis to thymine (T) in Sichuan Arabidopsis on the SVP coding sequence results in different leaf size and aridity indexes.²²⁴

Another new perspective of flowering time regulation is nutrient, more specifically nitrogen in the soil. Nitrogen regulates plant growth primarily in two ways. In one way as a nutrient element, inorganic nitrogen in the soil mainly exists in two forms, nitrate nitrogen and ammonium nitrogen. In another way as a signal affects the development stages of plants. Plants living in waterlogged soil, such as rice, mainly absorb reduced ammonium nitrogen, while plants living in aerobic soil, such as corn and wheat, mainly absorb oxidized nitrate nitrogen. From the perspective of flowering time regulation, nitrogen deficiency significantly accelerates flowering in plants, while nitrogen overdose probably delays flowering time and fruit maturity. A recent review paper pinpoints that multiple flowering time pathways involved in a nitrogen-dependent manner. Low nitrogen (N) mediated precocious flowering is because of induced expression of *CO*. But in *cry1* mutant, which is insensitive to N changes, the expression level of *CO* does not change with alterations in nitrogen concentration. Thus, transcriptional regulation of *CO* plays a crucial role in nitrogen-mediated flowering control.²²⁵ FLOWERING BHLH 4 (FBH4) is a highly phosphorylated protein, and its phosphorylation status remains a low level under low nitrogen conditions. The transcriptional activation activity of *CO* is regulated by the phosphorylation status of multiple serine/threonine residues of FBH4 and its subcellular localization. The reduced phosphorylation of FBH4 leads to its accumulation in the nucleus, thereby promoting the expression of *CO*. Additionally, low nitrogen conditions inhibit SnRK1 activity, which can directly phosphorylate FBH4, and decreased

levels of FBH4 phosphorylation active *CO* transcription.²²⁶ NRT1.13, also known as NPF4.4 is a potential transporter belonging to the *NRT1/PTR/NPF* family. Under normal-nitrate conditions, the *nrt1.13* mutant plants slightly delay flowering time up to 3 to 4 days compared to the wild-type Col-0 plants. Interestingly, under low-nitrate conditions, *nrt1.13* needs approximate 20 days to flowering, which allows growth of additional 14 rosette leaves compared with Col-0. In addition, the delayed flowering time under low-nitrate conditions lost in *flc nrt1.13* double mutant. Therefore, the nitrate-dependent flowering mediated by NRT1.13 is mainly achieved through the regulation of its downstream gene *FLC*.²²⁷ In rice, nitrogen induction of flowering is mediated by glutamine (Gln) rather than NH₄⁺. Mutants of *N-mediated heading date 1* (*Nhd1*) exhibit delayed flowering time, which is insensitive to nitrogen supply under both LD and SD conditions. Gln promotes the transcription levels of *Nhd1*, and accumulated Nhd1 directly activates the florigen gene *heading date 3a* (*Hd3a*), which could be a major part of nitrogen determinate heading date regulation mechanisms in rice.¹⁹³

Recently, N⁶-methyladenosine (m⁶A) mRNA modification has been found to be important for gene expression. *FIONA1* (*FIO1*), an unknown nucleus-localized m⁶A methyltransferase, determines m⁶A modifications in more than 2000 Arabidopsis transcripts. Interestingly, the flowering time of *fio1* mutant is significantly earlier under long days.²²⁸ In addition, a family of small proteins FLOWERING PROMOTING FACTOR1-like proteins, among which FPL1 and FPL7 in *Brachypodium distachyon*, have been identified as flowering time activators by accumulation of *BdFT1*.²²⁹

Neofunction and subfunction of classic flowering time genes

Beyond regulating flowering time, some FTG orthologs are also critical to subsequent developmental stages after floral transition. With the advancement in genome sequencing, gene editing and transformation technology, a series of unexpected findings have been made in different plant species, especially crops. Soybean, one of the most important oil crops, is a typical photoperiod-sensitive plants and its seed size is also photoperiod dependent. Generally, long-day and short-day preferable soybean plant species produce larger seeds under long-day and short-day conditions, respectively. In the short-day cultivar Williams 82, *GmCOL2b*, one of four CO orthologs, is required for seed size reduction under the long-day condition (Figure 4D). Similarly, in Arabidopsis, *CO* is expressed in different regions of developing seeds and directly represses *AP2* expression.²³⁰

Perennial *Populus* species exhibit a reduced growth rate and simultaneously begin to develop terminal buds in autumn when the length of days and levels of temperatures fall below a certain threshold. In the European aspen, there are three FT paralogs, *PtFT1*, *PtFT2a* and *PtFT2b* are three paralogs. Evolutionarily, *PtFT1* and *PtFT2* are the results of the ancient Salicoid whole-genome duplication event. Domestically, *PtFT2a* and *PtFT2b* have been further duplicated in some local species. Functionally, *PtFT1* is surprisingly induced before bud formation during winter and is necessarily involved in bud dormancy release. However, the expression of *PtFT2a* and *PtFT2b* is redundantly upregulated in bud dormancy release and bud flush, both of which are highly important for growth robustness (Figure 4D). Differently, *PtFT2b* has a stronger circadian rhythm. Sub-functionalization of *PtFTs* might explain how *Populus* trees take full advantage of the relative benefits of immediate reproduction balanced against survivorship and next-round reproduction.¹⁹² In the perennial woody apple trees (*Malus x domestica* Borkh.), the juvenile phase usually last four to eight years before flowering and fruiting. The apple genome contains MdFT1 and MdFT2 paralogs, both of which exhibit similar expression during the bloom phase. *In vitro* experiments show that MdFT1 interacts with MdWRKY6 to promote expression of *APPLE FLORICAULA LEAFY 1* (*AFL1*).²³¹

Below the ground, FT also plays a critical role. In soybean, Williams 82 formed infection threads and nodules formed only under light condition after being infected by *Bradyrhizobium* strain USDA110. According to the transcriptome data, light-induced genes, including a group of TGACG motif binding factors GmFTs and GmSTFs, move from shoots to roots for nodulation. GmSTF3 is phosphorylated by the rhizobium-activated calcium- and calmodulin-dependent protein kinase (CCaMK) and further recruits GmFT2a.²⁰⁷ In addition, the genetic mapping of two offspring from the same sub-progeny with nitrogen deficient or sufficient leaf phenotypes clearly

shows a major QTL responsible for shoot-mediated root nodulation. Through detailed genetic and molecular studies, a 43-bp insertion in the promoter region of *GmFT2a* leads to lower expression and inhibits its translocation from shoots to roots (Figure 4D). In roots, GmFT2a interacts with the nuclear factor GmNFYA-C, which activates the symbiotic signaling.²³² In Arabidopsis, comprehensive structural and biochemical analyses show that CO uses the B-Box and CCT domains to form a trimeric complex with the nuclear factors NF-YB and NF-YC. The complex specifically interacts with CO responsive elements (COREs) in the promoter of *FT*.²³²

Although some *FRI*-containing Arabidopsis ecotypes require a long period of cold treatment before floral status is acquired. *B. napus* varieties have three major ecotypes. There is essentially no cold requirement for spring types widely grown in North America, which are planted after spring and harvested before winter. Semi-winters domesticated in the last century in China only require a weak vernalization treatment and are grown in regions with mild winters. Most European rapeseeds are winter ecotypes, which need to experience an extended period of cold in winter prior to flowering in the coming spring. Thus, spring *B. napus* is annual and semi-winter and winter are biennial. Some of semi-winter *B. napus* cultivars in China prefer 8 to 10-hour day lengths for flowering promotion. Globally, in terms of vernalization requirements, accessions with different ecotypes and growth habitats might establish various regulatory patterns. For example, pangenome of *B. napus* facilitates characterization of ten *BnFLC* paralogs genes, which are localized to A1, A2, A3, A10, and C2, C3, C9 subgenomes. Different *BnFLC* paralogs have very different gene expression levels in three ecotypes. In the spring type, the total expression levels of *BnFLCs* are much lower and the expression of each *BnFLC* is quickly reduced upon cold treatment. Interestingly, the two closest paralogs *BnaC2.FLC* and *BnaC9.FLC* show relatively stable cold expression. Another two closest paralogs *BnaA2.FLC* and *BnaA10.FLC*, harbouring several different lengths of transposon insertions, have a moderate expression decay rate in response to the cold treatment.¹⁹⁸ Different gene expression regulatory elements,²³³ gene copy numbers, varied gene structures²³⁴ and expression patterns of *BnFLCs* likely indicate that *BnFLCs* undergo subfunctionalisation for ecotype formation and flowering time adaptation, which still need much further genetic evidence. More interestingly, by studying two diploid polycarpic perennial Arabidopsis species *Crucihimalaya himalaica* and *Erysimum nevadense*, *FLC* and *FLC*-like genes *FLOWERING LOCUS M (FLM)* and *MAF (MADS AFFECTING FLOWERING)* were required for vernalization and polycarpic perennial flowering. To overexpress either *EnFLC* or *EnFLM* in the winter-annual *Brassica napus* Zhong Shuang 11 (ZS11) could resume the growth of new axillary shoots with rosette leaves and even flowers after vernalization,²³⁵ which indicates the role of *FLC* and *FLC*-like genes in the transition from polycarpic perennial to biennial and annual flowering behavior in Brassicaceae (Figure 4D).

In the last five years, multiple new FTG and novel flowering time regulatory mechanisms have been reported. Scientists use a combination of genetic mapping, sequencing omics, evolutionary, biochemical and gene editing tools to uncover detailed functions of FTG in model plants and crops. Increased experimental and analytical data clearly show that FTG homologs and paralogs develop conserved and unexpected new functions in different plant species. Scientific advancements have provided great insights into crop modernization. For example, how to precisely adjust flowering time and reproductive maturation for widening planting zones is of great interest. FTG obviously could be widely used for the improvement of many agronomic traits,²³⁶ such as bud outgrowth, nitrogen usage and drought resistance. However, how FTG can potentially be applied to different plant species requires cross-discipline collaboration. Flowering time could change much earlier for summer high plants²³⁷ or be largely delayed in biennial *B. napus*,²³⁸ which raises major conflicts between global environmental effects and agriculture efforts. What would our generations do?

FLOWER DEVELOPMENT

The development of flowers starts soon after FMs emerge on the flanks of the SAM. In a typical flower, such as those of Arabidopsis, four types of floral organs (i.e., sepals, petals, stamens, and carpels) differentiate from the FMs and arrange in concentric whorls from the outermost to the center (Figure 5A). In different groups of flowering plants, however, flowers exhibit a

remarkable diversity in number, type, size, shape, color, fusion, and arrangement of floral organs and floral symmetry. Thanks to extensive studies in two pioneering model plants, Arabidopsis and snapdragon (*Antirrhinum majus*, Plantaginaceae), as well as quite a few species from the Asteraceae, Solanaceae, Ranunculaceae, Papaveraceae, Poaceae, and Orchidaceae, much has been learned about the molecular mechanisms underlying the development of diverse flowers in the past decades. We now know that no matter how diverse the flowers are, all floral organs undergo four key developmental processes, i.e., initiation, identity determination, morphogenesis and maturation (Figure 5A).²³⁹ It is the evolutionary modifications in these processes that have driven morphological changes of flowers, even the evolutionary novelties.²³⁹ To comprehensively understand the developmental mechanisms of flowers, we here first outline how a typical flower is made by using the Arabidopsis flower as an example and then explore how flowers with diverse morphology and structures are formed.

Initiation of Arabidopsis floral organs

In Arabidopsis, shortly after the formation of the FM, sixteen floral organ primordia develop centripetally from groups of founder cells through cell proliferation.²⁴⁰ During this process, the relative positions and total number of floral organs are determined. The precise positioning of floral organs largely depends on the antagonistic interplay between auxin and cytokinin (Table S5). First, auxin maxima determine initiation sites, mediated by genes controlling the biosynthesis and polar transport of auxin, such as the *YUC* and *PIN-FORMED (PIN)* gene family members. Inactivation of these genes generally produces flowers with fewer floral organs or even a naked pin-like stem, as seen in the triple or quadruple mutants of *yuc1*, *yuc2*, *yuc4*, and *yuc6*, or in the *pin1* mutant.^{241,242} Second, cytokinin signaling shapes inhibitory fields surrounding primordia to maintain auxin maxima. For instance, mutation of the cytokinin-signaling inhibitor *ARABIDOPSIS HISTIDINE PHOSPHOTRANSFER PROTEIN6 (AHP6)* produces additional sepals and petals and disrupts floral phyllotaxy.²⁴³

The total number of floral organs is mainly dependent on the stem cell activity in the FM. The stem cell activity is maintained by a *WUS-CLV3* homeostasis loop, in which *WUS* activates the expression of *CLV3*, but *CLV3* represses *WUS*.²⁴⁴ When *WUS* is inactivated, the FM becomes diminished, resulting in fewer floral organs,²⁴⁵ whereas when *CLV3* is silenced, the FM becomes enlarged, leading to an increase in floral organ number, especially stamens and carpels.²⁴⁴ The stem cell activity is terminated once all floral organs are initiated, which is timely controlled by *WUS* repressors, such as *AGAMOUS (AG)*, *KNUCKLES (KNU)*, *SUPERMAN (SUP)*, and *CRABS CLAW (CRC)*.²⁴⁶ Notably, while the expression of *AG* is activated by *WUS*, *AG* in turn either directly turns off the expression of *WUS*, or indirectly functions via *KNU* and *CRC*.^{246,247} As expected, inactivation of these repressors produces flowers with indeterminate FMs and extra floral organs.²⁴⁶

In addition, there are other factors affecting floral organ initiation. For instance, the founder cell identity genes, represented by *DORNROSCHEN (DRN)*, *DORNROSCHEN-LIKE (DRNL)*, and *PUCHI*, determine the fate of founder cells; collective mutation in these genes results in decreased floral organs.²⁴⁸ The organ boundary specification genes, represented by *CUC1* and *CUC2*, are required for organ separation; inactivation of the *CUC* genes causes the fusion of intra- or inter-whorl floral organs,²⁴⁹ while inactivation of their inhibitors (i.e., *miR164a*, *b*, and *c*) leads to additional sepals and petals.²⁵⁰

Identity determination of Arabidopsis floral organs

Among the sixteen floral organ primordia, the outermost four finally develop into sepals, the subsequent four into petals, the next six into stamens, and the innermost two into carpels. This precise patterning is governed by combinatorial action of four classes of floral organ identity genes, as described by the ABCE model. According to this model, A and E function genes specify sepals; A, B, and E function genes, petals; B, C, and E function genes, stamens; and C and E function genes, carpels.²⁵¹ Inactivation of these genes causes homeotic transformations between floral organs (Table S5). For example, loss-of-function mutations of B function genes result in the conversion of petals into sepals and stamen into carpels.²⁵² In Arabidopsis, the A function is conferred by *APETALA1 (AP1)* and *AP2*, the B function by *AP3* and *PISTILLATA (PI)*, the C function by *AG*, and the E func-

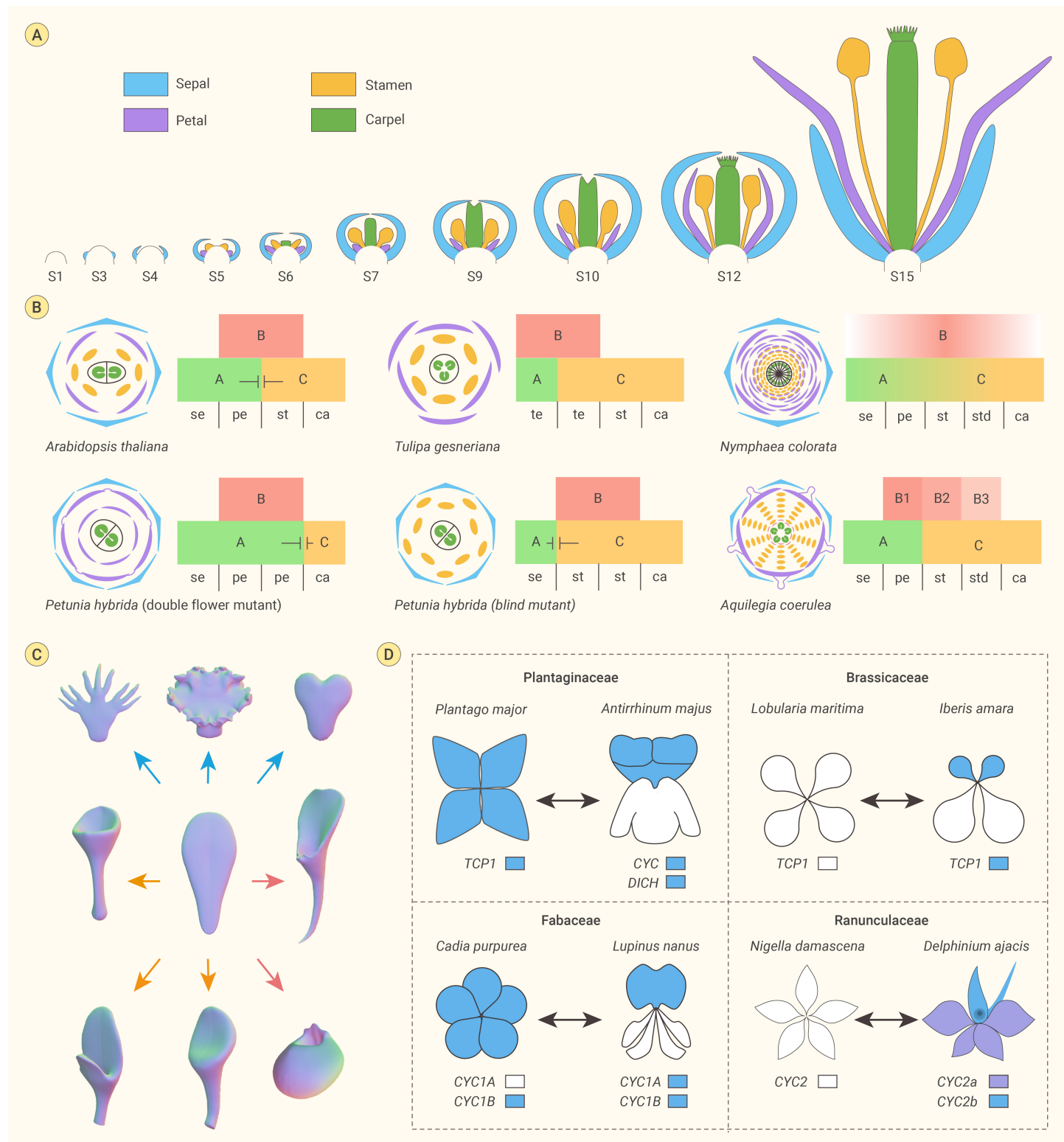


Figure 5. Development and evolution of flowers (A) A schematic diagram showing the development of an *Arabidopsis* flower. (B) Expression changes in floral organ identity genes result in flowers with different organ composition. In the models, A, B, and C represents different functions of floral organ identity genes. The negative regulatory relationships between A- and C-function genes are indicated by bars. Se, sepal; pe, petal; te, tepal; st, stamen; std, staminode; ca, carpel. (C) Modifications of the morphogenesis process result in petals with different shapes. Blue arrows point to petals with marginal elaborations; pink arrows point to petals with dorsal elaborations; and yellow arrows point to petals with ventral elaborations. (D) Changes in CYC-like genes result in floral symmetry transition. The filled colors represent the expression or functional domains of the focal genes, white indicates that the focal gene is not expressed.

tion by *SEP1*, *SEP2*, *SEP3*, and *SEP4*.²⁵³ Notably, of these genes, all but *AP2* encode MADS-box transcription factors that form specific protein complexes to specify floral organs.²⁵⁴ These complexes include AP1-SEPs-AP1-SEPs for sepals, AP3-PI-AP1-SEPs for petals, AP3-PI-AG-SEPs for stamens, and AG-SEPs-AG-SEPs for carpels; this is the so-called floral quartet model.²⁵¹

It should be noted that the precise spatial regulation of floral organ identity genes involves complex molecular mechanisms. First, cross-regulatory repression occurs among these genes.²⁵⁵ For instance, the A-function protein AP2 forms a protein complex with TOPLESS (TPL)-HISTONE DEACETYLASE19 (HDA19) to repress the expression of the B- and E- func-

tion genes (*AP3*, *PI* and *SEP3*) in sepals. The AP2-TPL-HDA19-complex, along with the AP1-LEUNIG (LUG)-SEUSS (SEU) complex, prevents the expression of the C-function gene *AG* from sepals and petals. The AG-SEP3-AG-SEP3 tetramer containing C- and E-function proteins represses the expression of the A-function gene *AP1* in stamens and carpels. Second, some boundary specification genes negatively regulate the expression of floral organ identity genes. For example, *RABBIT EARS* (*RBE*) restricts the expression of the C-function gene *AG* to stamens and carpels;²⁵⁶ *SUP* represses the expression of the B-function gene *AP3* in carpels;²⁵⁷ miR172 prevents the mRNA accumulation of the A-function gene *AP2* in the inner two whorls.²⁵⁸ Clearly, these multilayered regulatory networks set sharp boundaries that precisely determines types and respective numbers of the floral organs in Arabidopsis, revealing a complexity that far exceeds the ABC model.

Morphogenesis of Arabidopsis floral organs

The morphogenesis process involves division and expansion of cells, coupled with the establishment of polarities along the adaxial–abaxial, medial–lateral, and proximal–distal axes, which collectively determine the final size and shape of a floral organ. In a mature flower of Arabidopsis, the sepals and petals are laminar; the stamens consist of a filament supporting a pollen-producing anther, and the carpels fuse to form a gynoecium, which is composed of three distinct regions: a basal ovary containing ovules, a style, and an apical stigma. It is interesting that while these floral organs follow distinct developmental trajectories,^{259–262} the core regulatory programs governing their morphogenesis share remarkable similarities (Table S5).

In general, cell division and expansion underlie the growth rate of a floral organ, ultimately defining its final size.^{239,263} Specifically, cell proliferation which determines the total number of cells in an organ is positively controlled by *ANTEGUMENTA* (*ANT*), *AINTEGUMENTA-LIKE6* (*AIL6*), *ORGAN SIZE RELATED1* (*OSR1*), *GROWTH-REGULATING FACTOR* (*GRF*) and *GRF-INTERACTING FACTOR* (*GIF*), but negatively regulated by *BIG BROTHER* (*BB*), *DA1*, and *TEOSINTE BRANCHED/CYCLOIDEA/PCF4* (*TCP4*). Mutations in the positive regulators reduce cell numbers, leading to smaller floral organs, whereas inactivation of the negative ones increase cell numbers, resulting in larger organs. In contrast, cell expansion, which is responsible for cell size, is activated by *ARGOS-LIKE* (*ARL*) and *MED8*, but repressed by *AUXIN RESPONSE FACTOR8* (*ARF8*). Inactivation of these genes results in size reduction or enlargement of cells, thus affecting overall organ dimensions. Additionally, some genes, such as *MED25* and *BIG PETALp* (*BPEp*, a petal-specific transcript of *BPE*), repress both cell proliferation and expansion, thereby determining when the cells in floral organs stop growing.

The adaxial–abaxial, medial–lateral, and proximal–distal patterning determines the final shape and architecture of a floral organ.^{264–266} The adaxial identity is defined by *PHABULOSA* (*PHB*), *PHAVOLUTA* (*PHV*), *REVOLUTA* (*REV*), *ASYMMETRIC LEAVES1* (*AS1*), *AS2*, and transacting small interfering RNAs (ta-siRNAs). The abaxial fate is determined by *KANADI1* (*KAN1*), *KAN2*, *KAN3*, *YABBY3* (*YAB3*), *FILAMENTOUS FLOWER* (*FIL*), *ARF3*, *ARF4*, and miR165/166. In sepals, petals, and carpels of Arabidopsis, balanced expression of these adaxial and abaxial regulators establishes laminar structures.²⁶⁶ By contrast, stamen development involves rearrangement of adaxial–abaxial polarity genes, leading to the differentiation of the anther and the filament.²⁶⁷ Lateral outgrowth of sepals and petals is controlled by *WUSCHEL-RELATED HOMEBOX* (*WOX*) gene family members *WOX1* and *PRESSED FLOWER* (*PRS*); mutations of these genes result in extremely narrow sepals and petals.²⁶⁸ Distal growth of sepals, petals, and stamens is regulated by a C2H2 zinc finger transcription factor *JAGGED* (*JAG*); inactivation of this gene produces sepals and petals with a reduced distal part and a jagged margin and stamens with abnormal anthers.^{261,269,270} Notably, the medial–lateral and proximal–distal polarities of carpels are defined by distinct regulatory mechanisms compared to other floral organs, as detailed in Section “Fruit development”.

