Molecular Regulation of Maize Plant Height and Its Implications for Breeding Maize Tolerant to Enhanced Planting Density

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Abstract: The continuous increase in maize yield has become increasingly dependent on high-density planting, rendering plant height (PH) and plant architecture core traits that balance light interception efficiency and lodging resistance. Although many PH regulators have been well characterized, we are still confused on how to handle these regulators for maize breeding. At the same time, biotechnological advances have provided us with tools to address this issue. This review presents perspectives on integrating molecular information underlying maize PH regulation with these biotechnological tools to improve high-density tolerance in maize. We first compile an inventory of genes associated with maize PH and classify them into hormone-related and non-hormonal pathways, and reveal that loss-of-function mutations in most of these genes exhibit prominent allelic characteristics, including extreme dwarfism and pleiotropy. We discuss the underlying mechanisms of pleiotropy, which is important for successful editing aimed at achieving moderate dwarfism. Then, we discuss current strategies for high-density tolerance improvement, including genome editing strategies, introgression of dominance genes, multi-gene stacking, and phenotypic prediction, and offer key insights for their practical implementation. We offer insights and perspectives for future high-density tolerance improvement, which requires cell-resolution gene regulatory networks, AI-enabled precision genome editing, and phenotypic prediction models incorporating all environmental factors influencing plant growth. This review aims to provide guidance for researchers and breeders in translating molecular knowledge into improved high-density tolerance, lodging resistance, and yield gain.

Key words: maize; plant height; high-density tolerance; molecular mechanism; genome editing

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INTRODUCTION

The global population is projected to reach nearly 10 billion by 2050, demanding a remarkable increase in food production (Assefa et al., 2018). As a global staple crop, maize is expected to contribute substantially to meeting this ever-growing demand. A key to unlocking future yield gains lies in optimizing plant architecture for higher planting densities, a principle that was central to the Green Revolution's success in wheat and rice (Peng et al., 1999; Sasaki et al., 2002). This principle has been pursued by the global maize research community (Duvick, 2005). Increasing density deteriorates within-canopy light quality and intensifies competition among plants, triggering shade-avoidance responses (SAR) that promote internode elongation and increase lodging risk (Wang et al., 2016). Plant height (PH) is a pivotal

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trait correlated with both canopy structure and mechanical stability. Accordingly, an important breeding goal is semi-dwarf, density-adapted plant architecture that increases yield potential at the unit area level.

Many PH genes have been cloned in maize, and most of them are related to gibberellin (GA), auxin, and brassinosteroid (BR). Examples include the GA biosynthesis genes *anther ear1* (*An1*) (Bensen et al., 1995) and *DWARF1* (*D1*) (Chen et al., 2014), *brachytic2* (*Br2*), which modulates auxin transport (Multani et al., 2003), and the BR biosynthesis gene *brassinosteroid-deficient dwarf1* (*BRD1*) (Makarevitch et al., 2012). However, the deployment of these genes is often limited by (i) unfavorable pleiotropy, (ii) genetic-background dependence, (iii) frequent recessive inheritance, and (iv) linkage with undesirable loci. This creates a fundamental challenge for molecular breeders: How to handle a catalog of PH genes to design high-density tolerant maize ideotypes? The conventional approach to address this issue is "from find to validate to deploy" based on one or a few genes. With the advancement of biotechnology, we are now moving to a new approach of "from design to stack to test". This new approach requires us to: (i) deeply understand the gene regulatory networks (GRNs) that govern PH in concert with other agronomic traits, (ii) utilize genome editing to create tunable alleles that minimize negative pleiotropy, and (iii) integrate these modified alleles into multi-gene stacks whose performance in target environments can be predicted by advanced models.

In this review, we firstly retrospect the regulators of maize PH, and explore the mechanistic origins of pleiotropy. Then, we discuss the current strategies for developing semi-dwarf maize materials and varieties with enhanced high-density tolerance. Finally, we provide our opinions on how to accelerate the breeding process of density-tolerant semi-dwarf maize with the aid of biotechnological tools. The insights and perspectives synthesized in this review may serve as a valuable reference for maize geneticists and breeders alike.

Genetic Pathways Underlying Dwarfism in Maize

Let us begin with the current understanding of the regulatory mechanisms underlying PH in maize. PH is shaped by coordinated regulation of cell division, expansion, and differentiation. In maize, most cloned dwarfing genes fall into hormone-associated pathways, while some act through non-hormonal networks (Fig. 1; Table 1).

GA pathway

GA biosynthesis

GA biosynthesis starts with a cyclization step that

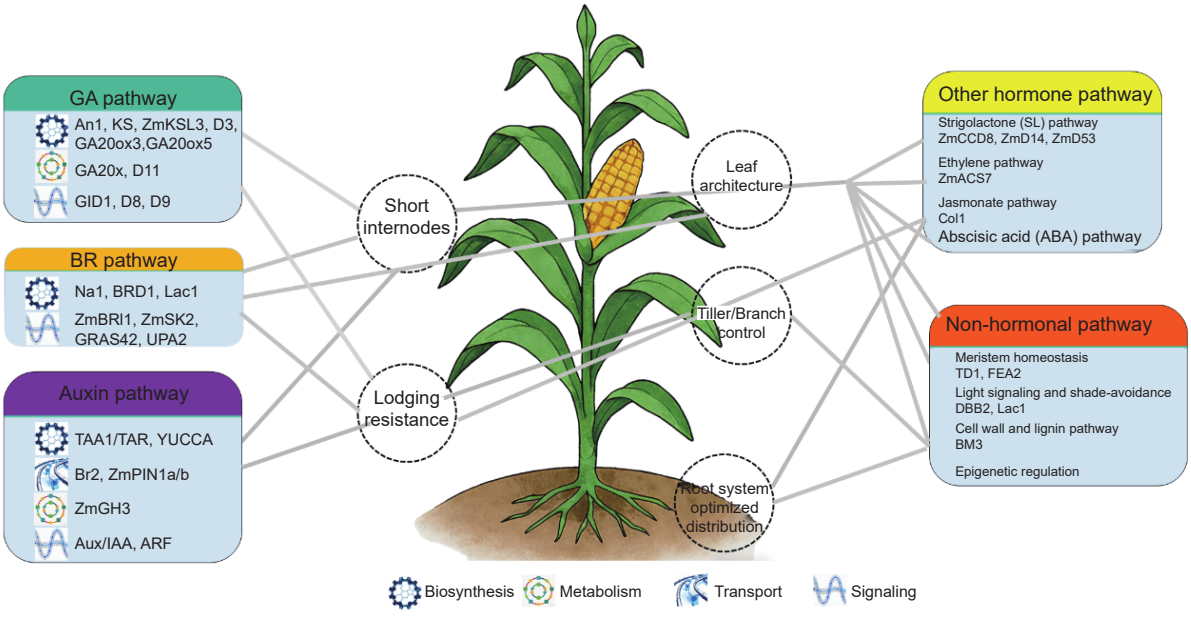

Fig. 1. Genetic pathways regulating maize dwarfism.
This diagram illustrates the major hormone pathways (gibberellin, GA; brassinosteroid, BR; auxin; strigolactone, SL; ethylene; jasmonate, JA; Abscisic acid, ABA) and non-hormonal regulators that modulate PH in maize.

converts geranylgeranyl diphosphate (GGPP) to ent-copalyl diphosphate (CPP), which is subsequently converted to ent-kaurene; ent-copalyl diphosphate synthase (CPS) catalyzes the first step (from GGPP to

Table 1. Key genes regulating PH and architecture in maize.

