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Suggested Citation:
Bahafid, E., Barr, Z. K., Burkart, R., Petrella, R., & Simon, R. (2025). Bridging cells and stages: Plasmodesmata for the coordination of plant development. Current Opinion in Plant Biology, 89, Article 102843. https://doi.org/10.1016/j.pbi.2025.102843

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Current Opinion in Plant Biology

Bridging cells and stages: Plasmodesmata for the coordination of plant development

Elmehdi Bahafid[1], Zoe Kathleen Barr[1], Rebecca Corinna Burkart[1], Rosanna Petrella[1] and Rüdiger Simon

Plants are multicellular organisms in which numerous specialized cell types must communicate to function as a unified system. Plant cells are enclosed by rigid walls, and therefore, intercellular communication requires the presence of plasmodesmata (PD), cytoplasmic channels bridging neighboring cells. These structures are crucial for coordinating developmental stages across tissues. To ensure proper growth and development, the movement of signaling molecules, RNAs, proteins, and nutrients through PD must be tightly controlled, underscoring the importance of regulating their selectivity.

Despite their essential role, direct evidence for PD involvement in developmental processes is limited and the mechanisms governing PD regulation remain incompletely understood. Recent studies suggest the existence of diverse regulatory mechanisms beyond the classical callose-based model, revealing a likely complex interplay of several PD regulators across development. In this review, we summarize recent findings on the role of PD in various plant developmental programs, discuss emerging regulatory mechanisms, and highlight how much remains to be discovered.

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Current Opinion in Plant Biology 2026, **89:**102843

This review comes from a themed issue on **Growth and development 2025**

Edited by **Daniel Kierzkowski** and **Margot Smit**

For complete overview of the section, please refer the article collection - Growth and development 2025