Maturation of Arabidopsis floral organs

The maturation process mainly involves two key developmental events. One is the differentiation of epidermal cells with diverse decorations, giving rise to floral organs with characteristic surface textures and color patterns.

The other is the formation of germlines in stamens and carpels, endowing male and female organs with reproductive abilities. Given that the germline specification will be covered in Section “Germline specification and fertilization”, this section focuses specifically on sepal and petal maturation.

In Arabidopsis, sepal maturation is characterized by the differentiation of giant pavement cells on the abaxial epidermis through endoreduplication.²⁷¹ The identity of giant cells is specified by HD-ZIP IV transcription factors *ARABIDOPSIS THALIANA MERISTEM IDENTITY1* (*ATML1*) and *HOMEODOMAIN GLABROUS11* (*HDG11*), as well as those encoding transmembrane proteins *ARABIDOPSIS CRINKLY4* (*ACR4*) and *DEFECTIVE KERNEL1* (*DEK1*).²⁷² These genes function upstream of the cell cycle inhibitor *LOSS OF GIANT CELLS FROM ORGANS* (*LGO*), which directly promotes endoreduplication and subsequent giant cell formation.²⁷²

Petal maturation involves the formation of conical cells on the adaxial epidermis and the whitening of the distal blade region. While the conical cell identity genes remain to be identified, several key genes governing their morphogenesis have been characterized (Table S5). For example, *KATANIN1* (*KTN1*) regulates the spatial organization of microtubule arrays during conical cell development; null mutation of *KTN1* leads to decreased tip sharpening of conical cells.²⁷³ *RHAMNOSIDE BIOSYNTHESIS1* (*RHM1*) mediates the synthesis of the pectic polysaccharide rhamnogalacturonan-I, essential for normal conical cell expansion; inactivation of *RHM1* causes helical twisting of the expanding conical cells.²⁷⁴ The petal whitening involves the chloroplast-to-leucoplast conversion.²⁷⁵ A recent study has shown that *CINCINNATA* (*CIN*)-like TCP proteins play key roles in controlling the petal color. In particular, *TCP4* promotes the petal whitening by directly repressing chlorophyll biosynthesis-related genes, such as *PROTOCHLOROPHYLLIDE REDUCTASE* (*PORB*), *DIVINYL REDUCTASE* (*DVR*), and *SUPPRESSOR OF OVEREXPRESSION OF CONSTANS 1* (*SOCT*).²⁷⁶

Developmental mechanisms underlying the diversity of flowers

As mentioned above, angiosperms exhibit fantastic floral diversity across multiple aspects. In particular, many plants have evolved flowers that differ significantly from those of Arabidopsis, including flowers with bilateral symmetry (or zygomorphy), undifferentiated perianth, elaborate petals, novel organ types, or lacking certain organ types (e.g., apetalous, unisexual, or perianthless flowers). Extensive investigations of species from representative flowering plant lineages clearly suggest that this diversity arises through modifications of the genetic programs involved in the four main floral organ developmental processes.²³⁹ Due to the space limitations, we present selected examples illustrating how changes of genes and programs involved in these processes result in diverse flowers.

The first group of examples illustrates the power of the dynamic regulation of the floral organ identity determination program (Figure 5B). It appears that many aspects of floral diversity can be attributed to boundary shifts of floral organ identity genes. For instance, in non-grass monocots (such as tulips and lilies of the Liliaceae), outward expansion of B-function genes into the first whorl leads to the generation of the undifferentiated, petaloid perianth, exemplifying the shifting boundary model.²⁷⁷ In some basal angiosperms (such as Amborellaceae and Nymphaeaceae), the expression gradients of floral organ identity genes result in gradual morphological transitions between adjacent floral parts, representing the fading borders model.²⁷⁸ Likewise, inward shift of the A-C boundary transforms stamens into petals, producing “double flowers”, as frequently seen in many ornamentals of *Prunus*, *Dianthus*, *Petunia*, *Rosa*, and *Nicotiana*.^{279,280} By contrast, outward expansion of the C-function genes convert petals into stamens, generating apetalous flowers, as seen in *Petunia hybrida blind* (*bl*) and snapdragon *fistulata* (*fis*) mutants and some ranunculaceous species.^{281,282} In addition, *AP3*-like B-function gene duplication followed by sub- or neo-functionalization leads to the production of brand-new floral organ types, such as the labella of orchids (Orchidaceae) and the staminodes of *Aquilegia*.^{283–285}

The second group of examples demonstrates how modifications of morphogenesis and maturation processes create flowers with elaborate floral organs (especially petals) in shape and color pattern (Figure 5C). For instance, during morphogenesis, heterogeneous cell division and expansion along petal edges lead to the formation of petals with teeth, lobes, fringes, or ruffles at the margin (i.e., marginal elaboration).^{263,286,287} By contrast, localized

cell division followed by anisotropic cell expansion results in spurred or pouched petals (i.e., dorsal elaboration).²⁸⁸⁻²⁹⁰ In addition, shifts in expression domains of adaxial and/or abaxial polarity genes generate various peltate petals (i.e., ventral elaboration).^{266,291} During maturation, co-option and dynamic regulation of programs involved in the differentiation of specialized cells (e.g., the pigment biosynthesis or trichome differentiation pathways) lead to the surface elaboration of floral organs, generating petals with different color patterns, trichome clusters, or hair pads.^{263,292} Notably, these mechanisms often combine to produce exceptionally complex petals, as seen in *Ophrys* (Orchidaceae) and *Nigella* (Ranunculaceae).^{293,294}

The third group of examples shows how floral diversity arises through rewiring of different programs or complicated interactions between processes, as exemplified by zygomorphic flowers (Figure 5D). According to the polar coordinate model, the establishment of floral zygomorphy depends on differential regulation of floral symmetry genes along the dorsoventral floral axis.²⁵² In snapdragon, which produces zygomorphic flowers, *CYCLOIDEA* (*CYC*) and *DICHOTOMA* (*DICH*) of the TCP family define the dorsal identity of the flower, while *DIVARICATA* (*DIV*) of the MYB family specifies the ventral identity of the flower (Table S5).²⁹⁵⁻²⁹⁷ Simultaneous mutations of *CYC* and *DICH* generate actinomorphic flowers with all petals and stamens becoming ventralized, whereas inactivation of *DIV* affects the ventral identity of the flower in the petal whorl.²⁹⁵ Evolutionary studies revealed that the *CYC*-like genes experienced multiple rounds of duplication events and some of the duplicate copies have been independently recruited into the floral regulatory networks of different lineages with zygomorphic flowers.²⁹⁸⁻³⁰⁵ Moreover, the *CYC*-like genes can regulate genes involved in different developmental processes of floral organs, eventually resulting in the evolution of diverse zygomorphic flowers.³⁰⁵⁻³⁰⁸

FRUIT DEVELOPMENT

Following floral organ specification, the development of the gynoecium and fruit marks the entrance of final reproduction stages. Fruits protect and nourish the developing seeds, and at maturation, render the seeds with diverse dispersal strategies. The development of the fruits is therefore characterized the angiosperms and considered to be adaptive in evolution. In a strict definition, fruit is derived from the ovary by differentiation and elaboration in post anthesis development stages. However, fruit can also be developed from flower associated organs, such as hypanthium and receptacles, or in an extreme case, the whole inflorescences. In the past decades, our understanding on the fruit development have been significantly advanced by the studies in model system, exemplified by *Arabidopsis* (dry fruit) and tomato (fleshy fruit). In this section, we summarize recent breakthroughs in identifying novel components of the molecular networks involved gynoecium patterning and fruit development in *Arabidopsis* (Figure 6 & Table S6).

From the floral meristem to a gynoecium primordium

In *Arabidopsis*, the gynoecium is composed of two congenitally fused carpels that develops in the middle of the FM. The initiation of the two carpel primordia terminates the FM. The carpel identity is determined by the C-class MADS-box gene, *AGAMOUS* (Figure 6A). Due to the functional importance of AG in specifying both the male and female reproductive organs, its expression is strictly restricted in the center of the FM by several transcription factors and chromatin remodelers. For example, during the vegetative growth phase, AG is repressed by Polycomb Repressive Complex 2 (PRC2) modifying histone 3 proteins at the lysine 27 position (H3K27me3) in the second intron locus.^{247,309,310} In the FM, AP2 recruits TPL and HDA19 to exclude the AG from the outer two whorls.³¹¹⁻³¹³

The initiation of AG expression of starts at stage 3 of flower development, long before the gynoecium primordium is visible as an elliptical flattened protrusion in the middle of the FM. At stage 3, WUS works with LEAFY (LFY) and PERANTHIA (PAN) activate the expression of AG.³¹⁴⁻³¹⁷ Expression of AG is indispensable for FM termination as *ag* mutation results in ectopic expression of WUS and indeterminate growth of the FM with a flower-in-flower phenotype.³¹⁸ AG shut down WUS and FM indeterminacy via looping the DNA at WUS locus or inducing repressive epigenetic modification on the histones of WUS chromatin (Figure 6A).³¹⁹⁻³²²

AG initiate the gynoecium developmental pathway by activating the

expression of *CRC*, which is subsequently detectable upon AG expression in the gynoecium primordium. CRC promotes the auxin biosynthesis by activating the auxin biosynthetic enzyme *YUC4* expression.³²² The auxin maxima generated by *YUC4* expression promote the cell differentiation and expansion during the early developmental stages of the gynoecium (Figure 6A). In addition, a yeast one-hybrid experiments show CRC associates with the promoters of 119 TFs including *ETTIN* (*ETT*), *FRUITFULL* (*FUL*), *NGATHA 2* (*NGA2*) and *JAGGED* (*JAG*), many of which play critical roles in gynoecium development.³²³

Gynoecium patterning along the polarity axes

After initiation, the gynoecium primordium continues to grow and differentiate into a single cylindrical tube shape. During the gynoecium patterning process, three growth axes can be observed with the asynchronous establishment of distinct tissues and cell types. The development of the replum in the middle region and two ovary locule and valves in the lateral defines the medio-lateral axis. The apical-basal axis is manifested by the differentiation of papillae cells, style, ovary and gynophore from the apex to the pedicel of the gynoecium (Figure 6C). The adaxial-abaxial axis distinguishes the inner and outer tissues of the valves similar to the leaf adaxial-abaxial axis. The medio-lateral and apical distal axis is established gradually during the gynoecium development process. In agreement with this scenario, a recently live-imaging analysis detected two orthogonal, time-shifted differentiation gradients: an early mediolateral gradient controls valve and replum morphogenesis and a late longitudinal gradient regulates style differentiation.²⁵⁹

Even though the gynoecium primordium terminates the FM, new tissues, such as septa, placenta and ovules continue to differentiation inside the gynoecium (Figure 6B). This capacity depends on the activity of Carpel Meristem Margin (CMM), which is a "meristematic-like zone" located in the medial region of the gynoecium. The re-establishment of the meristematic activity in the Carpel Margin Meristem (CMM) is co-opted by the ectopic expression of meristem identity genes and related hormone networks (Figure 6B). The expression of *STM*, required for stem cell maintenance in the SAM, is activated by CUC1 and CUC2, which encode a pair of NAC transcription factor that involved in SAM development and organ boundary definition.²⁴⁹ In the SAM, CUCs and *STM* constitute a double feedforward loop to sustain the stem cells.^{324,325} In the stem cells, *STM* promotes cytokinin biosynthesis but represses gibberellin activities to fine-tune the stem cell proliferation.^{169,170} Intriguingly, expression of *CUCs*, *STM* and cytokinin signal (visualized by *TCS:GFP*) are specifically localized in CMM, indicating the CMM cells are reminiscent of stem cells in the SAM (Figure 6C).³²⁶ Consistently, compromise either *STM*, *CUC2* function or cytokinin signaling pathway disrupt CMM identity, placenta development and ovule initiation, whilst exogenous cytokinin treatment leads to over proliferation of the CMMs.^{324,327,328} In the medial region, the replum is derived from the CMM and its identity is established by the BELL1-type homeodomain transcription factor, *REPLUMLESS* (*RPL*).³²⁹ *rpl* mutation leads to a reduction in replum size and ovule number, loss of the septum and carpel fusion defects.³²⁹ On the other hand, the development of valve identity in the lateral region is defined by the specific expression of two YABBY transcription factors, *FILAMENTOUS FLOWER* (*FIL*) and *YABBY 3* (*YAB3*), in the valves.³³⁰ The gynoecium from *fil; yab3* double mutant display severe defects in valve identities.³³⁰ In the valves, *FIL* and *YAB3* activate the expression of MADS-box transcription factor *FUL*, which specify valve identity and fruit growth by promoting cell elongation after pollination (discuss below).^{330,331} The expression domain of *RPL* and *YABs-FUL* transcription factors is mutually excluded in the respectively tissues. For example, in the medial region of the gynoecium, *RPL* is activated by the class I KNOX homeodomain transcription factor *BREVIPEDICELLUS* (*BP*) and *BP* subsequently binds to *RPL* to form a heterodimer, which in turn restricts the *YABs* gene expression to the lateral region.^{330,332} Similarly, expression of *FUL* in the valve region negatively regulates *RPL* genes.³³⁰ Therefore, tissue along the mediolateral axis of the ovary requires the maintenance of CMM activity and the antagonistic regulation of valve and replum identity genes.

Alongside the medio-lateral morphogenesis, the gynoecium primordium continues to grow both in longitudinally as a hollow tube, and then fused at the apex, where the papilla cells and style start to differentiate to manifest the apical-basal axis (Figure 6C). Gynoecium growth along the proximal-distal

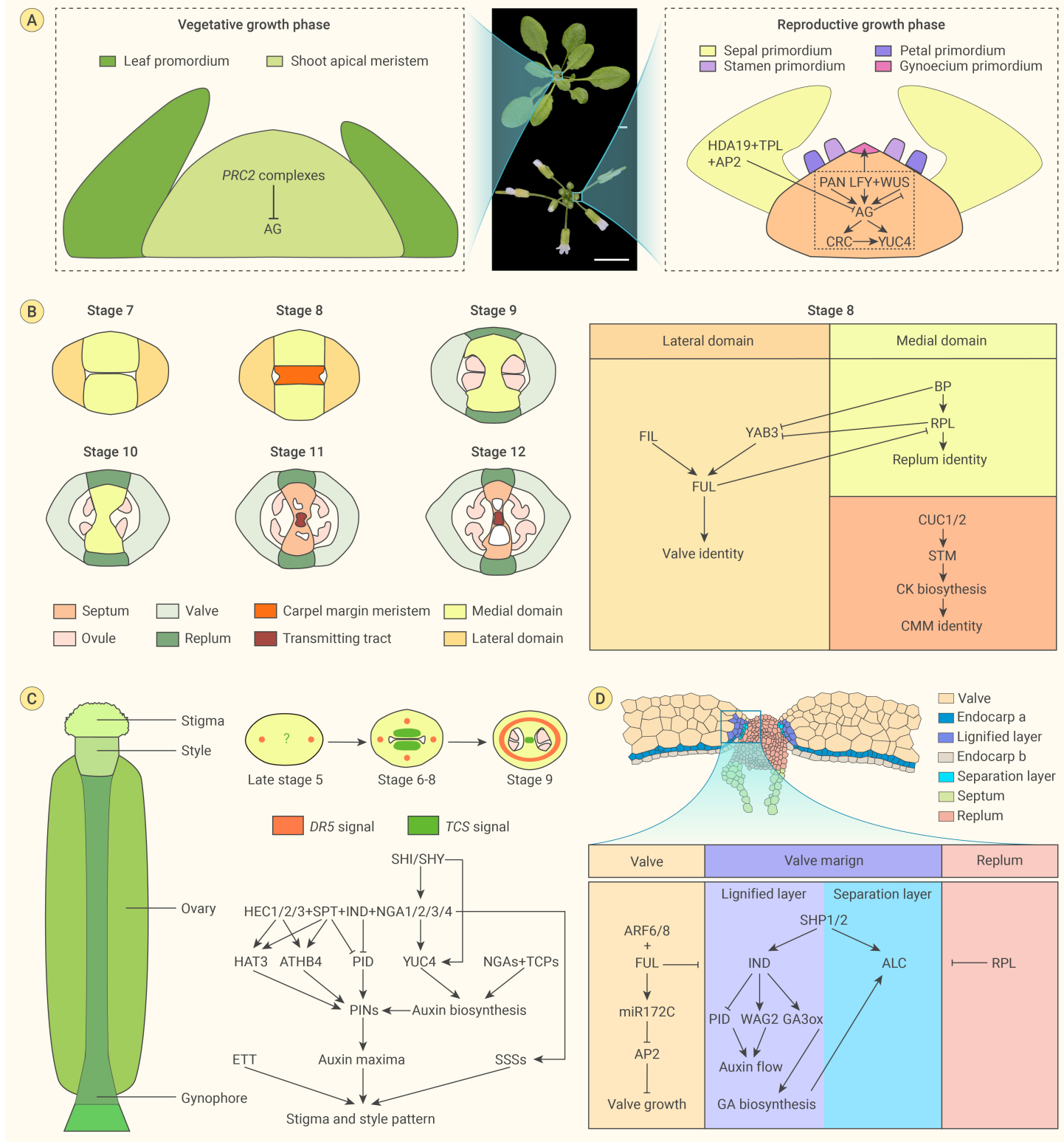


Figure 6. Developmental processes and GRNs involving in the gynoecium and fruit development (A) AG governing the initiation of gynoecium primordium. The left panel shows how AG is repressed in the vegetative growth phase. The right panel exhibits the GRNs induce AG expression and then gynoecium primordium initiation. (B) Complementary GRNs involving the differentiation of medial domain and lateral domain. The left panel represents the transections of gynoecium in different stages. The right panel shows the GRNs controlling the development along medial-lateral axis. (C) Auxin maximum pattern controls the differentiation of stigma and style along the proximal-distal axis. The cytokinesis and auxin dynamic are shown by gynoecium transections from stage 5 to 9. Lower right corner demonstrates GRNs determining the auxin maximum pattern which promotes the differentiation of stigma and style. (D) The differentiation of dehiscence zone in valve margin. The upper panel explains the dehiscence zone includes lignified layer and separation layer and GRNs is shown in bottom panel.

axis is promoted by the auxin maxima generated by local auxin biosynthesis and polar auxin transport.³²² Disruption of this auxin maxima in high-order *yuc* mutant or NPA-treatment abolish the differentiation of distinct tissues along the proximal-distal axis.^{333,334} The differentiation of style and stigma is

facilitated by the fusion of gynoecium apex. Studies of auxin signal reporter (DR5) revealed that the gradual fusion of the gynoecium apex is associated with a dynamic distribution of auxin signaling patterns (Figure 6C). Right upon gynoecium initiation, two DR5 foci are detected in the lateral region at

the primordium apex, manifesting the two carpel primordia. Later in development, two additional medial auxin foci are established and subsequently the auxin signaling pattern was transformed into a ring-form at the gynoecium apex (Figure 6C).^{335,336} bHLH transcription factors, SPATULA (SPT), INDEHISCENT (IND) and HECATE1/2/3 (HEC1/2/3) are indispensable for the gynoecium apex fusion as *spt*, *spt;ind* or *spt;hecs* mutations result in severe defects in the style with unfused apices. The stepwise dynamic auxin distribution is blocked at the two lateral foci stage in both *spt* and *ind;spt* mutants, therefore failing to process into the four-foci stage and prevented from reaching the ring-pattern auxin distribution required to style development.³³⁶ At the gynoecium apex, SPT and IND form heterodimers to repress the PINOID (PID) expression.³³⁷ PID encodes AGC3-type protein kinase that phosphorylate PIN proteins to facilitate their polar localization at the plasma membrane.³³⁸ As a result of this PID repression, PIN proteins become apolar localized leading to accumulation of auxin signaling at the medial region, which is subsequently transformed into a radial ring pattern at the gynoecium apex.^{328,337} In addition, SPT acts synergistically with HEC1-3 to regulate the expression of the adaxial regulators *HOMEOBOX ARABIDOPSIS THALIANA 3* (HAT3) and *ARABIDOPSIS THALIANA HOMEOBOX 4* (ATHB4), mediating the final step in the style radialisation process by establishing the ring-shaped auxin maximum (Figure 6C).³³⁹ Recently, it was uncovered that SPT activity was monitored at the protein level by O-glycosylation, indicating SPT activity is fine-tuned at multiple level to ensure the proper development of the style.³⁴⁰

Auxin maxima and the style development

Immediately after gynoecium fusion, the distal tissues, including style and stigmatic papillae cells, started to differentiate. It has long been suggested that the specification of style and stigma is associated with the auxin maxima at the gynoecium apex.³⁴¹ As discussed above, the ring-formed auxin distribution promotes the fusion of the gynoecium apex (Figure 6C). Subsequently, this ring auxin maxima is sustained by both polar auxin transport and local biosynthesis in the following developmental stages. Early in gynoecium development, IND and SPT was activated sequentially by HEC proteins and B3 transcription factor NGAs. Then IND-SPT-HECs-NGAs high-order protein complexes are formed to promote the apolar localization of PIN proteins, facilitating the establishment of the auxin ring.³⁴² In the separately but related pathway, NGAs was activated by the expression of *SHORT NTERNOSES/STYLISH* (SHI/STY), which encodes a zinc-finger transcription factor.³⁴³ NGAs subsequently cooperates with STY to induce auxin biosynthesis by activating *YUC4* expression at the gynoecium apex.^{333,342-345} In addition, NGA also physically associates with TCP transcription factors to control auxin biosynthesis at the gynoecium apex.³⁴⁶ With the action of both auxin transport and local synthesis, an auxin gradient is produced along the distal-proximal axis of the gynoecium with the maximum in the apex.³³⁴ The auxin signaling input was translated into transcriptional output by ARFs, which encode a family of transcription factors mediating auxin response.³⁴⁷ For example, *ARF3*, also known as *ETT*, is specifically expressed in the gynoecium apical region and in the replum.³⁴⁸ *ett* mutant gynoecia exhibit severe defects, including split style, polarity changes in the style and gynophore regions and a reduction in the ovary size.³⁴⁹ *ETT* is a unique ARF that different from others in structure by possessing the ETT-specific domain in the C terminal, which was recently demonstrated to interact with auxin directly.³⁴⁹ Surprisingly, *ETT* interacts with a plethora of transcription factors in an auxin-sensitive manner.^{349,350} Therefore, *ETT* could act as an interpreter of auxin levels in the apical part of the gynoecium and translate the subtle auxin signal difference into distinct protein interactome and downstream transcriptomes.³⁵¹ How *ETT* protein sensing the auxin gradient to elaborate the stigma, style and replum is an intriguing question that warrant further investigations. Finally, the growth of the style and stigma is regulated by *STIGMA AND STYLE STYLIST* (SSS) proteins. SSSs encode proteins belong to a previously uncharacterized family but are angiosperm-specific.³⁵² SSSs act downstream of NGA to direct the apical gynoecium patterning by controlling cell expansion and elongation.³⁵²

Fruit growth and dehiscence

The Arabidopsis gynoecium starts to grow along the longitudinal axis and becoming a fruit after pollination.³⁵³ The fruit growth is triggered by the

mobile signals generated from the fertilized ovules at stage 13.³⁵⁴ Recently, a live-imaging analysis revealed that the active growth of the fruit during the post-pollination stages was largely driven by extensive cell expansions, whilst cell divisions have little if any contributions.³⁵⁴ As noted above, FUL is a valve factor that positively regulates fruit growth by promoting cell elongation.³³¹ In the valve cells, FUL interacts with ARF6/8 to form heterodimers, which destabilize the *AP2* mRNA by direct activating the expression of *miR172C* (Figure 6D).³⁵⁵ Ectopic expression of a *miR172*-resistant version of *AP2* in the valves restricts fruit growth, which phenocopied the *ful* mutant.³⁵⁵ Recently, it was discovered that cytokinin has a negative effect on fruit growth. *CYTOKININ OXIDASE/DEHYDROGENASE 7* (*CKX7*), which encodes a cytokinin degradation enzyme, is actively transcribed in the fruit valves. Expression of *CKX7* is activated by the MADS-box transcription factor *SEED-STICK* (STK). As a result, both *stk* and *ckx7* mutant produce shorter fruits.³⁵⁶ STK was known by its function in funiculus development and ovule patterning.³⁵⁷ In this regard, it remains to be discriminate if the retard fruit growth in *stk* was originated from ovule defects or cytokinin accumulation or both.

