From Ear Development to Density Adaptation: Molecular Regulatory Networks and Genetic Improvement of Ear Traits in Maize

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Abstract: Maize, a crucial global food and feed crop, requires continuous yield improvement to ensure food security. High-density planting has become a core strategy for maize yield improvement. While modifying plant architecture to enhance density tolerance in maize has been well reviewed, the regulatory mechanisms of ear development under high-density conditions remain far less clear. Therefore, this review aims to deepen the understanding of the mechanisms regulating ear traits, providing key insights into how ear architecture adapts to high-density planting. It presents the ideal ear architecture for high-density planting and offers a comprehensive overview of current morphological, genetic, and molecular insights into ear development. Furthermore, this review systematically analyzes the functions, breeding implications, and high-density regulatory roles of key ear-trait regulators and associated physiological determinants. Finally, prospective research directions are outlined, focusing on regulatory networks, multi-omics integration, ideotype design, and precision breeding to guide the coordinated improvement of density tolerance. This review not only advances the mechanistic understanding of maize ear morphogenesis but also provides a theoretical framework for breeding elite maize adapted to a high-density cultivation system.

Key words: maize; ear architecture; high-density planting; ear development

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INTRODUCTION

Maize is one of the world's most important food and feed crops, and sustained yield improvement is essential for ensuring global food security (Tian et al., 2024). High-density planting is widely regarded as the optimal strategy to maximize maize yield (Yao et al., 2026; Zeng et al., 2025; Wang et al., 2025). However, once the planting density exceeds a critical threshold, plants perceive a decreased red:far-red ratio and subsequently activate a set of adaptive responses collectively termed the shade avoidance syndrome (SAS) (Warnasooriya and Brutnell, 2014; Franklin, 2008; Ruberti et al., 2012). These responses are commonly manifested as increased plant height, an extended anthesis-silking interval (ASI), reduced tassel and ear size, decreased grain number per ear, and even complete barrenness (Franklin, 2008; Keuskamp et al., 2010; Cui et al., 2015; Zhang et al., 2022). Therefore, crucial strategies for adapting maize to high-density planting include optimizing plant architecture, moderately reducing plant height, improving yield components, and enhancing resistance to biotic and abiotic stresses to boost population yield. Notably, ear traits are the most density-sensitive yield components (Wang et al., 2026; Xiao et al., 2025; Sheng et al., 2025; Jafari et al., 2024). Hence, a density-tolerant ear architecture is not merely a morphological target but a core determinant of yield stability in dense planting,

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making its genetic improvement key to breaking yield bottlenecks in maize. Our recent studies reveal marked differences in sensitivity to high-density stress among maize inbred lines. While sensitive lines exhibit poor grain filling, barren ear tips, and shorter ears, low-sensitivity lines maintain stable ear morphology under high planting densities (Fig. 1), indicating extensive natural diversity and complex genetic regulation underlying ear plasticity in response to dense planting.

Reviewing the regulatory mechanisms underlying density-tolerant ear architecture in maize has important implications for maize breeding. Decoding the genetic basis of density-tolerant ear architecture is essential for transforming maize breeding from empirical selection to precision design. The formation of a density-tolerant ear architecture reflects adaptive responses to high-density stress via fine-tuned development of tassels and ears, representing a typical morphological and physiological adaptation to SAS (Casal, 2013). Systematic investigation of this process will fill the knowledge gap in the cascaded regulatory mechanism linking population stress and individual development.

The establishment of maize ear architecture is a complex biological process involving the spatiotemporal coordination of tassel and ear differentiation, organ morphogenesis, and dynamic responses to environmental signals (Wang et al., 2024b). This process is precisely regulated by multiple layers of factors, including intrinsic genetic and molecular networks (e.g., key genes, hormone signaling) (Kong et al., 2023) and external environmental cues (e.g., light and nutrient stress) (Yu et al., 2026). To date, although some regulatory genes and pathways have been identified, our understanding remains unsystematic and fragmented, with three major shortcomings in current research: first, insufficient understanding of the spatiotemporal coordination between tassel and ear development under dense planting; second, a predominant focus on individual genes or single layers, lacking systematic integration of genetic basis and environmental interactions; third, a definition of "ideal density-tolerant ear architecture" that remains at the level of superficial morphological description, lacking a quantitative index system linked to physiological function and molecular mechanisms.

Given the above research status and key scientific questions, this review aims to re-evaluate the central role of density-tolerant ear architecture in high-density, high-yield maize production and to systematically integrate recent advances in related fields. First, we elaborate on the structural features and physiological development of the maize inflorescence, analyzing its response patterns under dense planting stress. We then summarize the genetic basis and molecular networks regulating ear development, and discuss the effects of environmental interactions. Furthermore, we review improvement

Fig. 1. Typical phenotype of different maize inbred lines under high-density planting.
Ears from three maize inbred lines are shown to illustrate their differential sensitivity to high-density planting. Typical stress phenotype under high-density conditions include poor or no grain filling (Inbred A), tip-barrenness (Inbred B), and slightly reduced ear size (Inbred C). Planting densities were 67 500 plants ha^{-1} (normal) and 135 000 plants ha^{-1} (high-density). Scale bars: 5 cm.

strategies for density-tolerant ear architecture based on both traditional breeding and modern biotechnology. This review serves to inform the mechanistic study of complex traits and to advance the molecular breeding for high-yielding, density-tolerant maize for high-density cultivation system.

Development of Maize Inflorescences and Their Roles in Shaping Ideal Ear Architecture Under High-Density Planting

Maize inflorescence development

Maize is a typical monoecious species that produces physically separate male (tassel) and female (ear) inflorescences. The tassel forms at the plant apex as a branched panicle, whereas the ear develops as a lateral branch in the leaf axil several nodes below the tassel and is an unbranched spadix. Despite their distinct morphologies, both inflorescences share a common early developmental program. A defining feature of the ear is the absence of branch meristem (BM) formation. This inherent determinacy establishes a compact architecture, which is a fundamental prerequisite for stable kernel set and yield under high-density planting.