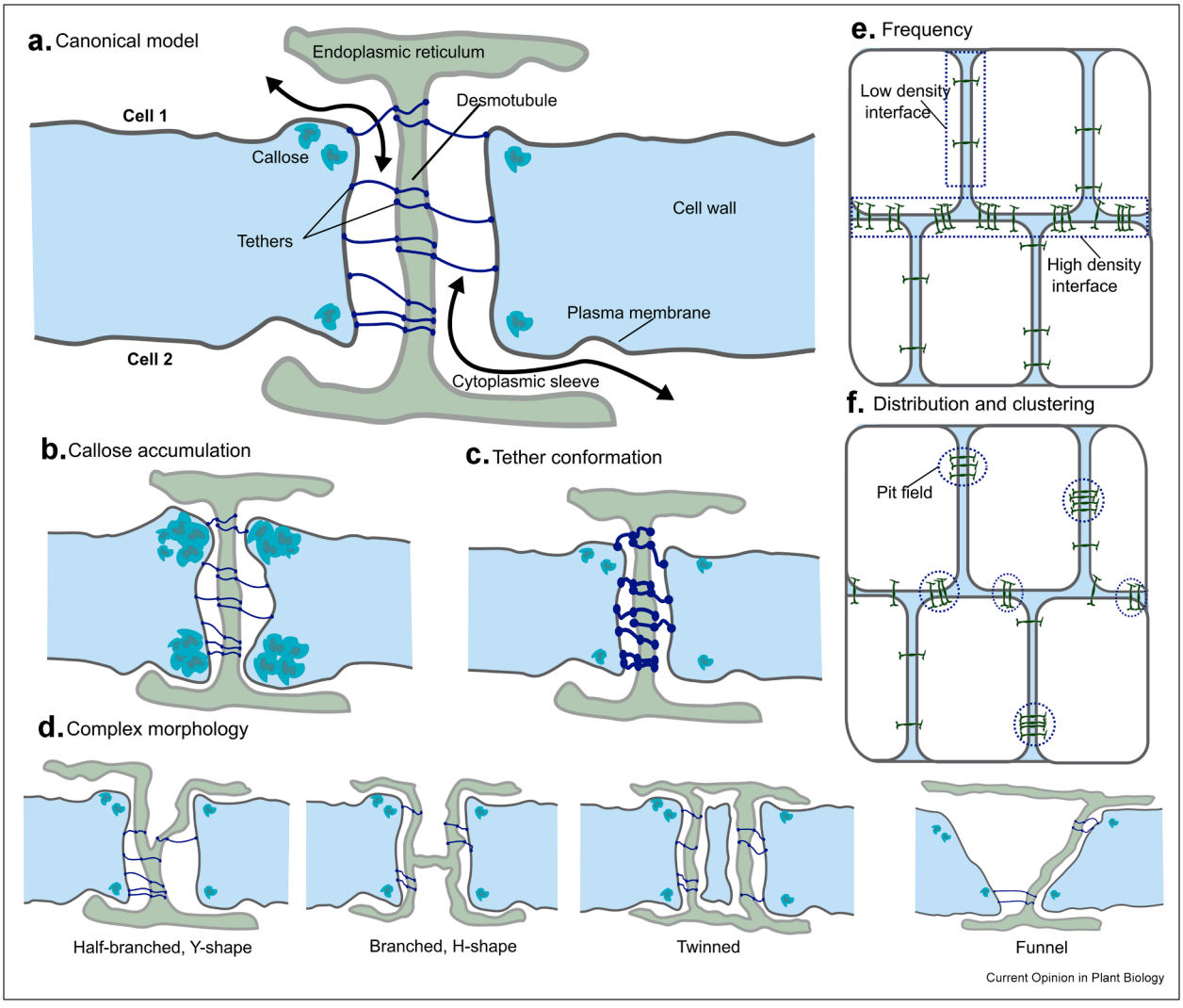
Available online 18 December 2025

https://doi.org/10.1016/j.pbi.2025.102843

Introduction

Plants develop most organs postembryonically, and the sessile nature of plantae requires robust coordination of growth and development in response to environmental cues. This coordination relies on both short- and long-range intercellular communication to convey positional and developmental information. Cell-to-cell communication in plants must overcome the fixed positioning of cells and the barrier of the rigid cell wall. Intercellular connectivity depends on two main pathways: apoplastic transport via the extracellular space and symplasmic transport via plasmodesmata (PD) [1,2]. PD are membrane-lined nanochannels traversing the cell wall to maintain cytoplasmic continuity. As specialized membrane contact sites, the canonically understood structure of the PD consists of plasma membrane–lined gaps in the cell wall with a highly constricted strand of endoplasmic reticulum (named the desmotubule) passing through the center (Figure 1a) [3,4]. PD are highly dynamic intercellular channels that enable the transport of ions, metabolites, RNAs, proteins, and signaling molecules between neighboring plant cells [5]. The extent of transport through PD (the trafficking capacity) depends on PD permeability. The regulation of PD permeability is varied (Figure 1) and occurs in response to developmental or environmental cues. There are several mechanisms known to adjust the size exclusion limit of individual PD. The best characterized is the deposition of the cell wall polysaccharide beta- 1,3- glucan (callose) (Figure 1b) which is modulated by PD-Localized Proteins (PDLPs), callose synthases, and beta-1,3- glucanases [6]. The presence and conformation of tethering proteins, including Multiple C2 Domain and Transmembrane region Proteins (MCTPs) [7,8], endoplasmic reticulum restriction [9], and varied structural morphologies (branching, twinning, and funnel-shaped) [1,10], can all regulate PD permeability by structural alteration (Figure 1c, d). At a tissue level, overall PD permeability is regulated by changes to the distribution (specific cell-interface positioning) and frequency of PD (Figure 1e, f) [8,11].

Figure 1



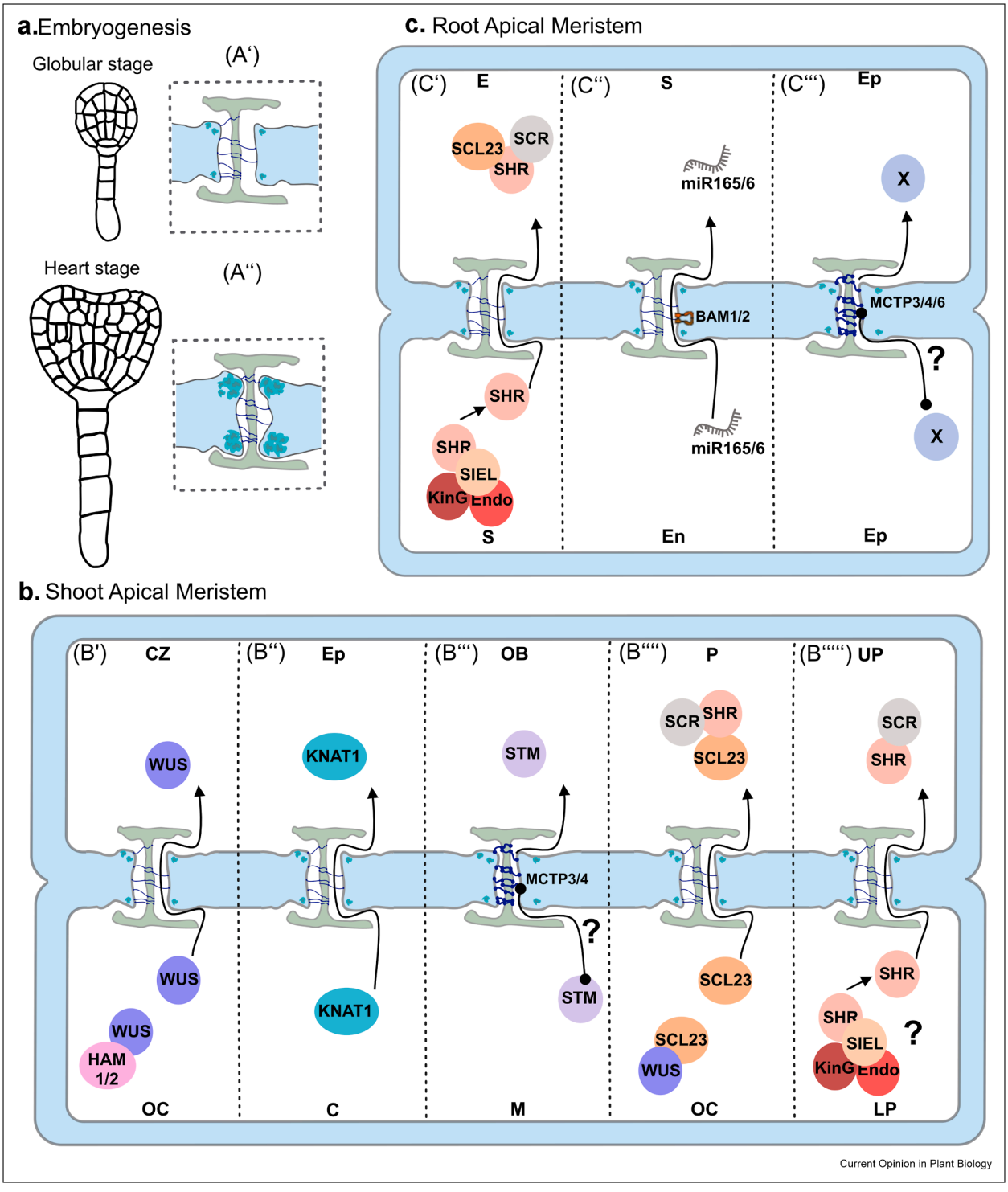
Schematic view of PD structure and varied regulation. (A) The canonical model of a simple PD. A gap in the cell wall allows flow through the cytoplasmic sleeve. The plasma membrane lines the cell wall, and a constricted tubular strand of endoplasmic reticulum passes centrally through the channel. Inside the cell wall, callose is accumulated. Tethering proteins connect the plasma membrane and endoplasmic reticulum. (B,C,D) Permeability regulation by alteration of individual PD. PD aperture is restricted by callose accumulation (B), the presence and conformation of membrane tethers (C), and the variation of morphology (D). Several conformations are illustrated: branching, twinning, and funnel shaped. (E,F) PD connections between neighboring cells illustrate regulation by PD frequency (E) and distribution pattern (F). Closely grouped PD show the clustering of PD into pit fields (E). PD, plasmodesma.