Gene	Pathway	Mutant Phenotype	Breeding Potential/Utilization Status	References
An1	GA biosynthesis	GA-responsive dwarfism	Limited due to severe pleiotropy, potential for weak allele editing	Bensen et al., 1995
D1	GA biosynthesis	Dwarfism	Similar to *An1*, requires fine-tuning	Chen et al., 2014
D3	GA biosynthesis	Severe GA deficiency	Suitable for partial loss-of-function editing	Winkler and Helentjaris, 1995
D5	GA biosynthesis	GA-deficient dwarfism	GA-deficient dwarfism	Fu et al., 2016
ZmGA20ox3	GA biosynthesis	Moderate dwarfism	Editing/miRNA suppression used in hybrid breeding	Zhang et al., 2020; Paciorek et al., 2022
GA2ox7.3	GA catabolism	Dwarfism, shortened internodes	Overexpression lines show potential for height control	Deng et al., 2024
D8/D9	GA signaling	GA-insensitive dwarfism	Edited alleles tested in multi-location trials with yield maintained	Harberd and Freeling, 1989; Lawit et al., 2010; Weers et al., 2024
GID1	GA signaling	Putative GA-insensitive dwarfism	Functional studies remain limited	Ueguchi-Tanaka et al., 2005
Na1	BR biosynthesis	Reduced stature, altered leaf morphology	Extreme dwarfism, potential for regulatory edits	Hartwig et al., 2011
BRD1	BR biosynthesis	Extreme dwarfism, stacked leaves	Not suitable for direct use, useful for mechanistic studies	Makarevitch et al., 2012
lac1	BR biosynthesis	Upright upper leaves, flat lower leaves	Increases yield at high densities; suitable for stacking	Tian et al., 2024
UPA2	BR signaling	Leaf angle reduction, improved high-density yield	Teosinte allele enhances density tolerance; used in stacking strategies	Tian et al., 2019
ZmBRI1	BR signaling	BR-insensitive dwarfism, upright leaves	RNAi lines show architectural changes	Kir et al., 2015
GRAS42	BR signaling	Semi-dwarf, upright leaf angle	Promising for combining dwarfism with upright leaf	Kaur et al., 2024
VT2	Auxin biosynthesis	Reduced internode, short stature	Not suitable for direct breeding, provides mechanistic insight	Phillips et al., 2011
Br2	Auxin transport	Shortened internodes, reduced height	Tunable editing alleles developed, promising for density tolerance	Multani et al., 2003; Zhao et al., 2025
ZmPIN1a/b	Auxin transport	Altered architecture, reduced height	Potential for fine-tuning via tissue-specific expression	Carraro et al., 2006; Gallavotti et al., 2008
ZmGH3	Auxin Metabolism	Modulated growth	Potential for stacking with other dwarf genes	Feng et al., 2015; Staswick et al., 2005
ZmCCD8	SL	shorter internodes, stem diameter	GR24-rescueable, potential for targeted modulation	Guan et al., 2012
ZmD14, ZmD53	SL signaling	SL-insensitive	*D14–D53* module as molecular leverage point	Liu et al., 2021a
ZmACS7	Ethylene	Semi-dwarf, shorter internodes	Corresponds to semidwarf3, useful for height control	Li et al., 2020a
COI1	JA	Shorter internodes, reduced height	Quadruple mutants promise for integrating stress responses	Feiz et al., 2024
VP14	ABA biosynthesis	ABA-deficient; affects primary root elongation and shoot elongation, indirectly influencing plant height	Modulate plant height via antagonistic interaction with GA; direct breeding application not yet established	Tan et al., 1997; Saab et al., 1992; Spollen et al., 2000; Yue et al., 2021

Continued Table

Gene	Pathway	Mutant Phenotype	Breeding Potential/Utilization Status	References
ZmPYL3	ABA signaling	Involved in ABA perception; influences cell expansion and internode growth	Core component of ABA signaling; can modulate growth-stress balance; potential editing target	Wang et al., 2014; Wang et al., 2018
ZmPP2Cs	ABA signaling	Negatively regulates ABA signaling; affects ABA sensitivity and plant height	Modulating ABA signaling threshold could optimize plant height; potential gene editing target	Wang et al., 2014; Wang et al., 2018
ZmSnRK2s	ABA signaling	Positively regulates ABA signaling; affects cell expansion and internode growth	As positive regulators, they can crosstalk with other hormone pathways to influence plant height	Wang et al., 2018
miR156	Epigenetic regulation	Overexpression delays vegetative phase change and alters plant height and architecture	Can optimize plant height and flowering time via SPL module; potential molecular breeding tool	Chuck et al., 2007
miR159	Epigenetic regulation	Regulates GAMYB transcription factors, thereby affecting gibberellin signaling and plant height	Indirect regulator of GA signaling; potential target for plant height modulation	Aya et al., 2009; Yu and Wang, 2020
miR164e	Epigenetic regulation	Cleaves *NAC32* transcript, relieving DELLA stabilization and promoting internode elongation; overexpression increases plant height	Precision editing target for plant height control; can be used to develop semi-dwarf materials	Peng et al., 2026
PAR1	Epigenetic regulation	Intronic Cacta transposon methylation controls expression: hypermethylation → normal height; hypomethylation → reduced height	Epiallele can be used to modulate plant height without complete knockout side effects	Li et al., 2025
ZmLSM2	Epigenetic regulation	Differential intronic DNA methylation affects RNA processing/maturation in vascular stem cells, influencing stalk strength and lodging resistance	Epigenetic modification can improve stalk strength and lodging resistance under high planting density	Zhang et al., 2026
TD1	Meristem homeostasis	Increased meristem size, slightly shorter	Moderate height effect, useful in combination	Bommert et al., 2005
FEA2	Meristem homeostasis	Enlarged meristems, fasciated ears	Moderate effect, potential for combination with other genes	Taguchi-Shiobara et al., 2001
DBB2	Light signaling	Reduced shade-induced elongation	High-density planting relevance; editing potential	Wang et al., 2025
ZmPHYC1/C2	Light signaling	Reduced height, attenuated shade avoidance	CRISPR-edited lines show SAR reduction under crowding	Li et al., 2020b
bm3	Cell wall/lignin	Altered lignin composition, digestibility	Indirect effect on height; useful in stacking for stalk strength	Vignols et al., 1995
ZmRPH1	Microtubule-associated	Reduced plant and ear height	Overexpression lines show dominant effect	Li et al., 2020c
UPA2	Leaf angle	Upright leaf angle	Teosinte allele, used in breeding for dense planting	Tian et al., 2019; Tian et al., 2024
ZMM28	Growth regulation	Increased yield, improved nitrogen use	Stacked with *D8* edits to recover yield	Wu et al., 2019; Fernandez et al., 2022

CPP), while ent-kaurene synthase (KS) catalyzes the second step (from CPP to ent-kaurene). Both CPS and KS belong to the diterpene synthase family, a group of cyclases that catalyze the formation of diterpenoid compounds. In maize, *An1* encodes CPS, and loss-of-function *An1* causes GA-responsive dwarfism accompanied by developmental abnormalities (Bensen et al., 1995). However, *an1* mutants still accumulate ~20% of wild-type ent-kaurene, implying there are additional CPS/KS-like activities. The redundancy of CPS/KS-like activities is supported by the identification of specific KS family members in maize. Kaurene synthase-like 3 (*ZmKSL3*), mapped to the classic GA-deficient dwarf-5 (*d5*) region, directly links diterpene synthase to PH regulation (Fu et al., 2016).

The next stage in GA biosynthesis is the early oxidation process. The classic *Dwarf3* (*D3*) gene encodes a cytochrome P450, and acts immediately downstream of the CPS/KS-mediated cyclization steps. It catalyzes the early oxidation of ent-kaurene to form ent-kaurenol, and subsequently ent-kaurenal, which are essential intermediates for the synthesis of bioactive GAs. Similar to the CPS/KS-mediated cyclization step, defects in this oxidation step exert a strong effect on PH but often have pleiotropic effects (Winkler and Helentjaris, 1995), as blocking this oxidation step prevents the formation of bioactive GAs.

Following the early oxidation step, the GA biosynthesis pathway proceeds to the late activation stage, catalyzed by GA20-oxidases (GA20ox) and

GA3-oxidases (GA3ox). GA20ox catalyzes the sequential oxidation of C_{20}-GA precursors to form C_{19}-GA intermediates, while GA3ox (Chen et al., 2014) further oxidizes these intermediates to produce bioactive GAs that directly regulate PH and broader developmental processes (Yamaguchi, 2008). Targeted suppression of *ZmGA20ox3* and *ZmGA20ox5* in vegetative tissues produces short-stature plants with improved standability while maintaining reproductive GA levels (Paciorek et al., 2022). The *GA20ox* family in maize comprises multiple members with distinct expression patterns and functional roles. *ZmGA20ox1*, *ZmGA20ox3*, and *ZmGA20ox5* are predominantly expressed in vegetative tissues, whereas *ZmGA20ox2* and *ZmGA20ox4* show higher expression in reproductive structures (Paciorek et al., 2022; Zhang et al., 2020). The multi-member structure also provides functional redundancy, buffering against complete loss-of-function mutations that would otherwise cause severe dwarfism.

GA catabolism and transport

In maize, GA2-oxidases (GA2ox) are key gibberellin deactivation enzymes that help maintain GA homeostasis by converting bioactive GAs and their immediate precursors into inactive forms. Dosage-based tests have shown that overexpression of a *GA2ox7.3* gene in maize caused dwarf plants and shortened internodes (Deng et al., 2024). GA and its intermediates can move between organs and compartments (Binenbaum et al., 2018). Mechanistic support for transporter-mediated routing has been demonstrated in *Arabidopsis*, including NPF (Nitrate transporter 1/Peptide transporter family) transporters mediating GA translocation and vacuolar hormone transport (Binenbaum et al., 2018). In maize, functional characterization of transport components remains limited.