Both inflorescences originate from a common developmental precursor, the inflorescence meristem (IM). In tassels, the IM is established when the shoot apical meristem (SAM) undergoes the floral transition (namely vegetative-to-reproductive transition) (Bäurle and Dean, 2006; Poethig, 2010). The SAM maintains a pool of totipotent and indeterminate stem cells that generate aboveground organs, including inflorescences, leaves, and stems. In ears, the IM is derived from an axillary meristem (AM), and its size and activity are critical determinants of final ear architecture. Specifically, the length and width of the ear IM directly influence ear length (EL) and kernel row number (KRN), respectively, thereby contributing to yield potential. During the development of maize inflorescence, the IM in tassels generates BMs, which further produce additional spikelet pair meristems (SPMs) along their flanks as well as from the IM or BMs, whereas the IM in ears directly produces SPMs. Each SPM forms two spikelet meristems (SMs), and each SM gives rise to two floral meristems (FMs), which develop into two florets (upper floret meristem, UFM; lower floret meristem, LFM) enclosed by glumes and later surrounded by lemma and palea (Fig. 2). Subsequently, sex differentiation diverges between tassels and ears. In tassel spikelets, stamen primordia preferentially develop into functional anthers, whereas the pistils of both florets undergo successive abortion after initiation, resulting in exclusively staminate flowers (Cheng et al., 1983). Conversely, in ear spikelets, the pistil primordia of the UFM rapidly develop into a functional carpel. Meanwhile, the stamens of the UFM and the pistil of the LFM gradually abort, ultimately producing a single pistillate flower (Cheng et al., 1983; Calderon-Urrea and Dellaporta, 1999; Tanaka et al., 2013; Li and Liu, 2017). Interestingly, simulated shade has been shown to accelerate floral transition and meristem progression in both tassels and ears (Fig. 2). This results in earlier anthesis and fewer tassel branches, but it also delays silking time due to a slower silk elongation rate, ultimately extending the ASI (Kong et al., 2023).

The anthesis-silking interval is a critical trait for ideal ear architecture

At flowering time, maize silks must elongate rapidly to emerge from the husks and intercept pollen released from the tassels. Delayed floret growth prolongs silk emergence, thereby increasing the ASI (Tang et al., 2023; Liu et al., 2021a; Bolaños and Edmeades, 1996). Under optimal conditions, commercial maize cultivars exhibit a short ASI, reflecting tight coordination between tassel and ear flowering. However, under stresses such as drought, shading, or high-density planting, the ASI of these cultivars increases significantly (Edmeades et al., 2000). This asynchrony reduces pollination efficiency and kernel set, weakens pollen competition, and limits assimilate allocation to developing florets, often resulting in barren ear tips, kernel gaps, and variable kernel size (Zhang et al., 2022). ASI is widely used to evaluate reproductive synchrony and density tolerance, with modern varieties showing smaller ASI increases and greater stability under elevated planting densities compared to older germplasm (Yao et al., 2026). Although numerous ASI-related quantitative trait loci (QTL) have been identified (Almeida et al., 2012; Khan et al., 2022; Lima et al., 2022; Fuad - Hassan et al., 2008; Turc et al., 2016), only a few underlying genes have been cloned. Transcriptomic analysis revealed that genes involved in cell growth and expansion regulate the ASI. Among these, *EXPANSIN4* plays a key role in regulating ASI (Liu et