It is well understood that PD are involved in cell-fate regulation and therefore integral to plant development [12,13]. However, we are far from understanding this role in detail. This review considers the varied PD regulatory mechanisms described across several specific developmental contexts. We propose that the requirement for controlled distribution of signals and nutrients appropriate to the specific developmental context necessitates the meditation of PD permeability via PD regulation mechanisms suited to the spatial–temporal context. To identify the requirement for and control of symplasmic transport, we examine PD regulation at major developmental stages and different organs.

Embryogenesis and meristems

During embryo formation, PD undergo pronounced structural and functional transitions [14]. In the early stages of embryogenesis, cells are symplasmically interconnected, facilitating unrestricted trafficking of macromolecules [15] (Figure 2A'). However, as the embryo develops from globular to heart stage, PD

Figure 2



Plasmodesmata (PD) function during embryogenesis and root and shoot apical meristem development. (A) During the globular stage, the embryo and suspensor form a single symplasmic continuum, indicated by open PD (A′). In the early heart stage, distinct symplasmic subdomains emerge within the embryo, illustrated by a closed PD (A″). (B) Examples of mobile macromolecular signals in the shoot apical meristem. WUS produced in the organizing center (OC), moves upward into the central zone (CZ) cells through PD (B′). KNAT1, produced in the cortical cells in the inflorescence stem (C), moves upward to the epidermal cells in the inflorescence stem (E) through PD (B″). Hypothetical model of STM interacts with MCTP3/4 at PD, facilitating its movement from the meristem (M) toward the organ boundary (OB) (B‴). SCL23 is produced in the organizing center (OC), moves outward into the organ primordia (P) cells where it forms a complex together with SHR and SCR (B⁗). A hypothetical model shows that SHR is expressed in the lower primordia cells (LP) and moves into the upper primordia cells (UP) where it interacts with SCR. In the LP, SIEL binds SHR to endosomes that travel along actin filaments and KinG, a microtubule-associated protein, transiently pauses their movement before SHR enters the endodermis (B⁗′). (C) Examples of mobile macromolecular signals in the root apical meristem. The transcription factor SHR is expressed in the stele (S) and moves into the

permeability becomes restricted, forming symplasmic domains associated with tissue patterning [14] (Figure 2a''). This transition shapes the auxin gradients critical for axis formation before the full establishment of PIN-FORMED (PIN) transporter polarity [5,16].

At the end of embryogenesis, the shoot apical meristem (SAM) and root apical meristem (RAM) are established, where stem cells drive postembryonic organogenesis. In the SAM, PD-mediated trafficking plays a central regulatory role. The transcription factor WUSCHEL (WUS) moves from the organizing center (OC) into stem cells located at the tip of the central zone, where it maintains stem cell identity and meristem organization [17,18]. HAIRY MERISTEM (HAM1/2) proteins interact with WUS in the OC to restrict its upward movement and control shoot stem cell production [19,20] (Figure 2c').