GA signaling

GA is perceived by soluble nuclear receptors of the GA insensitive dwarf 1 (GID1) family. In rice, bioactive GA binds GID1, and the GA–GID1 complex then promotes interaction with DELLA repressors, triggering DELLA ubiquitination and proteasomal degradation and thereby releasing GA-responsive growth programs (Ueguchi-Tanaka et al., 2005). However, maize *GID1* alleles are less commonly reported in the public literature. DELLA proteins repress GA responses, and stabilized DELLAs lead to GA-insensitive dwarfism. The maize genome contains two DELLA proteins, DWARF8 (D8) and DWARF9 (D9). The classical *d8* and *d9* mutants were among the first dominant dwarfing genes characterized in maize, exhibiting moderate to severe GA-insensitive dwarfism (Harberd and Freeling, 1989). The transcription factor ZmNAC32 interacts with D8 and protects it from degradation by masking the ubiquitination site (Peng et al., 2026). This stabilization of D8 suppresses the expression of growth-related genes, thereby inhibiting internode elongation. Meanwhile, miR164e negatively regulates this process by cleaving *ZmNAC32* transcripts, forming a "miR164e–NAC32–DELLA" regulatory module that precisely controls internode elongation and PH.

Beyond their role as repressors of GA signaling, DELLA proteins also function as central hubs that integrate multiple pathways. DELLAs physically interact with jasmonate–ZIM–domain (JAZ) proteins to coordinate growth with defense responses (Qi et al., 2014), repress auxin signaling by interacting with auxin response factor 6 (ARF6) (Ben-Targem et al., 2021), inhibit BR responses by binding to the transcription factor BZR1 (Li et al., 2012), integrate ABA signaling by upregulating the ABA transporter AIT1.1 (Shohat et al., 2020), and bind to phytochrome-interacting factors (PIFs) such as PIF3 and PIF4 to couple GA signaling with light perception (Li et al., 2016). In maize, the JA co-receptor F-box protein COI1 can directly trigger proteasome-dependent degradation of DELLA proteins (Feiz et al., 2024). In the *coi1* quadruple mutant, both D8 and D9 accumulate to high levels, leading to shortened internodes and reduced photosynthetic efficiency. These findings highlight DELLAs as valuable precise editing targets for optimizing plant architecture and stress tolerance in maize breeding.

BR pathway

The classical BR-deficient dwarf mutant in maize, *nana plant1* (*na1*), is caused by disruption of a DET2-like (de-etiolated 2) steroid 5α-reductase acting in early BR biosynthesis, displaying reduced stature and altered leaf morphology. *Na1* supplies upstream substrates and metabolic flux (Hartwig et al., 2011). *BR-deficient dwarf1* (*BRD1*) encodes a BR C-6 oxidase and catalyzes the terminal oxidation and activation step of late BR biosynthesis, converting intermediates into bioactive BRs. The null allele *brd1-*

m1 causes extreme dwarfism with stacked leaves and severe reproductive defects (Makarevitch et al., 2012). Brassinosteroid (BR)-mediated regulation of plant architecture is also evidenced by the *UPA1* and *UPA2* loci, which jointly control maize leaf angle (Tian et al., 2019). *UPA2* involves a polymorphism upstream of *ZmRAVL1*, which encodes a B3-domain transcription factor, and influences endogenous brassinosteroid content and leaf angle by regulating *BRD1* (underlies *UPA1*). The beneficial *UPA2* allele from teosinte boosts yield under dense planting. The *lac1* mutant displays a "smart canopy" with upright upper leaves and flat lower leaves (Tian et al., 2024). *LAC1* encodes a brassinosteroid C-22 hydroxylase responsible for BR biosynthesis, and its disruption reduces BR specifically in upper leaves via a *RAVL1*-mediated shade response. Field trials show that *lac1* increases yield at high densities, with further gains when combined with the teosinte *UPA2* allele.

Arabidopsis studies established the core BR signaling module that BR is perceived by the plasma-membrane receptor kinase BRI1 (BR insensitive1), which functions with co-receptors (often from the BAK1/SERK family). BR signaling inhibits the GSK3-like (glycogen synthase kinase3) kinase BIN2 (BR insensitive2), in part via BSU1-type (BRI1 suppressor1) phosphatases, allowing the accumulation and activation of BZR1/BZR2 (BR resistant1/2) transcription factors (Wang et al., 2006). In maize, RNAi knockdown of *ZmBRI1* homologs produces BR-insensitive phenotype characterized by shortened internodes, dark-green and upright/twisted leaves, reduced auricle formation, and altered patterns of cell division and elongation (Kir et al., 2015), and maize BIN2-like GSK3 kinases (ZmSK2: SHAGGY-like kinase 2) show strong developmental defects when perturbed (Wang et al., 2022). GRAS42 (GRAS transcription factor 42) is also a signaling component in maize, and GRAS42 loss results in semi-dwarf plants with shorter/wider leaves and a more upright leaf angle (Kaur et al., 2024), consistent with partial attenuation of BR signaling rather than complete BR deficiency.

Auxin pathway

The IPyA (indole-3-pyruvic acid) pathway is a two-step auxin biosynthetic route that involves two conserved enzyme families. Tryptophan aminotransferases (TAA1/TAR) enzymes convert tryptophan (Trp) into IPyA, and YUCCA flavin monooxygenases convert IPyA into auxin. In maize, *vanishing tassel2* (*VT2*) encodes a grass-specific TAA/TAR-type aminotransferase, and *vt2* mutants show insufficient endogenous auxin synthesis, and strong developmental defects, including reduced internode development and short stature (Phillips et al., 2011).

The *brachytic2* (*Br2*) gene encodes a ATP-binding cassette subfamily B transporter, and functions in intercalary meristems and nodal regions to export auxin into long-distance auxin streams. *Br2* loss reduces auxin export/transport, shortens lower internodes, and reduces PH (Multani et al., 2003). Interestingly, allelic variations at *Br2* in teosinte reduce internode elongation under competitive conditions (Wills et al., 2013), suggesting that *Br2* alleles from wild relatives could be mined for semi-dwarf variants. PIN proteins establish auxin efflux polarity and thus auxin gradients. *ZmPIN1a* and *ZmPIN1b* influence maize architecture via polar auxin transport (Carraro et al., 2006). Perturbations in PIN1-dependent auxin maxima formation and polar flux organization can affect plant growth and PH (Carraro et al., 2006; Gallavotti et al., 2008).

Auxin levels are buffered by conjugation. Stress response analysis of the *ZmGH3* (*gretchen hagen3*) family supports a role for *ZmGH3*-mediated conjugation in reducing free auxin pools and modulating growth and stress responses (Feng et al., 2015). Mechanistically, ZmGH3 proteins function as ATP-dependent auxin–amido synthetases that conjugate auxin to amino acids (e.g., auxin-Asp/auxin-Glu), thereby lowering the free auxin pool and reshaping auxin homeostasis and downstream growth programs (Staswick et al., 2005).

Auxin signaling relies on TIR1/AFB (Transport inhibitor response 1/auxin signaling F-box) F-box auxin co-receptors that promote ubiquitin-mediated degradation of Aux/IAA repressors, thereby releasing ARF (auxin response factor) transcription factors to activate auxin-responsive growth genes. In maize, many components of this nuclear signaling module are conserved, and pathway activity intersects nutrient and stress responses (Wang et al., 2023a). Using a synthetic nuclear auxin-response circuit, a study showed that all 16 maize Aux/IAA repressors can be degraded after auxin treatment, and that how fast they disappear depends on which receptor and which Aux/IAA are paired. The maize auxin receptor ZmAFB2/3b1 made the circuit respond to lower auxin

doses (i.e., higher sensitivity) than circuits built with *Arabidopsis* receptors (Ramos Báez et al. 2020). Together, these results indicate that natural or engineered changes within the TIR1/AFB–Aux/IAA–ARF core module can tune the strength and timing of auxin-driven transcription, which matters for plant architecture.

Other hormone pathways that regulate maize PH

Strigolactone (SL) pathway

SLs are carotenoid-derived hormones best known for suppressing axillary branching, but in maize SL deficiency also affects stem thickness, internode elongation, and adventitious root development. Knockout of the SL biosynthetic gene *ZmCCD8* (*carotenoid cleavage dioxygenase8*) caused pleiotropic architecture changes including reduced internode elongation and markedly reduced stem diameter, and these phenotypes can be rescued by GR24 (a synthetic strigolactone analog) (Guan et al., 2012).

