Biobreeding

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Review

Rose Genomics Research Associated with Important Agronomic and Ornamental Traits for Biobreeding: Progress, Challenges and Prospects

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ABSTRACT: Rose is a globally significant ornamental crop and an emerging genomics system for woody ornamental plants. In recent decades, the rapid evolution of high-throughput sequencing and robust technical platforms has yielded high-quality reference genomes and been propelling our understanding of rose biology to unprecedented depths. This review systematically synthesizes the current landscape of rose genomics, recent development of technical and resource platforms, and the molecular mechanisms underlying pivotal agronomic traits such as floral development, scent biosynthesis, petal lifespan, and stress resilience. Despite these strides, high genome heterozygosity, polyploidy, and recalcitrance to genetic transformation remain significant barriers. We discuss how integrating cutting-edge technologies, such as pangenomics, single-cell transcriptomics, AI-assisted genomic selection, and precise CRISPR-based editing, can bridge the gap between fundamental research and practical applications. Collectively, this review provides a strategic roadmap for accelerating the development of next-generation rose cultivars through trait-based biobreeding.

Keywords: Rose (*Rosa*); Functional genomics; Technical and resource platforms; Agronomic trait genes; Gene editing

1. Introduction

Roses (*Rosa* spp.), often celebrated as the “queen of flowers”, hold immense symbolic, cultural, and economic value worldwide, serving as a primary species for landscaping, cut flowers, scent production, and culinary applications [1]. Recently, the rose has emerged as a model system for woody ornamental plant genomics due to several unique biological and technical advantages. It possesses a moderately small

genome and an exceptionally rich germplasm comprising over 35,000 registered cultivars, which provides a vast reservoir of phenotypic variation [2–4]. Furthermore, the genus exhibits a complex evolutionary history characterized by interspecific hybridization and polyploidization [5,6].

The last decade has witnessed rapid advances in rose genomics, driven by the availability of high-quality, haplotype-resolved, and telomere-to-telomere (T2T) gapless, pangenome assemblies [7–10]. Coupled with available extensive genetic diversity resources and the development of efficient genetic transformation and CRISPR-based gene-editing systems, researchers are now equipped to dissect complex trait architectures [11–13]. Currently, rose genomic studies primarily focus on the construction of high-resolution genomic resources and transformation platforms, the functional analysis of key agronomic traits, and the characterization of underlying regulatory networks. In this review, we synthesize recent progress across these domains, presenting key findings that carry significant implications for genetic improvement. We also explore future prospects, highlighting the potential of these genomic insights to drive precision molecular breeding strategies in ornamental horticulture.

2. The Scope of Rose Genomics Research

The scope of rose genomics is defined by the need to decode an exceptionally complex genome to understand the molecular basis of key ornamental traits and enable precision genetic improvement. Modern cultivated roses (*Rosa* cvs.) primarily consist of the hybrid species, which emerged from independent domestication processes in both China and Europe [4]. The genus is characterized by reticulate evolution, featuring interspecific hybridization, introgression, and various levels of polyploidy, with most contemporary cultivars being tetraploid [14]. These evolutionary complexities have long posed substantial challenges for genetic analysis and breeding.

In response to these challenges, recent advancements in sequencing technologies and bioinformatics have greatly enhanced our ability to unravel the genetic makeup of roses. Following the trajectory of major cereal crops, the rose has achieved significant genomic milestones. The release of the first high-quality rose genome marked a turning point [1,15], and subsequent advances have led to the development of haplotype-resolved and telomere-to-telomere (T2T) gapless assemblies [8,16]. Comprehensive pangenomic analyses of roses have further uncovered widespread structural variation across the genus [17]. Together, these high-resolution maps provide the foundational framework for precise gene mining, annotation, and the identification of regulatory elements across the genome.

Built upon this robust genomic foundation, the primary scientific goal of rose genomic research is to understand how the complex, often highly heterozygous genome orchestrates plant development and produces the diverse phenotypes exhibited in over 35,000 registered cultivars [3]. This involves deciphering the information encoded in DNA sequences to reveal the molecular mechanisms underlying key ornamental traits, such as floral morphology, pigmentation, and fragrance biosynthesis, as well as developmental processes and responses to abiotic and biotic stresses.

An equally important goal is the translation of these findings into practical genetic improvement. By integrating germplasm mining with precision tools such as CRISPR-based gene editing and molecular design breeding, functional genomics provides the theoretical and technical support necessary to transition from traditional phenotypic selection to precision genetic engineering in ornamental horticulture. In essence, the scope of rose genomics research encompasses both fundamental discovery and its application, aiming to overcome evolutionary complexity through modern genomic approaches and ultimately to accelerate the breeding of superior rose cultivars.

3. Recent Development of Technical and Resource Platforms for Rose Genomics Research

Integrated technical platforms, encompassing genomic resources, genetic transformation, and gene editing, serve as the critical infrastructure for rose genomic studies. Recent collaborative efforts have yielded significant breakthroughs, providing a robust framework for elucidating gene–trait relationships at molecular resolution.

3.1. De Novo Assembly and Refinement of Rose Reference Genomes

A high-quality reference genome forms the foundation for deciphering the genetic basis of complex traits. Modern cultivated roses are mostly tetraploid hybrids that arose from intensive interspecific crosses between Chinese and European wild species and old cultivars, initiated in the eighteenth century and culminating in the first modern rose 'La France' in 1867 [2]. These roses combine key ornamental traits such as continuous flowering, diverse colors, and fragrances inherited from both eastern and western progenitors [4]. The rapid development of sequencing technologies has transitioned rose genomics from foundational diploid assemblies to comprehensive pangenome and haplotype-resolved frameworks.

In 2018, the first high-quality assemblies of the doubled haploid 'HapOB' provided critical insights into the mosaic domestication of modern roses and enabled the mapping of loci for continuous flowering and prickle density [1,15]. Recent efforts have advanced to haplotype-resolved and T2T assemblies. The haplotype-resolved genomes of the ancient diploid 'Chilong Hanzhu' [16] and the modern tetraploid 'Samantha' [7], one of the few phased chromosome-scale polyploid plant genomes reported so far, revealed frequent homoeologous exchanges. Genome analysis combined with a genome variation map of 233 *Rosa* accessions pinpointed the ancestral contributions of *R. chinensis* 'Old Blush' and *R. odorata* to modern roses and identified selective sweeps associated with continuous flowering, double flower, flower senescence, and disease resistance. This work provided an unprecedented resource to reconstruct the history of rose divergence, hybridization, and breeding. The pursuit of gapless genomes yielded the complete T2T assembly of *R. gigantea*, elucidating the expansion of phenylpropanoid biosynthesis networks that contribute to its unique tea scent [8], alongside a gap-free reference for *R. persica* that facilitates the use of wild genetic resources [9].

Moving beyond single reference genomes and addressing the scarcity of pangenomic resources for the *Rosa* subgenus, which comprises over 200 species, Zhang et al. constructed the first haplotype-resolved rose pangenome integrating 26 representative germplasms. These comprehensive resources captured the subgenus-wide genetic diversity and mapped over 1.7 million presence/absence variations. The reported structural variations (SVs) explain 'missing heritability' for core ornamental traits, such as an inversion-mediated *RcKSN* deletion for continuous flowering and transposable element-driven SVs modulating floral doubling and petal color [17]. Extensive introgression among intersectional accessions revealed by the pangenome further underscores the potential of cross-sectional breeding strategies.

Despite these milestones, several critical gaps remain. First, most reference genomes are derived from a narrow genetic background (e.g., 'HapOB' and 'Samantha'), which may not capture the full allelic diversity of polyploid cultivars. The bias toward diploid or doubled-haploid representatives, while technically convenient, risks overlooking haplotype-specific expression and homoeologous exchange patterns that are central to tetraploid rose biology. Second, functional annotation of SVs lags behind their discovery: most identified SVs are associated with traits via linkage rather than causal validation. Third, the complexity of polyploid subgenome coordination, such as homoeologous exchange frequencies and their phenotypic consequences, has only begun to be explored. The 'Samantha' genome revealed frequent exchanges, but how these vary across cultivars or affect trait stability is unknown. Future research may therefore prioritize: (i) generating haplotype-resolved assemblies from a phylogenetically diverse set of wild and cultivated polyploids; (ii) developing scalable SV-to-phenotype validation pipelines, including

CRISPR editing in polyploid backgrounds; (iii) integrating pangenomic data with spatial and temporal transcriptomes to dissect regulatory networks of SVs; and (iv) building user-friendly platforms for breeders to query SVs associated with target traits.

3.2. Genetic Transformation Platforms

Reliable genetic transformation is the cornerstone of functional validation and molecular breeding, yet its establishment in roses has historically been restricted by severe genotype dependency and recalcitrant regeneration [18]. Current methodologies predominantly utilize *Agrobacterium tumefaciens*-mediated gene delivery coupled with somatic embryogenesis, which has been refined through virulence gene augmentation (e.g., *virE*/*virG*) to achieve stable transformation in cultivars like 'Tineke' and 'Samantha' [11,12,19].

In parallel, microprojectile-mediated biolistic transformation has been applied to the rose genetic transformation as a complementary tool [20]. However, this technique faces substantial drawbacks, including high equipment costs, technical complexity in parameters optimization (e.g., microprojectile distance), frequent physical damage to target tissues that reduces cell viability, and a pronounced tendency for multi-copy transgene integration, which often leads to low transformation efficiencies [21]. Notably, only one rose cultivar, *Rosa hybrida* 'Glad Tidings', has reported multiple successful biolistic transformation events [20].