PD-dependent movement of the homeobox protein KNOTTED-LIKE 1 (KNAT1) from cortical cells in the inflorescence stem to epidermal cells is also essential for determining plant architecture and epidermal differentiation [21] (Figure 2c''). Similarly, the homeodomain transcription factor SHOOT MERISTEMLESS (STM) traffics through PD to maintain stem cell activity. STM movement between meristematic cells sustains meristem size, regulates axillary meristem initiation, and establishes organ boundaries, highlighting the importance of PD-mediated transport in SAM patterning [22] (Figure 2c'''). The regulation of STM mobility involves FT INTERACTING PROTEIN 3 and 4 (FTIP3/4, also known as MCTP3/4), which localize to PD [8,23]. MCTP3 and MCTP4 control intracellular distribution of STM by preventing its diversion to the plasma membrane, thereby promoting nuclear accumulation and restricting excessive intercellular diffusion essential for meristem maintenance [23] (Figure 2c'''). Furthermore, MCTP3/4 regulate protein mobility in the RAM [8], suggesting they likely fine-tune STM mobility in the SAM as well. Together, these findings indicate that PD-associated MCTPs integrate intracellular trafficking with intercellular transport.

Recent findings reveal the transcription factor SCARECROW-LIKE 23 (SCL23) is expressed in the OC and moves into the surrounding zones, suggestive of controlled cell-to-cell movement. The *scl23* mutant displays defects in meristem size and organ initiation, and physical as well as genetic interactions with WUS indicate that SCL23 contributes to stem cell maintenance. SCL23 moves into organ primordia, where it interacts with SHORT ROOT (SHR) and SCARECROW (SCR), previously considered root-specific transcription factors (Figure 2c''''). SHR is expressed in the lower layers of lateral organ primordia and moves upward to promote organ initiation. Therefore SHR localization overlaps with the auxin efflux carrier PIN1 and is promoted by auxin, linking SHR movement to auxin maxima formation [24] (Figure 2c''''). Altogether, PD in the SAM function as developmental gatekeepers, controlling the mobility of key regulators.

In the RAM, PD regulate stem cell niche maintenance and tissue patterning by mediating SHR movement from the stele to the endodermis. This process involves the kinesin KinG and SHR partner SIEL, which anchor SHR-associated endosomes to microtubules and actin, promoting pauses that enhance intercellular transport [25] (Figure 2b'). These findings suggest that a similar PD- and cytoskeleton-dependent mechanism may regulate SHR mobility in the SAM. PD-localized receptor-like kinases BAM1/2 promote the intercellular mobility of miR165/6, and *bam1bam2* mutants display aberrant xylem patterning due to defective small RNA trafficking [26] (Figure 2b''). Similar to the SAM, MCTPs are essential for RAM maintenance. Mutations in MCTP3/4/6 disrupt RAM organization and reduce intercellular movement, underscoring their role in maintaining functional PD conduits [8] (Figure 2b'''). Collectively, these findings reveal that multiple molecular layers control PD gating to fine-tune intercellular mobility important for the function and tissue patterning of the RAM.

Vegetative stage

Studies in vegetative tissues have majorly advanced the understanding of PD structure and regulation by identifying tissue-specific spatial−temporal regulation of PD. The overexpression of well-characterized PD components PDLP5 or PDLP6 had a cell type−specific effect on PD-mediated transport causing accumulation of anthocyanin, starch, and sucrose [27]. This included enlarged starch grains in mesophyll (PDLP5 and PDLP6) and bundle sheath (BS) cells (PDLP6 only) [27]. Gene expression and callose accumulation were tissue specific: PDLP6 in the leaf vasculature and PDLP5 in the epidermal and mesophyll cells. This was functionally associated with symplasmic transport by fluorescent tracer assays and delayed growth phenotypes [27]. Distribution of PD components is also cell-interface specific. In the *Arabidopsis* root, apicobasal walls have a higher PD frequency than longitudinal walls

endodermis (E) where it interacts with SCR and SCL23. In the stele, SIEL binds SHR to endosomes that travel along actin filaments, where KinG, a microtubule-associated protein, transiently pauses their movement before SHR enters the endodermis (C′). miR165/6 is produced in endoderm and moves inward through PD. BAM1 and BAM2, localized at these PD, facilitate its intercellular movement from the endodermis into the stele (C″). Hypothetical model of MCTP3/4/6 regulating the transport of protein X through PD in root meristem epidermal cells (C‴). SCR, SCARECROW; SHR, SHORT ROOT; STM, SHOOT MERISTEMLESS; WUS, WUSCHEL.

[8]. Here, SUPPRESSOR OF ACTIN 7 (SAC7) is a phosphatidylinositol 4-phosphate (PI4P) regulating PD by the PI4P-mediated stabilization of MCTPs. *SAC7* has tissue-specific expression preferentially in the trichoblast. SAC7 was enriched to PD and with polar localization to the apicobasal cortical region of the cell without lateral accumulation [8]. This is a newly proposed developmental regulation of PD established by tissue differentiation.

In the gametophytic tissue of *Marchantia polymorpha*, dormant gemmae have simple PD [28]. Complex PD frequency increases throughout gemma development and PD further accumulate in the main plant body thalli. Comparison of PD permeability throughout the juvenile-to-adult transition found consistent restriction of cell connectivity with a marginal increase in transport in the mature thalli [28]. This increased transport alongside maturation opposes vascular plants where the increase in complex PD structures (branched and funnel) occurring with tissue maturation is associated with reduced transport [10,29]. In *Arabidopsis*, pectin acyltransferase *POWDERY MILDEW RESISTANCE 5* (*PMR5*) aids cell wall loosing required for secondary PD formation [30]. *PMR5* mutation reduced artificial microRNA (miRNA) trafficking (epidermis-to-mesophyll movement) and impeded short interfering RNA (siRNA), Green Fluorescent Protein (GFP), and viral movement by decreasing the frequency of leaf secondary PD [30].