In maize, SL perception is mainly mediated by *ZmD14A*/*B*, which encode α/β-hydrolase–type strigolactone receptors. Upon binding SL, ZmD14 proteins undergo conformational changes and form a signaling complex with the SCF F-box protein, which then promotes ubiquitin–proteasome–mediated degradation of downstream repressors such as ZmD53 and SMXL proteins. ZmD53 is a central SL signaling repressor that interacts with ZmD14A/B in a GR24-dependent manner, and a gain-of-function *ZmD53* form produces strong SL-insensitive phenotypes (Liu et al., 2021a).

Ethylene pathway

Ethylene signaling can reduce cell elongation and often promotes radial thickening and lignification, potentially lowering PH while strengthening stalks. Consistent with this, transcriptomic studies of maize dwarfs frequently enrich ethylene-related regulators (Gao et al., 2024). At the gene-to-phenotype level, evidence is strongest for *ZmACS7* encoding aminocyclopropane-1-carboxylic acid synthase 7, and corresponding to the *semidwarf3* locus. A C-terminal change increases ZmACS7 protein stability, elevates ACC/ethylene accumulation, suppresses internode cell elongation, and ultimately reduces PH (Li et al., 2020a).

ABA pathway

ABA pathway genes regulate PH through three coupled layers: ABA biosynthesis, signal transduction, and crosstalk with other hormones. At the biosynthetic level, the *VP14* gene encodes a 9-cis-epoxycarotenoid dioxygenase, and genetic work demonstrates that *vp14* mutants are ABA-deficient, establishing *VP14* as a key ABA biosynthetic regulator in maize (Tan et al., 1997). Endogenous ABA accumulation helps sustain primary root elongation while limiting shoot elongation, and this process directly affects plant stature (Saab et al., 1992; Spollen et al., 2000).

At the signaling level, genetic complementation experiments reveal a core ABA signaling network involving ZmPYLs (receptors), clade A ZmPP2Cs (negative regulators), and ZmSnRK2s (positive kinases) (Wang et al., 2018), and specific receptor–phosphatase modules (*ZmPYL3–ZmPP2C*) were supported by biochemical validation in maize (Wang et al., 2014). These modules control ABA sensitivity thresholds, which influence cell expansion, meristem activity, and internode growth. ABA may participate in the regulation of vegetative growth in an antagonistic manner with GA (Yue et al., 2021). For instance, drought stress elevates ABA levels, which can suppress the expression of GA biosynthesis genes like *GA20ox*, leading to reduced bioactive GA and subsequently shorter internodes (Liu et al., 2024). Collectively, these findings indicate that ABA pathway genes can indirectly shape PH or growth by modulating the trade-off between growth and stress response.

Non-hormonal regulatory pathways

Meristem homeostasis

A major non-hormonal determinant of PH is the size and activity of stem-cell pools in the shoot apical meristem (SAM). CLAVATA–WUSCHEL (CLV–WUS) pathway is well known as the central regulatory pathway coordinating stem cell proliferation with differentiation in *Arabidopsis*. Among the core components, CLV1 is a leucine-rich repeat receptor kinase, CLV2 is a receptor-like protein, CLV3 is a small secreted peptide ligand, and *WUS* encodes a homeodomain transcription factor. In maize, CLV-like signaling acts as a brake on meristem proliferation, thereby influencing organ initiation and plant stature. *TD1* (*thick tassel dwarf1*) encodes a maize ortholog of *CLV1*; *td1* mutants show increased meristem size and altered inflorescence

traits, and importantly are slightly shorter than normal siblings (Bommert et al., 2005). *FEA2* (*fascinated ear2*) encodes a CLV2-like protein that restricts meristem proliferation; *fea2* mutants have enlarged meristems and fasciated ears, demonstrating CLV pathway conservation in maize (Taguchi-Shiobara et al., 2001). Weak or hypomorphic alleles in these genes can result in a modest increase in meristem size, leading to a slight increase in kernel row number without causing fasciation (Bommert et al., 2013). Concurrently, they may cause a minor reduction in PH due to altered internode patterning. Precise modulation of meristem homeostasis, perhaps through promoter editing to subtly reduce gene expression (Liu et al., 2021b), can simultaneously improve yield potential (more kernel rows) and lodging resistance (slightly shorter plants), a highly desirable combination for high-density ideotypes.

Light signaling and shade-avoidance networks

Under high density, SAR typically triggers internode elongation, and reduces yield stability. HY5 is a key transcription factor in light signaling, and DBB2 (dimerization partner of BBX proteins2) can physically bind HY5 and thereby inhibit HY5-driven transcription of the GA-catabolism gene *ZmGA2ox4*. Since *ZmGA2ox4* deactivates GA, lowering *ZmGA2ox4* expression would be expected to reduce GA breakdown, helping sustain higher GA levels and thus promote SAR-induced internode elongation (Wang et al., 2025). DBB2 also interacts with PIF4, a bHLH transcription factor, to enhance the transcriptional activation of cell elongation-related genes (Wang et al., 2025). Recent work has revealed additional layers of light–BR crosstalk relevant to high-density planting. In the *lac1* smart canopy mutant, phytochrome A (PHYA) photoreceptors accumulate under shade conditions and directly interact with the *RAVL1* transcription factor, promoting its degradation via the 26S proteasome (Tian et al., 2024). Since *RAVL1* activates *LAC1* expression and BR biosynthesis, this PHYA-mediated degradation reduces BR biosynthesis specifically in upper leaves, thereby decreasing upper leaf angle under dense stands. This mechanism provides a direct molecular link between light perception and BR-mediated architectural optimization, explaining how plants dynamically adjust leaf angles in response to crowding.

Epigenetic regulation: miRNAs and DNA methylation

Beyond direct genetic and hormonal controls, epigenetic mechanisms such as microRNAs (miRNAs) and DNA methylation add a fresh layer of PH determination. Several miRNAs have been implicated in the post-transcriptional regulation of genes governing maize architecture. For instance, the *miR156–SPL* module controls vegetative phase change and internode elongation, with *miR156* overexpression delaying reproductive transition and altering plant stature (Chuck et al., 2007), while *miR159* modulates the transcription of GAMYB-like transcription factors that mediate gibberellin signaling (Aya et al., 2009; Yu and Wang, 2020). Meanwhile, the *miR164e–NAC32* module modulates PH by post-translationally regulating DELLA protein stability; *miR164e* overexpression promotes internode elongation, whereas *NAC32* overexpression reduces PH (Peng et al., 2026).

DNA methylation regulates maize PH through multiple mechanisms. In the *PAR1* epiallele, intronic *cacta* transposon methylation controls gene expression: hypermethylation maintains normal PH, while hypomethylation reduces it (Li et al., 2025). Similarly, differential intronic DNA methylation of *ZmLSM2* modulates RNA processing and maturation within vascular stem cells, influencing stem mechanical strength and lodging resistance (Zhang et al., 2026). DNA methylation affects transcription factor binding at cis-elements, and such cis-regulatory variation explains ~72% of heritable phenotypic variation (Engelhorn et al., 2025). This collectively highlights that our understanding of the functional roles of DNA methylation remains in its infancy.

Cell wall and lignin pathways

Lignin biosynthesis mutants provide direct evidence that cell wall pathways alter stalk properties and sometimes growth allocation. The classic maize brown *midrib3* (*BM3*) mutation occurs in the gene encoding caffeic acid O-methyltransferase (COMT), altering lignin composition and digestibility (Vignols et al., 1995). Beyond its role in silage quality, *BM3* exemplifies a broader principle: modifications to lignin synthesis reshape the trade-off among PH, stalk stiffness, and biomass quality.

The molecular basis of pleiotropy in dwarfing genes

The successful deployment of dwarfing genes is often

hindered by pleiotropy, the phenomenon where a single gene or locus influences multiple unrelated traits. Understanding its molecular basis is key to designing better breeding strategies by excluding the causal genes or sequences based on its mechanism. The pleiotropic effects observed in maize dwarfing mutants can be broadly categorized into several mechanistic classes.

Pathway-level crosstalk

Genes in hormone pathways are often pleiotropic because the hormones usually regulate diverse processes beyond internode elongation (Fig. 2–A). For example, severe mutations in *An1* not only dwarf the plant but also affect floral development and fertility because GA is required for these processes (Bensen et al., 1995). DELLAs are well-established signaling hubs that integrate multiple hormone pathways (Daviere and Achard, 2016). Similarly, BR-deficient *brd1* mutants exhibit extreme dwarfism alongside altered leaf morphology and reproductive defects, reflecting BR's role in cell division and differentiation (Makarevitch et al., 2012).