In order to dispersal the seeds at maturation, the Arabidopsis fruits devise the dehiscent zones (DZs) in the valve margin to promote fruit dehiscence (Figure 6D). The DZ is observed as the separation layer (SL) and the lignified layer (LL) at the valve margin in the cross section of a mature fruit.³⁵³ The SL is characterized by a layer of isodiametric and parenchyma cells locate near to the replum, whilst the LL is positioned at the edge of the valve with cells developed rigid secondary walls (Figure 6D). In addition, the LL cells are connected with the lignified endocarp b (endb) layer of the valve to form a half-ring pattern in each valve.³⁵³ When the fruits mature, the valve parenchyma cells lose water and generate mechanical force that promote the valve to break at the LL, and hence, the pod dehiscence took place. In Arabidopsis, the differentiation of the DZ is governed by an intricate genetic regulatory network that has been recently extensively reviewed.³⁵⁸ In brief, the DZ identity is specified by the MADS-box transcription factor *SHATTER-PROOF1* and 2 (*SHP1/2*) and *IND*.³⁵⁹ The SL cell identity alone is established by the myb/bHLH transcription factor, *ALCATRAZ* (ALC).³⁶⁰ Expression of both *IND* and *ALC* depends on SHPs and all these four genes are negatively regulated by FUL in the valve and RPL in the replum, respectively, in a sandwich pattern to pattern the DZ (Figure 6D).³³⁰ In addition to these transcription factors, plant hormones have also been demonstrated to play a pivotal function in DZ differentiation. For example, IND mediates the PIN protein localization by dynamically regulating the expression AGC3-type protein kinase *PID* and *WAG2*. As a result, an auxin fluctuation is created during the DZ patterning process with a maximum required for early cell identity determination and a minimum for later cell differentiation.³⁶¹ Additionally, *GA3ox* was identified as another direct target of IND, which encode a metabolic enzyme involved in GA biosynthesis. In the DZ, high level GA destabilize DELLA repressors and release ALC in SL specification.³⁶² Moreover, it was recently discovered that TCS:GFP is specifically localized in the DZ and exogenous cytokinin (benzylaminopurine, BAP) application rescued the indehiscent fruit defect in both *ind* and *shp1*; *shp2* double mutant, indicating another potential output of the SHP-IND module is to monitor cytokinin homeostasis during DZ development.³⁶³ Finally, the maturation of DZ is promoted by the deposition of excessive secondary cell walls in the LL and cell disintegration in the SL, respectively (Figure 6D).³⁵⁸ These processes are mediated by the NAC transcription factor *NAC SECONDARY WALL THICKENING PROMOTING FACTOR1* (NST1) in the LL and the polygalacturonase (PG) proteins *ARABIDOPSIS DEHISCENCE ZONE POLYGALACTURONASE1* (ADPG1) and *ADPG2* in the SL, respectively.^{364,365} All together, these observations suggest the development of a simple DZ is coordinated by a complex genetic cascade with transcription factors intertwined with phytohormones. How these hormones are intercrossed to direct the DZ differentiation is an intriguingly topic that await further clarification.

GERMLINE SPECIFICATION AND FERTILIZATION

The process of sexual reproduction in flowering plants occurs within the diploid sporophyte and haploid gametophyte generations. Germline specification enables cells of the diploid sporophyte generation in plants to transition into the haploid gametophyte generation. This process ultimately leads to the production of female and male gametes (Figure 7 & Table S7). These

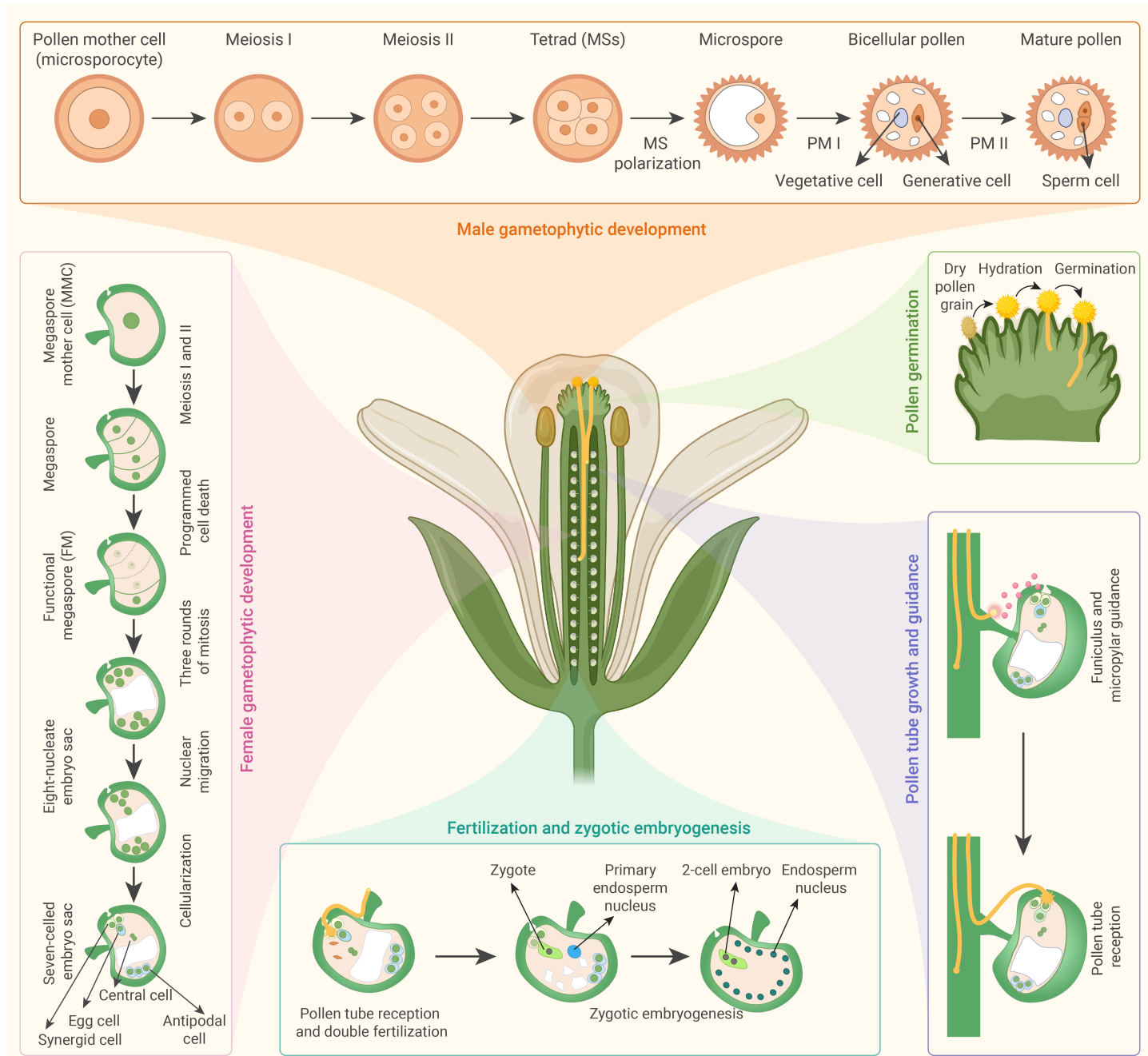


Figure 7. Gametophytic development and fertilization in Arabidopsis During microsporogenesis, the diploid pollen mother cell (microsporocyte, MS) undergoes two rounds of meiotic divisions, forming a tetrad with four haploid MSs. Subsequently, MSs are released from the tetrads. During microgametogenesis, MSs polarize and proceed to undergo highly asymmetric division, pollen mitosis I (PMI), culminating in the formation of two unequal daughter cells: a large VC and a small GC. The GC migrates into the VC to form a unique cell-within-a-cell structure. The GC undergoes the mitotic division, PMII, to produce two male gametes. During microsporogenesis, the MMC within the ovule undergoes meiosis, forming four aligned megaspores, with the one at the chalazal end survives and develops as the functional megaspore (FM) and the other three degenerate. The FM undergoes three rounds of nuclear division forming an eight-nucleated syncytium. After reposition of the two polar nuclei in the central part, cellularization partitions the syncytium into seven cells: an egg cell, a central cell, two synergid cells and three antipodal cells. Once landing on the stigma, compatible pollen hydrates to protrude pollen tube penetrating the papilla cell. In the style, the pollen tubes are attracted by attractants secreted by synergids to target the ovule. Upon contact communication of pollen tube with the receptive synergid, pollen tube and synergid collapse to enable the release of two sperm cells for double fertilization.

haploid gametes ultimately return to the sporophyte generation through fertilization. Germline specification and fertilization are crucial for the alternation of generations in plants. During this process, specific steps differentiate into distinct cell types. Although each of these cell types is transient, they are critical to the successful progression of sexual reproduction.³⁶⁶

The germline comprises cells destined for gamete production.¹¹ Germline specialization generates haploid male and female gametes. During fertilization, these gametes fuse, integrating genetic material from both parents to form diploid offspring. The sporophytic generation initiates with the zygote and persists until meiosis occurs in the megaspore mother cell (MMC) and

pollen mother cell (microsporocyte). This generation constitutes the predominant phase of angiosperm life cycles. Meiosis of the MMC and microsporocyte, generating haploid megaspores and microspores, respectively. During the gametophytic phase, the haploid megaspores and microspores undergo successive mitotic divisions, defining their developmental trajectory.^{8,11} The megaspores differentiate into the female gametophyte, whereas the microspores develop into the male gametophyte. The stage continues until fertilization occurs between the male and female gametophytes, culminating in the transition of the gametophytic generation into the sporophytic generation. This phenomenon of regular alternation between gametophytic and

sporophytic generations in the life cycle is called the alternation of generations.

Flowering plants possess multicellular gametophytes. In the anthers, microsporocytes undergo meiosis and mitosis to form pollen grains (male gametophyte), which contain two sperm cells (male gametes) that are enclosed in a pollen vegetative cell (Figure 7). The MMC, derived from a single subepidermal cell in the nucleus of the ovule embedded in the ovary of the carpel, undergoes meiosis to form a tetrad of megaspores (1n). In most flowering plants, three of these cells located at the distal end of the ovule primordium degenerate. The remaining chalazal cell subsequently undergoes three rounds of mitosis to form a multicellular embryo sac. This structure comprises two synergid cells, one egg cell, one central cell, and three antipodal cells, representing the predominant form of female gametophytes in flowering plants (Figure 7).^{8,10}

Fertilization in flowering plants begins with the pollination after anthesis, including sequential events of pollen adhesion, recognition, hydration, and germination on the stigma. Then, the germinated pollen tube traverses the stigma and style, growing through transmitting tissues. Guided by ovular signals, the pollen tube enters the embryo sac via the micropyle, ruptures, and releases two sperm cells. One sperm cell fuses with the egg cell to form the zygote, developing into the embryo, while the other one fuses with the central cell to develop into the endosperm (Figure 7).^{367,368} This fertilization process is known as double fertilization. The delivery of male gametes to the female gametophyte via the pollen tube to complete fertilization is referred to as siphonogamy. After double fertilization, the plant transitions from the gametophytic generation to the sporophytic generation.⁹ Ultimately, the fertilized ovule develops into seeds following seed development and morphogenesis. It subsequently acquires the ability to live independently after maturation and dehydration.³⁶⁹

Germline development and fertilization are governed by complex molecular and cellular mechanisms involving precisely coordinated signaling pathways and gene regulatory networks. Deciphering these mechanisms have provided fundamental insights into reproductive biology, including the origins of genetic diversity and evolutionary adaptation strategies. From an applied perspective, the knowledge enables the development of crops with enhanced yield, improved nutritional quality, and increased resilience to biotic and abiotic stressors. Furthermore, optimization of fertilization efficiency through targeted interventions can directly improve agricultural productivity, thereby support global food security and promote sustainable ecosystem management.

Male germline specification

In flowering plants, male germline development originates within the anther. Diploid microsporocytes arise in the anther locules. Following meiosis (two successive nuclear divisions), microsporocytes produce haploid tetrads of microspores through microsporogenesis. These microspores differentiate and are released from the tetrad (Figure 7). Upon release, the microspore undergoes rapid expansion. Small vacuoles within the cell coalesce into a dominant central vacuole, sequestering the nucleus to the periphery—a process termed microspore polarization (Figure 7). Microspore polarization is not merely a passive consequence of vacuolar expansion but an active process requiring microtubule-mediated cytoskeletal reorganization.³⁷⁰ Polarized microspores then undergo asymmetric pollen mitosis I (PM I), yielding two distinct cell types: a large vegetative cell (VC) and a smaller generative cell (GC). The GC subsequently becomes entirely enclosed within the VC cytoplasm, establishing a characteristic cell-within-cell configuration (Figure 7).^{8,371} Through symmetric pollen mitosis II (PM II), the GC divides to produce two sperm cells (Figure 7).⁸ These sperm cells are male gametes, and together with the VC, they constitute the male gametophyte (pollen grain). The sperm cells remain interconnected until fertilization. One sperm cell extends a cytoplasmic projection that anchors the vegetative nucleus, maintaining physical connectivity and forming a cellular structure called the male germ unit (MGU).^{372,373} The division of the generative cell into two sperm cells via PMII occurs within the pollen grains in approximately 30% of angiosperm species, whereas in the remaining species, this process takes place within the pollen tube. Consequently, anthers release either bicellular (containing a VC and a GC) or tricellular (containing a VC and two sperm cells) pollen grains.³⁷⁴

During microgametogenesis, cell division and fate determination are regulated by intricate mechanisms. The microspore serves as the germline progenitor, with PM I establishing cellular polarity. This process critically depends on microtubule-associated proteins and regulators. These components coordinate nuclear positioning and division plane orientation to establish the cell polarity required for asymmetric division and germ cell fate. Disruption of these mechanisms, as exemplified by *Arabidopsis* *MICROTUBULE ORGANIZATION 1* (*MOR1*) mutants, compromises division asymmetry and cytokinesis, leading to complete male germline abortion.^{370,375,376} Specifically, *MOR1*-deficient microspores fail to establish the polarized microtubule arrays required for asymmetric nuclear migration prior to PM I. This failure results in symmetric divisions that preclude germ cell differentiation.

Cell cycle control variations in vegetative and germ cells are crucial for germline specification and male gametophyte patterning. Key players include *Chromatin Assembly Factor 1* (*CAF1*), which is pivotal in DNA replication, facilitating the deposition of histones H3 and H4 onto DNA to assemble nucleosomes.³⁷⁷ Defects in *CAF1* result in cell cycle arrest, preventing progression of GC to the subsequent PM II stage and culminating in a single sperm cell.³⁷⁸ Additionally, the A-type cyclin-dependent kinase (CDKA;1) is essential for germ cell division in *Arabidopsis*, with *cdka;1* mutant displaying retarded S-phase and delayed germ cell entry into mitosis during pollen tube growth.³⁷⁹ The F BOX-LIKE 17 (*FBL17*) is expressed in male germ cells post-PM I and forms part of the SCF E3 ubiquitin ligase complex, targeting CDK inhibitors KIP-RELATED PROTEIN 6 (*KRP6*) and *KRP7* for degradation, thereby facilitating germ cell advancement through S-phase.³⁸⁰ The *fbl17* mutant exhibited a similar germ cell defect to *cdka;1*^{380,381} while CDKA's activity is hypothesized to be inhibited by *KRP6/7* in VCs, hindering their cell cycle progression. Germline-specific expression of *FBL17* thus differentially controls the cell cycle in GCs and VCs, facilitating germ cell progression through S-phase.

Unlike *FBL17* and CDKA, the RETINOBLASTOMA-RELATED (*RBR*) protein acts as a repressor, inhibiting vegetative cell re-entry into the cell cycle.³⁸² Loss of function mutation in *RBR* causes mild over-proliferation of VCs, and, to a lesser degree, GCs.³⁸² In dividing cells, CDKA forms complex with cyclin D2 to phosphorylate *RBR*, preventing its interaction with E2Fs transcription factors, culminating in relief of the inhibition of S-phase genes.³⁸³⁻³⁸⁶ Nevertheless, CDKA in the VC does not alleviate *RBR*-mediated S-phase progression inhibition, possibly due to high *KRP6* and *KRP7* levels in the VC. Conversely, VC proliferation relies on CDKA in *RBR*'s absence.³⁸⁶ The findings above demonstrate that precise regulation of the cell cycle in distinct cells during microgametogenesis is essential for cell division and fate determination.

Germline specification follows the asymmetric and then the symmetric mitosis, under the control of two R2R3 MYB protein DUO POLLEN 1 (*DUO1*) and *DUO3*.³⁸⁷⁻³⁸⁹ *DUO1*, specifically expressed in the male germline, is crucial for male germ cell differentiation.^{387,389,390} Defective *DUO1* results in arrested PM II of germ cells at G2-M transition, yielding non-fertile GC.^{387,389,390} *DUO1* influences the transcription of genes like *DUO1-ACTIVATED ZINC FINGER1* (*DAZ1*) and *DAZ2* in GCs, facilitating mitotic entry at the G2/M transition.³⁹¹ The *duo1* mutant also shows suppressed expression of male germline-specific genes such as *MALE-GAMETE-SPECIFIC HISTONE H3* (*MGH3*), *HAPLESS 2/GENERATIVE CELL SPECIFIC 1* (*HAP2/GCS1*), and *GAMETE EXPRESSED 2* (*GEX2*).³⁸⁷ *DUO1* and PCF (TCP)-DOMAIN FAMILY PROTEIN 20 (*TCP20*) transcription factor collectively regulate the expression of the G2/M regulator *CYCLIN B1;1* (*CYCB1;1*), which is necessary for germ cells to enter mitosis at G2-M transition.^{387,389,392} *DUO3* promotes germ cell mitotic entry independently of the *DUO1*-*CDPK1;1* pathway. *DUO1* and *DUO3* conjointly influence a subset of germline genes, underscoring their combined impact on sperm cell specification and fertilization.³⁸⁸ With the orchestration of these regulators, the GC proceeds to divide mitotically (PM II) producing two sperm cells, ready for the subsequent fertilization.

Female germline specification

In most angiosperms, a single subepidermal archesporial cell in the apical region of the ovule primordium gives rise to the MMC. The MMC undergoes meiosis, generating four haploid megaspores through megasporogenesis

(Figure 7).¹⁰ Three megaspores undergo programmed cell death (PCD) and degenerate, leaving a single functional megaspore (FM) (Figure 7). The FM then undergoes three mitotic nuclear divisions without cytokinesis. The first mitosis produces a binucleate embryo sac. A central vacuole subsequently develops between the nuclei, partitioning the cell into micropylar and chalazal domains. During the second mitosis, both nuclei divide synchronously, resulting in a tetranucleate embryo sac. Concurrently, the central vacuole expands further. The third mitosis generates an octonucleate embryo sac: three nuclei cluster at the micropylar pole, three at the chalazal pole, and two polar nuclei flank the central vacuole (Figure 7). Following this, one chalazal polar nucleus migrates toward the micropylar pole. Cellularization simultaneously transforms the syncytium into a seven-celled structure containing eight nuclei. The migrating polar nucleus fuses with its micropylar counterpart, forming a diploid secondary nucleus in the central cell. This completes the development of the seven-celled, eight-nucleate female gametophyte (FG), comprising two synergids and an egg cell at the micropylar pole, three antipodal cells at the chalazal pole, and a binucleate central cell (Figure 7).^{9,10,393} The synergid-egg cell complex (egg apparatus) and central cell collectively form the female germ unit (FGU). The egg cell and central cell serve as the two types of female gametes.³⁹⁴

The process of female germline specification is regulated by intricate mechanisms, primarily including transcriptional regulation, cell positional information, and phytohormone level, among others. Specific cell types possess distinct gene expression profiles to facilitate their specialized functions. Transcriptional regulation mediated by specific transcription factors is critical for the process of female germline specification. In Arabidopsis, *RWP-RK domain-containing transcription factors 1* (*RKD1*) and *RKD2*, preferentially expressed in the egg cell, regulate the expression of egg-cell-specific genes.³⁹⁵ Remarkably, ectopic expression of *RKD1* and *RKD2* in sporophytic cells can induce an egg cell-like transcriptome in Arabidopsis, indicating their significant role in egg cell identity.³⁹⁵ Disruptions in *RKD3* and *RKD4* affect egg cell specification and modify the transcriptomic landscape of ovule tissues. This demonstrates the RKD proteins' shared functions in egg cell development.^{395,396} Additionally, *MpRKDs* are reported to be crucial for egg cell differentiation or egg cell arrest in liverwort, suggesting a conserved function in egg cell specification during evolution.^{397,398}

In the process of female germline specification, positional information of individual cells plays a pivotal role in guiding their differentiation and fate determination (refer to a recent review³⁹⁹). This is exemplified by the significant impact of positional dynamics within the female gametophyte on determining egg cell fate.³⁹³ In the eight-nucleate syncytium, nuclei relocated to different positions acquire distinct cell identities. Nuclei from different origins assume identical identities when positioned similarly.³⁹³ This phenomenon is associated with auxin distribution, with the egg cell exhibiting higher auxin concentrations than synergids.³⁹³

Phytohormones orchestrate diverse aspects of plant growth and development, with their precise regulation being essential for cellular differentiation and fate determination. In the context of female germline specification, spatiotemporal modulation of phytohormone levels within tissues and individual cells is crucial for guiding cell fate decisions (refer to a recent review⁴⁰⁰). As an example, in Arabidopsis ovules, GA biosynthesis/signaling is confined to primordia. Overexpressing GA receptors/enzymes causes multiple enlarged MMCs. GA signaling degrades CK type-B *Arabidopsis* response regulators ARR1/10/12 (ARR1/10/12), which bind DELLAs. CK components are expressed in MMCs and sporophytic cells but are GA-suppressed; perturbations induce ectopic MMC-like cells. SHRUBBY (SHBY) interacts with GA/CK machinery to inhibit GA-induced degradation. CK restricts sub-epidermal cells from adopting MMC identity. GA and CK antagonistically balance degradation to regulate female germline specification.⁴⁰¹

Genetic mutants like *eostre*, *wyrd*, *Lachesis* (*lis*), *clotho* (*clo/gfa1*), *Atropos* (*ato*), and *Altered meristem program 1* (*amp1*) often develop multiple egg cells, indicating disruptions in cell fate determination.⁴⁰²⁻⁴⁰⁶ For instance, in *lis* mutants, accessory cells adopt a gametic cell fate, highlighting LIS's role in preventing such cellular fate transitions.^{402,404} *eostre* mutants demonstrate aberrant expression of *BEL1-LIKE HOMEODOMAIN 1* (*BLH1*) and interaction with *KNOTTED1-LIKE HOMEODOMAIN 3* (*KNAT3*), a class II KNOX gene.⁴⁰⁶ Conversely, mutations in *lis*, *ato*, and *clo* affect splicing machinery compo-

nents, suggesting a link between gametic fate restriction and post-transcriptional mechanisms.^{403,404}

Molecular regulation of fertilization

Upon deposition of pollen grains on the stigma of a flower, a tip-growing pollen tube emerges and elongates into the style of the pistil. This process marks the onset of pollen tube guidance (Figure 7).^{368,407} To date, insights into the molecular mechanisms governing these processes have been largely obtained from investigations in the model plant Arabidopsis. Stigma-pollen tube interaction factors, such as Rapid alkalization factor (RALF)-FERONIA (FER) peptide-receptor pairs, govern the stigma-pollen recognition for efficient pollen germination and exclude alien pollen.^{408,409} During the invasive growth of pollen tube, their integrity is controlled by the balance between the internal turgor and cell wall mechanics. The integrity of the pollen tube is maintained by cell surface proteins, like receptor kinases ANXUR1/2 (ANX1/2) and BUDDHA'S PAPER SEAL 1/2 (BUPS1/2), peptides RALF 4/19 and cell-wall-associated LEUCINE-RICH REPEAT/EXTENSINS (LRXs).^{410,411}