Phytohormones are major developmental regulators (reviewed [31]). In the *Arabidopsis* root, brassinosteroids are regulated by PD-mediated transport of brassinosteroid precursors with biosynthesis completed in neighboring cells [32]. PD permeability was decreased via callose accumulation in response to high brassinosteroid levels, potentially regulated by brassinosteroid signaling—triggered transcription [32](Figure 3B). The stress response hormone abscisic acid (ABA) induces callose synthesis to reduce intercellular trafficking and alter tissue development [33,34] (Figure 3a). ABA treatment of *Physcomitrium patens* protonema increased cell wall deposits at PD causing neck-region restriction or abscission of the PD membrane structure [35] which is consistent with the findings in floral induction, where ABA leads to temperature-dependent callose accumulation (see 'Flowering transition').

High frequency of PD at the mesophyll—bundle sheath (M—BS) interface was directly associated with net assimilation rate of CO_2 [36]. PD requirement for solute diffusion across the M—BS interface is high in C_4 plants where the BS cell wall is heavily suberized [37]. PD density was compared in monocots by measurement across developmental timepoints; PD frequency was higher at the M—BS interface in C_4 *Setaria viridis* than in C_3 *Oryza sativa* [38]. Similar comparison of M—BS, BS—BS, and M—M in *Gynandropsis gynandra* (C_4), *Tarenaya hassleriana* (C_3), and *Arabidopsis thaliana* (C_3) identified a 13-fold higher PD frequency at the mesophyll—BS interface in the C_4 than in C_3 species. This increase of PD occurs rapidly in response to light in cotyledons during dark-to-light transition [39]. Both studies identified an increase of pit fields coincided with shoot emergence further linking PD frequency and photosynthesis (Figure 3b) [38,39]. Conservation of high PD frequency in C_4 plants is proposed to support the exchange of metabolites where photosynthesis is partitioned across two cells [39].