Spatiotemporal expression pattern

A classic example is the maize *liguleless1* (*LG1*) gene, which encodes a squamosa promoter binding protein (SBP) domain transcription factor. *LG1* is expressed at the boundary between the leaf blade and sheath, where it directs ligule and auricle formation (Zhong et al., 2025). However, *LG1* is also expressed in spikelet primordia (Eveland et al., 2014). Mutations in *LG1* affect both leaf morphology and tassel structure, demonstrating that its spatial expression domains account for its pleiotropic effects. The transcription factor bZIP29 regulates PH and endosperm filling, which can be explained by its spatiotemporal expression and a large number of target downstream genes (Yang et al., 2022; Zhang et al., 2025). These findings indicate that genes expressed in multiple tissues, or at distinct developmental stages inevitably influence diverse traits (Fig. 2–B).

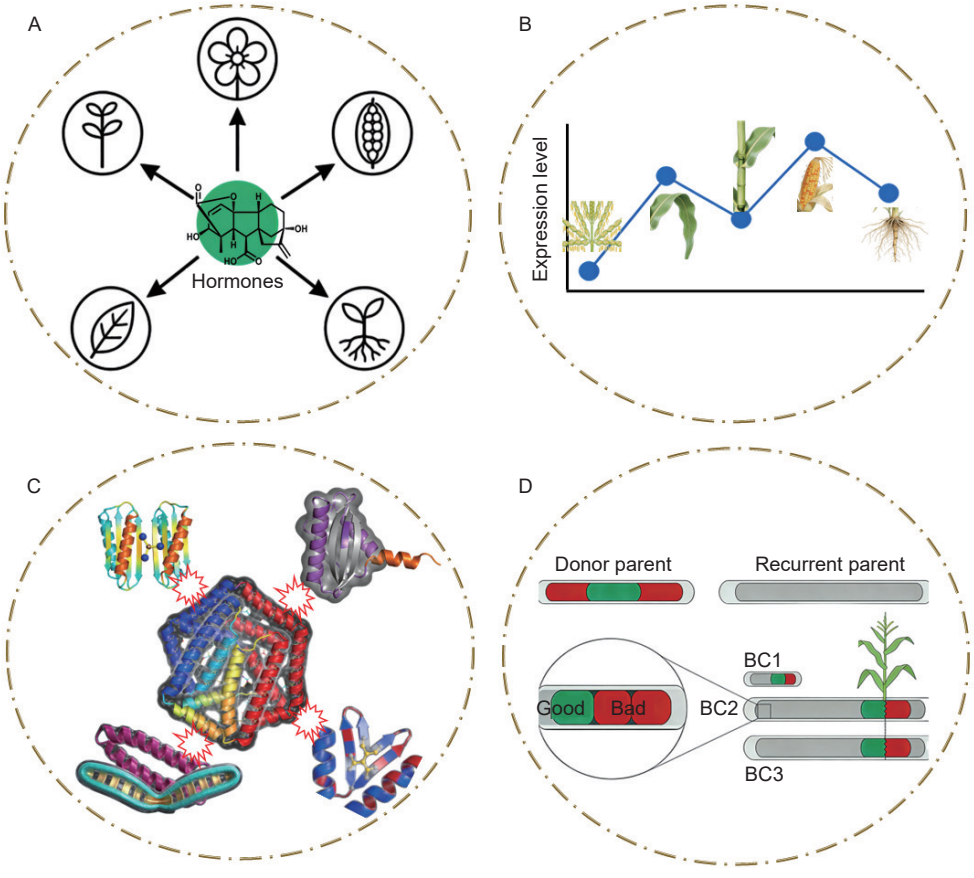

Fig. 2. The molecular basis of pleiotropy in dwarfing genes.
A. pathway-level crosstalk; **B**. spatiotemporal expression pattern; **C**. protein multifunctionality; **D**. linkage drag

Protein multifunctionality

Protein multifunctionality can arise from protein domain architecture, alternative splicing, post-translational modifications, or differential interaction partners. Many maize proteins contain multiple functional domains that mediate distinct activities. While loss-of-function of *ZmAMP1* leads to a severe dwarf phenotype (Lv et al., 2014), a missense SNP mutation in *ZmAMP1* underlies an early-flowering, semi-dwarf and lodging resistance phenotype (Wang et al., 2023b). The *knotted1* (*KN1*) gene, a founding member of the KNOX homeobox transcription factor family, binds a cis-regulatory element of *GA2ox1*, and directly regulates GA catabolism (Bolduc and Hake, 2009). At the same time, *KN1* exhibits pleiotropic effects on leaf morphology, meristem maintenance, and inflorescence architecture. The pleiotropy of *KN1* arises from this dual functionality: its DNA-binding activity governs distinct transcriptional programs (Bolduc et al., 2012), while its protein-interaction capacity allows it to interact with other proteins (Tsuda et al., 2017). In maize, protein multifunctionality represents an important mechanism underlying pleiotropy (Fig. 2–C).

Linkage drag

In breeding, when a favorable gene is introduced from a donor parent, tightly linked flanking regions carrying undesirable traits are co-inherited, creating the illusion of pleiotropy. For example, the rice *Ghd8* gene affects grain protein content in the background of *Ehd1* (wild-type allele), but is masked in the background of *ehd1* (mutant allele) due to the epistasis of *Ehd1*×*Ghd8* (Wei et al., 2024). A quantitative trait locus (QTL) cluster in a 5 Mb region around *TB1* is associated with tiller number, ear row number, flowering time, and yield (Bouchet et al., 2017), indicating tight linkage of multiple trait-associated genes in this region. Understanding linkage drag as a cause of pleiotropy is crucial for overcoming constraints in maize breeding (Fig. 2–D).

Strategies for deploying dwarf genes in high-density maize breeding

The translation of our molecular knowledge into improved varieties requires a strategic approach to deployment. Breeders now have an advanced toolbox at their disposal, with strategies ranging from single-gene precision editing to multi-gene stacking and genome-wide prediction. The choice among these is not one-size-fits-all but depends on the breeding objective, the genetic architecture of the target trait, and the specific gene's properties. This decision-making framework is illustrated in Fig. 3. For instance, if a known major gene like *Br2* has negative linkage drag, precision editing is the preferred path. If a dominant, non-pleiotropic allele is desired, a forward-genetics cloning strategy might be initiated. For complex traits like yield stability under density, multi-gene stacking combined with phenotypic prediction becomes essential. This section critically evaluates each strategy, not as an isolated case study, but as an interrelated option within this broader decision-making context.

Precision editing of known genes to break negative effects and linkage drag

Genome editing changes breeding deployment mainly in three ways. First, it enables the creation of allelic series, so that breeders can tune the degree of PH reduction. Second, it helps avoid linkage drag by introducing desired variants (Fig. 3–A). Third, precision editing of multifunctional genes can create new alleles that retain only favorable effects, avoiding the adverse phenotypic consequences. Here, we introduce the editing of some promising genes as examples.

Br2

Since a complete knockout of *Br2* by editing might mimic the strong dwarf phenotype. Editing by introducing a missense mutation in a non-critical domain, or by targeting the promoter to slightly reduce expression, can create weak or intermediate alleles. A previous study demonstrated that site-directed editing targeting the final exon of *Br2* can generate a series of dwarf mutant alleles, facilitating the development of dwarf germplasm with tunable dwarfing degrees (Zhao et al., 2025). This tunability allows breeders to select the exact degree of dwarfing that maximizes lodging resistance without incurring a yield penalty. Direct generation of *Br2* allelic editing variants in elite inbred lines can minimize background dependence and linkage drag. This approach can be combined with synergistic selection for yield-related and lodging resistance traits. Future deployment of *Br2* editing may focus on combining domain-targeted edits with promoter modifications to achieve tissue- or stage-specific expression, restricting moderate dwarfing to early vegetative growth.