Agrobacterium tumefaciens-mediated stable genetic transformation is convenient for gene function analysis. However, the current procedure for stable genetic transformation is time-consuming because of the two-step induction of somatic embryos and difficult shoot regeneration [12,22]. Moreover, a large input of materials and labor costs is required due to low transformation efficiency (less than 10%). Third, only a few varieties among the 35,000 rose cultivars have been successfully transformed. To bypass these bottlenecks, *Agrobacterium rhizogenes*-mediated hairy root transformation systems (HRTS) have emerged as highly efficient alternatives. Recent optimized HRTS protocols utilizing strains K599 or MSU440 have achieved transformation efficiencies up to 74.1% (the rate of transgenic roots) within 30 days across diverse diploid and tetraploid cultivars [23,24].

Despite the high efficiency of root transformation, it remains challenging to induce adventitious bud formation, either directly or indirectly, from hairy roots in roses, necessitating further investigation. First, root tissues are already committed to a differentiated state; their cell fates are less plastic than those of meristematic or juvenile tissues (e.g., leaf explants or somatic embryos). Re-programming differentiated root cells toward shoot organogenesis requires specific developmental regulators (such as *BBM* or *WUS*) that are typically not activated by standard HRTS protocols. Second, the *A. rhizogenes* Ri plasmid, which carries *rol* genes (e.g., *rolA*, *rolB*, *rolC*), integrates into the plant genome and disrupts endogenous hormone homeostasis—specifically, altering auxin/cytokinin ratios and promoting root proliferation while suppressing shoot formation. This inherent property of Ri T-DNA makes whole-plant regeneration from hairy roots exceptionally difficult in most dicot species, including roses. Consequently, although HRTS is a powerful tool for rapid *in vivo* functional validation in roots, it cannot serve as a direct route for generating stable transgenic shoots. This explains the persistent gap between root transformation efficiency and whole-plant regeneration success.

Although robust genetic transformation protocols for roses have been established, the efficient regeneration of whole transgenic plants remains a significant challenge. Internally, comprehensive screening of genotypes is essential to assess the regeneration potential across various rose varieties. Different plant tissues and organs (leaf, immature embryos, stem internodes, and roots) can be utilized for tissue culture and regeneration in roses. Understanding the molecular mechanisms underlying regeneration is key to developing genotype-independent protocols [12,22,25–27]. Externally, regeneration efficiency is strongly influenced by the induction medium composition, growth regulator combinations, and culture conditions, all requiring ongoing optimization [25]. Future research may prioritize the development of

genotype-independent regeneration frameworks by leveraging developmental regulators (e.g., *BBM* or *WUS* genes) to bypass traditional tissue culture, optimizing stress-mitigating culture environments, and exploring alternative organogenesis pathways from diverse explant types. Ultimately, bridging the gap between rapid *in vivo* validation and stable whole-plant regeneration will be essential to provide a high-throughput pipeline for the precision molecular breeding of next-generation rose cultivars.

3.3. Gene Editing Platforms

The CRISPR/Cas9 system has revolutionized targeted genome editing in roses, serving as a pivotal tool for precision trait modification [13]. Nevertheless, its deployment in woody ornamentals is frequently bottlenecked by variable targeting efficiencies and inherently protracted tissue culture cycles, characterized by recalcitrant regeneration.

Successful gene editing in plants necessitates the use of suitable single-guide RNAs (sgRNAs). However, the targets provided by conventional sgRNA design tools may not always perform optimally in plant systems [28,29]. To address this challenge, dual-platform strategies have been developed to enhance the efficiency and reliability of gene editing. First, a stable rose suspension cell line enables the rapid, high-throughput prescreening of sgRNAs and promoters. This system revealed substantial variation in editing efficiency among targets within the same gene, such as the ethylene signaling component *RhEIN2*, allowing researchers to identify optimal guides prior to stable transformation [13]. Second, the integration of CRISPR/Cas9 with HRTS provides a rapid *in vivo* functional validation pipeline. Using roots transformed with sgRNA expression cassettes, the efficiency of sgRNAs can be easily monitored in the short term, thereby reducing the time and resources wasted on non-functional sgRNAs. For example, the utilization of Arabidopsis *U6* promoters to drive sgRNAs targeting *PHYTOENE DESATURASE* (*RhPDS*) via HRTS resulted in the successful editing in over 62% of transgenic hairy roots, featuring a diverse array of indels [24].

While optimized gene editing platforms have significantly propelled rose genomics research forward, current applications remain largely restricted to single-gene edits or small fragment modifications. The progress of gene editing in this genus is further complicated by high levels of heterozygosity and the persistent challenge of chimerism in edited lines, both of which obscure phenotypic interpretation and hinder the generation of stable homozygous mutants. Consequently, achieving complex chromosomal engineering, overcoming broad genotype dependence, and generating transgene-free edited whole plants remain critical frontiers for precision molecular design breeding in roses [30–32].

4. Genes Controlling Important Agronomic and Ornamental Traits

The identification and characterization of genes governing pivotal agronomic and ornamental traits constitute the core of rose genomics research. In recent years, leveraging expanded genetic resources and multi-omics platforms, researchers have identified and characterized numerous key genes associated with floral morphology, developmental processes, scent biosynthesis, and stress responses. These studies provide a foundation for elucidating the underlying regulatory mechanisms of trait formation and critical molecular targets for rose trait-based breeding.

4.1. Flowering Time

The transition from vegetative growth to flowering is a critical developmental switch that determines reproductive success and, in roses, defines key ornamental and economic values such as targeted seasonal blooming and continuous flowering. This process is precisely modulated by a sophisticated regulatory network that integrates exogenous environmental cues (e.g., photoperiod) and endogenous signals (e.g., age and the gibberellin (GA) pathways), ultimately converging on core floral integrators [33–37] (Table 1).

The photoperiodic signaling pathway serves as the primary environmentally dependent module. The *PHYTOCHROME-INTERACTING FACTORs* (*PIFs*) and *CONSTANS* (*CO*) form a pivotal regulatory hub. Under non-inductive light conditions, RcPIFs physically interact with RcCO to form a repressive complex that suppresses downstream florigen activation [33]. This pathway is fine-tuned by light-mediated post-translational modifications (PTMs): favorable light promotes the nuclear accumulation of kinase OPEN STOMATA 1-Like (RcOST1L), which specifically phosphorylates RcPIF4, triggering its proteasomal degradation and thereby releasing *FLOWERING LOCUS T* (*RcFT*) to promote flowering [38,39]. Conversely, specific red-light environments trigger a cascade where SENSITIVITY TO RED LIGHT REDUCED 1 (RcSRR1) interferes with the COP9 SIGNALOSOME SUBUNIT 5B (RcCSN5B)-mediated deneddylation of CULLIN 4-RING E3 ubiquitin ligase (RcCRL4), promoting RcCO proteolysis to prevent precocious flowering [40]. Collectively, these findings suggest that the central floral regulators of the photoperiod pathway, CO/FT, likely function as the primary mediators in modulating responses to light. This role mirrors their well-established function in Arabidopsis, where they serve as key integrators of environmental signals to regulate flowering time [41,42].

Independent of external light signals, flowering in roses is governed by age-related and GA pathways through internal developmental cues. Within the age-related pathway, the conserved SQUAMOSA PROMOTER BINDING-LIKE (RcSPL) transcription factors serve as crucial activators. Notably, RcSPL1 forms an activation complex with TATA-BINDING PROTEIN-ASSOCIATED FACTOR 15b (RcTAF15b), which directly upregulates floral meristem identity genes in response to plant age [34]. In the GA pathway, the *TERMINAL FLOWER 1* (*TFL1*) homolog *RoKSN*, which forms a complex with the bZIP transcription factor FLOWERING LOCUS D (RoFD) and potentially competes with RoFT, acts as a core negative regulator controlling the perpetual flowering trait. Natural loss-of-function mutations in *RoKSN* release floral repression, enabling modern rose cultivars to develop their characteristic continuous flowering habit [35–37,43–45]. Additionally, PLANT AT-RICH SEQUENCE AND ZINC-BINDING PROTEIN 8 (RcPLATZ8) functions as a critical negative regulator of flowering time in roses, delaying floral transition through interactions with essential hormonal pathways [46]. However, the specific pathways by which PLATZ8 regulates rose flowering remain to be further explored.

Rose flowering research has revealed a regulatory architecture that is largely conserved with model annuals—such as the SPL and CO/FT modules—yet distinct in its management of polycarpy and domestication-driven traits. The most significant divergence is observed in the continuous flowering (CF) habit of modern cultivars, which contrasts with the ancestral once-flowering (OF) mode [44]. While the natural loss-of-function mutation in *RoKSN* is established as the primary genetic driver for the CF transition, the precise spatio-temporal integration of this mutation with fluctuating environmental signals remains a significant gap in our current understanding. Future endeavors must therefore transition toward deciphering the epigenetic mechanisms, such as chromatin remodeling and DNA methylation, that maintain the perennial flowering "memory" across seasons, while simultaneously exploring the environmental plasticity of the CF trait in response to global climate instability. Furthermore, leveraging high-resolution multi-omics, including single-cell and spatial transcriptomics, will be essential to resolve the cellular-level dynamics within the shoot apical meristem, ultimately providing the theoretical foundation for engineering "designer" cultivars with precisely tailored blooming periods through CRISPR-based precision biobreeding.