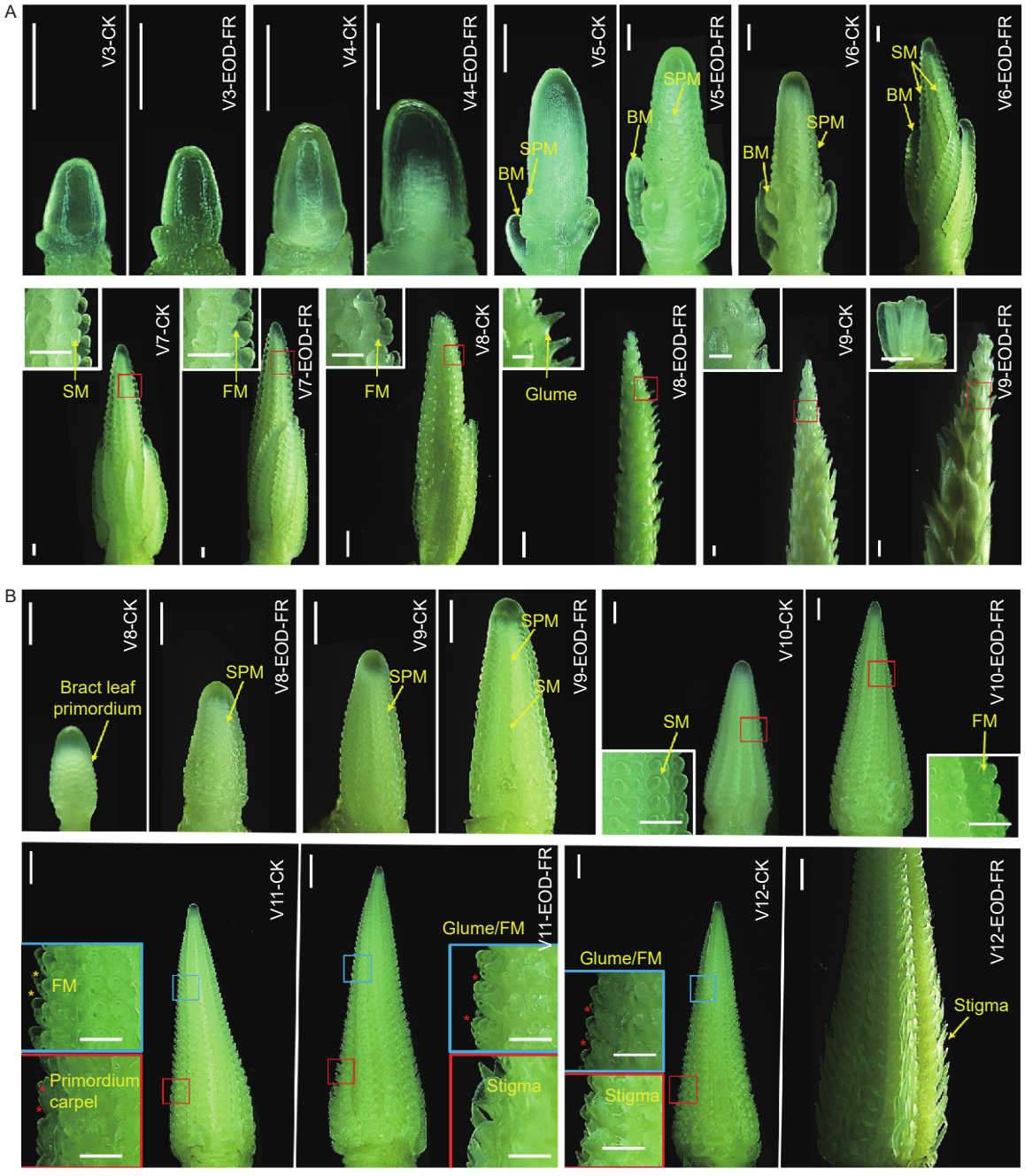

Fig. 2. Morphology of early maize tassel and ear development under simulated shade.
A. Stereomicroscope images of shoot apices (developing tassels) from vegetative stages V3 to V9, comparing control (left) and simulated shade-treated (right) plants, demonstrating precocious development upon shading. Scale bars: 300 μm (V3–V6); 500 μm (V7–V9); 300 μm (insets). **B**. Stereomicroscope images of developing ears from stages V8 to V12, comparing control (left) and simulated shade-treated (right) plants, showing accelerated ear development under shade. Scale bars: 500 μm (V8–V10); 1000 μm (V11–V12); 250 μm (insets). SAM, shoot apical meristem; IM, inflorescence meristem; SPM, spikelet pair meristem; BM, branch meristem, SM, spikelet meristem; FM, floral meristem. CK denotes the control (check) under normal light conditions; EOD-FR indicates end-of-day far-red light treatment, representing simulated shade conditions. Images are adapted from Kong et al. (2023).

al., 2021a). Increasing *ZmEXPA5* expression in transgenic maize decreases ASI and improves grain yield (Tao et al., 2023). Thus, ZmEXPs function as critical regulators of silk elongation. In addition, under shade stress, detasseling can shorten the ASI, enhance ear growth, and improve kernel number by redirecting assimilates to the ear (Gao et al., 2020). Nonetheless, the detailed developmental processes and mechanisms underlying ASI remain largely unclear. Overall, coordinated regulation of meristem

maintenance, vegetative-to-reproductive transition, IM activity and differentiation, floral development, and tassel–ear synchrony is essential for achieving stable and high maize yields.

An ideal density-tolerant ear architecture

Based on current density-tolerant maize germplasm and modern maize production demands (Chen et al., 2024; Wang et al., 2025), the ideal ear architecture for high-density planting should integrate several key traits: (I) a medium-sized, cylindrical ear with determinate inflorescence architecture, minimizing tapering and ensuring uniform kernel development; (II) a high kernel row number (18–20 rows) with deep flint-type kernels, strong grain-filling capacity, and a high shelling percentage; (III) a short and stable ASI under dense and stressful conditions to maintain reproductive synchrony; (IV) resistance to major ear rot pathogens to preserve grain quality and minimize mycotoxin contamination; and (V) rapid post-maturity moisture loss to facilitate direct kernel harvesting and lower drying costs. Collectively, optimal density tolerance arises from the precise integration of compact plant architecture, efficient grain filling, reproductive stability, rapid post-maturity moisture loss, and robust stress resistance.

Mechanisms of Inflorescence Development and Regulation in Maize

The development and differentiation of inflorescences determine floret number and fertility, ultimately contributing to maize yield. The key stages of inflorescence development encompass meristem activity, floral transition, IM stability and differentiation, and floral development. The size of the IM is closely associated with key ear traits, such as kernel row number and kernels per row (Wang et al., 2024a; Bommert et al., 2013a; Somssich et al., 2016). Meristem maintenance requires a precise balance between stem cell proliferation and differentiation (Zhang and Yuan, 2014). The genetic control governing the maintenance and development of the maize inflorescence is a highly coordinated process. It involves a complex network that includes the WUSCHEL–CLAVATA (WUS–CLV) feedback loop, specific transcription factors, the homeostasis of key phytohormones (auxin and cytokinin), and multiple signaling pathways, all of which collectively determine ear architecture and yield potential (Somssich et al., 2016; Kitagawa and Jackson, 2019; Chen and Gallavotti, 2021; Chaudhry et al., 2024; Du et al., 2022; Pautler et al., 2013).