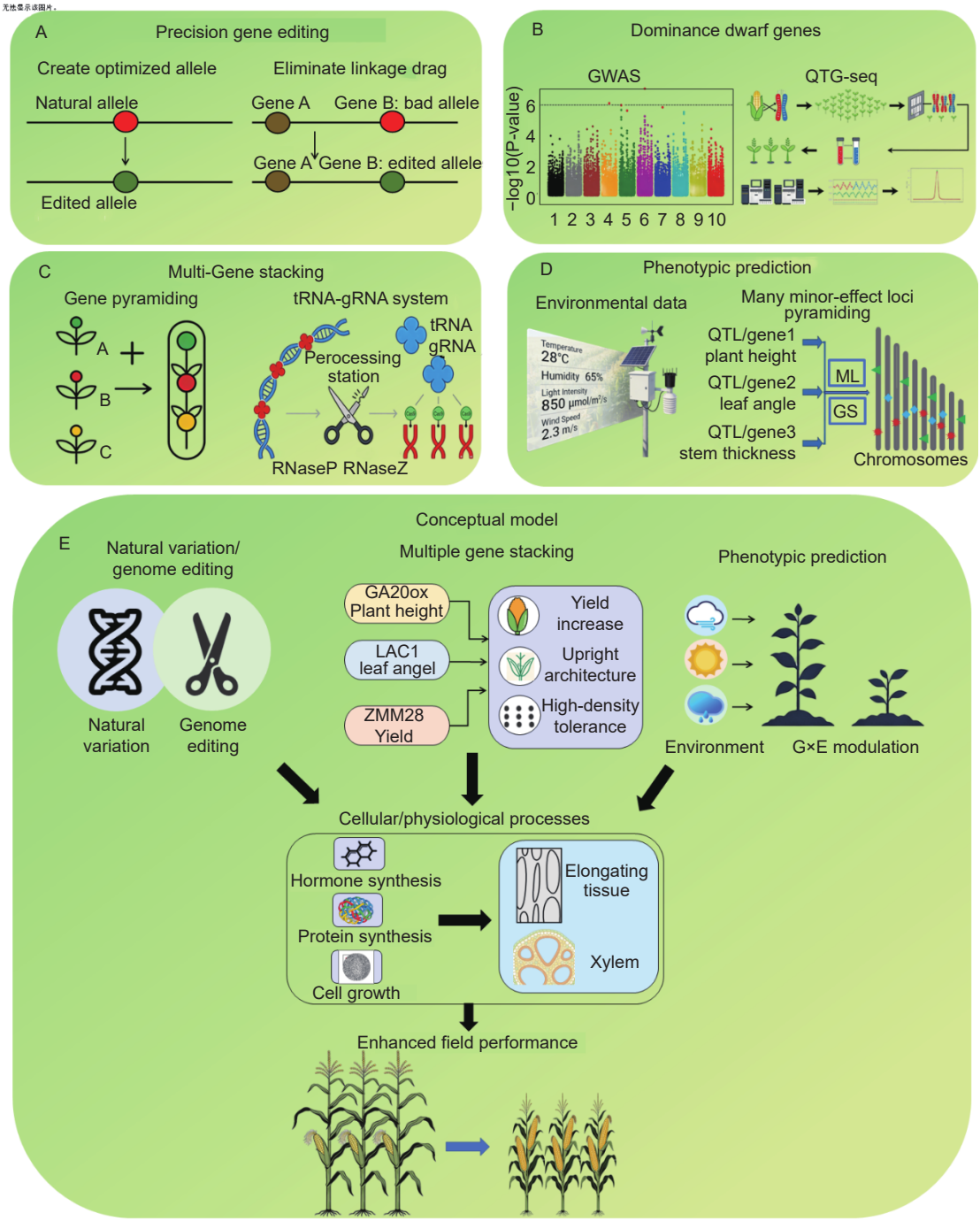

Fig. 3. Strategies for deploying dwarf genes in high-density maize breeding.
Four strategies are illustrated: **A**. precision editing of known genes to tune dwarfing degree; **B**. cloning and deployment of dominant/semi-dominant dwarf genes to simplify hybrid breeding; **C**. multi-gene stacking to break trade-offs; **D**. phenotypic prediction to guide selection under variable densities and environments; **E**. A conceptual model linking molecular regulators to high-density planting performance.

DELLA

d8 mutants behave as dominant GA-insensitive dwarfs (Harberd and Freeling, 1989), and influence developmental timing traits, highlighting both their breeding value and pleiotropy risk (Lawit et al., 2010). DELLA editing offers two advantages. First, dominant or semi-dominant effect can be achieved. Second, since DELLA proteins integrate GA signaling with other pathways, targeted editing of DELLA offers a feasible strategy to mitigate excessive elongation responses induced by dense planting and shade competition (Lawit et al., 2010). In

a field evaluation study, hybrids carrying edited *D8* were tested across 17 U.S. Corn Belt field locations from 2021 to 2022 at three planting densities (80 000, 100 000, 120 000 plants ha^{-1}). Across these densities, edited hybrids showed ~34% lower PH and 32% lower ear height, while maintaining grain yield comparable to isogenic comparators comparators, and significantly reduced lodging (Weers et al., 2024). This study provides a strong proof-of-concept that DELLA editing can deliver "shorter and more standable" hybrids without a yield penalty.

GA20ox family

Targeted engineering of *GA20ox* family members, like *ZmGA20ox3* and *ZmGA20ox5*, achieves moderate PH reduction while preserving normal reproductive development. The multi-member nature of the *GA20ox* family allows for this specificity, as suppressing a single isoform can avoid the global GA depletion caused by targeting a universally expressed biosynthesis gene like *An1* or *D3*. CRISPR/Cas9-mediated editing of *ZmGA20ox3* in maize generated semidwarf lines with concomitant reductions in endogenous GA levels (Zhang et al., 2020). Another notable example is a dominant miRNA-based suppression strategy that targets *ZmGA20ox3* and *ZmGA20ox5*, producing a short-stature architecture in hybrid germplasm (Paciorek et al., 2022). Using multi-member gene families to create tunable phenotypic effects is a recurring theme in crop breeding.

Other genes for editing

Tuning the perception of light signals and their consequent plant architecture responses can prevent maize from over-elongating upon neighbor detection. Through CRISPR/Cas9-mediated knockout and transgenic overexpression lines, *ZmPHYC1/ZmPHYC2* in maize have been demonstrated to regulate flowering time and PH, while conferring an attenuated SAR under shading conditions (Li et al., 2020b), indicating that genetic manipulation of *ZmPHYCs* can reduce PH and mitigate the SAR response in crowded planting environments.

While loss-of-function mutations in genes such as *An1*, *D3* and *Na1* result in extreme dwarfism, these genes remain potential targets for genome editing, as their partial loss-of-function or regulatory edits may generate graded reductions in PH. Moreover, promoter editing of CLE family genes has validated the feasibility of fine-tuning agronomic traits via targeted editing of regulatory regions (Liu et al., 2021b).

Cloning and deployment of dominant or semi-dominant dwarf genes

Placing a dominant or semi-dominant dwarf gene allele in a single parent can adjust hybrid performance, shorten breeding cycles, and simplify hybrid design. One of the best-studied dominant dwarf systems in maize is *D8* (Harberd and Freeling, 1989). Another representative case is *ZmRPH1* encoding microtubule-associated protein. Overexpression of *ZmRPH1* reduced both PH and ear height by shortening internodes (Li et al., 2020c). This study evaluated hybrids derived from crossing a tester to *ZmRPH1* overexpression lines and reported significantly reduced plant and ear height in hybrids, lower lodging rate, no obvious impact on flowering time and fertility, and in most cases, no significant reduction in yield traits, with density trials including 60 000, 90 000, and 120 000 plants ha^{-1}.

Despite the above cases, *de novo* cloning of dominant or semi-dominant dwarf genes is still necessary for maize breeding because the current PH-reducing alleles are either too pleiotropic (Lawit et al., 2010), too recessive for efficient hybrid deployment (Paciorek et al., 2022), or too background-dependent (Fei et al., 2022) to serve as breeding-grade tools. Although dominance QTL for dwarfism are frequently detected in maize (Chen et al., 2018; Chen et al., 2025), it is still difficult and time-consuming to clone the genes underlying these QTL using conventional map-based cloning approach. GWAS (genome-wide association study) can simultaneously fine-map multiple candidate genes (Korte and Farlow, 2013), enabling high-throughput cloning of genes underlying maize dwarfism (Fig. 3–B). However, dwarfism caused by rare alleles is often difficult to detect with GWAS, and such alleles are more readily cloned using biparental or other designed mapping populations derived from a limited number of parental lines. QTG-seq (quantitative trait gene sequencing) was proposed as a rapid strategy to clone QTGs segregating in a biparental population within only four generations, by combining QTL partitioning with bulked-segregant whole-genome sequencing and an optimized candidate-gene identification algorithm (Zhang et al., 2019). Next-generation bulked segregant analysis (NG-BSA) can integrate biological big data, machine-learning models, high-throughput phenotyping, and large population sizes, enabling

rapid and high-throughput cloning of QTGs in a streamlined workflow (Wang et al., 2023c).