Table 1. Examples of Genes Controlling Flowering Time Traits.

Trait	Gene	Encoding Product	References
Flowering time	*RcPIF1*	Phytochrome-interacting factors	[33,38]
	RcPIF3	Phytochrome-interacting factors	
	RcPIF4	Phytochrome-interacting factors	
	RcFT	Florigen proteins	

RcCO	CONSTANS transcription factor	
RcPIF4	Phytochrome-interacting factors	[39]
RcphyB	Phytochrome B	
RcOST1L	SNF1-RELATED PROTEIN KINASE	
RcFRSL3	FRS-like transcription factor	[40]
RcSRR1	Pioneer protein	
RcCSN5B	Constitutive photomorphogenesis 9 (COP9) signalosome subunit	
RcTAF15b	TATA-box binding protein associated factor	[34]
RcSPL1	SPL transcription factor	
RoKSN	Phosphatidylethanolamine-binding proteins	[35–37,43–45]
RcPLATZ8	PLATZ transcription factors	[46]

4.2. Flower Shape

Rose floral architecture, defined by petal size (fullness), petal number (single *vs*. double), and petal angle (openness), is a complex morphological trait governed by integrated networks of transcriptional regulators, phytohormones, and epigenetic modifiers [47,48].

Petal size relies on spatiotemporally regulated cell proliferation and expansion, driven by extensive hormonal crosstalk. Cytokinin (CTK) is essential for early cell division. The repressor MYB73, an R2R3-type MYB transcription factor, recruits a co-repressor TOPLESS and histone deacetylase HDA19 to reduce H3K9 acetylation at the *miR159* promoter, accelerating CTK degradation and thereby restricting petal size [47]. Conversely, RELATED TO AP2 4-LIKE (RhRAP2.4L) and the ARABIDOPSIS RESPONSE REGULATOR 1 (RhRR1)–SCARECROW-LIKE 28 (RhSCL28) module sustain cell division by repressing inhibitors and activating core *cyclins* (*RhCYCA1;1*/*B1;2*) in a CTK-dependent manner [48,49]. Cell expansion is synergistically modulated by other hormones. Ethylene restricts cell expansion through several mechanisms: it regulates the miR164-mediated expression of *NAC DOMAIN CONTAINING PROTEIN 100* (*RhNAC100*) [50], targets the DELLA protein *GA INSENSITIVE 1* (*RhGAI1*) via ETHYLENE INSENSITIVE 3 (EIN3) [51], and suppresses the expression of *RhPIP2;1* [52]. Additionally, the derepression of endogenous phytosulfokine (PSK) by the HD-Zip II transcription factor RhHAT9 stimulates cell proliferation [53]. Auxin coordinates the timing of expansion through the AUXIN RESPONSE FACTOR 2 (RhARF2)–RhMYB6 module [54]. Furthermore, jasmonate (JA) and GA orchestrate cell division and cellulose synthesis via transcription factors such as MYELOCYTOMATOSIS 2 (RhMYC2) and RhMYB70 [55,56]. Recent studies have introduced the concept of a post-translational "speed governor" that dynamically calibrates rose petal dimensions. While brassinosteroids (BRs) function as master accelerators of cell expansion via the transcription factor BRASSINAZOLE RESISTANT 1 (RhBZR1), which directly activates cell-wall remodeling genes such as *EXPANSIN 4* (*RhEXPA4)* and *PLASMA MEMBRANE INTRINSIC PROTEIN 1;1* (*RhPIP1;1*), this growth is precisely restrained by ethylene. Ethylene signaling triggers the activation of the MITOGEN ACTIVATED PROTEIN KINASE 7 (RhMPK7), which physically interacts with and phosphorylates RhBZR1 at the Ser-173 residue. This phosphorylation event effectively abolishes the DNA-binding affinity and transactivation potential of RhBZR1, thereby providing a molecular "brake" on BR-driven expansion rates. The discovery of the RhMPK7–RhBZR1 module reveals a sophisticated kinetic control mechanism that integrates growth-promoting and growth-inhibiting hormonal signals to maintain morphological homeostasis during flower development [57].

Homeotic transformations between stamens and petals primarily determine the commercially vital "double-flower" trait (increased petal number). *APETALA 2-LIKE* (*RcAP2L*) and *ASYMMETRIC LEAVES 1* (*RcAS1*) facilitate stamen-to-petal transitions, while AGAMOUS-LIKE 80 (RcAGL80) inhibits them [58]. Auxin homeostasis is critical in this organ specification: the DIOXYGENASE FOR AUXIN OXIDATION 1 (RhDAO1) prevents unwarranted homeotic conversion by catabolizing free IAA [59], whereas RhARF18

recruits the histone deacetylase RhHDA6 to directly suppress the C-class gene *AGAMOUS* (*RhAG*), promoting petaloid stamen development [60].

Flower opening, a critical developmental transition for pollination, is orchestrated by the synchronized expansion and movement of floral organs. In roses, the phytohormone ethylene drives petal movement by inducing asymmetric growth at the petal base. This process is mediated by the HD-Zip I transcription factor *PETAL MOVEMENT-RELATED PROTEIN 1* (*RhPMP1*), which serves as a direct transcriptional target of RhEIN3. RhPMP1 subsequently activates the expression of *ANAPHASE-PROMOTING COMPLEX 3b* (*RhAPC3b*), thereby enhancing endoreduplication levels in the parenchyma cells on the adaxial side of the petal (ADSP) base. The resulting localized cellular expansion provides the necessary mechanical force to drive the asymmetric growth and outward bending of petals [61].

The above studies collectively reveal that rose flower shape is controlled by a multi-layered network involving hormonal crosstalk (cytokinin, ethylene, brassinosteroids, auxin, and jasmonate), transcription factor cascades (MYB, AP2/ERF, HD-Zip, and ARF), and post-translational modifications (Figure 1; Table 2). However, several critical issues remain unresolved. First, the temporal dynamics of these regulators during petal development are poorly understood. Most studies report expression at one or two time points, but petal size and number are determined by the precise timing of cell division arrest and expansion onset. For instance, RhMYB73-mediated cytokinin degradation occurs early, whereas RhBZR1 activity is relevant later. How these temporal modules are coordinated, and what upstream signals trigger the transition, is unknown. Second, the spatial dimension is largely ignored. Petal size results from coordinated growth of adaxial, abaxial, and marginal regions; yet almost all molecular studies treat petals as homogeneous tissues. Single-cell transcriptomics or spatial *in situ* hybridization could resolve whether the same regulators act differentially across petal domains. Third, the interaction between petal number regulation (homeotic transformation) and petal size regulation (cell proliferation/expansion) is rarely addressed in the same genetic context. For example, double-flowered roses often have smaller individual petals, suggesting a trade-off or shared resource allocation, but the molecular link between *RhAG* suppression (which increases stamen-to-petal conversion) and cell-cycle regulators like *RhCYCA1;1* has not been explored. Fourth, while the RhMPK7–RhBZR1 phosphorylation module is an elegant example of post-translational "brake" control, it remains unclear whether similar mechanisms exist for other hormone pathways (e.g., auxin- or GA-mediated expansion). Moreover, the upstream signals that activate RhMPK7 specifically in petal cells are unknown.

Future work could integrate live imaging of petal growth with cell-type-specific transcriptomics to create a spatiotemporal atlas of floral organogenesis. Functional validation using conditional CRISPR (e.g., tissue-specific promoters) will help dissect the stage-specific roles of key regulators. Additionally, comparative studies between single and double flowers within the same genetic background could identify the molecular basis of petal number–size trade-offs.

Table 2. Examples of Genes Controlling Flower Shape Traits.

Trait	Gene	Encoding Product	References
Petal size	*MYB73*	R2R3-type MYB transcription repressor	[47]
	TPL	Transcriptional corepressor	
	HDA19	Histone deacetylase	
	miR159	microRNAs	
	CKX6	Cytokinin oxidase	
	RhRAP2.4L	AP2/ERF transcription factor	[48]
	RhARR14	Cytokinin two-component response regulator	
	RhKRP2	Kip-related protein	
	RhBPEub	bHLH transcription factor	
	RhSCL28	GRAS transcription factor	[49]
	RhRR1	Cytokinin two-component response regulator	

	RhNAC100	NAC transcription factor	[50]
	miR164	microRNAs	
	RhGAI1	GRAS transcription factors	[51]
	RhEIN3-3	ETHYLENE INSENSITIVE 3 transcription factor	
	RhPIP2;1	Aquaporin	[52]
	RhPSK3;1	PSK precursor	[53]
	RhHAT9	The homeodomain-leucine zipper (HD-Zip) transcription factor	
	RhARF2	Auxin response factor	[54]
	RhMYB6	R2R3 MYB transcription factor	
	RhMYC2	bHLH transcription factor	[55]
	RhLOG3	Cytokinin riboside 5′-monophosphate phosphoribohydrolase	
	RhMYB70	R2R3 MYB transcription factor	[56]
	RhGA3ox3	Gibberellin 3-oxidase	
	RhMPK7	Mitogen-activated protein kinase	[57]
	RhBZR1	BZR1 transcription factor	
Petal number	*RcAP2L*	AP2 domain transcription factor	[58]
	RcAS1	MYB transcription factor	
	RcAGL80	MADS-box transcription factor	
	RhARF18	Auxin-responsive transcription factor	[60]
	RhHDA6	Histone deacetylase	
	RhPILS1	Intracellular auxin carriers	
	RhDAO1	Auxin-catabolizing dioxygenase	[59]
Petal angle	*RhPMP1*	Homeodomain transcription factor	[61]
	RhAPC3b	Subunit of the Anaphase-Promoting Complex	