CLV–WUS pathway in inflorescence meristem maintenance

The function of CLAVATA receptors is evolutionarily conserved (Bommert et al., 2005b; Kitagawa and Jackson, 2019). Numerous studies have shown that the canonical CLV–WUS negative feedback loop represents a key genetic mechanism for maintaining the stability of the SAM and IM (Table 1) (Bommert et al., 2013a; Bommert et al., 2005b; Je et al., 2016; Je et al., 2018; Kerstetter et al., 1997; Liu et al., 2021b). Key regulators include THICK TASSEL DWARF1 (TD1) and FASCIATED EAR2 (FEA2), the maize orthologs of Arabidopsis CLV1 and CLV2, respectively (Bommert et al., 2005a; Bommert et al., 2013b; Taguchi-Shiobara et al., 2001); the CLV-type leucine-rich repeat (LRR) receptor-like protein FEA3 (Je et al., 2016); and four potential CLV3-like signaling factors—FON2-LIKE CLE PROTEIN1 (ZmFCP1) and the CLAVATA3/EMBRYO-SURROUNDING REGION (CLE) peptides ZmCLE7, ZmCLE14, and ZmCLE1E5 (Je et al., 2016; Liu et al., 2021b). Meanwhile, specific receptor-ligand interactions have been characterized in maize. These include the direct pairs ZmFCP1–FEA3 and ZmCLE14–TD1, while ZmCLE7 and ZmFCP1 peptides bind to the multi-component complexes FEA2–CT2–ZmGβ and FEA2–ZmCRN, respectively (Du et al., 2022). *fea3* was epistatic to *bam1d* in the control of IM size (Lindsay et al., 2026). Mutations in CLV pathway genes lead to overproliferated IMs, resulting in fasciated ears with excessive kernel rows, disorganized kernels, and shorter cobs, which ultimately reduce yield (Je et al., 2018; Taguchi-Shiobara et al., 2001; Wu et al., 2019b; Kim et al., 2022).While the density tolerance of such mutants is uncharacterized, fine-tuning IM activity through targeted mutations in coding or regulatory regions of these genes represents a promising strategy. It can increase kernel row number without compromising ear architecture, potentially boosting yield (Li et al., 2022; Liu et al., 2021b; Je et al., 2016; Bommert et al., 2013b).

ZmWUS1 and *ZmWUS2* are the maize orthologs of Arabidopsis WUS. *ZmWUS1* is expressed in the organizing center (OC)-like region of the IM, while the expression pattern of *ZmWUS2* remains unclear (Nardmann and Werr, 2006; Je et al., 2016; Bommert

et al., 2005a). *ZmWUS1* functions in both meristem maintenance and AM initiation. Its essential role in regulating meristem size is demonstrated by the dominant maize mutant *Barren inflorescence3* (*Bif3*). *ZmWUS2* and other *WUSCHEL-RELATED HOMEOBOX* (*WOX*) family members may act

Table 1. Maize genes involved in regulating inflorescence architecture and ear morphology.

Gene name	Protein	Pathway	Mutant phenotype	References
TD1	Leucine-rich repeat receptor-like kinase protein	CLV-WUS	Fasciated ear	Bommert et al., (2005b)
FEA2	Leucine-rich repeat receptor-like protein	CLV-WUS	Fasciated ear	Bommert et al., (2013a)
FEA3	Leucine-rich repeat receptor-like protein	CLV-WUS	Fasciated ear	Je et al., (2016)
FEA4	bZIP-transcription factor	Auxin	Fasciated ear	Pautler et al., (2015)
ZmFCP1	CLAVATA3/ESR-related (CLE) peptide	CLV-WUS	Fasciated ear	Je et al., (2016)
ZmCLE7	CLE peptide	CLV-WUS	Fasciated ear	Liu et al., (2021b)
ZmCLE1E5	CLE peptide	CLV-WUS	Enhance grain yield per ear	Liu et al., (2021b)
ZmCRN	Pseudokinase	CLV-WUS	Fasciated ear	Je et al., (2018)
CT2	Ga subunit	CLV-WUS	Fasciated ear	Bommert et al., (2013b)
ZmGB1	Gβ subunit	CLV-WUS	Fasciated ear	Wu et al., (2019b)
ZmWUS1-B/Bif3	Homeobox-transcription factor	CLV-WUS; Cytokinin	Barren inflorescence	Chen et al., (2021)
KN1	Homeobox-transcription factor	Hormone	Inflorescence and floral defects	Kerstetter et al., (1997)
ZmGRX2				
ZmGRX5	Glutaredoxins	Modulate FEA4 activity	Triple mutant suppresses meristem growth	Yang et al., (2021)
MSCA1				
BA1	bHLH-transcription factor	Auxin	Barren stalk	Gallavotti et al., (2004)
BA2	Nuclear factor	Auxin	Barren stalk	Yao et al., (2019)
BAF1	Transcriptional regulator	Regulate *BA1*	Ear shoots absent or scanty	Gallavotti et al., (2011)
BIF2	Serine/threonine protein kinase	Auxin	Barren inflorescence	Skirpan et al., (2009)
BIF4	AUX/IAA-transcription factor	Auxin	Barren inflorescence	Galli et al., (2015)
BIF1	AUX/IAA-transcription factor	Auxin	Barren inflorescence	Galli et al., (2015)
SPI1	Flavin monoxygenase	Auxin	Reduced tassel branch number; small ears	Gallavotti et al., (2008)
VT2	Tryptophan amino transferase	Auxin	Barren ear; barren stalk	Phillips et al., (2011)
NDL1	ATP-dependent metalloprotease	ROS and Auxin	Reduced tassel branch number; small ears	Liu et al., (2019)
UB2	SBP-transcription factor	UB2/UB3/TSH4	Unbranched tassel	Chuck et al., (2014)
UB3	SBP-transcription factor	UB2/UB3/TSH4; Cytokinin	Unbranched tassel	Chuck et al., (2014)
TSH4	SBP-box transcription factor	UB2/UB3/TSH4	Defective tassel	Chuck et al., (2010)
GIF1	GRF-interacting factor	GIF1-GRF complexes	Fasciated inflorescence meristems	Zhang et al., (2018)
RA1	Zinc-finger transcription factor	RA3/RA2-RA1	Ear and tassel many-branched	Vollbrecht et al., (2005)
RA2	LATERAL ORGAN BOUNDARIES (LOB)	RA3/RA2-RA1	Tassel many-branched	Bortiri et al., (2006)
RA3	Trehalose-6-phosphate phosphatase	RA3/RA2-RA1	Branched inflorescence	Satoh-Nagasawa et al., (2006)