Several issues should be considered when deploying dominant dwarf genes in commercial programs. First, dominance *per se* is not sufficient; the allele must exhibit stable expressivity across diverse environments and genetic backgrounds. Second, even dominant dwarf genes should be evaluated in hybrid combinations rather than inbred lines alone, because heterosis and epistasis can mask or modify dwarfing effects. Third, the genetic background should be considered: a major dominant dwarf gene provides the primary PH reduction, while polygenic modifiers compensate for yield penalties.

Multi-gene stacking to achieve short stalks without decreasing yield

In contrast to a single major dwarf gene approach, modern breeding increasingly treats semi-dwarfing as a platform trait that must be combined with yield-protecting and density-adaptive traits (Fig. 3–C). This is because strong dwarfing leads to yield penalties, while density tolerance depends on more than just shorter plants. For instance, *ZMM28* encodes a maize MADS-box transcription factor. Overexpression of *ZMM28* driven by a constitutive promoter (event DP202216) confers enhanced growth rate, increased photosynthetic capacity, improved nitrogen uptake and nitrogen utilization efficiency, and assimilatory capacity. Consequently, this led to stable yield increases across multiple environments without altering PH or flowering time. The combination of *D8* edits with DP202216 resulted in significant yield increase compared with *D8* edits alone. This complementarity arises because *ZMM28* boosts yield potential through enhanced source capacity, while *D8* edits reduce lodging risk by lowering PH, together addressing two independent limitations to high-density productivity (Wu et al., 2019; Fernandez et al., 2022).

Another practical ideotype development is to combine semi-dwarf and suitable leaf angle and shapes, where the dwarf alleles reduce lodging risk and the leaf trait alleles improve light penetration and radiation use efficiency at high density. A recent study presents a comprehensive genomics-guided breeding strategy for developing ideotype maize optimized for high-density planting (Yao et al., 2026). Through a combination of map-based cloning, GWAS, expression analysis, the authors identified and validated eight genes, including *ZmAIM1* and *ZmSINAT4* for leaf angle, *ZmCOL6* and *ZmPDS10* for both PH and ear height, *ZmKRP16* for PH, *ZmOXS3* and *ZmTAL1* for leaf width, and *ZmA3A1* for leaf length. By pyramiding favorable alleles of the eight genes, along with other genome-wide loci associated with the target traits, into parental lines of Yufeng303, a very popular hybrid in Northern China. They developed four new hybrids that are better adapted to high-density planting, and achieved 4.1–9.2% higher plot yields than Yufeng303 under high-density conditions. This work serves as a template for genes/genomics-informed hybrid improvement in maize, effectively bridging the gap between gene discovery and breeding gains.

Multiplex editing expressing multiple gRNAs in one transformation dramatically increases the probability of generating useful combinatorial genotypes. A key platform for multiplex CRISPR editing in plants is the tRNA–gRNA (tRNA-guided RNA) processing system (Xie et al., 2015), in which a single Pol III transcript is designed as a tandem array of tRNA–gRNA units. After transcription, the plant's endogenous tRNA maturation machinery (including RNase P and RNase Z) recognizes each tRNA segment and precisely cleaves at the tRNA boundaries, thereby releasing multiple functional gRNAs from one long precursor RNA. This design avoids building many separate U6:gRNA expression cassettes and provides a compact way to edit multiple genes in one transformation. In a maize implementation experiment (Qi et al., 2016), the authors optimized the architecture using maize glycine-tRNA as the processing element and demonstrated that the system worked robustly for multiplex mutagenesis, including expression cassettes containing up to four tRNA–gRNA units. Compared to single-gRNA strategies, the tRNA–gRNA system not only increased the number of target sites, but also enhanced mutagenesis efficiency.

Phenotypic prediction

As breeding shifts from single-gene manipulation to multi-allele stacks, classical marker-assisted selection becomes insufficient because many contributors are small-effect and background-dependent. Phenotypic prediction can be achieved through two methods: genomic selection (GS), which predicts breeding values using genome-wide markers, and machine learning (ML), which leverages multi-source data

(integrating genotypic, high-throughput phenotypic, and environmental information) to enhance predictive accuracy (Fig. 3–D). Phenotypic prediction can (i) guide parent selection, (ii) predict which allele combinations will perform optimally in specific environments, and (iii) increase field testing efficiency by evaluating only hybrids with higher predicted trait values.

Density tolerance is a complex trait that emerges from interactions among three key factors: plant architecture (e.g., PH, leaf angle), stress physiology (e.g., shade avoidance, lodging resistance), and environmental conditions (e.g., light quality, water availability, nitrogen status). Accurate prediction therefore requires models that capture both polygenic inheritance and genotype-by-environment (G × E) interactions. Methods that build kernels combining genomic relationships with environmental covariates can boost predictive ability and improve genetic gain within a given cost (Montesinos-López et al., 2022). In tropical maize, environment data-based kernels and optimized training-set strategies have been proposed for traits including PH and ear height (Gevartosky et al., 2023). Conceptually, robust selection needs models that generalize across those variables and factors because field performance under crowding is highly dependent on environment factors.

Conceptual model

We propose a conceptual model for dwarf gene deployment (Fig. 3–E). Taking *geneX* in the GA20ox family as an example, loss-of-function or weak *geneX* alleles (from mutation, RNAi, genome editing, or natural variation) reduce bioactive GA accumulation in internodes, suppressing intercalary meristem cell division and parenchyma cell elongation. On the one hand, the cellular-level changes reduce final plant height and lower the gravity center of the plant, which is the main determinant of stem lodging resistance under high planting density. On the other hand, the reduced plant height optimizes canopy structure, improves light penetration into the lower canopy layers, and reduces mutual shading between adjacent individuals under high planting density. These two synergistic effects collectively enhance the standability and yield stability under high planting density. The favorable *geneX* allele can be stacked with the *lac1* mutant allele. Plants carrying both alleles not only exhibit the favorable traits of reduced plant height and optimized canopy structure, but also retain the phenotype of the *lac1* mutant—a more upright leaf posture and smaller leaf angle. These traits further optimize the spatial distribution of light interception and increase the effective photosynthetic leaf area at the population level. Stacking multiple genes (such as *Zmm28*, *TD1,* etc.) allows the integration of their combined effects into GS or ML models, enabling accurate prediction of field performance. This model connects molecular regulatory mechanisms to the breeding goal of enhancing high-density tolerance, offering an actionable framework for the rational design of high-density tolerant ideotypes.

How dwarf genes are deployed mainly depends on the specific context. Precision editing is preferable for directly improving existing elite lines by creating tunable alleles of known genes and breaking linkage drag. Deploying dominant or semi-dominant genes enables the introduction of novel variation from diverse germplasm. Multi-gene stacking is suitable for building well-designed ideotypes where genes in multiple pathways must be modulated or pyramided simultaneously. Finally, phenotypic prediction allows breeders to select the best combinations of alleles for specific environments. The choice among these strategies should be informed by clear breeding objectives, the germplasm resources at hand, and insights into the underlying molecular genetics.

PERSPECTIVES

Elucidating fine-scale gene regulatory networks

Density-tolerant semi-dwarf breeding is related to multiple complex traits, each of which is regulated by many genes. Therefore, the next wave of progress will rely on establishing fine-scale gene regulation networks (GRNs) that connect (i) hormone pathway, (ii) shade-avoidance and light signaling, (iii) cell wall and vascular development for stalk strength, and (iv) reproductive sink formation for ear and kernel development. The smart canopy concept exemplifies the need for tissue- and cell-specific GRN resolution. In the *lac1* mutant, BR deficiency is not uniform but strongest in upper leaves due to differential regulation by *PHYA–RAVL1* interactions (Tian et al., 2019). This spatial specificity cannot be predicted from bulk transcriptome analysis.

Recent advances in single-cell (scRNA-seq) and spatial transcriptomics enable high-resolution dissection of gene expression. Spatial transcriptome

profiling of the developing ear and kernel links regulatory modules to reproductive sink traits that must remain robust under PH reduction (Wang et al., 2024; Fu et al., 2023). Single-cell and time-course meristem transcriptomes are revealing cell-level regulatory dynamics of inflorescence development and sex differentiation (Sun et al., 2024). Single-cell and spatial transcriptomics will be essential for mapping tissue-specific regulatory circuits and identifying nodes that can be tuned to optimize canopy architecture for high-density planting (Fig. 4–A). GRNs will become actionable when they can predict which genes or nodes can be tuned in internodes without sacrificing ear and kernel development, enabling tissue-specific stacking and rational selection of manipulation targets. The ideal targets should meet two criteria: (i) they function in specific tissues or developmental stages (e.g., internodes vs. ears), (ii) they have few paralogs or functional redundancy to minimize compensation effects.