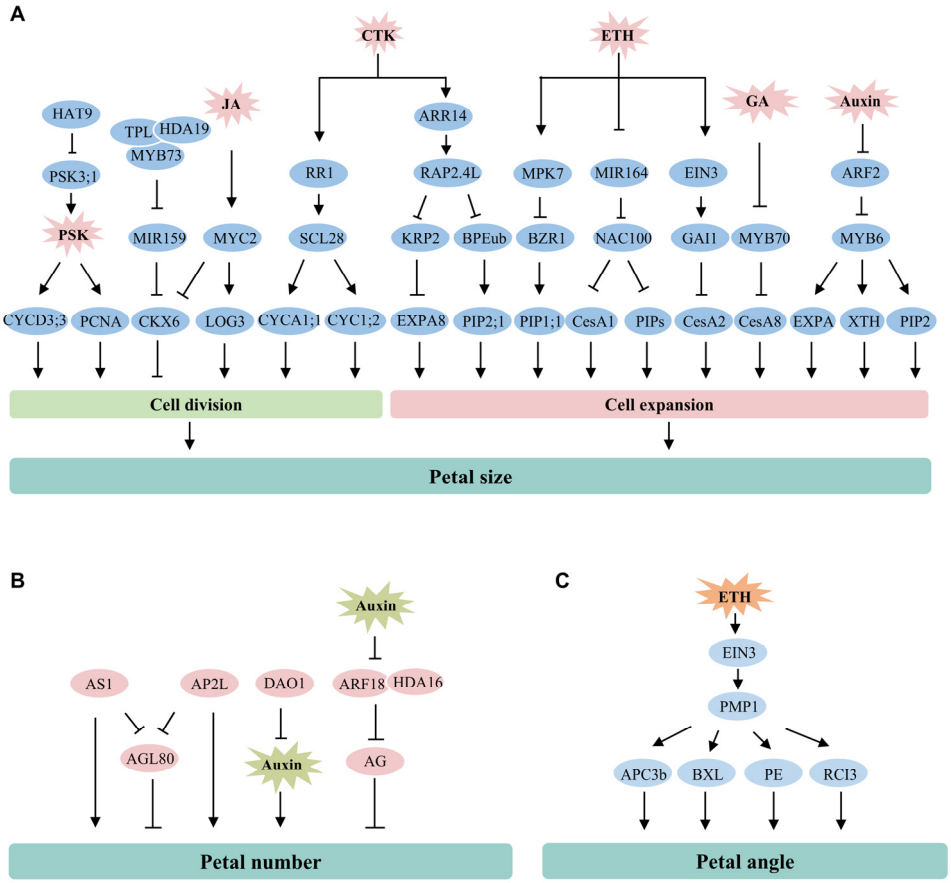

Figure 1. Regulatory Network of Petal Shape in Rose. Characterized genes related to petal size (**A**), petal number (**B**), and petal angle (**C**) in rose. Arrows indicate positive regulation, and T-shaped arrows indicate negative regulation.

4.3. Petal Senescence and Abscission

Flower senescence is a programmed cell death process characterized by the loss of petal function, typically categorized into wilting and abscission. In roses, these two processes often occur concurrently, serving as the primary determinants of postharvest longevity and commercial value [62]. These processes are orchestrated by hierarchical phytohormone networks, with ethylene acting as the central executioner through extensive transcriptional and post-translational crosstalk (Figure 2, Table 3) [63,64].

Ethylene is the master regulator of rose petal senescence; however, unlike many other ethylene-sensitive species, it does not exhibit a classic positive feedback loop in its own biosynthesis [63]. Instead, ethylene accelerates senescence by upregulating the expression of its own receptors and signal transduction components, such as *ETHYLENE RECEPTOR 3* (*RhETR3)* and *CONSTITUTIVE TRIPLE RESPONSE* (*RhCTR*) [65,66]. Beyond primary signaling, ethylene employs diverse downstream modules, including the induction of *CBL-INTERACTING PROTEIN KINASE 6* (*RhCIPK6*), whose silencing significantly delays senescence [67]. Ethylene further fine-tunes senescence through complex interactions with other phytohormones: it works synergistically with abscisic acid (ABA) and JA while antagonizing GA and CTK. Elevated ethylene simultaneously suppresses GA biosynthesis (via the HomeoBox 1 (RhHB1)–GIBBERELLIN 20 OXIDASE 1 (RhGA20ox1) axis) and disrupts GA signaling by inducing SENESCENCE-ASSOCIATED F-BOX (RhSAF), an SCF E3 ligase component that ubiquitinates GA receptors GIBBERELLIN INSENSITIVE DWARF 1 (RhGID1s) for proteasomal degradation [64,68]. Concurrently, ethylene amplifies senescence signals by activating the ABA biosynthesis gene *RhNCED1* via RhERF3 [69]. CTKs delay petal senescence through multifaceted mechanisms, including two ethylene-antagonizing pathways: (1) the RhHB6–PATHOGEN RELATED PROTEIN 10 (RhPR10.1) module elevates endogenous CTK content to suppress ethylene-promoted senescence [70], and (2) ETHYLENE RESPONSE FACTOR 113 (RhERF113) enhances floral CTK levels to counteract ethylene effects [71]. Concurrently, JA integrates with ethylene signaling via calcium-sensor relays: ethylene-activated CALCINEURIN B-LIKE PROTEIN 4 (RhCBL4)–RhCIPK3 phosphorylates JASMONATE ZIM-DOMAIN 5 (RhJAZ5) for 26S proteasomal degradation, derepressing JA responses to accelerate senescence, thus establishing an ethylene–JA molecular bridge [72]. Critically, the R2R3-MYB transcription factor RhMYB108 acts as a hormonal convergence node: highly expressed in ethylene/JA-treated or senescing petals, it directly activates senescence-associated genes, serving as a terminal executor for both hormones [73].

Reactive oxygen species (ROS) are pivotal senescence regulators, their levels determined by the balance between synthesis and scavenging. ROS generation originates from both apoplastic and mitochondrial compartments, where distinct regulatory modules orchestrate accumulation: (1) the HISTONE DEACETYLASE 15 (HDA15)–RELATED TO ABI3/VP1 2 (RAV2)–TOPLESS (TPL) complex represses *RESPIRATORY BURST OXIDASE HOMOLOG A* (*RBOHA*) expression via H3K9 deacetylation at its promoter [74]. Meanwhile, the WRKY DNA-BINDING PROTEIN 33 (WRKY33)–PLATZ9 transcription factor module inhibits *RBOHD* to decrease apoplastic ROS [75]; (2) concurrently, mitochondrial ROS production is restricted via PIF8–B-BOX DOMAIN PROTEIN 28 (BBX28)-mediated downregulation of *SUCCINATE DEHYDROGENASE 1* (*SDH1*), which perturbs electron transport chain flux [76]. Counteracting these processes, ROS scavenging relies on both enzymatic (e.g., catalase) and non-enzymatic (e.g., ascorbic acid) systems, and studies demonstrate that the transcription factor HB22 suppresses *GDP-L-GALACTOSE PHOSPHORYLASE 1* (*RhGGP1*), thereby depleting ascorbate pools and crippling ROS detoxification capacity, thereby accelerating petal senescence [77].

Autophagy has also emerged as a critical regulatory mechanism, particularly in response to external cues. Darkness triggers senescence through the transcription factors RhPIF4/8 and ELONGATED

HYPOCOTYL5 (RhHY5), which fine-tune autophagic activity [78]. This intricate interplay highlights the complex regulatory networks underlying the senescence process in rose petals.

Petal abscission in garden roses is intrinsically linked to their "self-cleaning" trait, orchestrated through interconnected ethylene and auxin signaling networks. Ethylene drives the dissolution of the abscission zone (AZ) through multiple coordinated mechanisms. These include the RhERF1/RhERF4-mediated pectin degradation pathway that loosens cell walls [79] and an ER-associated E2-E3 ubiquitin enzyme system regulating ethylene receptor turnover [80].

A breakthrough in understanding this process is the identification of the RhMPK3–LOB DOMAIN-CONTAINING PROTEIN 41 (RhLOB41)–RhWRKY9 module, which acts as a post-translational "gatekeeper" for ethylene-induced petal abscission [81]. In this pathway, the transcription factor RhWRKY9 serves as a dual-function executor: it concurrently activates ROS production by upregulating *RhRBOHD* and suppresses ROS scavenging by repressing *RhCAT2*. This coordinated "push-pull" mechanism generates a decisive ROS burst that locks the abscission process into an irreversible state. Ethylene primes this circuit by activating the RhMPK3 kinase, which phosphorylates the transcriptional repressor RhLOB41 at the Ser30 residue. This phosphorylation event creates a phosphodegron that targets RhLOB41 for autophagy-dependent degradation, thereby relieving the repression on *RhWRKY9*. The discovery of this MAPK-mediated autophagy pathway redefines ROS not merely as metabolic byproducts but as decisive signaling executors that ensure the directionality of developmental transitions in rose floral organs.