Continued Table

Gene name	Protein	Pathway	Mutant phenotype	References
REL2	Transcriptional co-repressor	REL2-RA1	Pleiotropic defects	Gregory et al., (2024)
KRN1/TS6	AP2 transcription factor		Defective tassel; tassel seed	Wang et al., (2019)
KRN2	WD40 protein	DUF1644-KRN2	Increased kernel row number	Chen et al., (2022)
KRN4		Regulat *UB3*		Liu et al., (2015)
KRN5b	Phosphatidylinositol 5-phosphatase	Phosphoinositide signaling	Disordered kernel rows	Shen et al., (2024)
ZmER1	LRR receptor-like serine/threonine-protein kinase	ER-EPFL signaling	Enlarged inflorescence meristems	Liu et al., (2026)
KNR6	Serine/threonine protein kinase	Auxin	RNAi lines had shorter ears	Jia et al., (2020)
ZmACO2	1-aminocyclopropane-1-carboxylate oxidase2	Ethylene	Increased ear length	Ning et al., (2021)
EAD1	Aluminum-activated malate transporter	ROS	Decreased ear length	Pei et al., (2022)
YIGE1	Unknown protein	Sugar and auxin	Decreased ear length	Luo et al., (2022)
YIGE2	Unknown protein	Auxin	Decreased ear length	Liu et al., (2024)

redundantly with *ZmWUS1* (Chen et al., 2021). Despite its importance, our incomplete understanding of the WUS regulatory pathway in maize necessitates further investigation into the functions of WUS homologs and their upstream regulators, especially under high-density planting. *KN1* is predominantly expressed in the vegetative meristem and directly targets genes involved in hormone pathways. Loss-of-function mutants display defects in meristem maintenance, resulting in reduced numbers of branches and spikelets (Kerstetter et al., 1997, Vollbrecht et al., 2000; Bolduc et al., 2012). REL2/RELK transcriptional corepressors regulate meristem size by modulating the CLV–WUS pathway and by maintaining hormone and redox homeostasis (Gregory et al., 2024; Robil and Tran, 2024). Heterozygosity at the *rel2* locus can significantly increase seed yield in certain hybrid combinations (Gregory et al., 2024), and similarly, heterozygous states of *ZmCRN* and *ZmCLE7* quantitatively increase KRN in both inbred lines and hybrids (Wang et al., 2024a).

Knowledge-driven genome editing has been successfully employed to create novel beneficial alleles, demonstrating that design-based genetic improvement is an effective strategy for manipulating ear traits (Li et al., 2025; Liu et al., 2021b). Future studies should prioritize elucidating how the WUS–CLV pathway interacts with light signaling to mediate shade-regulated inflorescence development under high-density planting. Within this framework, targeted editing of the CLV–WUS feedback loop represents a particularly promising approach for developing alleles that enhance yield by fine-tuning meristem size.

Hormonal regulation of inflorescence architecture

Phytohormones are essential for regulating inflorescence architecture (Barazesh and McSteen, 2008). Auxin plays a critical signaling role in initiating lateral organs and axillary meristems, as well as in determining floret number on the maize ear (Table 1). In maize, *SPARSE INFLORESCENCE1* (*SPI1*) and *VANISHING TASSEL2* (*VT2*) are the respective orthologs of two key Arabidopsis genes in the tryptophan-dependent auxin biosynthesis pathway: *YUCCA* (*YUC*) and *TRYPTOPHAN AMINOTRANSFERASE 1* (*TAA1*). Both *spi1* and *vt2* single mutants exhibit reduced free auxin levels and impaired initiation of AMs and lateral organs (Gallavotti et al., 2008; Phillips et al., 2011). Additionally, polar auxin transport, driven by specific influx and efflux carriers, establishes a heterogeneous auxin distribution within the meristem, creating local maxima that are essential for initiating lateral organs. Maize *BARREN INFLORESCENCE2* (*BIF2*) encodes a PINOID serine/threonine kinase that phosphorylates ZmPIN1a to regulate auxin redistribution. The *bif2* mutant exhibits suppressed initiation of both AMs and lateral primordia (Skirpan et al., 2009). Another potential regulator of polar auxin transport in maize is the *BARREN INFLORESCENCE1* (*BIF1*) gene. The

semi-dominant *Bif1* mutant shares phenotypic similarities with *bif2* (Barazesh et al., 2009; Skirpan et al., 2009). Additionally, several auxin signaling pathway genes have been reported to be involved in inflorescence development, including *BARREN STALK1* (*BA1*) and *BARREN STALK2* (*BA2*) in maize (Yao et al., 2019; Gallavotti et al., 2004). Several genes regulate *BA1* expression in maize, such as *Barren inflorescence2* (*Bif2*) and *Barren stalk fastigiate1* (*Baf1*). In particular, *Bif1* and *Bif4* are dominant alleles of Aux/IAA repressor genes, whose protein products interact with maize auxin response factors (ZmARFs) (Galli et al., 2015). BARREN STALK FASTIGIATE1 (BAF1) functions upstream of BA1 and plays a role in AM initiation. In *baf1* mutants, ears are either absent or develop abnormally as structures partially fused to the main stalk (Gallavotti et al., 2011).