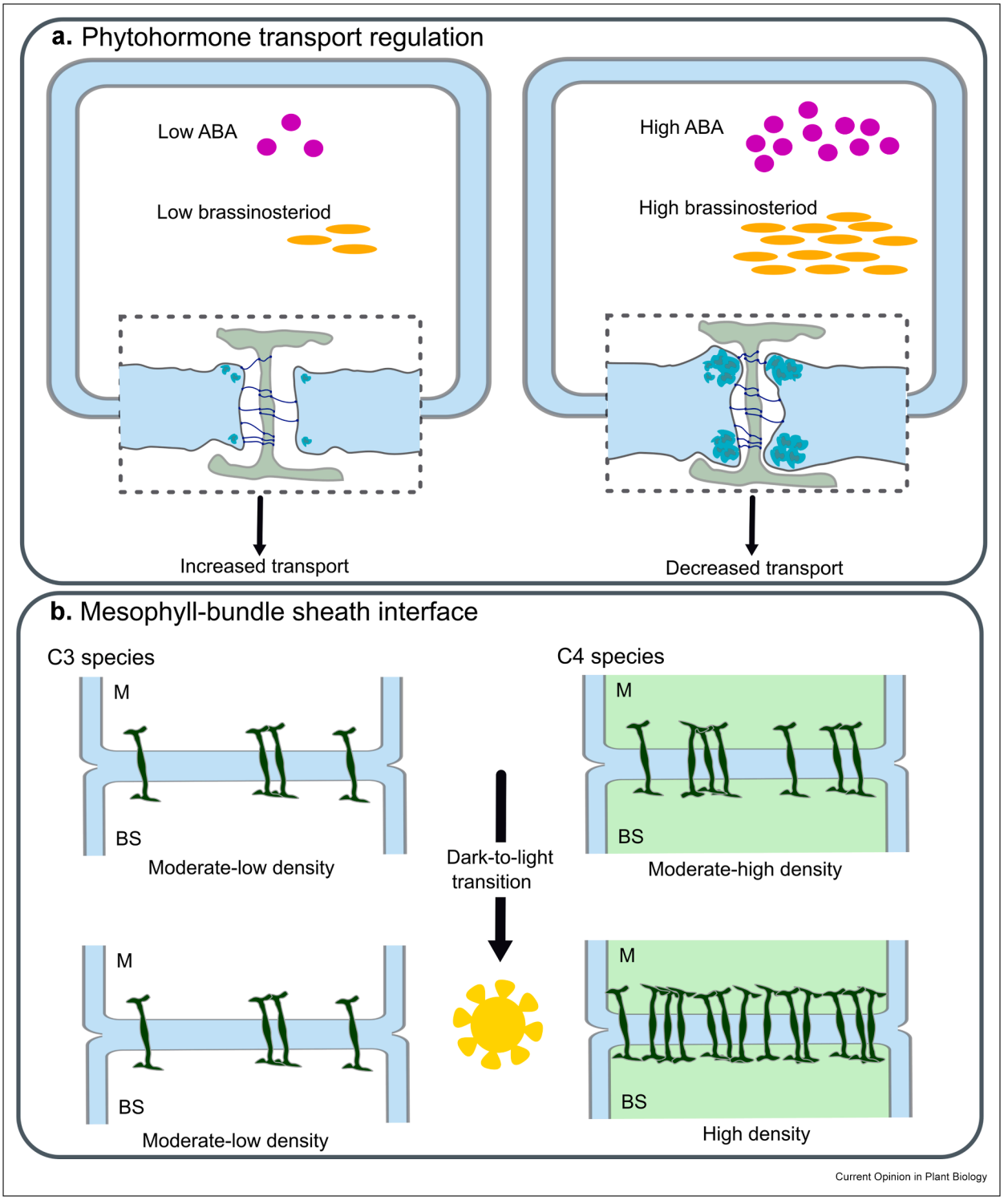
Flowering transition

In floral transition, major developmental reprogramming must be triggered by the movement of environmentally induced signals. In *A. thaliana*, a mobile flowering signal, originally termed florigen [40], was later identified as the FLOWERING LOCUS T (FT) protein [41]. FT travels from leaves to the SAM through PD after phloem unloading [42]. FT mobility has been reported in several plant species [43—46], underscoring a conserved role for symplasmic transport in floral induction. Control of PD gating has long been recognized as essential not only for floral transition but also for flower formation [47].

Recent findings reveal how strongly temperature modulates this system. Murata et al. [34] showed that low temperatures delay flowering by impairing FT transport to the SAM (Figure 4A). This effect arises from enhanced ABA signaling, which increases the expression of the callose synthases CalS1 and CalS7, promotes callose deposition, reduces PD permeability (Figure 3a) and thus restricts FT translocation, likely through its interaction with the MCTP FT INTERACTING PROTEIN 1 (FTIP1). This FT-FTIP1 interaction was previously shown to mediate FT translocation from companion cells to sieve elements via PD, enabling leaf-to-SAM transport [48]. These results provide a mechanistic link between temperature signaling, callose homeostasis, and PD regulation.

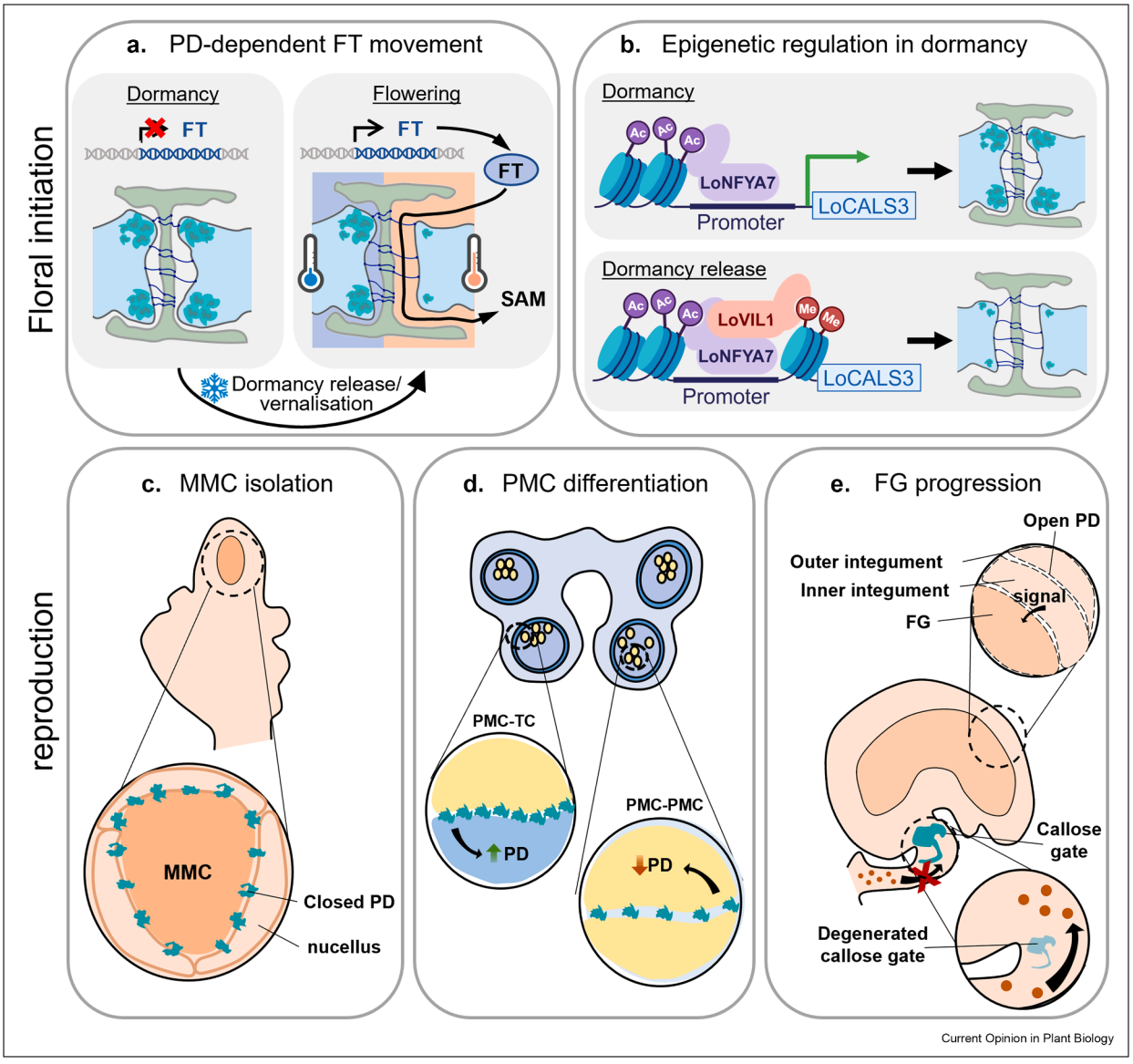
Both vernalization and dormancy are strongly temperature dependent to prevent premature flowering. In birch, dormancy was linked to callose-mediated PD closure and subsequent shutdown of symplasmic transport, which is later reversed by chilling [49]. A similar process promotes dormancy during short days in the *Populus* species hybrid aspen. Here, ABA induces CALS1-dependent PD closure through the ortholog of Arabidopsis floral repressor SHORT VEGETATIVE PHASE, named SVL, blocking movement of growth-promoting signals like FT1 and simultaneously suppresses gibberellic acid (GA), thereby promoting photoperiodic dormancy [50,51]. Furthermore, *FT1* downregulation during short days maintains dormancy,