After passing through the solid style, pollen tubes enter the transmitting tissue that undergoes cell death in the central part to form a "tunnel". Within this tunnel, pollen tubes continuously perceive and respond to guiding signals from female gametophytes for precise targeting (Figure 7). MILDEW RESISTANCE LOCUS O (MLO) family members MLO5, 9, and 15 identified as key regulators for cytosolic Ca²⁺ gradient regulation.⁴¹² Mutations in *MLO5* and 9 results in highly coiled pollen tubes on the septum surface outside the micropyle and impaired the cytosolic Ca²⁺ level,⁴¹² implicating the role of MLOs in coupling ovular guiding signals and Ca²⁺ signaling. This polarized growth culminates in the pollen tube arrival at the receptive synergid and subsequent release of two sperm cells. Secreted peptides, such as the synergids-derived LURES, XIUIQUS, TICKETS, NON-DEFENSIN PEPTIDE ATTRACTANT 1 (NPA1) and central cell-derived SALVAGERS, have been identified as guiding signals.⁴¹³⁻⁴¹⁹ These series of guiding signals direct pollen tube exit from the transmitting tissue towards the ovule and into the embryo sac (Figure 7). Synergids and the central cell serve as primary sources of these pollen tube attractants.^{414,420} These signals are perceived by receptor kinases localized on the pollen tube plasma membrane.^{421,422} Under conditions of failed double fertilization, the persistent synergid attracts another pollen tube. When both synergids are consumed and double fertilization still fails, the central cell releases stored SALVAGERS to attract additional pollen tubes for seed production salvage.^{408,414,423}

Upon synergid entry, the pollen tube bursts, releasing its cytoplasm and two sperm cells, an event known as pollen tube reception (Figure 7).³⁶⁷ FER, ANJEA (ANJ), HERCULES RECEPTOR KINASE (HERK) and LORELEI (LRE), and the pollen tube derived ligands RALFs are critical in this process, as their mutants exhibit pollen tube overgrowth around the synergid cells without pollen tube burst.⁴²⁴⁻⁴²⁷ The compatible synergid-pollen tube interaction induces characterized Ca²⁺ spikes in both synergids. The receptive synergid undergoes degeneration, while the remaining synergid (known as persistent synergid) remains intact.⁴²⁸ The Ca²⁺ spikes are mediated by FER and its downstream MLO protein NORTIA (NTA) that is recruited to the synergid plasma membrane upon effective synergid-pollen tube interaction.⁴²⁹

Following release, the sperm cells migrate to the interspace between the egg cell and central cell (Figure 7). Sperm cell positioning is vital for successful double fertilization, where simultaneous attachment of two sperms to the egg cell leads to one sperm being repositioned towards the central cell.^{373,430} During sperm contact, the egg cell secretes the cysteine-rich peptide EGG CELL 1 (EC1) to induce the secretion of fusogen HAP2/GCS1 to the sperm cell surface. HAP2/GCS1 then acts together with the four-pass transmembrane protein DUF679 DOMAIN MEMBRANE PROTEIN 8/9 (DMP8/9) to facilitate gamete fusion in Arabidopsis.⁴³¹⁻⁴³³ Before fusion, the sperm-specific protein GEX2 is crucial for gamete attachment, with its disruption leading to attachment failure.^{434,435} Sperm cell surface cation/H⁺ transporters CATION/H⁺ EXCHANGER 17/18/19 (CHX17/18/19) simultaneously affect the sperm cell shape and its adhesion with the female gametes in an independent pathway from GEX2 and DMP8/9.⁴³⁶ Following adhesion, fusogen protein HAP2/GCS1 on the sperm plasma membrane form a trimer to bring the two membranes of the male and female gametes to merge into one.^{431,437}

Different mechanisms at different stage of male-female interaction during

fertilization are employed to prevent entry of more than one pollen tube, preventing polyspermy that changes the offspring ploidy and viability. RALFs derived from the pollen tube collaborate with their receptors, FER, ANJ, and HERK1 on the septum surface, to regulate single pollen tube exit from the transmitting tract.⁴³⁸ Synergid cells that successfully trigger pollen tube burst induced NO production from the synergid to prevent additional pollen tube entry through inactivating LURE1.⁴³⁹ The fertilization of both female gametes both contribute to terminating pollen tube attraction. After successful fertilization, the egg cell secretes proteinase EGG CELL-SPECIFIC 1/2 (ECS1/2) to cleave LURE1.⁴⁴⁰ The fertilized central cell fuses with the persistent synergid to terminate synergid-based pollen tube attraction following double fertilization.⁴⁴¹ Additionally, ARABINO GALACTAN PROTEIN/JAGGER (AGP4/JAGGER) is also required for the elimination of the persistent synergid.⁴⁴² After fertilization, SALVAGERS are secreted to the seed coat or maintained in the endosperm, instead of being secreted to attract pollen tubes,⁴¹⁴ may act as another layer to prevent polytubey after fertilization recovery.

FUTURE DIRECTIONS AND APPLICATIONS

Plant development represents a highly coordinated process governed by genetic programs and environmental interactions. Significant advances have been made in deciphering the roles of phytohormones, transcriptional regulators, and signaling networks across various developmental stages. However, many fundamental aspects remain incompletely understood. For instance, floral development involves extreme complexity in organ formation and intricate crosstalk among genetic, hormonal, and environmental factors, posing substantial research challenges.^{443,444} Furthermore, the field's heavy reliance on a few model systems (e.g., *Arabidopsis*) constrains the comprehension of floral architecture diversity, particularly in basal angiosperms and early-diverging eudicots. Critical knowledge gaps persist in germline development and fertilization, including the mechanisms of nuclear positioning and cell fate determination within the embryo sac, molecular drivers of germ cell migration, signaling cascades triggered by female-secreted peptides guiding pollen tube growth, and processes governing sperm cell movement and fusion with female gametes.

Translational applications of plant developmental biology are expanding rapidly, particularly in designing crops with improved architecture and resilience. Modulating branching patterns, plant height, and inflorescence structure can enhance photosynthetic efficiency, reduce lodging, and facilitate mechanized harvesting.⁴⁴⁵⁻⁴⁴⁷ For instance, promoter engineering of *IPA1* in rice helped break the trade-off between tiller number and panicle size. Recently, *BRD3*, a gene involved in brassinosteroid metabolism, was identified as responsible for spikelet clustering in a specific rice germplasm. A chromosomal structural variation fine-tunes tissue-specific brassinosteroid levels, mitigating the antagonism between grain number and grain size, thereby increasing yield.⁴⁴⁸ Similarly, a large deletion on the short arm of wheat chromosome results in the loss of three closely associated genes modulating the balance between GA and BR signaling. This mutation confers a beneficial semi-dwarf trait while increasing grain weight and nitrogen use efficiency.⁴⁴⁹ In maize, the natural mutant *lac1* exhibits a "smart canopy" architecture, characterized by upright upper leaves, less erect middle leaves, and relatively flat lower leaves. It optimizes light capture and photosynthetic efficiency under high planting densities, ultimately enhancing yield.⁴⁵⁰ Synthetic apomixis has emerged as a highly promising tool for rapidly fixing elite agronomic traits in crop species. By enabling the clonal propagation of seeds through asexual reproduction, this technology allows the preservation of superior genetic combinations, such as high yield, disease resistance, or stress tolerance, over multiple generations without segregation.⁴⁵¹

Emerging technologies are accelerating trait design and breeding programs. For example, a breeding robot named GEAIR (Genome Editing combined with AI-based Robotics) leverages knowledge of floral development to open a new venue for AI-assisted breeding and intelligent cultivation.⁴⁵² Nevertheless, several challenges remain. A primary hurdle is identifying key genes and *cis*-regulatory elements essential for target traits. Integrating multi-omics approaches, including genomics, transcriptomics, proteomics, and metabolomics, will provide systems-level insights into the regulatory and metabolic networks steering plant growth and adaptation. Additionally, developing efficient gene editing systems is critical, particularly

in situ knock-in strategies and tissue-culture-free heritable genome editing technologies to facilitate broader application across species. Overcoming the haploid induction bottleneck remains imperative for achieving efficient synthetic apomixis.⁴⁵³

Looking forward, a more comprehensive understanding of the antagonistic and synergistic relationships between agronomic traits, coupled with advanced technologies such as genome editing, apomixis, and AI-assisted breeding systems, will be essential to achieve predictive crop design. In summary, plant development research has revealed a complex tapestry of genetic and environmental interactions that shape the plant life cycle. The profound insights from this field not only deepen our fundamental knowledge but also offer significant practical potential. Leveraging a whole-life-cycle perspective on plant development will enable the engineering of more resilient crops, enhance agricultural productivity, and contribute to sustainable ecosystem management. As research continues to unravel developmental complexities, it becomes increasingly evident that plants hold solutions to some of humanity's most pressing challenges. The integration of artificial intelligence, spatial transcriptomics,⁴⁵⁴⁻⁴⁵⁸ and streamlined gene delivery and editing systems,⁴⁵⁹⁻⁴⁶¹ is expected to establish a new paradigm in plant developmental biology, heralding a transformative phase characterized by groundbreaking discoveries and technological innovations.

REFERENCES

- Goldberg R. (1988). Plants: Novel developmental processes. *Science* **240**:1460–1467. DOI:10.1126/science.3287622
- Sussex M. (1989). Developmental programming of the shoot meristem. *Cell* **56**:225–229. DOI:10.1016/0092-8674(89)90895-7
- Sussex I. and Kerk N. (2001). The evolution of plant architecture. *Curr. Opin. Plant Biol.* **4**:33–37. DOI:10.1016/S1369-5266(00)00132-1
- Wang B., Smith S., and Li J. (2018). Genetic regulation of shoot architecture. *Annu. Rev. Plant Biol.* **69**:437–468. DOI:10.1146/annurev-arplant-042817-040422
- Lü T., Gao F., Su T., et al. (2025). Developmental genetics of fruit diversity in Brassicaceae. *Curr. Opin. Plant Biol.* **85**:102707. DOI:10.1016/j.pbi.2025.102707
- Sauquet H., von Balthazar M., Magallón S., et al. (2017). The ancestral flower of angiosperms and its early diversification. *Nat. Commun.* **8**:16047. DOI:10.1038/ncomms16047
- Freytes S., Canelo M., and Cerdan P. (2021). Regulation of flowering time: When and where. *Curr. Opin. Plant Biol.* **63**:102049. DOI:10.1016/j.pbi.2021.102049
- Billey E., Hafidh S., Cruz-Gallardo I., et al. (2021). LARP6C orchestrates posttranscriptional reprogramming of gene expression during hydration to promote pollen tube guidance. *Plant Cell* **33**:2637–2661. DOI:10.1093/plcell/koab131
- Yang W., Shi D. and Chen Y. (2010). Female gametophyte development in flowering plants. *Annu. Rev. Plant Biol.* **61**:89–108. DOI:10.1146/annurev-arplant-042809-112203
- Hater F., Nakel T. and Gross-Hardt R. (2020). Reproductive multitasking: The female gametophyte. *Annu. Rev. Plant Biol.* **71**:517–546. DOI:10.1146/annurev-arplant-081519-035943
- Berger F. and Twell D. (2011). Germline specification and function in plants. *Annu. Rev. Plant Biol.* **62**:461–484. DOI:10.1146/annurev-arplant-042110-103824
- Dresselhaus T., Sprunck S. and Wessel G. (2016). Fertilization mechanisms in flowering plants. *Curr. Biol.* **26**:R125–R139. DOI:10.1016/j.cub.2015.12.032
- Hafidh S. and Honys D. (2021). Reproduction multitasking: The male gametophyte. *Annu. Rev. Plant Biol.* **72**:581–614. DOI:10.1146/annurev-arplant-080620-021907
- Chaudhury A., Ming L., Miller C., et al. (1997). Fertilization-independent seed development in *Arabidopsis thaliana*. *Proc. Natl. Acad. Sci. USA* **94**:4223–4228. DOI:10.1073/pnas.94.8.4223
- Durbak A., Yao H. and McSteen P. (2012). Hormone signaling in plant development. *Curr. Opin. Plant Biol.* **15**:92–96. DOI:10.1016/j.pbi.2011.12.004
- Alvarez-Buylla E., Benitez M., Corvera-Poire A., et al. (2010). Flower development. *Arabidopsis Book* **8**:e0127. DOI:10.1199/tab.0127
- Prunet N. and Meyerowitz E. (2016). Genetics and plant development. *C.R. Biol.* **339**:240–246. DOI:10.1016/j.crv.2016.05.003
- Thomson B. and Wellmer F. (2019). Molecular regulation of flower development. *Plant Development and Evolution* **131**:185–210. DOI:10.1016/bs.ctdb.2018.11.007
- Zik M. and Irish V. (2003). Flower development: Initiation, differentiation, and diversification. *Annu. Rev. Cell Dev. Biol.* **19**:119–140. DOI:10.1146/annurev.cellbio.19.111301.134635
- Cheng Q., Li J. and Wang B. (2024). *ABP1/ABIs* and *TMKs* form receptor complexes to perceive extracellular auxin and trigger fast phosphorylation responses. *Innov. Life* **2**:100063. DOI:10.59717/j.xinn-life.2024.100063
- Dong Y., Su N. and Zhang Y. (2024). New wine in an old bottle: *ABCB19*, known as auxin exporter, also exports brassinosteroids. *Innov. Life* **2**:100072. DOI:10.59717/j.xinn-life.2024.100072

22. Franklin K. (2009). Light and temperature signal crosstalk in plant development. *Curr. Opin. Plant Biol.* **12**:63–68. DOI:10.1016/j.pbi.2008.09.007
23. Pierik R., Fankhauser C., Strader L., et al. (2021). Architecture and plasticity: Optimizing plant performance in dynamic environments. *Plant Physiol.* **187**:1029–1032. DOI:10.1093/plphys/kiab402
24. Sultan S. (2010). Plant developmental responses to the environment: Eco-devo insights. *Curr. Opin. Plant Biol.* **13**:96–101. DOI:10.1016/j.pbi.2009.09.021
25. Wang Q. and Lin C. (2020). Mechanisms of Cryptochrome-Mediated Photoresponses in Plants. *Annu. Rev. Plant Biol.* **71**:103–129. DOI:10.1146/annurev-arplant-050718-100300
26. Legris M., Ince Y. C. and Fankhauser C. (2019). Molecular mechanisms underlying phytochrome-controlled morphogenesis in plants. *Nat. Commun.* **10**:5219. DOI:10.1038/s41467-019-13045-0
27. Kami C., Lorrain S., Hornitschek P., et al. (2010). Light-regulated plant growth and development. *Curr. Top. Dev. Biol.* **91**:29–66. DOI:10.1016/S0070-2153(10)91002-8
28. Smith H. (1970). Phytochrome and photomorphogenesis in plants. *Nature* **227**:665–668. DOI:10.1038/227665a0
29. Christie J., Arvai A., Baxter K., et al. (2012). Plant UVR8 photoreceptor senses UV-B by Tryptophan-mediated disruption of cross-dimer salt bridges. *Science* **335**:1492–1496. DOI:10.1126/science.1218091
30. Li J., Li G., Wang H., et al. (2011). Phytochrome signaling mechanisms. *Arabidopsis Book* **9**:e0148. DOI:10.1199/tab.0148
31. Hiltbrunner A., Viczian A., Bury E., et al. (2005). Nuclear accumulation of the phytochrome A photoreceptor requires PHY1. *Curr. Biol.* **15**:2125–2130. DOI:10.1016/j.cub.2005.10.042
32. Klose C., Viczian A., Kircher S., et al. (2015). Molecular mechanisms for mediating light-induced nucleo/cytoplasmic partitioning of phytochrome photoreceptors. *New Phytol.* **206**:965–971. DOI:10.1111/nph.13207
33. Christie J., Reymond P., Powell G., et al. (1998). Arabidopsis NPH1: A flavoprotein with the properties of a photoreceptor for phototropism. *Science* **282**:1698–1701. DOI:10.1126/science.282.5394.1698
34. Lariguet P., Schepens I., Hodgson D., et al. (2006). PHYTOCHROME KINASE SUBSTRATE 1 is a phototropin 1 binding protein required for phototropism. *Proc. Natl. Acad. Sci. USA* **103**:10134–10139. DOI:10.1073/pnas.0603799103
35. Ahmad M. and Cashmore A. (1993). HY4 gene of *A. thaliana* encodes a protein with characteristics of a blue-light photoreceptor. *Nature* **366**:162–166. DOI:10.1038/366162a0
36. Ahmad M., Jarillo J., Smirnova O., et al. (1998). Cryptochrome blue-light photoreceptors of Arabidopsis implicated in phototropism. *Nature* **392**:720–723. DOI:10.1038/33701
37. Guo H., Yang H., Mockler T., et al. (1998). Regulation of flowering time by Arabidopsis photoreceptors. *Science* **279**:1360–1363. DOI:10.1126/science.279.5355.1360
38. Yanovsky M. and Kay S. (2002). Molecular basis of seasonal time measurement in Arabidopsis. *Nature* **419**:308–312. DOI:10.1038/nature00996
39. Zeng D., Lv J., Li X., et al. (2025). The Arabidopsis blue-light photoreceptor CRY2 is active in darkness to inhibit root growth. *Cell* **188**:60–76. DOI:10.1016/j.cell.2024.10.031
40. Huang Y., Baxter R., Smith B., et al. (2006). Crystal structure of cryptochrome 3 from Arabidopsis thaliana and its implications for photolyase activity. *Proc. Natl. Acad. Sci. USA* **103**:17701–17706. DOI:10.1073/pnas.0608554103
41. Baudry A., Ito S., Song Y., et al. (2010). F-box proteins FKF1 and LKP2 act in concert with ZEITLUPE to control Arabidopsis clock progression. *Plant Cell* **22**:606–622. DOI:10.1105/tpc.109.072843
42. Song Y., Estrada D., Johnson R., et al. (2014). Distinct roles of FKF1, GIGANTEA, and ZEITLUPE proteins in the regulation of CONSTANS stability in Arabidopsis photoperiodic flowering. *Proc. Natl. Acad. Sci. USA* **111**:17672–17677. DOI:10.1073/pnas.1415375111
43. Wu D., Hu Q., Yan Z., et al. (2012). Structural basis of ultraviolet-B perception by UVR8. *Nature* **484**:214–U296. DOI:10.1038/nature10931
44. Wang Q., Zuo Z., Wang X., et al. (2016). Photoactivation and inactivation of Arabidopsis cryptochrome 2. *Science* **354**:343–347. DOI:10.1126/science.aaf9030
45. Shao K., Zhang X., Li X., et al. (2020). The oligomeric structures of plant cryptochromes. *Nat. Struct. Mol. Biol.* **27**:480–488. DOI:10.1038/s41594-020-0420-x
46. Ma L., Wang X., Guan Z., et al. (2020). Structural insights into BIC-mediated inactivation of Arabidopsis cryptochrome 2. *Nat. Struct. Mol. Biol.* **27**:472–479. DOI:10.1038/s41594-020-0410-z
47. Li H., Burgie E., Gannam Z., et al. (2022). Plant phytochrome B is an asymmetric dimer with unique signalling potential. *Nature* **604**:127–133. DOI:10.1038/s41586-022-04529-z
48. Wang Z., Wang W., Zhao D., et al. (2024). Light-induced remodeling of phytochrome B enables signal transduction by phytochrome-interacting factor. *Cell* **187**:6235–6250. DOI:10.1016/j.cell.2024.09.005
49. Zhang Y., Lin X., Ma C., et al. (2023). Structural insights into plant phytochrome A as a highly sensitized photoreceptor. *Cell Res.* **33**:806–809. DOI:10.1038/s41422-023-00858-4
50. Huq E. and Quail P. (2002). PIF4, a phytochrome-interacting bHLH factor, functions as a negative regulator of phytochrome B signaling in Arabidopsis. *EMBO J.* **21**:2441–2450. DOI:10.1093/emboj/21.10.2441
51. Ma D., Li X., Guo Y., et al. (2016). Cryptochrome 1 interacts with PIF4 to regulate high temperature-mediated hypocotyl elongation in response to blue light. *Proc. Natl. Acad. Sci. USA* **113**:224–229. DOI:10.1073/pnas.1511437113
52. Pedmale U., Huang S., Zander M., et al. (2016). Cryptochromes interact directly with PIFs to control plant growth in limiting blue light. *Cell* **164**:233–245. DOI:10.1016/j.cell.2015.12.018
53. Lau O. and Deng X. (2010). Plant hormone signaling lightens up: Integrators of light and hormones. *Curr. Opin. Plant Biol.* **13**:571–577. DOI:10.1016/j.pbi.2010.07.001
54. Gao H., Song W., Severing E., et al. (2022). PIF4 enhances DNA binding of CDF2 to co-regulate target gene expression and promote Arabidopsis hypocotyl cell elongation. *Nat. Plants* **8**:1082–1093. DOI:10.1038/s41477-022-01213-y
55. Oh E., Zhu J. and Wang Z. (2012). Interaction between BZR1 and PIF4 integrates brassinosteroid and environmental responses. *Nat. Cell Biol.* **14**:802–809. DOI:10.1038/ncb2545
56. Martinez C., Espinosa-Ruiz A., de Lucas M., et al. (2018). PIF4-induced BR synthesis is critical to diurnal and thermomorphogenic growth. *EMBO J.* **37**:e99552. DOI:10.15252/emboj.201899552
57. Li K., Yu R., Fan L., et al. (2016). DELLA-mediated PIF degradation contributes to coordination of light and gibberellin signalling in Arabidopsis. *Nat. Commun.* **7**:11868. DOI:10.1038/ncomms11868
58. Zhang X., Ji Y., Xue C., et al. (2018). Integrated regulation of apical hook development by transcriptional coupling of EIN3/EIL1 and PIFs in Arabidopsis. *Plant Cell* **30**:1971–1988. DOI:10.1105/tpc.18.00018
59. Xu F., He S., Zhang J., et al. (2018). Photoactivated CRY1 and phyB interact directly with AUX/IAA proteins to inhibit Auxin signaling in Arabidopsis. *Mol. Plant* **11**:523–541. DOI:10.1016/j.molp.2017.12.003
60. Mao Z., He S., Xu F., et al. (2020). Photoexcited CRY1 and phyB interact directly with ARF6 and ARF8 to regulate their DNA-binding activity and auxin-induced hypocotyl elongation in Arabidopsis. *New Phytol.* **225**:848–865. DOI:10.1111/nph.16194
61. Liang T., Mei S., Shi C., et al. (2018). UVR8 interacts with BES1 and BIM1 to regulate transcription and photomorphogenesis in Arabidopsis. *Dev. Cell* **44**:512–523. DOI:10.1016/j.devcel.2017.12.028
62. Wang W., Lu X., Li L., et al. (2018). Photoexcited CRYPTOCHROME1 interacts with dephosphorylated BES1 to regulate brassinosteroid signaling and photomorphogenesis in Arabidopsis. *Plant Cell* **30**:1989–2005. DOI:10.1105/tpc.17.00994
63. Liu B., Zuo Z., Liu H., et al. (2011). Arabidopsis cryptochrome 1 interacts with SPA1 to suppress COP1 activity in response to blue light. *Genes Dev.* **25**:1029–1034. DOI:10.1101/gad.2025011
64. Favory J., Stec A., Gruber H., et al. (2009). Interaction of COP1 and UVR8 regulates UV-B-induced photomorphogenesis and stress acclimation in Arabidopsis. *EMBO J.* **28**:591–601. DOI:10.1038/emboj.2009.4
65. Osterlund M., Hardtke C., Wei N., et al. (2000). Targeted destabilization of HY5 during light-regulated development of Arabidopsis. *Nature* **405**:462–466. DOI:10.1038/35013076
66. Seo H., Watanabe E., Tokutomi S., et al. (2004). Photoreceptor ubiquitination by COP1 E3 ligase desensitizes phytochrome A signaling. *Genes Dev.* **18**:617–622. DOI:10.1101/gad.1187804
67. Weidler G., Zur Oven-Krockhaus S., Heunemann M., et al. (2012). Degradation of Arabidopsis CRY2 is regulated by SPA proteins and phytochrome A. *Plant Cell* **24**:2610–2623. DOI:10.1105/tpc.112.098210
68. Liu Q., Wang Q., Liu B., et al. (2016). The blue light-dependent polyubiquitination and degradation of Arabidopsis Cryptochrome2 requires multiple E3 ubiquitin ligases. *Plant Cell Physiol.* **57**:2175–2186. DOI:10.1093/pcp/pcw134
69. Jang I., Henriques R., Seo H., et al. (2010). Arabidopsis PHYTOCHROME INTERACTING FACTOR proteins promote phytochrome B polyubiquitination by COP1 E3 ligase in the nucleus. *Plant Cell* **22**:2370–2383. DOI:10.1105/tpc.109.072520
70. Miao L., Zhao J., Yang G., et al. (2022). Arabidopsis cryptochrome 1 undergoes COP1 and LRBs-dependent degradation in response to high blue light. *New Phytol.* **234**:1347–1362. DOI:10.1111/nph.17695
71. Dong J., Ni W., Yu R., et al. (2017). Light-dependent degradation of PIF3 by SCF^{EBF1/2} promotes a photomorphogenic response in Arabidopsis. *Curr. Biol.* **27**:2420–2430. DOI:10.1016/j.cub.2017.06.062
72. Shi H., Shen X., Liu R., et al. (2016). The red light receptor phytochrome B directly enhances substrate-E3 ligase interactions to attenuate ethylene responses. *Dev. Cell* **39**:597–610. DOI:10.1016/j.devcel.2016.10.020
73. Ren H., Han J., Yang P., et al. (2019). Two E3 ligases antagonistically regulate the UV-B response in Arabidopsis. *Proc. Natl. Acad. Sci. USA* **116**:4722–4731. DOI:10.1073/pnas.1816268116
74. Zhang Q., Lin L., Fang F., et al. (2023). Dissecting the functions of COP1 in the UVR8 pathway with a COP1 variant in Arabidopsis. *Plant J.* **113**:478–492. DOI:10.1111/tpj.16059
75. Ni W., Xu S., Tepperman J., et al. (2014). A mutually assured destruction mechanism attenuates light signaling in Arabidopsis. *Science* **344**:1160–1164. DOI:10.1126/science.1250778
76. Chen Y., Hu X., Liu S., et al. (2021). Regulation of Arabidopsis photoreceptor CRY2 by