Cytokinins (CKs) play a regulatory role in inflorescence development by promoting cell proliferation (Du et al., 2022; Chaudhry et al., 2024). *UNBRANCHED2* (*UB2*) and *UB3* are duplicated SBP-box transcription factor genes that redundantly control KRN formation. They function by limiting cell differentiation to the lateral domains of the IM through cytokinin biosynthesis and signaling pathways. Notably, maize *UB3*, a direct target of GIF1, modulates branching via cytokinin pathways. Together with *UB2* and *TSH4* (Chuck et al., 2010), it represses lateral primordia initiation, in part by directly targeting the promoters of cytokinin-related genes such as *LOG1* and type-A RESPONSE REGULATORs (*RRs*) (Du et al., 2017; Hake et al., 2020; Chuck et al., 2014; Zhang et al., 2018). Cytokinin establishes a positive feedback loop by inducing *WUS* expression through both CLV-dependent and CLV-independent pathways. This induction is mediated by type-B *RRs*, which are primary cytokinin-response transcription factors that bind directly to the *WUS* promoter to activate it (Kitagawa and Jackson, 2019). Notably, *ZmWUS1-B* overexpression is driven by its promoter containing multimerized type-B *RRs* binding sites. Ultimately, cytokinin hypersensitivity triggers stem cell overproliferation and reorganizes the *Bif3* inflorescence meristem, leading to ball-shaped ears and severe yield loss (Chen et al., 2021). Furthermore, studies indicate that WUS directly regulates additional cytokinin- and auxin-related genes (Kitagawa and Jackson, 2019; Zhao, 2010; Zhang and Yuan 2014). *ABPH1* encodes a cytokinin-inducible type-A *RRs*. It functions as a negative regulator of SAM size and a positive regulator of *PIN1* expression, revealing a complex interaction between auxin and cytokinin signaling during phyllotactic patterning (Giulini et al., 2004; Lee et al., 2009). Therefore, optimizing the cytokinin/auxin balance at specific growth stages presents a viable strategy to enhance crop yield by increasing grain number.

Role of physiological metabolites in inflorescence development

The dynamic changes of physiological substances during the formation of maize inflorescence involve coordinated shifts in carbon-nitrogen metabolism, metabolites, reactive oxygen species (ROS) and small molecules (Table 1). These can directly or indirectly reflect maize inflorescence development, and understanding them offers critical guidance for improving density-tolerant inflorescence architecture in maize. The experiments in maize cultivation show that carbon and nitrogen metabolism play important roles in regulating maize inflorescence development (Ge et al., 2008; Lai et al., 2026; Wang et al., 2026). Trehalose is the first identified carbohydrate that negatively shapes inflorescence architecture, especially its branching (Satoh-Nagasawa et al., 2006). In addition, malate is an important metabolite involved in shaping tassel and ear architecture. During ear inflorescence development, malate accumulates and modulates ROS production and cell proliferation, thereby influencing maize EL (Pei et al., 2022). High ROS levels perturb auxin homeostasis, leading to defects in reproductive organogenesis (Liu et al., 2019). The spatiotemporal accumulation of ROS, including superoxide anion (O^{2-}), hydrogen peroxide (H_2O_2) and hydroxyl radicals, can restrict meristem and floret development. Acting redundantly with ZmGRX2 and ZmGRX5, the maize CC-type glutaredoxin MSCA1 modulates the redox state and activity of the transcription factor FEA4, thereby regulating its function as a repressor of inflorescence meristem development (Yang et al., 2021). In addition, inositols, small molecules synthesized from the glycolytic metabolite glucose 6-phosphate and functioning as cytosolic solutes, have been shown to negatively regulate kernel number in maize ears (Shen et al., 2024).

Under high-density planting conditions, shade stress and resource competition alter the levels and

spatiotemporal distribution of key phytohormones (e.g., auxin, cytokinin) and the levels of essential metabolites (e.g., sugars, reactive oxygen species, inositols) within the developing maize ear. These changes directly affect meristem activity, floret initiation, silk elongation, and grain filling. However, these critical dynamics and their regulatory mechanisms have received limited attention and remain unclear. Therefore, elucidating how hormonal and metabolic reprogramming regulates ear plasticity under dense planting is essential for understanding the physiological and molecular basis of density tolerance and for breeding maize varieties with stable, high-yielding ear architecture adapted to high-density planting systems.

Genetic Dissection and Breeding Perspectives of Maize Ear-Related Traits

Maize ear architecture, a primary determinant of yield under dense planting, is a complex trait controlled by multiple quantitative trait loci (QTLs). Maize ear architecture is highly amenable to QTL analysis. Maize inbred lines exhibit substantial natural variation in key ear-related traits, including kernel row number, kernels per row, ear diameter, and kernel depth (Upadyayula et al., 2006; Li et al., 2018; Jiang et al., 2023). Several of these QTLs have been positionally cloned. The genetic basis and molecular regulation of KRN and EL, two key determinants of maize yield, have been systematically investigated (Table 1). While the functions of several cloned genes are well-characterized under standard conditions, a critical gap exists in understanding their specific roles and regulatory dynamics in response to high-density stress (Li et al., 2025).

Key genes regulating KRN

Several genes regulating KRN, a major yield component, show promise for breeding, such as *KRN1*, *KRN2*, *KRN4*, *KRN5b*, *ZmERECTA1* (*ZmER1*), and *qKRN5.04*. *KRN1* likely corresponds to *indeterminate spikelet 1* (*ids1*)/*Tassel seed6*, encoding an AP2-domain protein homologous to the wheat domestication gene *Q* (Wang et al., 2019b). *KRN2* is a key domestication gene encoding a WD40-domain protein. The maize allele of *KRN2* is favorable for KRN, and knockout of *KRN2* can increase KRN without compromising other agronomic traits, highlighting its potential for maize improvement (Chen et al., 2022). *KRN4* fine-tunes *UB3* expression by recruiting a UB2-centered transcriptional complex via chromatin looping (Hake et al., 2020; Liu et al., 2015). This *UB2/UB3* module is implicated in the shade avoidance response, suggesting *KRN4* may modulate meristem activity under light competition, offering a pathway to stabilize KRN in dense canopies. *KRN5b* encodes an inositol polyphosphate 5-phosphatase (5PTase) affecting kernel number by regulating the initiation and maintenance of reproductive axillary meristems (Shen et al., 2024). Given the role of inositol phosphates in stress signaling (Jia et al., 2019), *KRN5b* merits investigation for its potential in maintaining fertile florets under density stress. Additionally, mutants of *ZmER1* exhibit compact architecture, enlarged IMs, and increased KRN. The enlarged IM phenotype in *Zmer1* mutants is suppressed by mutations in *ZmWUS1*. The weak alleles of *Zmer1* have been associated with improved agronomic traits like leaf angle and KRN (Liu et al., 2026), positioning it as an integrative target for simultaneous improvement of plant and ear architecture.