Counteracting these abscission-promoting processes, auxin signaling maintains petal attachment. AUXIN RESPONSE FACTOR 7 (RhARF7) promotes attachment by upregulating the *SUCROSE TRANSPORTER 2* (*RhSUC2*) to ensure nutrient supply to the AZs [82], while silencing *INDOLEACETIC ACID-INDUCED PROTEIN 16* (*RhIAA16*) promotes petal abscission [83]. This antagonistic interaction creates a dynamic equilibrium where ethylene drives abscission through cell wall remodeling and targeted ROS bursts, while auxin preserves attachment via metabolic regulation and gene repression, collectively determining the precise timing of petal shedding.

The regulatory framework for rose petal senescence is now well-defined, with ethylene as the central executioner operating through unique features (no auto-feedback loop) and complex hormonal crosstalk. The identification of the RhMPK3–RhLOB41–RhWRKY9 module as a post-translational "gatekeeper" for abscission represents a major conceptual advance. However, several contradictions and gaps warrant critical examination. First, the role of ABA in rose petal senescence remains ambiguous. While one study shows that ethylene activates ABA biosynthesis via RhERF3–RhNCED1 [69], others report that ABA treatment alone does not induce petal senescence in rose as strongly as in Arabidopsis or tomato. This suggests that ABA acts as an amplifier rather than an initiator. No direct ABA signaling components (e.g., PYL receptors, PP2Cs) have been functionally characterized in rose petals. Second, the antagonism between ethylene and GA involves both biosynthetic suppression and receptor degradation (RhSAF–RhGID1). However, the relative contribution of these two branches under natural senescence conditions (versus exogenous hormone treatments) is unclear. Are they redundant or sequentially activated? Third, while ROS are established as executors, the spatiotemporal pattern of different ROS species (H_2O_2 *vs*. O_2^- *vs*. singlet oxygen) has not been resolved. The RhMPK3–RhWRKY9 module induces a "burst", but which ROS species dominate and how they trigger cell death specifically in petal cells (versus leaf cells) is unknown. Fourth, autophagy is implicated in dark-induced senescence, but its interaction with ethylene signaling is unexplored. Does ethylene activate autophagy, or do they operate in parallel? The discovery of autophagic degradation of RhLOB41 suggests a direct link, yet general autophagy markers (*ATG* genes) have not been systematically studied in rose petals.

Compared to leaf senescence, rose petal senescence is remarkably rapid and irreversible. The absence of a positive feedback loop in ethylene biosynthesis may explain why roses do not exhibit the autocatalytic ethylene bursts observed in carnation or petunia, thereby allowing more precise temporal control. This

difference likely evolved to facilitate the "self-cleaning" trait, where petals abscise quickly after anthesis to prevent fungal growth on decaying tissue. However, the molecular reason for the lack of auto-feedback is unknown, perhaps due to a non-functional EIN3-binding site in *RhACS* promoters.

Future research could: (i) use systems biology approaches (time-series transcriptome + metabolome + hormone profiling) to model the dynamic network; (ii) apply single-nucleus RNA-seq to dissect cell-type-specific senescence programs in the abscission zone versus blade; (iii) engineer rose lines with inducible manipulation of key nodes (e.g., RhMPK3, RhWRKY9) to test causality; and (iv) screen for natural variation in senescence speed across germplasm to identify alleles that delay petal senescence without affecting flower opening.

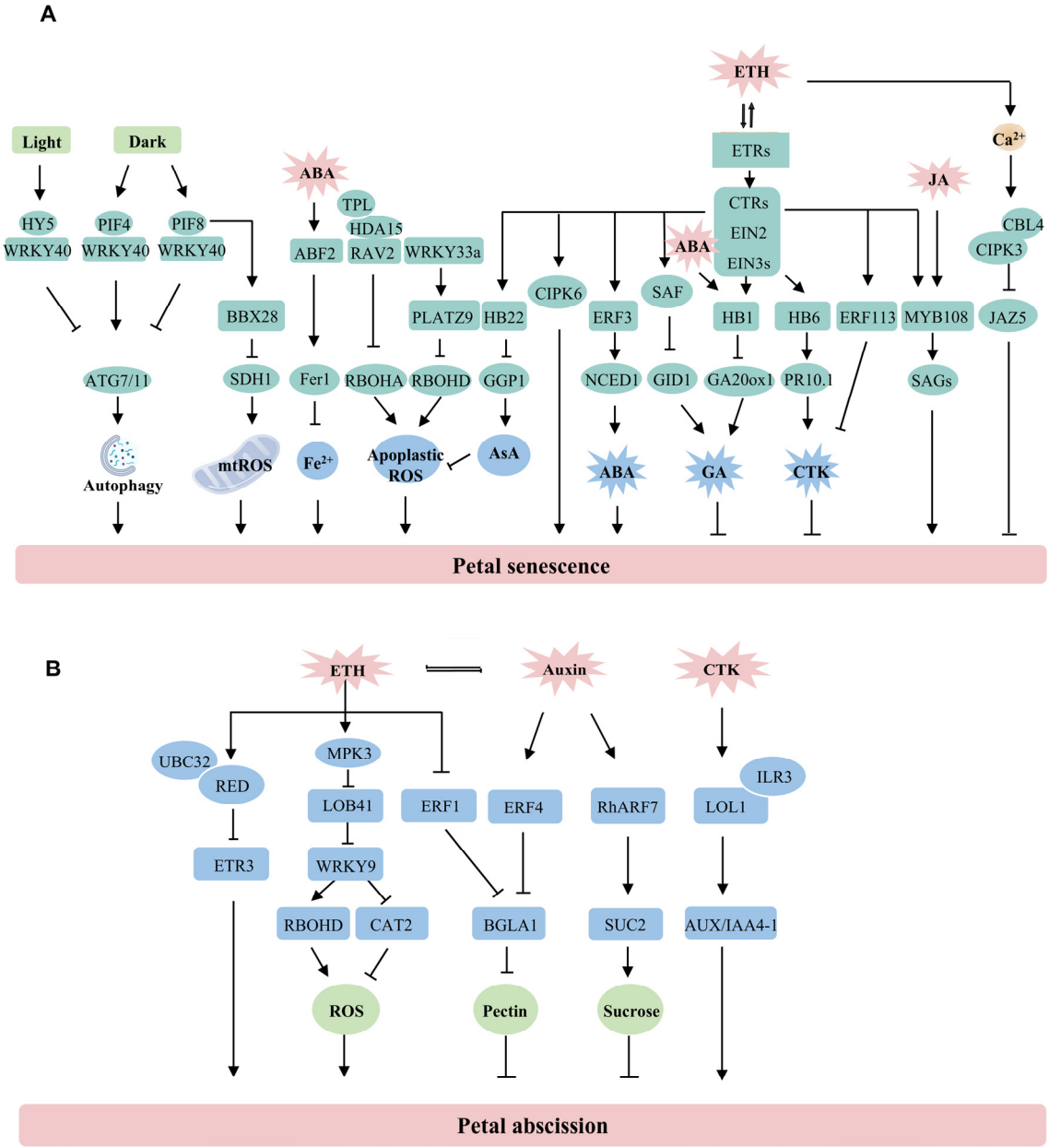

Figure 2. Regulatory Network of Petal Senescence in Rose. Characterized genes related to petal senescence (**A**) and abscission (**B**) in rose. Arrows indicate positive regulation, and T-shaped arrows indicate negative regulation.

Table 3. Examples of Genes Controlling Petal Senescence and Abscission Traits.

Trait	Gene	Encoding Product	References
Petal senescence	*RhHB1*	Homeodomain-leucine zipper I transcription factor	[68]
	RhGA20ox1	GA20 oxidase	
	RhSAF	F-box protein	[64]
	RhGID1	Gibberellic acid receptor	
	RhERF3	Ethylene-responsive factors	[69]
	RhNCED1	9-cis-epoxycarotenoid dioxygenase	
	RhHB6	Homeodomain-Leu zipper I transcription factor	[70]
	RhPR10.1	Pathogenesis-related protein	
	RhERF113	Ethylene response factor	[71]
	RhCIPK3	Calcineurin B-like protein (CBL) interacting protein kinase	[72]
	RhJAZ5	Jasmonic acid response repressor	
	RhCBL4	Calcineurin B-like protein	
	RhCIPK6	Calcineurin B-like protein (CBL) interacting protein kinase	[67]
	RhMYB108	R2R3-MYB transcription factor	[73]
	RhHDA15	Histone deacetylase	[74]
	RhRAV2	RAV transcription factor	
	RhRbohA1/2	Respiratory burst oxidase homolog protein	
	RhWRKY33a	WRKY transcription factor	[75]
	RhPLATZ9	Plant AT-rich sequence and zinc-binding protein	
	RhBBX28	B-box protein	[76]
	RhPIF8	Phytochrome-interacting factor	
	RhHB22	HOMEODOMAIN-LEUCINE ZIPPER (HD-ZIP) II transcription factor	[77]
	RhGGP1	GDP-l-galactose phosphorylase	
	RhHY5	bZIP-type transcription factor	[78]
	RhPIF4	Phytochrome-interacting factor	
	RhPIF8	Phytochrome-interacting factor	
Petal abscission	*RhERF1*	Ethylene response factor	[79]
	RhERF4	Ethylene response factor	
	RhBGLA1	Beta-glucanase	
	RED	E3 ligase RING finger	[80]
	UBC32	E2 partner	
	RhMPK3	Mitogen-activated protein kinase	[81]
	RhLOB41	LOB transcription factor	
	RhWRKY9	WRKY transcription factor	
	RhRBOHD	NADPH oxidase	
	RhCAT2	Catalase	
	RhARF7	AUXIN response factor	[82]
	RhSUC2	Sucrose transporter	
	RhIAA16	Indoleacetic acid-induced protein	[83]

4.4. Petal Color

Petal color is a defining ornamental trait in roses, determined predominantly by the accumulation of flavonoids (mainly anthocyanins for pink/red hues) and carotenoids (for yellow hues), while orange petals result from their synergistic accumulation [84,85].