- two distinct E3 ubiquitin ligase. *Nat. Commun.* **12**:2155. DOI:10.1038/s41467-021-22410-x
77. Ma L., Li X., Zhao Z., et al. (2021). Light-Response Bric-A-Brack/Tramtrack/Broad proteins mediate cryptochrome 2 degradation in response to low ambient temperature. *Plant Cell* **33**:3610–3620. DOI:10.1093/plcell/koab219
 78. Jiang L., Zhang S., Niu Y., et al. (2025). Photoexcited CRY1 physically interacts with ATG8 to regulate selective autophagy of HY5 and photomorphogenesis in *Arabidopsis*. *Plant Cell* **37**:koaf196. DOI:10.1093/plcell/koaf196
 79. Kathare P., Xin R., Ganesan A., et al. (2022). SWAP1-SFPS-RR1 splicing factor complex modulates pre-mRNA splicing to promote photomorphogenesis in *Arabidopsis*. *Proc. Natl. Acad. Sci. USA* **119**:e2214565119. DOI:10.1073/pnas.2214565119
 80. Yan T., Heng Y., Wang W., et al. (2022). SWELLMAP 2, a phyB-interacting splicing factor, negatively regulates seedling photomorphogenesis in *Arabidopsis*. *Front. Plant Sci.* **13**:836519. DOI:10.3389/fpls.2022.836519
 81. Dong J., Chen H., Deng X., et al. (2020). Phytochrome B induces intron retention and translational inhibition of PHYTOCHROME-INTERACTING FACTOR3. *Plant Physiol.* **182**:159–166. DOI:10.1104/pp.19.00835
 82. Zhao Z., Dent C., Liang H., et al. (2022). CRY2 interacts with CIS1 to regulate thermosensory flowering via FLM alternative splicing. *Nat. Commun.* **13**:7045. DOI:10.1038/s41467-022-34886-2
 83. Jiang B., Zhong Z., Gu L., et al. (2023). Light-induced LLPS of the CRY2/SPA1/FIO1 complex regulating mRNA methylation and chlorophyll homeostasis in *Arabidopsis*. *Nat. Plants.* **9**:2042–2058. DOI:10.1038/s41477-023-01580-0
 84. Wang X., Jiang B., Gu L., et al. (2021). A photoregulatory mechanism of the circadian clock in *Arabidopsis*. *Nat. Plants* **7**:1397–1408. DOI:10.1038/s41477-021-01002-z
 85. Yang J., Li L., Li X., et al. (2023). The blue light receptor CRY1 interacts with FIP37 to promote N⁶-methyladenosine RNA modification and photomorphogenesis in *Arabidopsis*. *New Phytol.* **237**:840–854. DOI:10.1111/nph.18583
 86. Li X., Liang T. and Liu H. (2022). How plants coordinate their development in response to light and temperature signals. *Plant Cell* **34**:955–966. DOI:10.1093/plcell/koab302
 87. Jung J., Domijan M., Klose C., et al. (2016). Phytochromes function as thermosensors in *Arabidopsis*. *Science* **354**:886–889. DOI:10.1126/science.aaf6005
 88. Legris M., Klose C., Burgie E., et al. (2016). Phytochrome B integrates light and temperature signals in *Arabidopsis*. *Science* **354**:897–900. DOI:10.1126/science.aaf5656
 89. Hahn J., Kim K., Qiu Y., et al. (2020). Increasing ambient temperature progressively disassembles *Arabidopsis* phytochrome B from individual photobodies with distinct thermostabilities. *Nat. Commun.* **11**:1660. DOI:10.1038/s41467-020-15526-z
 90. Fujii Y., Tanaka H., Konno N., et al. (2017). Phototropin perceives temperature based on the lifetime of its photoactivated state. *Proc. Natl. Acad. Sci. USA* **114**:9206–9211. DOI:10.1073/pnas.1704462114
 91. Ma L., Li X., Zhao Z., et al. (2021). Light-Response Bric-A-Brack/Tramtrack/Broad proteins mediate cryptochrome 2 degradation in response to low ambient temperature. *Plant Cell* **33**:3610–3620. DOI:10.1093/plcell/koab219
 92. Saitoh A., Takase T., Abe H., et al. (2021). ZEITLUPE enhances expression of PIF4 and YUC8 in the upper aerial parts of *Arabidopsis* seedlings to positively regulate hypocotyl elongation. *Plant Cell Rep.* **40**:479–489. DOI:10.1007/s00299-020-02643-8
 93. Hayes S., Sharma A., Fraser D., et al. (2017). UV-B perceived by the UVR8 photoreceptor inhibits plant thermomorphogenesis. *Curr. Biol.* **27**:120–127. DOI:10.1016/j.cub.2016.11.004
 94. Sasaki A., Ashikari M., Ueguchi-Tanaka M., et al. (2002). Green revolution: A mutant gibberellin-synthesis gene in rice - new insight into the rice variant that helped to avert famine over thirty years ago. *Nature* **416**:701–702. DOI:10.1038/416701a
 95. Davière J., Wild M., Regnault T., et al. (2014). Class I TCP-DELLA interactions in inflorescence shoot apex determine plant height. *Curr. Biol.* **24**:1923–1928. DOI:10.1016/j.cub.2014.07.012
 96. Ueguchi-Tanaka M., Nakajima M., Katoh E., et al. (2007). Molecular interactions of a soluble gibberellin receptor, GID1, with a rice DELLA protein, SLR1, and gibberellin. *Plant Cell* **19**:2140–2155. DOI:10.1105/tpc.106.043729
 97. Van de Velde K., Thomas S., Heyse F., et al. (2021). N-terminal truncated RHT-1 proteins generated by translational reinitiation cause semi-dwarfing of wheat Green Revolution alleles. *Mol. Plant* **14**:679–687. DOI:10.1016/j.molp.2021.01.002
 98. Liao Z., Yu H., Duan J., et al. (2019). SLR1 inhibits MOC1 degradation to coordinate tiller number and plant height in rice. *Nat. Commun.* **10**:2738. DOI:10.1038/s41467-019-10667-2
 99. Guo H., Li L., Ye H., et al. (2009). Three related receptor-like kinases are required for optimal cell elongation in *Arabidopsis thaliana*. *Proc. Natl. Acad. Sci. USA* **106**:7648–7653. DOI:10.1073/pnas.0812346106
 100. Zhiponova M., Vanhoutte I., Boudolf V., et al. (2013). Brassinosteroid production and signaling differentially control cell division and expansion in the leaf. *New Phytol.* **197**:490–502. DOI:10.1111/nph.12036
 101. Castorina G. and Consonni G. (2020). The role of brassinosteroids in controlling plant height in Poaceae: A genetic perspective. *Int. J. Mol. Sci.* **21**:1191. DOI:10.3390/Ijms21041191
 102. Hong Z., Ueguchi-Tanaka M., Umemura K., et al. (2003). A rice brassinosteroid-deficient mutant, *ebisu dwarf* (*d2*), is caused by a loss of function of a new member of cytochrome P450. *Plant Cell* **15**:2900–2910. DOI:10.1105/tpc.014712
 103. Tanabe S., Ashikari M., Fujioka S., et al. (2005). A novel cytochrome P450 is implicated in brassinosteroid biosynthesis via the characterization of a rice dwarf mutant, *dwarf11*, with reduced seed length. *Plant Cell* **17**:776–790. DOI:10.1105/tpc.104.024950
 104. Lee S., Hwang J., Joo S., et al. (2010). Biosynthesis and metabolism of dolichosterone in *Arabidopsis thaliana*. *Bull. Korean Chem. Soc.* **31**:3475–3478. DOI:10.5012/bkcs.2010.31.11.3475
 105. He J., Gendron J., Sun Y., et al. (2005). BZR1 is a transcriptional repressor with dual roles in brassinosteroid homeostasis and growth responses. *Science* **307**:1634–1638. DOI:10.1126/science.1107580
 106. Tong H., Liu L., Jin Y., et al. (2012). DWARF AND LOW-TILLERING acts as a direct downstream target of a GSK3/SHAGGY-like kinase to mediate brassinosteroid responses in rice. *Plant Cell* **24**:2562–2577. DOI:10.1105/tpc.112.097394
 107. Vert G. and Chory J. (2006). Downstream nuclear events in brassinosteroid signalling. *Nature* **441**:96–100. DOI:10.1038/nature04681
 108. Li L., Yu X., Thompson A., et al. (2009). *Arabidopsis* MYB30 is a direct target of BES1 and cooperates with BES1 to regulate brassinosteroid-induced gene expression. *Plant J.* **58**:275–286. DOI:10.1111/j.1365-313X.2008.03778.x
 109. Yin Y., Vafeados D., Tao Y., et al. (2005). A new class of transcription factors mediates brassinosteroid-regulated gene expression in *Arabidopsis*. *Cell* **120**:249–259. DOI:10.1016/j.cell.2004.11.044
 110. Tong H. and Chu C. (2018). Functional specificities of brassinosteroid and potential utilization for crop improvement. *Trends Plant Sci.* **23**:1016–1028. DOI:10.1016/j.tplants.2018.08.007
 111. Niu M., Wang H., Yin W., et al. (2022). Rice DWARF AND LOW-TILLERING and the homeodomain protein OSH15 interact to regulate internode elongation via orchestrating brassinosteroid signaling and metabolism. *Plant Cell* **34**:3754–3772. DOI:10.1093/plcell/koac196
 112. Tong H., Xiao Y., Liu D., et al. (2014). Brassinosteroid regulates cell elongation by modulating gibberellin metabolism in rice. *Plant Cell* **26**:4376–4393. DOI:10.1105/tpc.114.132092
 113. Unterholzner S., Rozhon W., Papacek M., et al. (2015). Brassinosteroids are master regulators of gibberellin biosynthesis in *Arabidopsis*. *Plant Cell* **27**:2261–2272. DOI:10.1105/tpc.15.00433
 114. Bai M., Shang J., Oh E., et al. (2012). Brassinosteroid, gibberellin and phytochrome impinge on a common transcription module in *Arabidopsis*. *Nat. Cell Biol.* **14**:810–U878. DOI:10.1038/ncb2546
 115. Gallego-Bartolomé J., Minguet E., Grau-Enguix F., et al. (2012). Molecular mechanism for the interaction between gibberellin and brassinosteroid signaling pathways in *Arabidopsis*. *Proc. Natl. Acad. Sci. USA* **109**:13446–13451. DOI:10.1073/pnas.1119992109
 116. Chen Y., Fan X., Song W., et al. (2012). Over-expression of OsPIN2 leads to increased tiller numbers, angle and shorter plant height through suppression of OsLAZY1. *Plant Biotechnol. J.* **10**:139–149. DOI:10.1111/j.1467-7652.2011.00637.x
 117. Multani D., Briggs S., Chamberlin M., et al. (2003). Loss of an MDR transporter in compact stalks of maize *br2* and sorghum *dw3* mutants. *Science* **302**:81–84. DOI:10.1126/science.1086072
 118. Li A., Hao C., Wang Z., et al. (2022). Wheat breeding history reveals synergistic selection of pleiotropic genomic sites for plant architecture and grain yield. *Mol. Plant* **15**:504–519. DOI:10.1016/j.molp.2022.01.004
 119. Hirano K., Yoshida H., Aya K., et al. (2017). SMALL ORGAN SIZE 1 and SMALL ORGAN SIZE 2/DWARF AND LOW-TILLERING form a complex to integrate auxin and brassinosteroid signaling in rice. *Mol. Plant* **10**:590–604. DOI:10.1016/j.molp.2016.12.013
 120. Tian Z., Chen B., Li H., et al. (2024). Strigolactone-gibberellin crosstalk mediated by a distant silencer fine-tunes plant height in upland cotton. *Mol. Plant* **17**:1539–1557. DOI:10.1016/j.molp.2024.08.007
 121. Lazar G. and Goodman H. (2006). MAX1, a regulator of the flavonoid pathway, controls vegetative axillary bud outgrowth in *Arabidopsis*. *Proc. Natl. Acad. Sci. USA* **103**:472–476. DOI:10.1073/pnas.0509463102
 122. Lin H., Wang R., Qian Q., et al. (2009). DWARF27, an iron-containing protein required for the biosynthesis of strigolactones, regulates rice tiller bud outgrowth. *Plant Cell* **21**:1512–1525. DOI:10.1105/tpc.109.065987
 123. Liu F., Wang P., Zhang X., et al. (2018). The genetic and molecular basis of crop height based on a rice model. *Planta* **247**:1–26. DOI:10.1007/s00425-017-2798-1
 124. Schumacher K., Schmitt T., Rossberg M., et al. (1999). The *Lateral suppressor* (*Ls*) gene of tomato encodes a new member of the VHLID protein family. *Proc. Natl. Acad. Sci. USA* **96**:290–295. DOI:10.1073/pnas.96.1.290
 125. Li X., Qian Q., Fu Z., et al. (2003). Control of tillering in rice. *Nature* **422**:618–621. DOI:10.1038/nature01518
 126. Zhu Y. and Wagner D. (2020). Plant inflorescence architecture: The formation, activity, and fate of axillary meristems. *Cold Spring Harbor Perspect. Biol.* **12**:a034652. DOI:10.1101/cshperspect.a034652
 127. Shi B., Zhang C., Tian C., et al. (2016). Two-step regulation of a meristematic cell population acting in shoot branching in *Arabidopsis*. *PLoS Genet.* **12**:e1006168. DOI:10.1371/journal.pgen.1006168
 128. Fletcher L., Brand U., Running M., et al. (1999). Signaling of cell fate decisions by

- CLAVATA3 in *Arabidopsis* shoot meristems. *Science* **283**:1911–1914. DOI:10.1126/science.283.5409.1911
129. Shao G., Lu Z., Xiong J., et al. (2019). Tiller bud formation regulators *MOC1* and *MOC3* cooperatively promote tiller bud outgrowth by activating *FON1* expression in rice. *Mol. Plant* **12**:1090–1102. DOI:10.1016/j.molp.2019.04.008
130. Lu Z., Shao G., Xiong J., et al. (2015). *MONOCULM 3*, an ortholog of *WUSCHEL* in rice, is required for tiller bud formation. *J. Genet. Genomics* **42**:71–78. DOI:10.1016/j.jgg.2014.12.005
131. Brand U., Grünwald M., Hobe M., et al. (2002). Regulation of *CLV3* expression by two homeobox genes in *Arabidopsis*. *Plant Physiol.* **129**:565–575. DOI:10.1104/pp.001867
132. Keller T., Abbott J., Moritz T., et al. (2006). *Arabidopsis* *REGULATOR OF AXILLARY MERISTEMS1* controls a leaf axil stem cell niche and modulates vegetative development. *Plant Cell* **18**:598–611. DOI:10.1105/tpc.105.038588
133. Müller D., Schmitz G. and Theres K. (2006). *Blind* homologous *R2R3 Myb* genes control the pattern of lateral meristem initiation in *Arabidopsis*. *Plant Cell* **18**:586–597. DOI:10.1105/tpc.105.038745
134. Schmitz G., Tillmann E., Carriero F., et al. (2002). The tomato *Blind* gene encodes a MYB transcription factor that controls the formation of lateral meristems. *Proc. Natl. Acad. Sci. USA* **99**:1064–1069. DOI:10.1073/pnas.022516199
135. Jelfetz D., David-Schwartz R., Borovsky Y., et al. (2011). *CaBLIND* regulates axillary meristem initiation and transition to flowering in *pepper*. *Planta* **234**:1227–1236. DOI:10.1007/s00425-011-1479-8
136. Guo D., Zhang J., Wang X., et al. (2015). The WRKY transcription factor *WRKY71/EXB1* controls shoot branching by transcriptionally regulating *RAX* Genes in *Arabidopsis*. *Plant Cell* **27**:3112–3127. DOI:10.1105/tpc.15.00829
137. Yang F., Wang Q., Schmitz G., et al. (2012). The bHLH protein ROX acts in concert with RAX1 and LAS to modulate axillary meristem formation in *Arabidopsis*. *Plant J.* **71**:61–70. DOI:10.1111/j.1365-313X.2012.04970.x
138. Doebley J., Stec A. and Hubbard L. (1997). The evolution of apical dominance in maize. *Nature* **386**:485–488. DOI:10.1038/386485a0
139. Hubbard L., McSteen P., Doebley J., et al. (2002). Expression patterns and mutant phenotype of *teosinte branched1* correlate with growth suppression in maize and teosinte. *Genetics* **162**:1927–1935. DOI:10.1093/genetics/162.4.1927
140. Aguilar-Martínez J. A., Poza-Carrión C. and Cubas P. (2007). *Arabidopsis* *BRANCHED1* acts as an integrator of branching signals within axillary buds. *Plant Cell* **19**:458–472. DOI:10.1105/tpc.106.048934
141. Minakuchi K., Kameoka H., Yasuno N., et al. (2010). *FINE CULM1 (FC1)* works downstream of strigolactones to inhibit the outgrowth of axillary buds in rice. *Plant Cell Physiol.* **51**:1127–1135. DOI:10.1093/pcp/pcq083
142. Braun N., de Saint Germain A., Pillot J. P., et al. (2012). The pea TCP transcription factor PsBRC1 acts downstream of strigolactones to control shoot branching. *Plant Physiol.* **158**:225–238. DOI:10.1104/pp.111.182725
143. Xie Y. R., Liu Y., Ma M. D., et al. (2020). *FHY3* and *FAR1* integrate light and strigolactone signaling to regulate branching. *Nat. Commun.* **11**:1955. DOI:10.1038/S41467-020-15893-7
144. Otori K., Tanabe N., Tamoi M., et al. (2019). *Sugar Transporter Protein 1 (STP1)* contributes to regulation of the genes involved in shoot branching via carbon partitioning in *Arabidopsis*. *Biosci. Biotechnol. Biochem.* **83**:472–481. DOI:10.1080/09168451.2018.1550355
145. Dun E. A., de Saint Germain A., Rameau C., et al. (2012). Antagonistic action of strigolactone and cytokinin in bud outgrowth control. *Plant Physiol.* **158**:487–498. DOI:10.1104/pp.111.186783
146. Wang L., Wang B., Yu H., et al. (2020). Transcriptional regulation of strigolactone signalling in *Arabidopsis*. *Nature* **583**:277–281. DOI:10.1038/s41586-020-2382-x
147. Hu J., Ji Y. Y., Hu X. T., et al. (2020). *BES1* functions as the co-regulator of *D53-like* SMXLs to inhibit *BRC1* expression in strigolactone-regulated shoot branching in *Arabidopsis*. *Plant Commun.* **1**:100014. DOI:10.1016/J.Xplc.2019.100014
148. González-Grandío E., Pajaro A., Franco-Zorrilla J. M., et al. (2017). Absciscic acid signaling is controlled by a *BRANCHED1/HD-ZIP I* cascade in *Arabidopsis* axillary buds. *Proc. Natl. Acad. Sci. USA* **114**:E245–E254. DOI:10.1073/pnas.1613199114
149. Liu X., Hu Q., Yan J., et al. (2020). γ -Carotene isomerase suppresses tillering in rice through the coordinated biosynthesis of strigolactone and abscisic acid. *Mol. Plant* **13**:1784–1801. DOI:10.1016/j.molp.2020.10.001
150. Luo L., Takahashi M., Kameoka H., et al. (2019). Developmental analysis of the early steps in strigolactone-mediated axillary bud dormancy in rice. *Plant J.* **97**:1006–1021. DOI:10.1111/tpj.14266
151. Zhou A., Kane A., Wu S., et al. (2025). Evolution of interorganismal strigolactone biosynthesis in seed plants. *Science* **387**:eadp0779. DOI:10.1126/science.adp0779
152. Abe S., Sado A., Tanaka K., et al. (2014). Carlactone is converted to carlactonoic acid by MAX1 in *Arabidopsis* and its methyl ester can directly interact with AtD14 in vitro. *Proc. Natl. Acad. Sci. USA* **111**:18084–18089. DOI:10.1073/pnas.1410801111
153. Jiang L., Liu X., Xiong G. S., et al. (2013). *DWARF 53* acts as a repressor of strigolactone signalling in rice. *Nature* **504**:401–405. DOI:10.1038/nature12870
154. Soundappan I., Bennett T., Morffy N., et al. (2015). *SMAX1-LIKE/D53* family members enable distinct MAX2-dependent responses to strigolactones and karrikins in *Arabidopsis*. *Plant Cell* **27**:3143–3159. DOI:10.1105/tpc.15.00562
155. Wang L., Wang B., Jiang L., et al. (2015). Strigolactone signaling in *Arabidopsis* regulates shoot development by targeting *D53-like* SMXL repressor proteins for ubiquitination and degradation. *Plant Cell* **27**:3128–3142. DOI:10.1105/tpc.15.00605
156. Zhou F., Lin Q. B., Zhu L. H., et al. (2013). D14-SCF(D3)-dependent degradation of D53 regulates strigolactone signalling. *Nature* **504**:406–410. DOI:10.1038/nature12878
157. Song X., Lu Z., Yu H., et al. (2017). *IPA1* functions as a downstream transcription factor repressed by D53 in strigolactone signaling in rice. *Cell Res.* **27**:1128–1141. DOI:10.1038/cr.2017.102
158. Li Q., Yu H., Chang W., et al. (2024). SMXL5 attenuates strigolactone signaling in *Arabidopsis thaliana* by inhibiting SMXL7 degradation. *Mol. Plant* **17**:631–647. DOI:10.1016/j.molp.2024.03.006
159. Zhang J., Wang B., Xu H., et al. (2025). Root microbiota regulates tiller number in rice. *Cell* **188**:3152–3166. DOI:10.1016/j.cell.2025.03.033
160. Domagalska M. and Leyser O. (2011). Signal integration in the control of shoot branching. *Nat. Rev. Mol. Cell Biol.* **12**:211–221. DOI:10.1038/nrm3088
161. Tanaka M., Takei K., Kojima M., et al. (2006). Auxin controls local cytokinin biosynthesis in the nodal stem in apical dominance. *Plant J.* **45**:1028–1036. DOI:10.1111/j.1365-313X.2006.02656.x
162. Hayward A., Stinberg P., Beveridge C., et al. (2009). Interactions between auxin and strigolactone in shoot branching control. *Plant Physiol.* **151**:400–412. DOI:10.1104/pp.109.137646
163. Prusikiewicz P., Crawford S., Smith R., et al. (2009). Control of bud activation by an auxin transport switch. *Proc. Natl. Acad. Sci. USA* **106**:17431–17436. DOI:10.1073/pnas.0906696106
164. Shinohara N., Taylor C. and Leyser O. (2013). Strigolactone can promote or inhibit shoot branching by triggering rapid depletion of the auxin efflux protein PIN1 from the plasma membrane. *PLoS Biol.* **11**:e1001474. DOI:10.1371/journal.pbio.1001474
165. Fang Z., Ji Y., Hu J., et al. (2020). Strigolactones and brassinosteroids antagonistically regulate the stability of the *D53-OsBZR1* complex to determine *FC1* expression in rice tillering. *Mol. Plant* **13**:586–597. DOI:10.1016/j.molp.2019.12.005
166. Hu J., Sun S. and Wang X. (2020). Regulation of shoot branching by strigolactones and brassinosteroids: Conserved and specific functions of *Arabidopsis* *BES1* and rice *BZR1*. *Mol. Plant* **13**:808–810. DOI:10.1016/j.molp.2020.03.008
167. Wang Y., Sun S., Zhu W. J., et al. (2013). Strigolactone/MAX2-induced degradation of brassinosteroid transcriptional effector *BES1* regulates shoot branching. *Dev. Cell* **27**:681–688. DOI:10.1016/j.devcel.2013.11.010
168. Wang J., Tian C., Zhang C., et al. (2017). Cytokinin signaling activates *WUSCHEL* expression during axillary meristem initiation. *Plant Cell* **29**:1373–1387. DOI:10.1105/tpc.16.00579
169. Jasinski S., Piazza P., Craft J., et al. (2005). *KNOX* action in *Arabidopsis* is mediated by coordinate regulation of cytokinin and gibberellin activities. *Curr. Biol.* **15**:1560–1565. DOI:10.1016/j.cub.2005.07.023
170. Yanai O., Shani E., Dolezal K., et al. (2005). *Arabidopsis* *KNOX1* proteins activate cytokinin biosynthesis. *Curr. Biol.* **15**:1566–1571. DOI:10.1016/j.cub.2005.07.060
171. Lo S., Yang S., Chen K. T., et al. (2008). A novel class of gibberellin 2-Oxidases control semidwarfism, tillering, and root development in rice. *Plant Cell* **20**:2603–2618. DOI:10.1105/tpc.108.060913
172. Cao D., Chabikwa T., Barbier F., et al. (2023). Auxin-independent effects of apical dominance induce changes in phytohormones correlated with bud outgrowth. *Plant Physiol.* **192**:1420–1434. DOI:10.1093/plphys/kiad034
173. Takei K., Sakakibara H., Taniguchi M., et al. (2001). Nitrogen-dependent accumulation of cytokinins in root and the translocation to leaf: Implication of cytokinin species that induces gene expression of maize response regulator. *Plant Cell Physiol.* **42**:85–93. DOI:10.1093/PCP/Pce009
174. Foo E., Yoneyama K., Hugill C. J., et al. (2013). Strigolactones and the regulation of pea symbioses in response to nitrate and phosphate deficiency. *Mol. Plant* **6**:76–87. DOI:10.1093/mp/sss115
175. Yoneyama K., Xie X. N., Kusumoto D., et al. (2007). Nitrogen deficiency as well as phosphorus deficiency in sorghum promotes the production and exudation of 5-deoxystrigol, the host recognition signal for arbuscular mycorrhizal fungi and root parasites. *Planta* **227**:125–132. DOI:10.1007/s00425-007-0600-5
176. Hu Q., Liu H., He Y., et al. (2024). Regulatory mechanisms of strigolactone perception in rice. *Cell* **187**:7551–7567. DOI:10.1016/j.cell.2024.10.009
177. Wu K., Wang S., Song W., et al. (2020). Enhanced sustainable green revolution yield via nitrogen-responsive chromatin modulation in rice. *Science* **367**:eaaz2046. DOI:10.1126/science.aaz2046
178. Zhang J., Zhang Y., Chen J., et al. (2024). Sugar transporter modulates nitrogen-determined tillering and yield formation in rice. *Nat. Commun.* **15**:9233. DOI:10.1038/s41467-024-53651-1
179. Liu Y., Wang H., Jiang Z., et al. (2021). Genomic basis of geographical adaptation to soil nitrogen in rice. *Nature* **590**:600–605. DOI:10.1038/s41586-020-03091-w
180. de Jong M., George G., Ongaro V., et al. (2014). Auxin and strigolactone signaling are required for modulation of *Arabidopsis* shoot branching by nitrogen supply. *Plant Physiol.* **166**:384–U549. DOI:10.1104/pp.114.242388
181. Hou M. M., Luo F. F., Wu D. X., et al. (2021). *OsPIN9*, an auxin efflux carrier, is required for the regulation of rice tiller bud outgrowth by ammonium. *New Phytol.* **229**:935–949. DOI:10.1111/nph.16901
182. Zhou J., Jiao F., Wu Z., et al. (2008). *OsPHR2* is involved in phosphate-starvation