Key genes regulating EL

Ear length is highly sensitive to density stress, often resulting in barren tips. Genes in the ethylene and auxin biosynthesis pathway emerge as critical regulators. *KNR6* encodes a serine/threonine protein kinase that regulates kernel number per row by controlling both floret number and EL. It functions in auxin-dependent inflorescence development by mediating the phosphorylation of an Arf GTPase-activating protein (Jia et al., 2020). Ethylene serves as a key signaling hormone involved in inflorescence development in maize. *ZmACS7* encodes 1-aminocyclopropane-1-carboxylic acid (ACC) synthase, a rate-limiting enzyme in ethylene biosynthesis. A gain-of-function mutation in *ZmACS7* leads to increased ethylene production, which reduces plant height, and decreased ear size and grain yield (Li et al., 2020a). Similarly, *ZmACO2* encodes ACC oxidase 2 (ACO2), which catalyzes the final step of ethylene biosynthesis and negatively regulates EL and grain yield. *ZmACO2* loss-of-function lines showed an increase in ear length by 6.8%−7.2% (Ning et al., 2021). Targeting these genes provides a strategy to mitigate the negative impact of density-accelerated ethylene synthesis on ear development. Beyond hormones, *EAD1* regulates maize ear development by mediating malate delivery to the apical regions of immature ears (Pei et al., 2022). *YIGE1* positively

regulates EL and grain yield by influencing IM length and floret number, likely through sugar and auxin pathways. Its paralog, *YIGE2*, also promotes ear development and EL via the auxin pathway. These two genes redundantly regulate IM development and EL (Luo et al., 2022; Liu et al., 2024). Their role in maintaining IM activity and floret survival under low light or nutrient competition warrants exploration.

Integrating genetic insights for density-tolerant breeding

The accrued knowledge reveals that many key ear trait genes, such as those in the *UB2/UB3* and ethylene pathways, are also hubs in light and stress signaling networks. This convergence provides a molecular basis for their high breeding value. The strategic manipulation of these genes—for example, using weak alleles (e.g., *ZmER1*), promoter engineering to fine-tune expression (e.g., *ZmACO2*), or knockouts to alleviate negative regulation (e.g., *KRN2*, *ZmACO2*)—enables the precision engineering of ear architecture. The future challenge and opportunity lie in systematically testing the effects of these genetic variants under high-density competitive environments and pyramiding them to achieve synergistic improvements in both constitutive yield potential and inducible stress resilience, ultimately accelerating the molecular design of maize ideotypes for high-density planting systems.

Molecular Insights and Challenges in Maize Ear Development Under High-Density Planting

Yield constraints under high-density planting

Increasing planting density is an effective strategy to enhance grain yield and biomass at the population level (Yan et al., 2024). However, high-density planting conditions often induce SAS, leading to limited water and nutrient availability, lower canopy and soil temperatures, and reduced light interception per plant. These constraints impair photosynthesis and grain filling, ultimately restricting individual plant performance and yield potential (Zhang et al., 2025; Pagano et al., 2007). Consequently, modern breeding programs increasingly prioritize population-level performance over individual plant traits. Under dense planting, kernel number and weight are the main determinants of yield. However, altered plant architecture frequently results in barren ear tips, primarily due to delayed ear development driven by strong SAS and prolonged ASI (Cui et al., 2015). In addition, reproductive asynchrony between pollen shedding and silking increases kernel abortion and barrenness (Wang et al., 2026). High density also reduces grain-filling rate and duration, decreasing kernel weight and promoting abortion in the upper ear, thereby leading to yield loss (Shah et al., 2021).

Light signaling and shade avoidance in inflorescence development

A major constraint on high-density tolerance in maize is its shade response, mediated by phytochromes (phys) and PHYTOCHROME-INTERACTING FACTORs (PIFs) (Warnasooriya and Brutnell 2014; Franklin 2008; Ruberti et al., 2012). In maize, there are six phytochrome genes, *ZmPHYA1*, *ZmPHYA2*, *ZmPHYB1*, *ZmPHYB2*, *ZmPHYC1*, and *ZmPHYC2*, that regulate photomorphogenesis and SAS (Sheehan et al., 2007; Sheehan et al., 2004; Wu et al., 2019a; Li et al., 2020b). Loss of *ZmPHYB1* and *ZmPHYB2* accelerates flowering, whereas overexpression of *ZmPHYB1* results in reduced shade responsiveness at the seedling stage and decreased plant and ear heights at the maturity stage (Zhao et al., 2022). However, the *Zmphyc1 c2* double mutant exhibits moderate early flowering under LD conditions and reduced shade sensitivity, while *ZmPHYC2* overexpression moderately reduces plant stature (Li et al., 2020b).

Recent transcriptomic analyses further show that simulated shade accelerates inflorescence development across all stages (Fig. 2). Key regulatory hubs, including *UB2*, *UB3*, and *TSH4*, likely function downstream of the ZmphyBs-ZmPIFs-ZmMIR156 signaling module to integrate light and hormone cues. Under shade conditions, genes involved in floral initiation (*MADS4*/*15*), branch meristem formation (*UB2*/*UB3*/*TSH4*/*ZFL1*/*ZFL2*/*SPI1*), spikelet pair meristem formation (*RA1*/*RA2*/*RA3*), and floral meristem formation (*ZAG1*) are expressed earlier (Kong et al., 2023; Vollbrecht et al., 2005; Bortiri et al., 2006; Satoh-Nagasawa et al., 2006). These findings highlight extensive crosstalk between light signaling, hormonal regulation, and developmental programs. However, the precise genetic regulatory network underlying maize inflorescence development under high-density planting remains unclear, and identifying key meristem regulators continues to be a major challenge.