Substantial progress has been made in the cloning and functional characterization of petal color genes in roses, unveiling the complex molecular networks governing this trait (Table 4). The pigmentation process is tightly controlled by MYB-dominated transcriptional networks. RcMYB1 acts as a master regulator directly driving *ANTHOCYANIN BIOSYNTHESIS GENEs* (*ABGs*) [86]. Light signals extensively modulate

this network: under illumination, RhHY5 represses the negative regulator *RhMYB3b* while inducing the positive regulator *RhMYB114a*. In low light, RhHY5 degradation shifts the balance, allowing RhMYB3b to competitively bind *RhbHLH3* and *ABG* promoters, thereby halting pigmentation [87]. Environmental stresses, such as drought, also trigger anthocyanin accumulation via a feedforward loop involving RcMYB75 and the GLUTATHIONE S-TRANSFERASE F11 (RcGSTFL11) [88]. Beyond anthocyanins, regulators like RhMYB3/30/305 and RhERF23 have been identified as key drivers of carotenoid biosynthesis during petal development [89], while ARF8 integrates auxin signaling into color regulation by targeting *CHALCONE SYHTHASE a/c* (*RhCHSa/c*) [90]. The above research emphasizes that, consistent with the anthocyanin and carotenoid regulatory mechanisms observed in many other plant species [91,92], members of the MYB transcription factor family are the key regulators of rose flower color. This highlights the evolutionary conservation of the MYB-mediated pathway across different plant taxa.

The MYB-bHLH-WD40 (MBW) complex is clearly the master regulator of anthocyanin biosynthesis in roses, consistent with other eudicots. Light regulation via RhHY5 and the competitive inhibition by RhMYB3b provides a mechanistic explanation for shade-induced color fading. The discovery of m6A-mediated stabilization of *ANTHOCYANIDIN SYNTHASE* (*RhANS*) mRNA adds an epitranscriptomic layer [93]. However, critical gaps remain. First, the interplay between anthocyanin and carotenoid pathways, which determines orange/red-yellow shades, is almost completely unstudied in roses. Do regulatory factors (e.g., MYBs) coordinate both pathways? Are there antagonistic interactions? In many species, high anthocyanin accumulation suppresses carotenoid visibility, but in orange roses both pigments coexist. The regulatory logic behind this coexistence is unknown. Second, although RcMYB1 is a master activator, its upstream regulators have not been identified. Does RhHY5 directly activate *RcMYB1*, or is the effect indirect via RhMYB114a? Promoter binding assays are needed. Third, epigenetic regulation beyond m6A (e.g., histone acetylation, DNA methylation) has not been explored. Given that petal color often varies with temperature and developmental stage, epigenetic plasticity is likely involved. Fourth, most studies are confined to a few cultivars (e.g., ‘Old Blush’ and ‘Samantha’). The allelic diversity of *MYB* genes across the genus is unknown; yet natural color variation from pale pink to deep crimson likely involves cis-regulatory changes in these transcription factors (TFs).

Compared to model systems like petunia or snapdragon, rose color research lags in the identification of specific MYB-bHLH partners. In petunia, different MYBs specify different pigment types (e.g., AN2 for anthocyanins and PH4 for vacuolar pH). In roses, only RcMYB1 and RhMYB3b have been characterized; the putative bHLH partner (RhbHLH3) has been mentioned but not fully validated. Moreover, the transport of anthocyanins into vacuoles—mediated by GSTs and MATE transporters—has only been touched upon (RcGSTFL11). The full complement of transporters and their regulation is missing.

Future studies could: (i) perform genome-wide association (GWAS) on a diverse panel for color traits to pinpoint causal *MYB* alleles; (ii) use CRISPR to simultaneously knock out and knock in specific MBW components to create novel colors; (iii) investigate the metabolic flux between anthocyanin and carotenoid branches using isotope labeling; and (iv) explore the role of m6A and other RNA modifications in other color-related genes.

Table 4. Examples of Genes Controlling Flower Color Traits.

Trait	Gene	Encoding Product	References
Flower color	*RcMYB1*	MYB transcription factor	[86]
	RhHY5	bZIP-type transcription factor	[87]
	RhMYB3b	MYB transcription factor	
	RhMYB114a	MYB transcription factor	
	RcMYB75	MYB transcription factor	[88]
	RcGSTFL11	Glutathione S-transferase	

	RhMYB3	MYB transcription factor	[89]
	RhMYB305	MYB transcription factor	
	RhMYB30	MYB transcription factor	
	RhERF23	Ethylene response factor	
	RhARF8	Auxin response factor	[90]
	RhANS	Anthocyanidin synthase	[93]
	RhALKBH10A	m^6A demethylase	
	RhALKBH10B	m^6A demethylase	

4.5. Floral Scent

The floral scent in roses is primarily derived from terpenoids, phenylpropanoids/benzenoids, and fatty acid derivatives [94]. The intricate bouquet of rose fragrances is released through distinct spatial and temporal patterns, contributing significantly to the overall sensory experience. In *Rosa gigantea*—a pivotal species in rose cultivation—the primary scent-emitting organs include petals, stamens, and pistils, each playing a unique role in forming spatially differentiated "scent-release units". Petals are particularly rich in isoeugenol, which imparts sweet, aromatic notes to the fragrance profile. Meanwhile, stamens contain high concentrations of eugenol, adding spicy-sweet undertones, while pistils synthesize methyleugenol, further enhancing the complexity of the scent [9]. Interestingly, although the two epidermal layers of the petal exhibit different cell morphologies, both are capable of producing and emitting volatile compounds [95,96]. Petal development typically progresses through two distinct phases after initiation in most plants. The first phase is characterized by slow growth, primarily driven by cell division, while the second phase involves rapid expansion due to cell enlargement. In roses, floral scent production peaks during this second stage, which usually coincides with the semi-opening state of the flower [97]. Notably, changes in individual major compounds align closely with fluctuations in the total scent profile throughout flower development [98]. Several scent-related genes are expressed during this process, including *RrAAT* [99], *RhPAAS* [100], and *OOMT2* [101]. In roses, the circadian clock plays a pivotal role in regulating the emission of most floral volatiles. These include terpenoids such as geraniol, E-citral, β-cubebene, geranyl acetate, and germacrene D [102,103], as well as phenylpropanoids/benzenoids like 2-phenylethanol (2-PE), phenylacetaldehyde (PAA), and 3,5-dimethoxytoluene (DMT) [100,102,104], and fatty acid derivatives such as hexyl acetate [102]. However, the regulatory effects of the circadian clock on these scent compounds vary significantly, with some showing little to no circadian influence. Take, for example, geranyl acetate and germacrene D. The emission of geranyl acetate peaks early in the light period, driven by the circadian-regulated expression of its biosynthetic gene *RhAAT* and the availability of its substrate, geraniol. Yet, under constant light conditions, this emission ceases due to the suppression of geraniol production. In contrast, the emission of germacrene D also displays a diurnal peak but lacks rhythmic gene expression (*RhGDS*) or endogenous accumulation. Instead, its oscillation is directly dependent on light exposure and vanishes entirely under continuous illumination [103]. This distinction underscores that synchronized daily scent emissions in roses are governed by independently evolved mechanisms: circadian coordination for geranyl acetate versus direct photoregulation for germacrene D. Such findings highlight the intricate and multifaceted regulation of volatile compounds in roses.

Terpenoids (e.g., geraniol, citronellol, and nerol) form the foundation of the classic "rose scent". A landmark paradigm shift in monoterpene biosynthesis was the discovery of NUDIX HYDROLASE 1 (RhNUDX1), a cytoplasmic Nudix hydrolase that synthesizes geraniol independently of traditional TERPENE SYHTHASES (TPS) [105]. Terpene production is highly dynamic: the RrMYB4–RrTPS31 module regulates sesquiterpenoids [106], while environmental stresses (like salinity) can paradoxically enhance scent emission via the RrbHLH105-mediated activation of geraniol synthesis [107].

Phenylpropanoids, notably 2-phenylethanol (2-PE), impart sweet and tea-like notes. 2-PE is synthesized via a two-step pathway involving PLP-dependent PHENYLACETALDEHYDE SYNTHASE (RhPAAS) and PHENYLACETALDEHYDE REDUCTASE (PAR), with specific *RhPAAS* allelic variations directly dictating production levels [108]. The diversification of rose scent is also driven by evolutionary novelties; for example, the *de novo*-originated gene *SCREP* acts as a functional regulator that shapes the scent profile by inhibiting eugenol synthesis [109], while various O-methyltransferases (OMTs) and acetyltransferases further decorate these volatiles to create the immense complexity of rose fragrance [110] (Table 5).