Figure 3



Variation of PD regulation mechanisms in vegetative tissue. (A) High levels of phytohormones correlate to increased callose and decreased cell-to-cell connectivity. ABA and brassinosteroid are represented in magenta and orange respectively. (B) PD frequency at the mesophyll–bundle (M–BS) sheath interface. In C3 plants, PD frequency is consistently moderate to low. In C4 plants, an already high PD frequency increases during dark-to-light transition. ABA, abscisic acid; PD, plasmodesma.

Figure 4



Dynamics of PD regulation during flowering and reproduction. (A) During dormancy, FT is repressed and symplasmic movement inhibited through callose accumulation and PD closure. Prolonged chilling induces floral transition, where FT is expressed and can move through open PD toward the SAM and initiate flowering. (B) Callose accumulation at PD is epigenetically regulated in dormant Lilly buds. During dormancy, the transcription factor LoNFYA7 binds to the *LoCALS3* promoter, promoting H3K9 acetylation and thus activating LoCALS3 expression, leading to PD closure. After prolonged chilling, LoVIL1 expression increases and interacts with LoNFYA7, inducing H3K27 methylation at the *LoCALS3* promoter, reducing *LoCALS3* expression. This decreases callose levels, opens PD, and restores symplasmic transport, thus leading to dormancy release. (C) Megaspore mother cell (MMC) is characterized by a deposition of callose to moderate communication with the neighboring cells and initiate germline program. (D) On the onset of meiosis, domains of the developing anthers are characterized by different accumulation of callose at the cell wall. High and low levels of callose determine increased PD frequency at pollen mother cell–tapetum (PMC–TC) cells junctions and decreased PD frequency at PMC–PMC junctions, respectively. (E) Top, in the ovule, unknown sporophyte signals traveling through PD are necessary to support female gametophyte (FG) progression. Bottom, on the phloem end, callose gate is preventing nutrient flow prior fertilization. Degradation of callose gate after fertilization supports seeds and embryo development. FT, FLOWERING LOCUS T; PD, plasmodesma; SAM, shoot apical meristem. (Figure partially created using BioRender https://BioRender.com/lbie2su).

while chilling reactivates *FT1* and induces expression of glucanases of the glucan hydrolase family 17, which degrade callose, reopen PD, and restore symplasmic transport, enabling FT1 movement to the SAM and subsequent flowering (Figure 4a). GA can mimic this effect [52]. More recently, the transcription factor low-

temperature-induced MADS-box1 (LIM1) was identified in hybrid aspen [53]. LIM1 promotes low-temperature-related dormancy release by reducing callose accumulation, activating FT1, and regulating GA signaling, with GA acting downstream of LIM1 and FT1 [53].

Vernalization is well known to involve epigenetic regulation. Intriguingly, recent findings indicate PD regulators can also be epigenetically controlled. In dormant lily buds, a regulatory network is described, in which NUCLEAR FACTOR-YA7 (LoNFYA7) and a VERNALIZATION INSENSITIVE 3-LIKE 1 homolog regulate the callose synthase *LoCALS3* promoter through opposing chromatin states depending on chilling. These modifications preserve PD closure in dormant buds but allow reopening after cold, thereby controlling symplasmic transport via an inheritable PD state (Figure 4b). This discovery highlights an additional epigenetic layer linking temperature, PD regulation, and flowering time.