- signaling and excessive phosphate accumulation in shoots of plants. *Plant Physiol.* **146**:1673–1686. DOI:10.1104/pp.107.111443
183. Yuan K., Zhang H., Yu C., et al. (2023). Low phosphorus promotes *NSP1-NSP2* heterodimerization to enhance strigolactone biosynthesis and regulate shoot and root architecture in rice. *Mol. Plant* **16**:1811–1831. DOI:10.1016/j.molp.2023.09.022
 184. Liu Y., Jafari F. and Wang H. (2021). Integration of light and hormone signaling pathways in the regulation of plant shade avoidance syndrome. *Abiotech.* **2**:131–145. DOI:10.1007/s42994-021-00038-1
 185. Waite J. and Dardick C. (2018). *TILLER ANGLE CONTROL 1* modulates plant architecture in response to photosynthetic signals. *J. Exp. Bot.* **69**:4935–4944. DOI:10.1093/jxb/ery253
 186. Nakamura M., Nishimura T. and Morita M. T. (2019). Gravity sensing and signal conversion in plant gravitropism. *J. Exp. Bot.* **70**:3495–3506. DOI:10.1093/jxb/erz158
 187. Wang W., Gao H., Liang Y., et al. (2022). Molecular basis underlying rice tiller angle: Current progress and future perspectives. *Mol. Plant* **15**:125–137. DOI:10.1016/j.molp.2021.12.002
 188. Li P., Wang Y., Qian Q., et al. (2007). *LAZY1* controls rice shoot gravitropism through regulating polar auxin transport. *Cell Res.* **17**:402–410. DOI:10.1038/cr.2007.38
 189. Chen J., Yu R., Li N., et al. (2023). Amyloplast sedimentation repolarizes LAZYs to achieve gravity sensing in plants. *Cell* **186**:4788–4802. DOI:10.1016/j.cell.2023.09.014
 190. Liu L., Zhu Y., Shen L., et al. (2013). Emerging insights into florigen transport. *Curr. Opin. Plant Biol.* **16**:607–613. DOI:10.1016/j.pbi.2013.06.001
 191. Wötzel S., Andreoli M., Albani M., et al. (2022). *Arabis alpina*: A perennial model plant for ecological genomics and life-history evolution. *Mol. Ecol. Resour.* **22**:468–486. DOI:10.1111/1755-0998.13490
 192. André D., Marcon A., Lee K., et al. (2022). *FLOWERING LOCUS T* paralogs control the annual growth cycle in *Populus* trees. *Curr. Biol.* **32**:2988–2996. DOI:10.1016/j.cub.2022.05.023
 193. Zhang S., Huang G., Zhang Y., et al. (2023). Sustained productivity and agronomic potential of perennial rice. *Nat. Sustainability* **6**:81–88. DOI:10.1038/s41893-022-00997-3
 194. Wu C., Cheng Z. and Gao J. (2024). Mysterious Bamboo flowering phenomenon: A literature review and new perspectives. *Sci. Total Environ.* **911**:168695. DOI:10.1016/j.scitotenv.2023.168695
 195. Wu T., Liu Z., Yu T., et al. (2024). Flowering genes identification, network analysis, and database construction for 837 plants. *Hortic. Res.* **11**:uhae013. DOI:10.1093/hr/uhae013
 196. Bouché F., Lobet G., Tocquin P., et al. (2016). FLOR-ID: An interactive database of flowering-time gene networks in *Arabidopsis thaliana*. *Nucleic Acids Res.* **44**:D1167–D1171. DOI:10.1093/nar/gkv1054
 197. Xu Y., Kong X. D., Guo Y., et al. (2023). Structural variations and environmental specificities of flowering time-related genes in *Brassica napus*. *Theor. Appl. Genet.* **136**:42. DOI:10.1007/s00122-023-04326-W. DOI:10.1007/s00122-023-04326-w
 198. Calderwood A., Lloyd A., Hepworth J., et al. (2021). Total *FLC* transcript dynamics from divergent paralogue expression explains flowering diversity in *Brassica napus*. *New Phytol.* **229**:3534–3548. DOI:10.1111/nph.17131
 199. Wei H., Wang X., Xu H., et al. (2020). Molecular basis of heading date control in rice. *Abiotech* **1**:219–232. DOI:10.1007/s42994-020-00019-w
 200. Zhou S., Zhu S., Cui S., et al. (2021). Transcriptional and post-transcriptional regulation of heading date in rice. *New Phytol.* **230**:943–956. DOI:10.1111/nph.17158
 201. Jing Y., Guo Q., Zha P., et al. (2019). The chromatin-remodelling factor *PICKLE* interacts with *CONSTANS* to promote flowering in *Arabidopsis*. *Plant Cell Environ.* **42**:2495–2507. DOI:10.1111/pce.13557
 202. Jing Y., Guo Q. and Lin R. (2019). The chromatin-remodeling factor *PICKLE* antagonizes polycomb repression of *FT* to promote flowering. *Plant Physiol.* **181**:656–668. DOI:10.1104/pp.19.00596
 203. Chen Y., Song S., Gan Y., et al. (2020). SHAGGY-like kinase 12 regulates flowering through mediating *CONSTANS* stability in *Arabidopsis*. *Sci. Adv.* **6**:eaaw0413. DOI:10.1126/sciadv.aaw0413
 204. Lee N., Ozaki Y., Hempton A., et al. (2023). The *FLOWERING LOCUS T* gene expression is controlled by high-irradiance response and external coincidence mechanism in long days in *Arabidopsis*. *New Phytol.* **239**:208–221. DOI:10.1111/nph.18932
 205. Zhu Y., Klasfeld S. and Wagner D. (2021). Molecular regulation of plant developmental transitions and plant architecture via PEPB family proteins: An update on mechanism of action. *J. Exp. Bot.* **72**:2301–2311. DOI:10.1093/jxb/eraa598
 206. Zhu Y., Liu L., Shen L., et al. (2016). *NaKRI* regulates long-distance movement of *FLOWERING LOCUS T* in *Arabidopsis*. *Nat. Plants* **2**:16075. DOI:10.1038/Nplants.2016.75
 207. Wang T., Guo J., Peng Y., et al. (2021). Light-induced mobile factors from shoots regulate rhizobium-triggered soybean root nodulation. *Science* **374**:65–71. DOI:10.1126/science.abh2890
 208. Zhang Y., Hua C., Kiang J., et al. (2024). A dephosphorylation-dependent molecular switch for *FT* repression mediates flowering in *Arabidopsis*. *Plant Commun.* **5**:100779. DOI:10.1016/j.xplc.2023.100779
 209. Zhu Y., Klasfeld S., Jeong C., et al. (2020). *TERMINAL FLOWER 1-FD* complex target genes and competition with *FLOWERING LOCUS T*. *Nat. Commun.* **11**:5118. DOI:10.1038/s41467-020-18782-1
 210. Martignago D., Falavigna V., Lombardi A., et al. (2023). The bZIP transcription factor *AREB3* mediates *FT* signalling and floral transition at the *Arabidopsis* shoot apical meristem. *PLoS Genet.* **19**:e1010766. DOI:10.1371/journal.pgen.1010766
 211. Dong L., Cheng Q., Fang C., et al. (2022). Parallel selection of distinct *Tof5* alleles drove the adaptation of cultivated and wild soybean to high latitudes. *Mol. Plant* **15**:308–321. DOI:10.1016/j.molp.2021.10.004
 212. Zheng S., Hu H., Ren H., et al. (2019). The *Arabidopsis* H3K27me3 demethylase JUMONJI 13 is a temperature and photoperiod dependent flowering repressor. *Nat. Commun.* **10**:1303. DOI:10.1038/s41467-019-09310-X
 213. Susila H., Juric S., Liu L., et al. (2021). Florigen sequestration in cellular membranes modulates temperature-responsive flowering. *Science* **373**:1137–1141. DOI:10.1126/science.abh4054
 214. Barnes A., Rodríguez-Zapata F., Juárez-Núñez K., et al. (2022). An adaptive teosinte mexicana introgression modulates phosphatidylcholine levels and is associated with maize flowering time. *Proc. Natl. Acad. Sci. USA* **119**:e2100036119. DOI:10.1073/pnas.2100036119
 215. Yu Y., Qiao L., Chen J., et al. (2020). *Arabidopsis* *REM16* acts as a B3 domain transcription factor to promote flowering time via directly binding to the promoters of *SOC1* and *FT*. *Plant J.* **103**:1386–1398. DOI:10.1111/tpj.14807
 216. Shen L., Zhang Y. and Sawetallake N. (2022). A Molecular switch for *FLOWERING LOCUS C* activation determines flowering time in *Arabidopsis*. *Plant Cell* **34**:818–833. DOI:10.1093/plcell/koab286
 217. Li T., Wang Y., Natran A., et al. (2024). C-TERMINAL DOMAIN PHOSPHATASE-LIKE 3 contributes to GA-mediated growth and flowering by interaction with DELLA proteins. *New Phytol.* **242**:2555–2569. DOI:10.1111/nph.19742
 218. Ortuño-Miquel S., Rodríguez-Cazorla E., Zavala-Gonzalez E., et al. (2019). *Arabidopsis* *HUA ENHANCER 4* delays flowering by upregulating the MADS-box repressor genes *FLC* and *MAF4*. *Sci. Rep.* **9**:1478. DOI:10.1038/s41598-018-38327-3
 219. Yuan C., Hu Y., Liu Q., et al. (2023). *MED8* regulates floral transition in *Arabidopsis* by interacting with *FPA*. *Plant J.* **116**:1234–1247. DOI:10.1111/tpj.16419
 220. Wang Y., Tao S., Wang W., et al. (2020). Molecular variation in a functionally divergent homolog of *FCA* regulates flowering time in *Arabidopsis thaliana*. *Nat. Commun.* **11**:5830. DOI:10.1038/s41467-020-19666-0
 221. Li X. D., Chen L. M., Yao L., et al. (2022). Calcium-dependent protein kinase CPK32 mediates calcium signaling in regulating *Arabidopsis* flowering time. *Natl. Sci. Rev.* **9**:nwab180. DOI:10.1093/nsr/nwab180
 222. Zhu P., Lister C. and Dean C. (2021). Cold-induced *Arabidopsis* *FRIGIDA* nuclear condensates for *FLC* repression. *Nature* **599**:657–661. DOI:10.1038/s41586-021-04062-5
 223. Fulgione A., Neto C., Elfarargi A., et al. (2022). Parallel reduction in flowering time from de novo mutations enable evolutionary rescue in colonizing lineages. *Nat. Commun.* **13**:1461. DOI:10.1038/s41467-022-28800-Z
 224. Guo X., Liang R., Lou S., et al. (2023). Natural variation in the *SVP* contributes to the pleiotropic adaption of *Arabidopsis thaliana* across contrasted habitats. *J. Genet. Genomics* **50**:993–1003. DOI:10.1016/j.jgg.2023.08.004
 225. Yuan S., Zhang Z., Zheng C., et al. (2016). *Arabidopsis* cryptochrome 1 functions in nitrogen regulation of flowering. *Proc. Natl. Acad. Sci. USA* **113**:7661–7666. DOI:10.1073/pnas.1602004113
 226. Sanagi M., Aoyama S., Kubo A., et al. (2021). Low nitrogen conditions accelerate flowering by modulating the phosphorylation state of *FLOWERING BHLH 4* in *Arabidopsis*. *Proc. Natl. Acad. Sci. USA* **118**:e2022942118. DOI:10.1073/pnas.2022942118
 227. Chen H., Lin S., Cheng L., et al. (2021). Potential transceptor *AtNRT1.13* modulates shoot architecture and flowering time in a nitrate-dependent manner. *Plant Cell* **33**:3595–3595. DOI:10.1093/plcell/koab172
 228. Xu T., Wu X., Wong C., et al. (2022). *FIONA1*-mediated m⁶A modification regulates the floral transition in *Arabidopsis*. *Adv. Sci.* **9**:2103628. DOI:10.1002/Adv.202103628
 229. Liu S., Chen S., Zhou Y., et al. (2023). *VERNALIZATION1* represses *FLOWERING PROMOTING FACTOR1-LIKE1* in leaves for timely flowering in *Brachypodium distachyon*. *Plant Cell* **35**:3697–3711. DOI:10.1093/plcell/koad190
 230. Yu B., He X., Tang Y., et al. (2023). Photoperiod controls plant seed size in a *CONSTANS*-dependent manner. *Nat. Plants* **9**:343–354. DOI:10.1038/s41477-023-01350-y
 231. Zuo X., Wang S., Liu X., et al. (2024). *FLOWERING LOCUS T1* and *TERMINAL FLOWER1* regulatory networks mediate flowering initiation in apple. *Plant Physiol.* **195**:580–597. DOI:10.1093/plphys/kiad086
 232. Li X., Zhou H., Cheng L., et al. (2022). Shoot-to-root translocated GmNN1/FT2a triggers nodulation and regulates soybean nitrogen nutrition. *PLoS Biol.* **20**:e3001739. DOI:10.1371/journal.pbio.3001739
 233. Schiessl S., Quezada-Martinez D., Tebartz E., et al. (2019). The vernalisation regulator *FLOWERING LOCUS C* is differentially expressed in biennial and annual *Brassica napus*. *Sci. Rep.* **9**:14911. DOI:10.1038/s41598-019-51212-X
 234. Yin S., Wan M., Guo C., et al. (2020). Transposon insertions within alleles of

- BnaFLC.A10* and *BnaFLC.A2* are associated with seasonal crop type in rapeseed. *J. Exp. Bot.* **71**:4729–4741. DOI:10.1093/jxb/eraa237
235. Zhai D., Zhang L., Li L., et al. (2024). Reciprocal conversion between annual and polycarpic perennial flowering behavior in the Brassicaceae. *Cell* **187**:3319–3337. DOI:10.1016/j.cell.2024.04.047
 236. Blümel M., Dally N. and Jung C. (2015). Flowering time regulation in crops – what did we learn from *Arabidopsis*. *Curr. Opin. Biotechnol.* **32**:121–129. DOI:10.1016/j.copbio.2014.11.023
 237. Franks S. J., Sim S. and Weis A. E. (2007). Rapid evolution of flowering time by an annual plant in response to a climate fluctuation. *Proc. Natl. Acad. Sci. USA* **104**:1278–1282. DOI:10.1073/pnas.0608379104
 238. O'Neill C., Lu X., Calderwood A., et al. (2019). Vernalization and floral transition in autumn drive winter annual life history in oilseed rape. *Curr. Biol.* **29**:4300–4306. DOI:10.1016/j.cub.2019.10.051
 239. Shan H., Cheng J., Zhang R., et al. (2019). Developmental mechanisms involved in the diversification of flowers. *Nat. Plants* **5**:917–923. DOI:10.1038/s41477-019-0498-5
 240. Chandler J. (2011). Founder cell specification. *Trends Plant Sci.* **16**:607–613. DOI:10.1016/j.tplants.2011.08.005
 241. Cheng Y., Dai X. and Zhao Y. (2006). Auxin biosynthesis by the YUCCA flavin monooxygenases controls the formation of floral organs and vascular tissues in *Arabidopsis*. *Genes Dev.* **20**:1790–1799. DOI:10.1101/gad.1415106
 242. Okada K., Ueda J., Komaki M., et al. (1991). Requirement of the auxin polar transport system in early stages of *Arabidopsis* floral bud formation. *Plant Cell* **3**:677–684. DOI:10.1105/tpc.3.7.677
 243. Besnard F., Refahi Y., Morin V., et al. (2014). Cytokinin signalling inhibitory fields provide robustness to phyllotaxis. *Nature* **505**:417–421. DOI:10.1038/nature12791
 244. Schoof H., Lenhard M., Haecker A., et al. (2000). The stem cell population of *Arabidopsis* shoot meristems is maintained by a regulatory loop between the *CLAVATA* and *WUSCHEL* genes. *Cell* **100**:635–644. DOI:10.1016/S0092-8674(00)80700-x
 245. Laux T., Mayer K., Berger J., et al. (1996). The *WUSCHEL* gene is required for shoot and floral meristem integrity in *Arabidopsis*. *Development* **122**:87–96. DOI:10.1242/dev.122.1.87
 246. Sun B. and Ito T. (2015). Regulation of floral stem cell termination in *Arabidopsis*. *Front. Plant Sci.* **6**:17. DOI:10.3389/fpls.2015.00017
 247. Pelayo M., Yamaguchi N. and Ito T. (2021). One factor, many systems: The floral homeotic protein *AGAMOUS* and its epigenetic regulatory mechanisms. *Curr. Opin. Plant Biol.* **61**:102009. DOI:10.1016/j.pbi.2021.102009
 248. Chandler J. and Werr W. (2017). *DORNROSCHEN*, *DORNROSCHEN-LIKE*, and *PUCHI* redundantly control floral meristem identity and organ initiation in *Arabidopsis*. *J. Exp. Bot.* **68**:3457–3472. DOI:10.1093/jxb/erx208
 249. Aida M., Ishida T., Fukaki H., et al. (1997). Genes involved in organ separation in *Arabidopsis*: An analysis of the cup-shaped cotyledon mutant. *Plant Cell* **9**:841–857. DOI:10.1105/tpc.9.6.841
 250. Sieber P., Wellmer F., Gheyselinck J., et al. (2007). Redundancy and specialization among plant microRNAs: Role of the *MIR164* family in developmental robustness. *Development* **134**:1051–1060. DOI:10.1242/dev.02817
 251. Theissen G. (2001). Development of floral organ identity: Stories from the MADS house. *Curr. Opin. Plant Biol.* **4**:75–85. DOI:10.1016/S1369-5266(00)00139-4
 252. Coen E. and Meyerowitz E. (1991). The war of the whorls: Genetic interactions controlling flower development. *Nature* **353**:31–37. DOI:10.1038/353031a0
 253. Causier B., Schwarz-Sommer Z. and Davies B. (2010). Floral organ identity: 20 years of ABCs. *Semin. Cell Dev. Biol.* **21**:73–79. DOI:10.1016/j.semdb.2009.10.005
 254. Becker A. and Theissen G. (2003). The major clades of MADS-box genes and their role in the development and evolution of flowering plants. *Mol. Phylogenet. Evol.* **29**:464–489. DOI:10.1016/S1055-7903(03)00207-0
 255. O'Maolighigh D., Graciet E. and Wellmer F. (2014). Gene networks controlling *Arabidopsis thaliana* flower development. *New Phytol.* **201**:16–30. DOI:10.1111/nph.12444
 256. Krizek B., Lewis M. and Fletcher J. (2006). *RABBIT EARS* is a second-whorl repressor of *AGAMOUS* that maintains spatial boundaries in *Arabidopsis* flowers. *Plant J.* **45**:369–383. DOI:10.1111/j.1365-3113X.2005.02633.x
 257. Prunet N., Yang W., Das P., et al. (2017). *SUPERMAN* prevents class B gene expression and promotes stem cell termination in the fourth whorl of *Arabidopsis thaliana* flowers. *Proc. Natl. Acad. Sci. USA* **114**:7166–7171. DOI:10.1073/pnas.1705977114
 258. Wollmann H., Mica E., Todesco M., et al. (2010). On reconciling the interactions between *APETALA2*, *miR172* and *AGAMOUS* with the ABC model of flower development. *Development* **137**:3633–3642. DOI:10.1242/dev.036673
 259. Gómez-Felipe A., Branchini E., Wang B., et al. (2024). Two orthogonal differentiation gradients locally coordinate fruit morphogenesis. *Nat. Commun.* **15**:2912. DOI:10.1038/S41467-024-47325-1
 260. Roeder A. (2021). *Arabidopsis* sepals: A model system for the emergent process of morphogenesis. *Quant. Plant Biol.* **2**:e14. DOI:10.1017/qpb.2021.12
 261. Sauret-Gueto S., Schiessl K., Bangham A., et al. (2013). *JAGGED* controls *Arabidopsis* petal growth and shape by interacting with a divergent polarity field. *PLoS Biol.* **11**:e1001550. DOI:10.1371/journal.pbio.1001550
 262. Silveira S., Le Gloanec C., Gomez-Felipe A., et al. (2022). Live-imaging provides an atlas of cellular growth dynamics in the stamen. *Plant Physiol.* **188**:769–781. DOI:10.1093/plphys/kiab363
 263. Fu X., Shan H., Yao X., et al. (2022). Petal development and elaboration. *J. Exp. Bot.* **73**:3308–3318. DOI:10.1093/jxb/erac092
 264. Fukushima K. and Hasebe M. (2014). Adaxial-abaxial polarity: The developmental basis of leaf shape diversity. *Genesis* **52**:1–18. DOI:10.1002/dvg.22728
 265. Walcher-Chevillet C. and Kramer E. (2016). Breaking the mold: Understanding the evolution and development of lateral organs in diverse plant models. *Curr. Opin. Genet. Dev.* **39**:79–84. DOI:10.1016/j.gde.2016.06.005
 266. Cheng J., Yao X., Li X., et al. (2023). Diversification of ranunculaceous petals in shape supports a generalized model for plant lateral organ morphogenesis and evolution. *Sci. Adv.* **9**:eadf8049. DOI:10.1126/sciadv.adf8049
 267. Lian H., Li X., Liu Z., et al. (2013). *HYL1* is required for establishment of stamen architecture with four microsporangia in *Arabidopsis*. *J. Exp. Bot.* **64**:3397–3410. DOI:10.1093/jxb/ert178
 268. Vandenbussche M., Horstman A., Zethof J., et al. (2009). Differential recruitment of transcription factors for lateral development and organ fusion in *Petunia* and *Arabidopsis*. *Plant Cell* **21**:2269–2283. DOI:10.1105/tpc.109.065862
 269. Dinneny J., Weigel D. and Yanofsky M. (2006). *NUBBIN* and *JAGGED* define stamen and carpel shape in *Arabidopsis*. *Development* **133**:1645–1655. DOI:10.1242/dev.02335
 270. Dinneny J., Yadegari R., Fischer R., et al. (2004). The role of *JAGGED* in shaping lateral organs. *Development* **131**:1101–1110. DOI:10.1242/dev.00949
 271. Roeder A., Chickarmane V., Cunha A., et al. (2010). Variability in the control of cell division underlies sepal epidermal patterning in *Arabidopsis thaliana*. *PLoS Biol.* **8**:e1000367. DOI:10.1371/journal.pbio.1000367
 272. Roeder A., Cunha A., Ohno C., et al. (2012). Cell cycle regulates cell type in the *Arabidopsis* sepal. *Development* **139**:4416–4427. DOI:10.1242/dev.082925
 273. Ren H., Dang X., Cai X., et al. (2017). Spatio-temporal orientation of microtubules controls conical cell shape in *Arabidopsis thaliana* petals. *PLoS Genet.* **13**:e1006851. DOI:10.1371/journal.pgen.1006851
 274. Saffer A., Carpita N. and Irish V. (2017). Rhamnose-containing cell wall polymers suppress helical plant growth independently of microtubule orientation. *Curr. Biol.* **27**:2248–2259 e2244. DOI:10.1016/j.cub.2017.06.032
 275. Pyke K. and Page A. (1998). Plastid ontogeny during petal development in *Arabidopsis*. *Plant Physiol.* **116**:797–803. DOI:10.1104/pp.116.2.797
 276. Zheng X., Lan J., Yu H., et al. (2022). *Arabidopsis* transcription factor TCP4 represses chlorophyll biosynthesis to prevent petal greening. *Plant Commun.* **3**:100309. DOI:10.1016/j.xplc.2022.100309
 277. Bowman J. (1997). Evolutionary conservation of angiosperm flower development at the molecular and genetic levels. *J. Biosci.* **22**:515–527. DOI:10.1007/Bf02703197
 278. Buzgo M., Soltis P. and Soltis D. (2004). Floral developmental morphology of *Amborella trichopoda* (Amborellaceae). *Int. J. Plant Sci.* **165**:925–947. DOI:10.1086/424024
 279. Cirilli M., Rossini L., Chiozzotto R., et al. (2022). Less is more: Natural variation disrupting a *miR172* gene at the *di* locus underlies the recessive double-flower trait in peach (*P. persica* L. Batsch). *BMC Plant Biol.* **22**:318. DOI:10.1186/s12870-022-03691-w
 280. Gattolin S., Cirilli M., Chessa S., et al. (2020). Mutations in orthologous *PETALOSA TOE*-type genes cause a dominant double-flower phenotype in phylogenetically distant eudicots. *J. Exp. Bot.* **71**:2585–2595. DOI:10.1093/jxb/eraa032
 281. Cartolano M., Castillo R., Efremova N., et al. (2007). A conserved microRNA module exerts homeotic control over *Petunia hybrida* and *Antirrhinum majus* floral organ identity. *Nat. Genet.* **39**:901–905. DOI:10.1038/ng2056
 282. Duan X., Zhao C., Jiang Y., et al. (2020). Parallel evolution of apetalous lineages within the buttercup family (Ranunculaceae): Outward expansion of *AGAMOUS1*, rather than disruption of *APETALA3-3*. *Plant J.* **104**:1169–1181. DOI:10.1111/tjp.14985
 283. Hsu H., Chen W., Shen Y., et al. (2021). Multifunctional evolution of *B* and *AGL6* MADS box genes in orchids. *Nat. Commun.* **12**:902. DOI:10.1038/s41467-021-21229-w
 284. Hsu H., Hsu W., Lee Y., et al. (2015). Model for perianth formation in orchids. *Nat. Plants* **1**:15046. DOI:10.1038/Nplants.2015.46
 285. Sharma B. and Kramer E. (2013). Sub- and neo-functionalization of *APETALA3* paralogs have contributed to the evolution of novel floral organ identity in *Aquilegia* (columbine, Ranunculaceae). *New Phytol.* **197**:949–957. DOI:10.1111/nph.12078
 286. Koyama T., Ohme-Takagi M. and Sato F. (2011). Generation of serrated and wavy petals by inhibition of the activity of TCP transcription factors in *Arabidopsis thaliana*. *Plant Signal. Behav.* **6**:697–699. DOI:10.4161/psb.6.5.14979
 287. Zheng G., Wei W., Li Y., et al. (2019). Conserved and novel roles of *miR164-CUC2* regulatory module in specifying leaf and floral organ morphology in strawberry. *New Phytol.* **224**:480–492. DOI:10.1111/nph.15982
 288. Ballerini E., Min Y., Edwards M., et al. (2020). *POPOVICH*, encoding a C2H2 zinc-finger transcription factor, plays a central role in the development of a key innovation, floral nectar spurs, in *Aquilegia*. *Proc. Natl. Acad. Sci. USA* **117**:22552–22560. DOI:10.1073/pnas.2006912117
 289. Puzey J., Gerbode S., Hodges S., et al. (2012). Evolution of spur-length diversity in