The discovery of RhNUDX1 as a non-canonical monoterpene synthase was a paradigm shift, demonstrating that Nudix hydrolases can generate geraniol independently of the classical TPS family. This finding has since been extended to other plants, but roses remain the only ornamental where this pathway is functionally validated. The circadian *vs*. light-directed regulation of different volatiles (geranyl acetate *vs*. germacrene D) reveals that scent emission is not a single "clock-controlled" output but a collection of independently evolved mechanisms. However, several major gaps remain. First, the regulatory networks upstream of scent biosynthesis genes are poorly defined. Only a few TFs have been identified (RrMYB4, RrbHLH105). The signaling pathways that link circadian clock components (e.g., LHY, CCA1, TOC1) to *RhNUDX1* or *RhAAT* promoters are completely unknown. Second, the subcellular organization of scent biosynthesis is understudied. RhNUDX1 is cytoplasmic, while TPS enzymes are typically plastidial. How are precursors (e.g., geranyl diphosphate) supplied to the cytoplasm? Do specific transporters exist? Third, the ecological role of induced scent under stress (e.g., salinity-induced geraniol increase) is hypothesized as pollinator attraction or defense, but no behavioral assays have been performed. Fourth, the genetic basis of scent variation among cultivars remains largely unexplored. While *RhPAAS* alleles correlate with 2-PE levels, most modern roses have lost strong scent due to selection for vase life and disease resistance. The specific mutations that cause scent loss have not been identified at the sequence level.

Table 5. Examples of Genes Controlling Flower Scent Traits.

Trait	Gene	Encoding Product	References
Floral scent	*RhNUDX1*	Nudix hydrolase	[105]
	RrTPS31	Terpene synthases	[106]
	RrMYB4	MYB transcription factor	
	RrbHLH105	bHLH transcription factor	[107]
	RrTPS5	Terpene synthase	
	RrTPS8	Terpene synthase	
	PAAS	Phenylacetaldehyde synthase	[108]
	SCREP	Transcription factor	[109]
	POMT	Phloroglucinol O-methyltransferase	[110]

4.6. Biotic Stress

Cultivated roses (*Rosa* cvs.) are consistently confronted with a myriad of disease and pest challenges that significantly hinder their growth, yield, and overall quality. Among the most pervasive pests are spider mites, aphids, and thrips, which collectively impose substantial stress on rose plants. Despite their detrimental impact, current pest management strategies predominantly rely on chemical control measures. However, molecular-level investigations into these biotic interactions are still relatively limited, leaving a notable gap in our understanding of the underlying mechanisms. On the disease front, key pathogens, notably downy mildew (*Peronospora sparsa* Berk), gray mold (*Botrytis cinerea*), powdery mildew (*Podosphaera pannosa*), and black spot (*Diplocarpon rosae*), cause substantial yield and quality losses through defoliation and direct tissue damage [111–113].

Recent transcriptomic advances have elucidated the complex regulatory networks underpinning rose immunity (Table 6), particularly against gray mold. Transcription factors act as central orchestrators in this defense response. The WRKY family plays a prominent role; for example, RcWRKY41 plays an important positive regulatory function in the resistance of rose petals against grey mold [111], RhWRKY13 modulates hormonal balance by enhancing CTK content and reducing ABA signaling to promote disease protection [114], and RhWRKY30 reinforces cell wall integrity by promoting lignin accumulation [115]. Similarly, AP2/ERF family members (such as RcERF099 and RhERF005) and bZIP transcription factors (like RhbZIP17) positively regulate disease resistance via defense gene expression and lignin biosynthesis [115–117]. Conversely, genes such as *RcbHLH112* have been identified as susceptibility factors that facilitate *B. cinerea* infection [118]. Defense is further fine-tuned by PTMs and receptor kinases; notably, the CALCIUM-DEPENDENT PROTEIN KINASE 5 (RcCDPK5) and the WALL-ASSOCIATED KINASE 4 (RcWAK4) significantly enhance resistance [119,120].

For foliar diseases, susceptibility to powdery mildew is strongly mediated by specific allelic variants within clade V of the *MILDEW RESISTANCE LOCUS O* (*RhMLO*) gene family [112], as well as splicing variants of *CONSTITUTIVE EXPRESSER OF PATHOGENESIS-RELATED GENES 5* (*RcCPR5*) [121]. Resistance to black spot is primarily conferred by nucleotide-binding leucine-rich repeat (NLR) proteins, with well-characterized loci including *RNA-DEPENDENT RNA POLYMERASE 1* (*rdr1*) and the tetraploid-associated resistance locus *rdr3* [113,122]. The ongoing evolutionary arms race is highlighted by *Marssonina rosae* effectors, such as SECRETED EFFECTOR PROTEIN 20 (MrSEP20) and MrSEP43, which actively suppress rose immunity by targeting host photosystem subunits and an orphan defense protein, respectively [123,124].

Transcriptomic studies have identified numerous TFs (WRKY, ERF, and bZIP) and signaling components (CDPK and WAK) that positively or negatively regulate resistance, primarily against *Botrytis cinerea*. The discovery of *Marssonina* effectors (MrSEP20, MrSEP43) that suppress host immunity provides insights into the molecular arms race. However, the field is heavily skewed toward gray mold, with limited research on black spot and powdery mildew—the two most economically damaging diseases in outdoor roses. This bias is partly methodological: *B. cinerea* is easy to inoculate and has a well-annotated genome, whereas *Diplocarpon rosae* (black spot) and *Podosphaera pannosa* (powdery mildew) are obligate or hemibiotrophic pathogens that are difficult to maintain and manipulate under laboratory conditions. Consequently, most studies on black spot resistance rely on QTL mapping (*rdr1* and *rdr3*) without cloned *R* genes, and the molecular function of these loci remains unknown. For powdery mildew, the *RhMLO* susceptibility alleles are characterized, but no corresponding resistance (*R*) genes have been cloned from wild species, despite known race-specific resistance in *R. roxburghii* and *R. multiflora*. Another critical gap is the lack of studies on pest resistance (aphids, thrips, and spider mites) at the molecular level; almost all pest management remains chemical. Moreover, the interaction between biotic stress responses and other traits (e.g., fragrance, senescence) is rarely examined. For example, ethylene is central to both senescence and defense; how roses balance these two outputs under pathogen attack is unexplored. Compared to major crops (rice and wheat), where *R* genes and NLR networks are extensively characterized, rose disease resistance research is at an early stage, lacking cloned *R* genes, effector-triggered immunity models, or durable resistance strategies. The reliance on chemical control is unsustainable, but the molecular tools for marker-assisted selection of polygenic resistance are still missing.

Table 6. Examples of Genes Controlling Biotic Resistance Traits.

Trait	Gene	Encoding Product	References
Gray mold resistance	*RcWRKY41*	WRKY transcription factor	[111]
	RhWRKY13	WRKY transcription factor	[114]
	RhCKX3	Cytokinin oxidase	
	RhABI4	APETALA2 (AP2)-type transcription factor	
	RhbZIP17	Basic leucine zipper transcription factor	[115]
	RhCAD1	Cinnamyl alcohol dehydrogenase	
	RhWRKY30	WRKY transcription factor	
	RcERF099	Ethylene response factor	[116]
	RhERF005	Ethylene response factor	[117]
	RhCCCH12	Zinc-finger transcription factor	
	RhPR10.1	Pathogenesis-related (PR) proteins	
	RcbHLH112	Basic/helix-loop-helix (bHLH) transcription factor	[118]
	RcNAC51	NAC transcription factors	[119]
	RcERF80	Ethylene response factor	
	RcCDPK5	Calcium-dependent protein kinase	
	RcWAK4	Wall-associated kinase	[120]
Powdery mildew resistance	*RhMLO1*	Membrane protein	[112]
	RhMLO2	Membrane protein	
	RcCPR5	Nucleoporin	[121]
Black spot disease resistance	*Rdr1*	TIR-NBS-LRR (TNL) type disease resistance protein	[113]
	Rdr3	TIR-NBS-LRR (TNL) type disease resistance protein	[122]
	RcPsaL	Photosystem I (PSI) subunits	[123]
	RcPsbX	Photosystem II (PSII) subunits	
	RcBROG	Orphan protein	[124]

4.7. Abiotic Stress

Abiotic stresses—predominantly extreme temperatures, drought, and salinity—critically impair rose growth, productivity, and ornamental quality. Over the past decade, significant progress has been made in identifying the molecular determinants of abiotic stress resilience in roses (Table 7).

Low-temperature stress (5–15°C) is a primary driver of impaired flower opening during winter, primarily by inducing the homeotic transformation of stamens into petaloid structures—a phenomenon known as stamen petaloidy. Based on recent studies, Han et al. have revealed that the A-class floral identity gene *APETALA* 2 homolog, *RcAP2*, plays a pivotal role in regulating the number of rose petals derived from stamens while also mediating responses to temperature fluctuations [125]. Notably, the R2R3-MYB transcription factor gene *RhMYB17* shows increased expression under low-temperature conditions. RhMYB17 acts as a transcriptional activator for *RhAP2* and *RhAP2L*, thereby facilitating the transformation of stamens into petals at low temperatures [126]. In addition, low temperature affects petal development through epigenetic regulation. Specifically, cold stress recruits the TELOMERE REPEAT BINDING FACTORS (RcTRB2)–CURLY LEAF (RcCLF) complex to deposit H3K27me3 epigenetic marks on the C-class floral identity gene *RcAG*, suppressing its expression to increase petal number [127]. Together, the above research indicates that the floral organ identity genes *AP2*, *AP2L*, and *AG* play an important role in mediating the influence of low temperature on the number of petals.

High temperatures in summer can cause the abnormal development of roses, such as bent peduncles [128], leaf yellowing and withering [129], decreased flower size, and other physiological diseases [130], which seriously affect their ornamental quality and economic value. Heat stress tolerance is orchestrated by HEAT STRESS TRANSCRIPTION FACTORs (HSFs), including RhHSFA7 and RcHSF30, which

activate *HEAT SHOCK PROTEINs* (e.g., *RcHSP18.1*) and *Bcl2-ASSOCIATED ATHANOGENE* (*BAG*) family genes, thereby conferring thermotolerance [131,132].