Reproductive stage

Plant reproduction relies on the correct establishment and progression of female and male germlines, eventually giving rise to haploid gametophytes, main players during the fertilization process. In ovule development, the female gametophyte is established by differentiation of the megaspore mother cell (MMC), a subepidermal cell in the distal domain [54]. A distinct feature of the MMC is callose deposits in the cell wall. Transition from meiosis to mitosis of the functional megaspore requires callose degradation, generating the mature female gametophyte [55]. In *Arabidopsis*, callose in the MMC cell wall moderates symplasmic connectivity to ensure gametophyte development, regulating genes involved in chromatin regulation, meiosis, and cell cycle progression [56] (Figure 4c). This suggests that callose accumulation is also critical for the establishment of the MMC's unique epigenetic state, necessary to guide subsequent meiosis [57]. This resembles the epigenetically heritable PD state seen in lily bud dormancy (see 'Flowering transition'), pointing at a general possibility of epigenetic PD regulation. Symplasmic signaling is required for sporophytic–gametophytic communication during ovule development (Figure 4E). Female germline progression was disrupted in mutant studies (*CHOLINE TRANSPORTER-LIKE1*(*CTL1*) or callose synthase (*cals3m)*) where PD morphology was altered specifically at the integuments [58].

Male gametophyte development shows parallels. In *O. sativa*, dynamic control of the cellulose-to-callose ratio maintains the optimal cell-to-cell connection at tapetum–pollen mother cell (PMC) and PMC–PMC interfaces (Figure 4d) [59]. Meiotic entry and proper extracellular spacing of PMCs require reduced symplasmic connectivity achieved by callose-moderated reduction of PD frequency rather than callose deposition alone. Here, callose is proposed to act as a stabilizing factor of PD. As anther development progresses, callose further regulates the optimal extracellular distance and cell connectivity among the central anther locular cells.

Upon fertilization, the ovules turn into a seed, harboring the embryo [60]. A novel plant tissue, termed the callose gate, was reported at the phloem end of the ovule [61]. At fertilization, genetic pathways trigger callose degradation and nutrient flow (Figure 4E). Mutants of a specific PD-associated glucanase (At_BGp-pap) produced a permanent closed-state of the callose gate and ultimately seed abortion. In parallel, overexpression produced a permanently open state, high nutrient uptake, and increased seed size. The regulation mechanism for the callose gate structure is therefore associated with PD regulatory components, connecting symplasmic nutrient flow and seed viability.

PD-associated callose turnover also affects organ size in fruit. The quantitative trait locus–associated gene FRUIT WEIGHT *2.2* (FW2.2) was previously characterized as a negative regulator of cell division and thereby fruit weight in tomato [62]. Recently, FW2.2 was identified as a PD-localized protein, interacting with callose synthases altering PD permeability via callose accumulation [63]. Mutants and overexpression lines are characterized by increased and reduced callose deposition, respectively, causing altered PD permeability. However, the authors speculate the activity of FW2.2 is spatiotemporally limited because tomato plants engineered with ectopic expression or full knock-out of *FW2.2* did not exhibit changes in fruit weight.

PD regulation also plays a central role in cotton fiber development. Fiber elongation is associated with transient callose-dependent PD closure and branching, synchronized with solute transporter expression and cell wall–loosening activity [64]. A recent mechanohydraulic model demonstrates PD dynamics, in combination with aquaporins, generate peaks in turgor and osmotic pressure that drive fiber expansion [65]. Furthermore, PLASMODESMATA CALLOSE BINDING (PDCB) proteins, which modulate PD permeability, have been implicated in this process. GhPDCB9, specifically expressed in fiber cells, was identified as a positive regulator of fiber length [66].

Future directions and challenges

The moderated intercellular transport of signals: transcription factors, phytohormones, RNA, and nutrients plays a central role in the control of plant development, from early embryogenesis and meristem formation to

flowering and reproduction. This review has highlighted PD as complex, multilayered regulatory hubs, coordinating cell-to-cell connectivity. Newly discovered regulatory mechanisms like epigenetics [56,67], MCTP activity, and PD complexity are expanding our understanding of PD control far beyond the classical callose-based model [8]. Recognizing the significance of these new mechanisms is essential to fully understand how PD function underpins plant development at every stage of the life cycle.

Although remarkable progress has been made in uncovering the mechanisms of plant-to-plant communication and the role of PD in coordinating intercellular signaling, our understanding of these processes at specific stages of plant development remains fragmentary. This gap largely reflects the technical difficulties of accessing and preserving plasmodesmal structures while maintaining their physiological integrity in native development-specific contexts. As developmental complexity increases, resolving cell-to-cell communication at appropriate spatial and temporal scales becomes increasingly challenging. Bridging this knowledge gap will require the application of emerging tools in live-cell imaging, single-cell and spatial omics, and correlative microscopy to capture plasmodesmal dynamics *in situ* [8,35,68]. Such approaches will reveal how intercellular communication is maintained, remodeled, or restricted throughout the plant life cycle, ultimately advancing a truly cell-resolved view of plant development and signaling.

Author contributions

E.B., Z.K.B., R.C.B., and R.P. contributed equally to conceptualization, original draft preparation, visualization, reviewing, and editing. Funding acquisition, project administration, supervision, and reviewing, and editing were done by R.S.

Funding

We are grateful for funding through the ERC Synergy grant 'SYMPORE,' grant number 951292.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgements

Due to space limitations, we apologise to colleagues where their work was not cited. We thank the ERC SymPore Consortium for thoughtful discussion.

Data availability

No data was used for the research described in the article.

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* of special interest
** of outstanding interest

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