AI-enabled precision in genome editing

Genome editing can already generate semi-dwarf alleles, but three bottlenecks remain: (i) generating edited populations and screening individual plants with desirable phenotypic traits requires a large number of editing events, (ii) editing may introduce off-targets and unintended changes, and (iii) combinatorial effects associated with multi-gene stacking should be evaluated in multi-environment trials (Jinek et al., 2012; Gehrke et al., 2018; Weers et al., 2024). These limitations can be addressed via AI-aided allele design.

AI is accelerating precision allele design by enabling both protein-level engineering and expression-level optimization (Fig. 4–B). At the protein level, by modeling three-dimensional structures and protein interactions, researchers can predict how specific amino acid substitutions alter protein function. For example, AlphaFold 3-guided editing of *D9* generated a mutant with reduced gibberellin sensitivity, achieving moderate dwarfism without yield penalties (Zheng et al., 2026). At the expression level, deep learning frameworks trained on epigenomic and expression data can predict how promoter variants affect transcription (Jaganathan et al., 2025), making it possible to design synthetic or edited promoters that drive expression in specific developmental contexts. Future breeding can integrate AI-driven protein engineering and expression-level optimization. By systematically evaluating whether

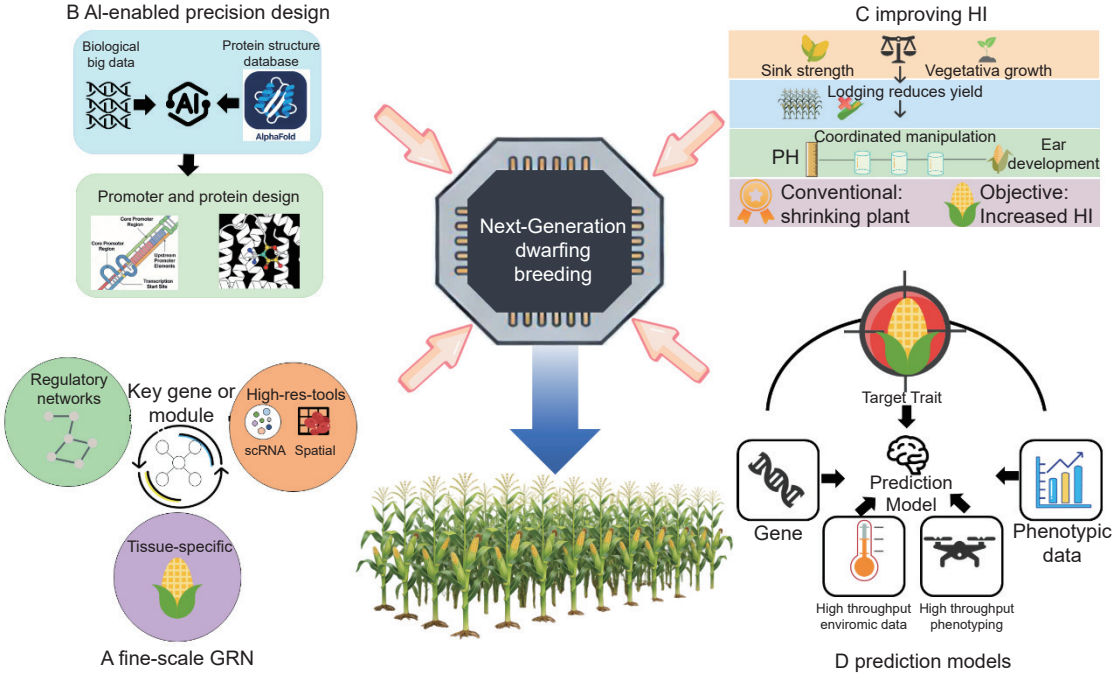

Fig. 4. Perspectives on maize density-tolerant breeding.
Four research directions can be focused: **A**. Elucidation of gene regulatory networks at cellular resolution; **B**. AI-enabled precision genome editing to minimize negative pleiotropic effects; **C**. Combining moderate dwarfism with stalk strength and robust ear development to improve harvest index; **D**. Phenotypic prediction incorporating genomic, phenomic, and enviromic data to forecast performance under high-density environments.

target genes are amenable to protein-level modification or expression-level tuning, multiple favorable alleles can be efficiently pyramided for elite crop improvement. AI can simulate the combinatorial effects of pyramiding multiple edited genes or regulatory network nodes (such as *GA20ox* governing plant height and *LAC1* controlling leaf angle) to predict field performance prior to material generation. This paradigm shifts breeding from trial-and-error gene stacking to rational, network-guided design.

Improving harvest index (HI)

A biological challenge for semi-dwarf, density-tolerant maize is avoiding trade-offs between ear sink strength and vegetative growth. Under high density, lodging can significantly reduce harvestable yield, so reducing plant and ear height can protect yield. However, if dwarfing influences reproductive development, the protected yield may still decline (Jin et al., 2023). Future progress will likely hinge on the coordinated manipulation of PH, lodging resistance, and ear development (Fig. 4–C).

Two practical trends support this direction. First, treating stalk strength and PH as equal targets without leading to yield reduction; transcriptomic studies are identifying pathways that can be stacked with PH alleles to reduce lodging (Xie et al., 2022). Second, nitrogen and density interact strongly with lodging and yield formation, so genetic gains must be validated under realistic N regimes. Field evidence indicates that optimized N management can improve culm mechanical strength and lodging resistance at high density (Ahmad et al., 2023). In summary, preferable multi-allele stacks will reduce plant and ear height and improve stalk biomechanics while maintaining yield performance, increasing HI under dense stands rather than simply shrinking the plant.

Phenotypic prediction by integrating high-throughput, continuous growth-related environmental factors

A key direction is incorporating real-time enviromics and high-throughput phenomics into prediction models, moving from "G only" or "G × E as a black box" toward mechanism-informed, data-rich prediction. Enviromics moves beyond simple location descriptors to a comprehensive characterization of the environment (Fig. 4–D). For instance, rather than just "Location A", enviromics data would include real-time data of: (i) light quality and quantity: daily light integral and the ratio of red to far-red light (R:FR). (ii) thermal time: growing degree days. (iii) water stress indices: cumulative precipitation, evapotranspiration, and soil moisture. (iv) soil characteristics: texture, pH, organic matter, and nutrition availability (Ahmad et al., 2023). These high-dimensional enviromic data can be structured into environmental covariance matrices or environmental kernels (Resende et al., 2025), enabling ML models to capture complex, nonlinear relationships among environmental factors, genetic markers, and phenotypes.

The advancement of real-time, high-throughput phenotyping (HTP) technologies, including unmanned aerial vehicles (UAVs), ground-based robots, and spectral sensors, has reshaped data collection by generating continuous, time-resolved phenotypic measurements throughout the growing season (Song et al., 2021). Integrating these rich HTP data with genomic information can enhance prediction accuracy and accelerate breeding decisions. The key challenge is how to integrate phenomic data into predictive models. A common strategy is to reduce dimensionality using functional data analysis (Vieu, 2018), which summarizes temporal variation into a few scores that serve as input for prediction models. Another strategy is phenomic prediction, where HTP-derived traits are used as inputs in a standard GS model to directly predict target traits. This approach has been shown to achieve comparable or even higher prediction accuracy than genomic prediction for traits like grain yield (Zhu et al., 2022). When both phenomic and genomic predictions are combined via weighting, accuracy improves further (Jackson et al., 2023). Together, these advanced approaches align with the "genomics–phenomics–enviromics triangle" concept (Crossa et al., 2021), in which predictive accuracy depends on optimizing all three data types within an integrated framework.

Synthesizing these perspectives, we propose that a future maize breeding paradigm for density tolerance can integrate four elements: (i) GRN-guided target prioritization, (ii) AI-empowered precision editing to create allelic series, (iii) stacking favorable alleles, whether from natural variation or genome editing, to enhance HI, and (iv) phenomics–enviromics–genomics prediction to optimize selection across environments. The ultimate goal is ideotypes with moderate stature reduction, enhanced lodging resistance, optimized source-sink relationships, and stable high yield under dense stands. Achieving this will require sustained collaboration among molecular

geneticists, breeders, physiologists, and computational biologists.

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DECLARATION OF COMPETING INTEREST

The authors declare no conflicts of interest.

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