The role of source–sink–transportation coordination in ear development

In addition to light signaling and the shade avoidance response, source–sink–transport coordination is a major constraint on high-density tolerance in maize. Photoassimilates produced in leaves are translocated to the ear and developing grains via the vascular bundle system, thereby supporting ear development and grain filling (Chang et al., 2017). However, high yield depends not merely on strong individual performance of the source, sink, or transport pathways, but on their coordinated interplay. An ideal plant architecture for dense planting should feature a compact plant with erect upper leaves to minimize shading of lower leaves and maintain high photosynthetic capacity, making leaf angle control a promising breeding target (Tian et al., 2024; Tian et al., 2019; Jafari et al., 2024; Sheng et al., 2025). In maize breeding, modern hybrids have displayed reduced relative ear height and upper leaf angles to adapt to increasing planting densities in China (Yao et al., 2026), indicating that the ideal plant architecture is undergoing refinement. High-density planting often induces leaf senescence. Therefore, research is needed to explain the mechanisms that delay leaf senescence, extend functional leaf longevity, and improve high-density tolerance and photosynthetic capacity (He et al., 2022; Zhao et al., 2025). Regarding sink strength, optimizing resource allocation requires minimizing unproductive sinks (e.g., barren stalks) and increasing productive ear number per unit area. High density frequently induces barren stalks and defective ear/tassel development, phenotypes reminiscent of *ba1*, *ba2*, and *baf1* mutants (Gallavotti et al., 2004; Yao et al., 2019), which are caused by weakened and unbalanced assimilate distribution. To counteract kernel abortion caused by insufficient sucrose supply under density stress, enhancing the activity of vascular sucrose transporters (e.g., SWEET and SUT families) represents a strategy to boost photoassimilate partitioning and kernel set. These arguments are supported by studies on carbohydrate-transport mutants, such as *ZmSWEET13a/b/c* and *ZmSUT1* (Yang et al., 2026; Baker et al., 2016; Braun, 2022; Julius et al., 2017; Ren et al., 2026; Bezrutczyk et al., 2018).

Perspective and strategies

Density-tolerant ear architecture is a core trait that coordinates the growth of individual plants and population structure to achieve high and stable yield under high-density planting systems. Considering the practical challenges confronting maize breeding and production—such as the trade-off between high density and inferior grain quality, increasing extreme climatic stresses, the limitations of fragmented basic research, and the mismatch between technological advances and breeding practice—future research should adopt an integrated, multi-dimensional strategy (Hultgren et al., 2025; Anderson et al., 2020; Shah et al., 2021). A comprehensive framework centering on in-depth mechanism elucidation, stress adaptation improvement, and breeding system optimization should be established to drive the research and application of density-tolerant ear architecture toward precision, coordination, and sustainability, thereby supporting the high-quality development of the maize industry.

Using integrated multi-omics, single-cell RNA sequencing, and spatial transcriptomic technologies, future studies should systematically dissect the spatiotemporal coordination of ear and tassel differentiation and development under high-density shade stress, clarify the relationships between ear morphological structure and physiological function, and identify critical physiological targets and molecular modules governing shade tolerance and kernel abortion resistance (Wang et al., 2024b; Sun et al., 2024). Focusing on the bottleneck of "high density but inferior quality," further investigation should explore the genetic linkage and coordinated regulatory networks between density-tolerant ear architecture, lodging resistance, and disease resistance, with the goal of identifying key genes and QTLs that simultaneously control density tolerance, stress resistance, and grain quality (Liu et al., 2025). Furthermore, future research should focus on the physiological responses and molecular regulatory mechanisms of maize ear development under combined high-temperature and high-density stresses, identify key functional genes conferring tolerance to combined stresses, and clarify the regulatory pathways underlying heat tolerance in ear morphology and physiology. Clarifying the interactive effects of key environmental factors under stress across regions, developing region-specific cultivation systems, and ensuring yield stability under high density by optimizing genotype–management–environment synergy are critical steps forward (Zhang et al., 2020;

Edwards 2016).

Ultimately, interdisciplinary integration should be strengthened to establish an ideal integrated model of plant and ear architecture, and to optimize the modern breeding system. The realization of genuine density tolerance relies on the synergistic interaction between plant architecture (plant height and leaf morphology) and ear architecture (ear structure, developmental characteristics) (Xiao et al., 2025; Jafari et al., 2024). Improvement targeting only ear morphology can hardly fully exploit the yield potential under dense planting. Future efforts should integrate knowledge of ear and tassel differentiation, genetic and molecular regulation, and environmental responses to define the optimal parameters of ideal plant and ear architecture under varied density conditions and ecological regions, and to construct an integrated plant-architecture and ear-architecture ideotype system. Meanwhile, deep integration of modern breeding tools, including marker-assisted selection, gene editing, haploid-inducer mediated genome editing, and genomic selection, with mechanistic research will optimize breeding strategies for density-tolerant ear architecture and establish standardized evaluation systems (Li et al., 2022; Tian et al., 2024; Chen et al., 2022; Escamilla et al., 2025; Yao et al., 2026; Wang et al., 2019a). These efforts will promote the transition of maize density-tolerance breeding from empirical selection to precision design, and accelerate the development and deployment of elite maize varieties with high density tolerance, high yield, and wide adaptability.

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DATA AND CODE AVAILABILITY

Data sharing not applicable to this article as no data sets were generated or analyzed during this study.

DECLARATION OF COMPETING INTEREST

The authors declare no conflict of interest.

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