- Aquilegia* petals is achieved solely through cell-shape anisotropy. *Proc. R. Soc. B Biol. Sci.* **279**:1640–1645. DOI:10.1098/rspb.2011.1873
290. Zhang R., Min Y., Holappa L., et al. (2020). A role for the auxin response factors *ARF6* and *ARF8* homologs in petal spur elongation and nectary maturation in *Aquilegia*. *New Phytol.* **227**:1392–1405. DOI:10.1111/nph.16633
291. Yao X., Zhang W., Duan X., et al. (2019). The making of elaborate petals in *Nigella* through developmental repatterning. *New Phytol.* **223**:385–396. DOI:10.1111/nph.15799
292. Fairnie A., Yeo M., Gatti S., et al. (2022). Eco-Evo-Devo of petal pigmentation patterning. *Essays Biochem.* **66**:753–768. DOI:10.1042/Ebc20220051
293. Bradshaw E., Rudall P., Devey D., et al. (2010). Comparative labellum micromorphology of the sexually deceptive temperate orchid genus *Ophrys*: Diverse epidermal cell types and multiple origins of structural colour. *Bot. J. Linn. Soc.* **162**:504–540. DOI:10.1111/j.1095-8339.2010.01033.x
294. Zhang R., Fu X., Zhao C., et al. (2020). Identification of the key regulatory genes involved in elaborate petal development and specialized character formation in *Nigella damascena* (Ranunculaceae). *Plant Cell* **32**:3095–3112. DOI:10.1105/tpc.20.00330
295. Galego L. and Almeida J. (2002). Role of *DIVARICATA* in the control of dorsoventral asymmetry in *Antirrhinum* flowers. *Genes Dev.* **16**:880–891. DOI:10.1101/gad.221002
296. Luo D., Carpenter R., Copsey L., et al. (1999). Control of organ asymmetry in flowers of *Antirrhinum*. *Cell* **99**:367–376. DOI:10.1016/S0092-8674(00)81523-8
297. Luo D., Carpenter R., Vincent C., et al. (1996). Origin of floral asymmetry in *Antirrhinum*. *Nature* **383**:794–799. DOI:10.1038/383794a0
298. Busch A., Horn S., Mühlhausen A., et al. (2012). Corolla monosymmetry: Evolution of a morphological novelty in the Brassicaceae family. *Mol. Biol. Evol.* **29**:1241–1254. DOI:10.1093/molbev/msr297
299. Busch A. and Zachow S. (2009). Flower symmetry evolution: Towards understanding the abominable mystery of angiosperm radiation. *Bioessays* **31**:1181–1190. DOI:10.1002/bies.200900081
300. Chapman M. A., Tang S., Draeger D., et al. (2012). Genetic analysis of floral symmetry in Van Gogh's sunflowers reveals independent recruitment of *CYCLOIDEA* genes in the Asteraceae. *PLoS Genet.* **8**:e1002628. DOI:10.1371/journal.pgen.1002628
301. Howarth D., Martins T., Chimney E., et al. (2011). Diversification of *CYCLOIDEA* expression in the evolution of bilateral flower symmetry in Caprifoliaceae and *Lonicera* (Dipsacales). *Ann. Bot.* **107**:1521–1532. DOI:10.1093/aob/mcr049
302. Pabón-Mora N., Madrigal Y., Alzate J. F., et al. (2020). Evolution of Class II TCP genes in perianth bearing Pipeales and their contribution to the bilateral calyx in *Aristolochia*. *New Phytol.* **228**:752–769. DOI:10.1111/nph.16719
303. Sengupta A. and Hileman L. (2022). A CYC-RAD-DIV-DRIF interaction likely pre-dates the origin of floral monosymmetry in Lamiales. *EvoDevo* **13**:3. DOI:10.1186/s13227-021-00187-w
304. Zhang W., Kramer E. and Davis C. (2010). Floral symmetry genes and the origin and maintenance of zygomorphy in a plant-pollinator mutualism. *P. Natl. Acad. Sci. USA* **107**:6388–6393. DOI:10.1073/pnas.0910155107
305. Zhao H., Liao H., Li S., et al. (2023). Delphinieae flowers originated from the rewiring of interactions between duplicated and diversified floral organ identity and symmetry genes. *Plant Cell* **35**:994–1012. DOI:10.1093/plcell/koac368
306. Citerne H., Jabbour F., Nadot S., et al. (2010). The evolution of floral symmetry. *Adv. Bot. Res.* **54**:85–137. DOI:10.1016/S0065-2296(10)54003-5
307. Su S., Xiao W., Guo W., et al. (2017). The *CYCLOIDEA-RADIALIS* module regulates petal shape and pigmentation, leading to bilateral corolla symmetry in *Torenia fournieri* (Linderniaceae). *New Phytol.* **215**:1582–1593. DOI:10.1111/nph.14673
308. Zhao Y., Broholm S., Wang F., et al. (2020). TCP and MADS-box transcription factor networks regulate heteromorphic flower type identity in *Gerbera hybrida*. *Plant Physiol.* **184**:1455–1468. DOI:10.1104/pp.20.00702
309. Hohenstatt M., Mikulski P., Komarynets O., et al. (2018). *PWWP-DOMAIN INTERACTOR OF POLYCOMB1* interacts with polycomb-group proteins and histones and regulates *Arabidopsis* flowering and development. *Plant Cell* **30**:117–133. DOI:10.1105/tpc.17.00117
310. Zhou Y., Wang Y., Krause K., et al. (2018). Telobox motifs recruit CLF/SWN-PRC2 for H3K27me3 deposition via TRB factors in *Arabidopsis*. *Nat. Genet.* **50**:638–644. DOI:10.1038/s41588-018-0109-9
311. Drews G., Bowman J. and Meyerowitz E. (1991). Negative regulation of the *Arabidopsis* homeotic gene *AGAMOUS* by the *APETALA2* product. *Cell* **65**:991–1002. DOI:10.1016/0092-8674(91)90551-9
312. Huang Z., Shi T., Zheng B., et al. (2017). *APETALA2* antagonizes the transcriptional activity of *AGAMOUS* in regulating floral stem cells in *Arabidopsis thaliana*. *New Phytol.* **215**:1197–1209. DOI:10.1111/nph.14151
313. Krogan N., Hogan K. and Long J. (2012). *APETALA2* negatively regulates multiple floral organ identity genes in *Arabidopsis* by recruiting the co-repressor TOPLESS and the histone deacetylase HDA19. *Development* **139**:4180–4190. DOI:10.1242/dev.085407
314. Brand U., Fletcher J., Hobe M., et al. (2000). Dependence of stem cell fate in *Arabidopsis* on a feedback loop regulated by CLV3 activity. *Science* **289**:617–619. DOI:10.1126/science.289.5479.617
315. Cao X., He Z., Guo L., et al. (2015). Epigenetic mechanisms are critical for the regulation of *WUSCHEL* expression in floral meristems. *Plant Physiol.* **168**:1189–1196. DOI:10.1104/pp.15.00230
316. Lohmann J., Hong R., Hobe M., et al. (2001). A molecular link between stem cell regulation and floral patterning in *Arabidopsis*. *Cell* **105**:793–803. DOI:10.1016/S0092-8674(01)00384-1
317. Maier A., Stehling-Sun S., Wollmann H., et al. (2009). Dual roles of the bZIP transcription factor *PERIANTHIA* in the control of floral architecture and homeotic gene expression. *Development* **136**:1613–1620. DOI:10.1242/dev.033647
318. Yanofsky M., Ma H., Bowman J., et al. (1990). The protein encoded by the *Arabidopsis* homeotic gene *agamous* resembles transcription factors. *Nature* **346**:35–39. DOI:10.1038/346035a0
319. Liu X., Kim Y., Müller R., et al. (2011). *AGAMOUS* terminates floral stem cell maintenance in *Arabidopsis* by directly repressing *WUSCHEL* through recruitment of polycomb group proteins. *Plant Cell* **23**:3654–3670. DOI:10.1105/tpc.111.091538
320. Shang E., Wang X., Li T., et al. (2021). Robust control of floral meristem determinacy by position-specific multifunctions of *KNUCKLES*. *Proc. Natl. Acad. Sci. USA* **118**:e2102826118. DOI:10.1073/pnas.2102826118
321. Sun B., Xu Y., Ng K., et al. (2009). A timing mechanism for stem cell maintenance and differentiation in the *Arabidopsis* floral meristem. *Genes Dev.* **23**:1791–1804. DOI:10.1101/gad.1800409
322. Yamaguchi N., Huang J., Tatsumi Y., et al. (2018). Chromatin-mediated feed-forward auxin biosynthesis in floral meristem determinacy. *Nat. Commun.* **9**:5290. DOI:10.1038/s41467-018-07763-0
323. Gross T. and Becker A. (2021). Transcription factor action orchestrates the complex expression pattern of *CRABS CLAW* in *Arabidopsis*. *Genes* **12**:1663. DOI:10.3390/genes12111663
324. Scofield S., Dewitte W. and Murray J. (2007). The *KNOX* gene *SHOOT MERISTEMLESS* is required for the development of reproductive meristematic tissues in *Arabidopsis*. *Plant J.* **50**:767–781. DOI:10.1111/j.1365-313X.2007.03095.x
325. Spinelli S., Martin A., Viola I., et al. (2011). A mechanistic link between *STM* and *CUC1* during *Arabidopsis* development. *Plant Physiol.* **156**:1894–1904. DOI:10.1104/pp.111.177709
326. Marsch-Martínez N. and de Folter S. (2016). Hormonal control of the development of the gynoecium. *Curr. Opin. Plant Biol.* **29**:104–114. DOI:10.1016/j.pbi.2015.12.006
327. Kamiuchi Y., Yamamoto K., Furutani M., et al. (2014). The *CUC1* and *CUC2* genes promote carpel margin meristem formation during *Arabidopsis* gynoecium development. *Front. Plant Sci.* **5**:165. DOI:10.3389/fpls.2014.00165
328. Reyes-Olalde J., Zúñiga-Mayo V., Serwatowska J., et al. (2017). The bHLH transcription factor *SPATULA* enables cytokinin signaling, and both activate auxin biosynthesis and transport genes at the medial domain of the gynoecium. *PLoS Genet.* **13**:e1006726. DOI:10.1371/journal.pgen.1006726
329. Roeder A., Ferrándiz C. and Yanofsky M. (2003). The role of the *REPLUMLESS* homeodomain protein in patterning the *Arabidopsis* fruit. *Curr. Biol.* **13**:1630–1635. DOI:10.1016/j.cub.2003.08.027
330. Dinneny J., Weigel D. and Yanofsky M. (2005). A genetic framework for fruit patterning in *Arabidopsis thaliana*. *Development* **132**:4687–4696. DOI:10.1242/dev.02062
331. Gu Q., Ferrándiz C., Yanofsky M., et al. (1998). The *FRUITFULL MADS-box* gene mediates cell differentiation during *Arabidopsis* fruit development. *Development* **125**:1509–1517. DOI:10.1242/dev.125.8.1509
332. Marsch-Martínez N., Zúñiga-Mayo V., Herrera-Ubaldo H., et al. (2014). The NTT transcription factor promotes replum development in *Arabidopsis* fruits. *Plant J.* **80**:69–81. DOI:10.1111/tpj.12617
333. Cheng Y., Dai X. and Zhao Y. (2006). Auxin biosynthesis by the YUCCA flavin monooxygenases controls the formation of floral organs and vascular tissues in *Arabidopsis*. *Genes Dev.* **20**:1790–1799. DOI:10.1101/gad.1415106
334. Nemhauser J., Feldman L. and Zambryski P. (2000). Auxin and ETTIN in *Arabidopsis* gynoecium morphogenesis. *Development* **127**:3877–3888. DOI:10.1242/dev.127.18.3877
335. Larsson E., Roberts C., Claes A., et al. (2014). Polar auxin transport is essential for medial versus lateral tissue specification and vascular-mediated valve outgrowth in *Arabidopsis* gynoecia. *Plant Physiol.* **166**:1998–U1237. DOI:10.1104/pp.114.245951
336. Moubayidin L. and Ostergaard L. (2014). Dynamic control of auxin distribution imposes a bilateral-to-radial symmetry switch during gynoecium development. *Curr. Biol.* **24**:2743–2748. DOI:10.1016/j.cub.2014.09.080
337. Girin T., Paicu T., Stephenson P., et al. (2011). *INDEHISCENT* and *SPATULA* interact to specify carpel and valve margin tissue and thus promote seed dispersal in *Arabidopsis*. *Plant Cell* **23**:3641–3653. DOI:10.1105/tpc.111.090944
338. Huang F., Zago M., Abas L., et al. (2010). Phosphorylation of conserved PIN motifs directs *Arabidopsis* PIN1 polarity and auxin transport. *Plant Cell* **22**:1129–1142. DOI:10.1105/tpc.109.072678
339. Carabelli M., Turchi L., Morelli G., et al. (2021). Coordination of biradial-to-radial symmetry and tissue polarity by HD-ZIP II proteins. *Nat. Commun.* **12**:4321. DOI:10.1038/s41467-021-24550-6
340. Jiang Y., Curran-French S., Koh S., et al. (2024). O-glycosylation of the transcription factor *SPATULA* promotes style development in *Arabidopsis*. *Nat. Plants* **10**:283–299. DOI:10.1038/s41477-023-01617-4

341. Ostergaard L. (2009). Don't 'leaf' now. *The making of a fruit. Curr. Opin. Plant Biol.* **12**:36–41. DOI:10.1016/j.pbi.2008.09.011
342. Ballester P., Martínez-Godoy M., Ezquerro M., et al. (2021). A transcriptional complex of NGATHA and bHLH transcription factors directs stigma development in *Arabidopsis*. *Plant Cell* **33**:3645–3657. DOI:10.1093/plcell/koab236
343. Alvarez J. P., Goldshmidt A., Efroni I., et al. (2009). The NGATHA distal organ development genes are essential for style specification in *Arabidopsis*. *Plant Cell* **21**:1373–1393. DOI:10.1105/tpc.109.065482
344. Martínez-Fernández I., Sanchis S., Marini N., et al. (2014). The effect of NGATHA altered activity on auxin signaling pathways within the *Arabidopsis* gynoecium. *Front. Plant Sci.* **5**:210. DOI:10.3389/fpls.2014.00210
345. Sohlberg J., Myrenäs M., Kuusk S., et al. (2006). *STY1* regulates auxin homeostasis and affects apical-basal patterning of the *Arabidopsis* gynoecium. *Plant J.* **47**:112–123. DOI:10.1111/j.1365-313X.2006.02775.x
346. Wang Y., Wang N., Lan J., et al. (2024). *Arabidopsis* transcription factor *TCP4* controls the identity of the apical gynoecium. *Plant Cell* **36**:2668–2688. DOI:10.1093/plcell/koae107
347. Li S., Xie Z., Hu C., et al. (2016). A review of Auxin Response Factors (ARFs) in plants. *Front. Plant Sci.* **7**:47. DOI:10.3389/fpls.2016.00047
348. Sessions A., Nemhauser J., McColl A., et al. (1997). *ETTIN* patterns the *Arabidopsis* floral meristem and reproductive organs. *Development* **124**:4481–4491. DOI:10.1242/dev.124.22.4481
349. Kuhn A., Harbrough S., McLaughlin H., et al. (2020). Direct ETTIN-auxin interaction controls chromatin states in gynoecium development. *eLife* **9**:e51787. DOI:10.7554/eLife.51787
350. Simonini S., Deb J., Moubayidin L., et al. (2016). A noncanonical auxin-sensing mechanism is required for organ morphogenesis in *Arabidopsis*. *Genes Dev.* **30**:2286–2296. DOI:10.1101/gad.285361.116
351. Simonini S., Bencivenga S., Trick M., et al. (2017). Auxin-induced modulation of ETTIN activity orchestrates gene expression in *Arabidopsis*. *Plant Cell* **29**:1864–1882. DOI:10.1105/tpc.17.00389
352. Li W., Huang X., Zou J., et al. (2020). Three STIGMA AND STYLE STYLISTS pattern the fine architectures of apical gynoecium and are critical for male gametophyte-pistil interaction. *Curr. Biol.* **30**:4780–4788. DOI:10.1016/j.cub.2020.09.006
353. Roeder A. and Yanofsky M. (2006). Fruit development in *Arabidopsis*. *Arabidopsis Book* **4**:e0075. DOI:10.1199/tab.0075
354. Ripoll J., Zhu M., Brocke S., et al. (2019). Growth dynamics of the *Arabidopsis* fruit is mediated by cell expansion. *Proc. Natl. Acad. Sci. USA* **116**:25333–25342. DOI:10.1073/pnas.1914096116
355. Ripoll J., Bailey J., Mai Q., et al. (2015). microRNA regulation of fruit growth. *Nat. Plants* **1**:15036. DOI:10.1038/Nplants.2015.36
356. Di Marzo M., Herrera-Ubaldo H., Caporali E., et al. (2020). *SEEDSTICK* controls *Arabidopsis* fruit size by regulating cytokinin levels and *FRUITFULL*. *Cell Rep.* **30**:2846–2857. DOI:10.1016/j.celrep.2020.01.101
357. Petrella R., Caselli F., Roig-Villanova I., et al. (2020). BPC transcription factors and a Polycomb Group protein confine the expression of the ovule identity gene *SEEDSTICK* in *Arabidopsis*. *Plant J.* **102**:582–599. DOI:10.1111/tjp.14673
358. Balanzà V., Roig-Villanova I., Di Marzo M., et al. (2016). Seed abscission and fruit dehiscence required for seed dispersal rely on similar genetic networks. *Development* **143**:3372–3381. DOI:10.1242/dev.135202
359. Liljgren S., Roeder A., Kempin S., et al. (2004). Control of fruit patterning in *Arabidopsis* by *INDEHISCENT*. *Cell* **116**:843–853. DOI:10.1016/S0092-8674(04)00217-X
360. Rajani S. and Sundaresan V. (2001). The *Arabidopsis* myc/bHLH gene *ALCATRAZ* enables cell separation in fruit dehiscence. *Curr. Biol.* **11**:1914–1922. DOI:10.1016/S0960-9822(01)00593-0
361. Sorefan K., Girin T., Liljgren S., et al. (2009). A regulated auxin minimum is required for seed dispersal in *Arabidopsis*. *Nature* **459**:583–U114. DOI:10.1038/nature07875
362. Arnaud N., Girin T., Sorefan K., et al. (2010). Gibberellins control fruit patterning in *Arabidopsis thaliana*. *Genes Dev.* **24**:2127–2132. DOI:10.1101/gad.593410
363. Marsch-Martinez N., Ramos-Cruz D., Reyes-Oralde J., et al. (2012). The role of cytokinin during *Arabidopsis* gynoecia and fruit morphogenesis and patterning. *Plant J.* **72**:222–234. DOI:10.1111/j.1365-313X.2012.05062.x
364. Mitsuda N. and Ohme-Takagi M. (2008). *NAC* transcription factors *NST1* and *NST3* regulate pod shattering in a partially redundant manner by promoting secondary wall formation after the establishment of tissue identity. *Plant J.* **56**:768–778. DOI:10.1111/j.1365-313X.2008.03633.x
365. Ogawa M., Kay P., Wilson S., et al. (2009). *ARABIDOPSIS DEHISCENCE ZONE POLYGALACTURONASE1 (ADPG1)*, *ADPG2*, and *QUARTET2* are Polygalacturonases required for cell separation during reproductive development in *Arabidopsis*. *Plant Cell* **21**:216–233. DOI:10.1105/tpc.108.063768
366. Huang X. and Sun M. (2025). Cell fate determination during sexual plant reproduction. *New Phytol.* **245**:480–495. DOI:10.1111/nph.20230
367. Johnson M., Harper J. and Palanivelu R. (2019). A fruitful journey: Pollen tube navigation from germination to fertilization. *Annu. Rev. Plant Biol.* **70**:809–837. DOI:10.1146/annurev-arplant-050718-100133
368. Li H., Meng J. and Yang W. (2018). Multilayered signaling pathways for pollen tube growth and guidance. *Plant Reprod.* **31**:31–41. DOI:10.1007/s00497-018-0324-7
369. Goldberg R., de Paiva G. and Yadegari R. (1994). Plant embryogenesis: Zygote to seed. *Science* **266**:605–614. DOI:10.1126/science.266.5185.605
370. Twell D., Park S., Hawkins T., et al. (2002). MOR1/GEM1 has an essential role in the plant-specific cytokinetic phragmoplast. *Nat. Cell Biol.* **4**:711–714. DOI:10.1038/ncb844
371. Russell S. and Jones D. (2015). The male germline of angiosperms: Repertoire of an inconspicuous but important cell lineage. *Front. Plant Sci.* **6**:173. DOI:10.3389/fpls.2015.00173
372. McCue A., Cresti M., Feijó J., et al. (2011). Cytoplasmic connection of sperm cells to the pollen vegetative cell nucleus: Potential roles of the male germ unit revisited. *J. Exp. Bot.* **62**:1621–1631. DOI:10.1093/jxb/err032
373. Sprunck S. (2020). Twice the fun, double the trouble: Gamete interactions in flowering plants. *Curr. Opin. Plant Biol.* **53**:106–116. DOI:10.1016/j.pbi.2019.11.003
374. Brewbaker J. (1967). The distribution and phylogenetic significance of binucleate and trinucleate pollen grains in the angiosperms. *Am. J. Bot.* **54**:1069–1083. DOI:10.1002/j.1537-2197.1967.tb10735.x
375. Whittington A., Vugrek O., Wei K., et al. (2001). MOR1 is essential for organizing cortical microtubules in plants. *Nature* **411**:610–613. DOI:10.1038/35079128
376. Park S., Howden R. and Twell D. (1998). The *Arabidopsis thaliana* gametophytic mutation *gemini pollen1* disrupts microspore polarity, division asymmetry and pollen cell fate. *Development* **125**:3789–3799. DOI:10.1242/dev.125.19.3789
377. Zhou H., Madden B., Muddiman D., et al. (2006). Chromatin assembly factor 1 interacts with histone H3 methylated at lysine 79 in the processes of epigenetic silencing and DNA repair. *Biochemistry* **45**:2852–2861. DOI:10.1021/bi0521083
378. Chen Z., Hui J., Ingouff M., et al. (2008). Chromatin assembly factor 1 regulates the cell cycle but not cell fate during male gametogenesis in *Arabidopsis thaliana*. *Development* **135**:65–73. DOI:10.1242/dev.010108
379. Iwakawa H., Shinmyo A. and Sekine M. (2006). *Arabidopsis* *CDKA1*, a *cdc2* homologue, controls proliferation of generative cells in male gametogenesis. *Plant J.* **45**:819–831. DOI:10.1111/j.1365-313X.2005.02643.x
380. Kim H., Oh S., Brownfield L., et al. (2008). Control of plant germline proliferation by SCF^{FB17} degradation of cell cycle inhibitors. *Nature* **455**:1134–1137. DOI:10.1038/nature07289
381. Gusti A., Baumberger N., Nowack M., et al. (2009). The *Arabidopsis thaliana* F-Box protein FB17 is essential for progression through the second mitosis during pollen development. *PLoS One* **4**:e4780. DOI:10.1371/journal.pone.0004780
382. Chen Z., Hafidh S., Poh S., et al. (2009). Proliferation and cell fate establishment during *Arabidopsis* male gametogenesis depends on the Retinoblastoma protein. *Proc. Natl. Acad. Sci. USA* **106**:7257–7262. DOI:10.1073/pnas.0810992106
383. Boniotti M. and Gutierrez C. (2001). A cell-cycle-regulated kinase activity phosphorylates plant retinoblastoma protein and contains, in *Arabidopsis*, a CDKA/cyclin D complex. *Plant J.* **28**:341–350. DOI:10.1046/j.1365-313X.2001.01160.x
384. Desvoyes B., de Mendoza A., Ruiz-Trillo I., et al. (2014). Novel roles of plant RETINOBLASTOMA-RELATED (RBR) protein in cell proliferation and asymmetric cell division. *J. Exp. Bot.* **65**:2657–2666. DOI:10.1093/jxb/ert411
385. Desvoyes B. and Gutierrez C. (2020). Roles of plant retinoblastoma protein: Cell cycle and beyond. *EMBO J.* **39**:e105802. DOI:10.15252/embj.2020105802
386. Gombos M., Raynaud C., Nomoto Y., et al. (2023). The canonical E2Fs together with RETINOBLASTOMA-RELATED are required to establish quiescence during plant development. *Commun. Biol.* **6**:903. DOI:10.1038/s42003-023-05259-2
387. Brownfield L., Hafidh S., Borg M., et al. (2009). A plant germline-specific integrator of sperm specification and cell cycle progression. *PLoS Genet.* **5**:e1000430. DOI:10.1371/journal.pgen.1000430
388. Brownfield L., Hafidh S., Durbarray A., et al. (2009). *Arabidopsis* DUO POLLEN3 is a key regulator of male germline development and embryogenesis. *Plant Cell* **21**:1940–1956. DOI:10.1105/tpc.109.066373
389. Durbarray A., Vizir I. and Twell D. (2005). Male germ line development in *Arabidopsis* duo pollen mutants reveal gametophytic regulators of generative cell cycle progression. *Plant Physiol.* **137**:297–307. DOI:10.1104/pp.104.053165
390. Rotman N., Durbarray A., Wardle A., et al. (2005). A novel class of MYB factors controls sperm-cell formation in plants. *Curr. Biol.* **15**:244–248. DOI:10.1016/j.cub.2005.01.013
391. Borg M., Rutley N., Kagale S., et al. (2014). An EAR-dependent regulatory module promotes male germ cell division and sperm fertility in *Arabidopsis*. *Plant Cell* **26**:2098–2113. DOI:10.1105/tpc.114.124743
392. Li C., Potuschak T., Colón-Carmona A., et al. (2005). *Arabidopsis* TCP20 links regulation of growth and cell division control pathways. *Proc. Natl. Acad. Sci. USA* **102**:12978–12983. DOI:10.1073/pnas.0504039102
393. Sun Y., Wang X., Pan L., et al. (2021). Plant egg cell fate determination depends on its exact position in female gametophyte. *Proc. Natl. Acad. Sci. USA* **118**:e2017488118. DOI:10.1073/pnas.2017488118
394. Huang B. and Russell S. (1992). Female germ unit: Organization, isolation, and function. *Int. Rev. Cytol.* **140**:233–293. DOI:10.1016/S0074-7696(08)61099-2
395. Koszegi D., Johnston A., Rutten T., et al. (2011). Members of the RKD transcription factor family induce an egg cell-like gene expression program. *Plant J.* **67**:280–291. DOI:10.1111/j.1365-313X.2011.04592.x
396. Tedeschi F., Rizzo P., Rutten T., et al. (2017). RWP-RK domain-containing transcription factors control cell differentiation during female gametophyte

- development in *Arabidopsis*. *New Phytol.* **213**:1909–1924. DOI:10.1111/nph.14293
397. Koi S., Hisanaga T., Sato K., et al. (2016). An evolutionarily conserved plant RKD factor controls germ cell differentiation. *Curr. Biol.* **26**:1775–1781. DOI:10.1016/j.cub.2016.05.013
398. Rövekamp M., Bowman J. and Grossniklaus U. (2016). *Marchantia* MpRKD regulates the gametophyte-sporophyte transition by keeping egg cells quiescent in the absence of fertilization. *Curr. Biol.* **26**:1782–1789. DOI:10.1016/j.cub.2016.05.028
399. Cai H., Ma S., Su H., et al. (2022). Positional signals establishment in the regulation of female germline specification. *Seed Biol.* **1**:0006. DOI:10.48130/SeedBio-2022-0006
400. Cai H., Liu L., Ma S., et al. (2023). Insights into the role of phytohormones in plant female germline cell specification. *Curr. Opin. Plant Biol.* **75**:102439. DOI:10.1016/j.pbi.2023.102439
401. Cai H., Liu K., Ma S., et al. (2025). Gibberellin and cytokinin signaling antagonistically control female-germline cell specification in *Arabidopsis*. *Dev. Cell* **60**:706–722. DOI:10.1016/j.devcel.2024.11.009
402. Gross-Hardt R., Kägi C., Baumann N., et al. (2007). *LACHESIS* restricts gametic cell fate in the female gametophyte of *Arabidopsis*. *PLoS Biol.* **5**:494–500. DOI:10.1371/journal.pbio.0050047
403. Moll C., von Lyncker L., Zimmermann S., et al. (2008). *CLO/GFA1* and *ATO* are novel regulators of gametic cell fate in plants. *Plant J.* **56**:913–921. DOI:10.1111/j.1365-3113.2008.03650.x
404. Völz R., von Lyncker L., Baumann N., et al. (2012). *LACHESIS*-dependent egg-cell signaling regulates the development of female gametophytic cells. *Development* **139**:498–502. DOI:10.1242/dev.075234
405. Kong J., Lau S. and Jürgens G. (2015). Twin plants from supernumerary egg cells in *Arabidopsis*. *Curr. Biol.* **25**:225–230. DOI:10.1016/j.cub.2014.11.021
406. Pagnussat G., Yu H. and Sundaesana V. (2007). Cell-fate switch of synergid to egg cell in *Arabidopsis* *eostre* mutant embryo sacs arises from misexpression of the BEL1-like homeodomain gene *BLH1*. *Plant Cell* **19**:3578–3592. DOI:10.1105/tpc.107.054890
407. Cheung A., Duan Q., Li C., et al. (2022). Pollen-pistil interactions: It takes two to tangle but a molecular cast of many to deliver. *Curr. Opin. Plant Biol.* **69**:102279. DOI:10.1016/J.Pbi.2022.102279
408. Lan Z., Song Z., Wang Z., et al. (2023). Antagonistic RALF peptides control an intergeneric hybridization barrier on Brassicaceae stigmas. *Cell* **186**:4773–4787. DOI:10.1016/j.cell.2023.09.003
409. Liu C., Shen L., Xiao Y., et al. (2021). Pollen PCP-B peptides unlock a stigma peptide-receptor kinase gating mechanism for pollination. *Science* **372**:171–175. DOI:10.1126/science.abc6107
410. Ge Z., Bergonci T., Zhao Y., et al. (2017). *Arabidopsis* pollen tube integrity and sperm release are regulated by RALF-mediated signaling. *Science* **358**:1596–1599. DOI:10.1126/science.aao3642
411. Mecchia M., Santos-Fernandez G., Duss N., et al. (2017). RALF4/19 peptides interact with LRX proteins to control pollen tube growth in *Arabidopsis*. *Science* **358**:1600–1603. DOI:10.1126/science.aao5467
412. Meng J., Liang L., Jia P., et al. (2020). Integration of ovular signals and exocytosis of a Ca²⁺ channel by MLOs in pollen tube guidance. *Nat. Plants* **6**:143–153. DOI:10.1038/s41477-020-0599-1
413. Marshall E., Costa L. and Gutierrez-Marcos J. (2011). Cysteine-Rich Peptides (CRPs) mediate diverse aspects of cell-cell communication in plant reproduction and development. *J. Exp. Bot.* **62**:1677–1686. DOI:10.1093/jxb/err002
414. Meng J., Xu Y., Wang W., et al. (2023). Central-cell-produced attractants control fertilization recovery. *Cell* **186**:3593–3605. DOI:10.1016/j.cell.2023.06.024
415. Meng J., Zhang M., Yang W., et al. (2019). TICKET attracts pollen tubes and mediates reproductive isolation between relative species in Brassicaceae. *Sci. China-Life Sci.* **62**:1413–1419. DOI:10.1007/s11427-019-9833-3
416. Okuda S., Tsutsui H., Shiina K., et al. (2009). Defensin-like polypeptide LUREs are pollen tube attractants secreted from synergid cells. *Nature* **458**:357–361. DOI:10.1038/nature07882
417. Takeuchi H. and Higashiyama T. (2012). A species-specific cluster of defensin-like genes encodes diffusible pollen tube attractants in *Arabidopsis*. *PLoS Biol.* **10**:e1001449. DOI:10.1371/journal.pbio.1001449
418. Wang W.-Q., Meng J.-G., Yang F., et al. (2024). A non-defensin peptide NPA1 attracts pollen tube in *Arabidopsis*. *Seed Biology* **3**:e003. DOI:10.48130/seedbio-0024-0001
419. Zhong S., Liu M., Wang Z., et al. (2019). Cysteine-rich peptides promote interspecific genetic isolation in *Arabidopsis*. *Science* **364**:eaau9564. DOI:10.1126/science.aau9564
420. Higashiyama T., Yabe S., Sasaki N., et al. (2001). Pollen tube attraction by the synergid cell. *Science* **293**:1480–1483. DOI:10.1126/science.1062429
421. Takeuchi H. and Higashiyama T. (2016). Tip-localized receptors control pollen tube growth and LURE sensing in *Arabidopsis*. *Nature* **531**:245–248. DOI:10.1038/nature17413
422. Wang T., Liang L., Xue Y., et al. (2016). A receptor heteromer mediates the male perception of female attractants in plants. *Nature* **531**:241–244. DOI:10.1038/nature16975
423. Meng J., Li S. and Li H. (2024). Central cell: The key to determine persistent pollen tube attraction or termination. *Sci. China-Life Sci.* **67**:2030–2032. DOI:10.1007/s11427-023-2534-2
424. Capron A., Gourgues M., Neiva L., et al. (2008). Maternal control of male-gamete delivery in *Arabidopsis* involves a putative GPI-anchored protein encoded by the *LORELEI* gene. *Plant Cell* **20**:3038–3049. DOI:10.1105/tpc.108.061713
425. Duan Q., Kita D., Johnson E., et al. (2014). Reactive oxygen species mediate pollen tube rupture to release sperm for fertilization in *Arabidopsis*. *Nat. Commun.* **5**:3129. DOI:10.1038/Ncomms4129
426. Galindo-Trigo S., Blanco-Tourinan N., DeFalco T., et al. (2020). RLK1L receptor-like kinases HERK1 and ANJEA are female determinants of pollen tube reception. *EMBO Rep.* **21**:e48466. DOI:10.15252/embr.201948466
427. Escobar-Restrepo J., Huck N., Kessler S., et al. (2007). The FERONIA receptor-like kinase mediates male-female interactions during pollen tube reception. *Science* **317**:656–660. DOI:10.1126/science.1143562
428. Ngo Q., Vogler H., Litiuiev D., et al. (2014). A calcium dialog mediated by the FERONIA signal transduction pathway controls plant sperm delivery. *Dev. Cell* **29**:491–500. DOI:10.1016/j.devcel.2014.04.008
429. Kessler S., Shimosato-Asano H., Keinath N., et al. (2010). Conserved molecular components for pollen tube reception and fungal invasion. *Science* **330**:968–971. DOI:10.1126/science.1195211
430. Huang J., Ju Y., Wang X., et al. (2015). A one-step rectification of sperm cell targeting ensures the success of double fertilization. *J. Integr. Plant Biol.* **57**:496–503. DOI:10.1111/jipb.12322
431. Johnson M., von Besser K., Zhou Q., et al. (2004). *Arabidopsis* *hapless* mutations define essential gametophytic functions. *Genetics* **168**:971–982. DOI:10.1534/genetics.104.029447
432. Sprunck S., Rademacher S., Vogler F., et al. (2012). Egg cell-secreted EC1 triggers sperm cell activation during double fertilization. *Science* **338**:1093–1097. DOI:10.1126/science.1223944
433. Wang W., Xiong H. X., Zhao P., et al. (2022). DMP8 and 9 regulate HAP2/GCS1 trafficking for the timely acquisition of sperm fusion competence. *Proc. Natl. Acad. Sci. USA* **119**:e2207608119. DOI:10.1073/pnas.2207608119
434. Misamore M., Gupta S. and Snell W. (2003). The *Chlamydomonas* Fus1 protein is present on the mating type *plus* fusion organelle and required for a critical membrane adhesion event during fusion with *minus* gametes. *Mol. Biol. Cell* **14**:2530–2542. DOI:10.1091/mbc.E02-12-0790
435. Mori T., Igawa T., Tamiya G., et al. (2014). Gamete attachment requires GEX2 for successful fertilization in *Arabidopsis*. *Curr. Biol.* **24**:170–175. DOI:10.1016/j.cub.2013.11.030
436. Chen S., Wang L., Jia P., et al. (2022). Osmoregulation determines sperm cell geometry and integrity for double fertilization in flowering plants. *Mol. Plant* **15**:1488–1496. DOI:10.1016/j.molp.2022.07.013
437. Mori T., Kuroiwa H., Higashiyama T., et al. (2006). GENERATIVE CELL SPECIFIC 1 is essential for angiosperm fertilization. *Nat. Cell Biol.* **8**:64–71. DOI:10.1038/ncb1345
438. Zhong S., Li L., Wang Z., et al. (2022). RALF peptide signaling controls the polytubey block in *Arabidopsis*. *Science* **375**:290–296. DOI:10.1126/science.abl4683
439. Duan Q., Liu M., Kita D., et al. (2020). FERONIA controls pectin- and nitric oxide-mediated male-female interaction. *Nature* **579**:561–566. DOI:10.1038/s41586-020-2106-2
440. Yu X., Zhang X., Zhao P., et al. (2021). Fertilized egg cells secrete endopeptidases to avoid polytubey. *Nature* **592**:433–437. DOI:10.1038/s41586-021-03387-5
441. Maruyama D., Völz R., Takeuchi H., et al. (2015). Rapid elimination of the persistent synergid through a cell fusion mechanism. *Cell* **161**:907–918. DOI:10.1016/j.cell.2015.03.018
442. Pereira A., Nobre M., Pinto S., et al. (2016). "Love is strong, and you're so sweet": JAGGER is essential for persistent synergid degeneration and polytubey block in *Arabidopsis thaliana*. *Mol. Plant* **9**:601–614. DOI:10.1016/j.molp.2016.01.002
443. Chen D., Yan W., Fu L., et al. (2018). Architecture of gene regulatory networks controlling flower development in *Arabidopsis thaliana*. *Nat. Commun.* **9**:4534. DOI:10.1038/S41467-018-06772-3
444. Smith S. (2016). Pleiotropy and the evolution of floral integration. *New Phytol.* **209**:80–85. DOI:10.1111/nph.13583
445. Bai Y., Gheyret G., Zhang H., et al. (2024). Trait-based neighbourhood effects modulate the growth-weather relationships of subtropical trees. *Innov. Life* **2**:100106. DOI:10.59717/j.xinn-life.2024.100106
446. Cao X. and Ren B. (2024). Legume nitrogen fixation under nitrate has "FUN" with zinc. *Innov. Life* **2**:100092. DOI:10.59717/j.xinn-life.2024.100092
447. Liu Q. and Fu X. (2023). Can heterotrimeric G proteins improve sustainable crop production and promote a more sustainable Green Revolution. *Innov. Life* **1**:100024. DOI:10.59717/j.xinn-life.2023.100024
448. Zhang X., Meng W., Liu D., et al. (2024). Enhancing rice panicle branching and grain yield through tissue-specific brassinosteroid inhibition. *Science* **383**:eadk8838. DOI:10.1126/science.adk8838
449. Song L., Liu J., Cao B., et al. (2023). Reducing brassinosteroid signalling enhances grain yield in semi-dwarf wheat. *Nature* **617**:118–124. DOI:10.1038/s41586-023-06023-6
450. Tian J., Wang C., Chen F., et al. (2024). Maize smart-canopy architecture enhances yield at high densities. *Nature* **632**:576–584. DOI:10.1038/s41586-024-07669-6

451. Li T., Wang C., Pan J., et al. (2025). Exploring potential strategies for haploid induction based on double fertilization in plants. *Plant Biotechnol. J.* **23**:4000–4016. DOI:10.1111/pbi.70197
452. Xie Y., Zhang T., Yang M., et al. (2025). Engineering crop flower morphology facilitates robotization of cross-pollination and speed breeding. *Cell* **188**:5809–5830. DOI:10.1016/j.cell.2025.07.028
453. Li S-Z, Wang J, Jia S-G, et al. (2023). Synthetic apomixis: From genetic basis to agricultural application. *Seed Biol.* **2**:10. DOI:10.48130/SeedBio-2023-0010
454. Shao Z., Qian T., Sun T., et al. (2025). Spatial-temporal large models: A super hub linking multiple scientific areas with artificial intelligence. *The Innovation* **6**:100763. DOI:10.1016/j.xinn.2024.100763
455. Xu Y., Liu X., Cao X., et al. (2021). Artificial intelligence: A powerful paradigm for scientific research. *The Innovation* **2**:100179. DOI:10.1016/j.xinn.2021.100179
456. Xu Y., Wang F., An Z., et al. (2023). Artificial intelligence for science-bridging data to wisdom. *The Innovation* **4**:100525. DOI:10.1016/j.xinn.2023.100525
457. Xu Y., Wang F. and Zhang T. (2024). Artificial intelligence is restructuring a new world. *The Innovation* **5**:100725. DOI:10.1016/j.xinn.2024.100725
458. Ma X.-K., Yu Y., Huang T., et al. (2024). Bioinformatics software development: Principles and future directions. *The Innovation Life* **2**:100083. DOI:10.59717/j.xinn-life.2024.100083
459. Cao X., Xie H., Song M., et al. (2023). Cut-dip-budding delivery system enables genetic modifications in plants without tissue culture. *The Innovation* **4**:100345. DOI:10.1016/j.xinn.2022.100345
460. Cao X., Xie H., Song M., et al. (2023). Extremely simplified cut-dip-budding method for genetic transformation and gene editing in *Taraxacum kok-saghyz*. *Innov. Life* **1**:100040. DOI:10.59717/j.xinn-life.2023.100040
461. Han X., Chen Y., Liu R., et al. (2024). Engineering hfCas12Max for improved gene editing efficiency. *Innov. Life* **2**:100067. DOI:10.59717/j.xinn-life.2024.100067

FUNDING AND ACKNOWLEDGMENTS

This study was supported by grants from the Strategic Priority Research Program of Chinese Academy of Sciences (XDB1090000), the National Key R&D Program of China (2024YFA1306700), the National Natural Science Foundation of China (32170247, 32221001, 32370231), and the Agricultural Science and Technology Innovation Program of the Chinese Academy of Agricultural Sciences (CAAS-ASTIP-2024-IVF). The founders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.

AUTHOR CONTRIBUTIONS

X.L. and H.T.L. wrote the “Photomorphogenesis: To live or to die?” section. R.Y.L. and B.W. wrote the “Plant height regulation” and “Shoot branching” sections. X.C.W. and Y.Z. wrote the “Transition to flowering can occur once or repeatedly” section. H.Y.S., S.X.L. and H.Z.K. wrote the “Flower development” section. S.Y.C. and H.J.L. wrote the “Germline specification and fertilization” section. Y.D. and H.R.S. wrote the “Fruit development” section. C.H.T., H.J.L., and B.W. wrote the introduction and perspective section. M.L., S.S. and J.Y.L. gave suggestions on the manuscript. C.H.T. integrated all the sections and finalized the draft with the help of all other authors. All authors contributed to the manuscript and approved the final version.

DECLARATION OF INTERESTS

The authors declare no competing interests.

DATA AND CODE AVAILABILITY

Not applicable.