Roses (*Rosa* sp.) are extensively utilized for both indoor and outdoor landscaping worldwide. Despite their widespread application, roses frequently encounter drought and salinity stress during cultivation and in outdoor environments. These abiotic stresses significantly impede their growth and development, ultimately leading to reduced productivity, particularly in saline or semiarid regions [133]. TFs serve as central hubs in gene regulatory networks, where ABA and drought stress have been shown to increase the expression of *RcNAC091* and *RcWRKY71*, with RcNAC091 directly binding to the *RcWRKY71* promoter to activate its transcription, thereby enhancing the expression of stress-related and ABA-related genes, which subsequently improves drought tolerance through inducing stomatal changes and promoting ROS scavenging [134]. Another transcription factor, *RcMYB8*, is upregulated under salt and drought stress conditions. It regulates the expression of *RcPR5*/*1* and *RcP5CS1*, which influence the Na^+/K^+ balance through callose accumulation, thereby enhancing salt stress resistance. Additionally, RcMYB8 affects proline accumulation, which improves drought tolerance [133]. The co-expression of *MtDREB1C* and *RcXET* genes further enhances drought tolerance in transgenic plants compared to wild-type controls [135]. In the salt-tolerant germplasm *Rosa rugosa*, RrWRKY1 and the bHLH factor UNFERTILIZED EMBRYO SAC 12 (RrUNE12) further mitigate salt stress by modulating stress-responsive pathways and promoting ascorbate synthesis to alleviate oxidative damage [136,137]. Additionally, the salinity-induced bHLH transcription factor *RcbHLH59*, expressed in both rose leaves and roots, activates the RcbHLH59–RcPRs signaling module. This regulatory circuit specifically enhances salt tolerance by modulating callose deposition to maintain Na^+/K^+ homeostasis, thereby preserving ionic balance under osmotic stress [138]. Based on the above molecular evidence, tolerance to drought and salinity involves the coordinated activation of ABA signaling pathways, osmotic adjustment mechanisms, and ROS scavenging systems, a conserved adaptive strategy observed in both monocot and dicot species (e.g., Arabidopsis, rice, and tomato) [139,140].

Beyond transcriptional control, PTMs and epigenetic co-regulation fine-tune stress responses. Under drought, the plasma membrane aquaporin RhPIP2;1 undergoes phosphorylation at Ser273, triggering the dissociation of the RhPTM C-terminal domain, which subsequently translocates to the nucleus to suppress carbohydrate metabolism and prioritize survival [141]. Conversely, the PROTEIN PHOSPHATASE 2C (RcPP2C24) acts as a negative regulator of drought signaling by inhibiting stomatal closure [142]. Moreover, epigenetic adaptation is involved in the stress response through MEDIATOR SUBUNIT 15a (RhMED15a) and RhMED15a-like, which act as enhancers of drought tolerance in rose, probably by modulating the expression of drought-related genes [143,144]. In salt stress, the TspO/MBR DOMAIN-CONTAINING MEMBRANE PROTEIN (TSPO)-mediated degradation of the ethylene receptor RhETR3 enhances ethylene signaling and reduces ROS accumulation, demonstrating the critical role of protein turnover in rapid adaptation [145].

Cut roses for commercial production are usually harvested at an open bud stage and are extremely susceptible to dehydration damage during postharvest handling, resulting in abnormal flower opening and senescence [146], involving multiple transcription factors and genes that play pivotal roles in regulating tolerance mechanisms. Among these, RhNAC3, a stress-associated NAC transcription factor, enhances dehydration tolerance by modulating osmotic stress-related genes to maintain cellular osmotic balance [147]. In parallel, NAC-LIKE, ACTIVATED BY APETALA3/PISTILLATA (RhNAP), contributes to dehydration tolerance and senescence regulation through the modulation of CTK catabolism, specifically by promoting the expression of *RhCKX6* [148]. Additionally, RhbHLH92 positively regulates dehydration tolerance via interaction with RhMYB123, forming a regulatory module that amplifies stress responses in rose petals [149]. RhNAC2 and RhEXPA4 are also critical for dehydration tolerance during petal expansion; RhNAC2 regulates stress-responsive pathways, while RhEXPA4 mediates cell wall loosening to facilitate cellular adaptation to water deficit [146]. Collectively, these findings underscore the intricate molecular

networks encompassing transcriptional regulation, hormone metabolism, and cell wall dynamics that govern dehydration tolerance in roses. Beyond dehydration tolerance, effective rehydration and recovery are equally crucial for postharvest quality. The scaffold protein CASP-LIKE PROTEIN 1D1 (RhCASPL1D1) stabilizes aquaporins such as RhPIP2, thereby facilitating water uptake after dehydration [150]. Concurrently, the MPK6 and RhMKK9 kinase cascades precisely regulate rehydration-induced ethylene production by promoting the spatio-temporal accumulation of ACC synthase [151,152]. These kinase-mediated adjustments exemplify the dynamic PTMs-driven metabolic shifts required for roses to recover from postharvest dehydration. Through the integration of these mechanisms, roses achieve a balance between stress adaptation and recovery, highlighting the complexity of their molecular resilience strategies.

Research on abiotic stress in roses has identified key transcription factors (NAC, WRKY, MYB, bHLH, and HSF) and post-translational mechanisms (phosphorylation of RhPIP2;1, TSPO-mediated receptor degradation) that confer tolerance to low/high temperature, drought, salinity, and postharvest dehydration. A recurring theme is the convergence on ABA signaling, ROS scavenging, and osmotic adjustment—mechanisms conserved across angiosperms. However, several critical issues emerge. First, most studies are conducted under controlled growth chamber conditions using severe, acute stress (e.g., sudden 4 °C, 200 mM NaCl, or complete water withdrawal). These conditions poorly mimic field environments, where stress is often gradual, fluctuating, and combined (e.g., heat + drought). The relevance of identified genes under realistic field scenarios has rarely been tested. Second, the crosstalk between different abiotic stresses is largely ignored. For example, low temperature induces stamen petaloidy through *RcAP2* and *RcAG* suppression; but does the same pathway respond to heat stress (which typically reduces petal number)? Direct comparisons are missing. Third, epigenetic memory of stress, which is known in Arabidopsis and rice, has only been touched upon in roses (RhMED15a). Do drought-induced DNA methylation changes persist and prime stress responses in subsequent generations? No studies have addressed this in roses. Fourth, the trade-off between stress tolerance and ornamental quality (flower size, fragrance, vase life) is rarely quantified. Rose stress research has benefitted from knowledge transfer from model plants (e.g., the role of HSFs, NACs, and aquaporins), but unique aspects such as low-temperature-induced floral homeotic transformation are not observed in *Arabidopsis*, highlighting the need for rose-specific models. However, the field lacks a centralized mutant collection or overexpression library for rapid functional screening.

Future studies could: (i) adopt multi-stress field trials with controlled environmental monitoring to validate candidate genes; (ii) apply multi-omics (transcriptome + metabolome + epigenome) under realistic stress regimes; (iii) develop high-throughput phenotyping platforms for stress-related traits (e.g., leaf wilting, ion leakage, flower abortion); (iv) use genome editing to create loss-of-function alleles of negative regulators (e.g., *RcPP2C24*) and evaluate trade-offs; and (v) investigate the role of graft-transmissible signals (rootstock-to-scion) in stress tolerance, as roses are frequently grafted.

Table 7. Examples of Genes Controlling Abiotic Resistance Traits.

Trait	Gene	Encoding Product	References
Low temperature	*RcAP2*	AP2 transcription factor	[125]
	RhMYB17	R2R3-MYB transcription factor	[126]
	RcAG	C-type MADS-box transcription factor	[127]
	RcTRBs	Telomere repeat binding factors	
	RcCLF	The PRC2 complex component	
Heat tolerance	*RhHsfA7*	Heat shock transcription factors	[131]
	RcHSF30	Heat shock transcription factors	[132]
	RcHSP18.1	Heat shock protein	
	RcBAG6	Bcl-2-associated athanogene protein	
Drought tolerance	*RcNAC091*	NAM/ATAF1/2/CUC2 (NAC) transcription factors	[134]
	RcWRKY71	WRKY transcription factor	

	RcMYB8	MYB transcription factor	[133]
	RcP5C51	Pyrroline-5-carboxylate synthase	
	MtDREB1C	DRE-binding (DREB) proteins	[135]
	RcXET	Xyloglucan endotransglycosylase	
	RhPIP2;1	Aquaporin	[141]
	RhPTM	Membrane-tethered MYB transcription factor	
	RcPP2C24	Protein phosphatase	[142]
	RhMED15a	Mediator subunit	[143]
	RhMED15a-like	Mediator subunit	[144]
Salt tolerance	*RrWRKY1*	WRKY transcription factors	[136]
	RrUNE12	bHLH transcription factor	[137]
	RrGGP2	GDP-L-galactose pyrophosphatase	
	RcbHLH59	Basic helix–loop–helix (bHLH) transcription factor	[138]
	RcPRs	Pathogenesis-related (PR) genes	
	RhETR3	Ethylene receptor	[145]
	RhTSPO	Tryptophan-rich sensory protein/mitochondrial benzodiazepine receptor	
Dehydration tolerance	*RhNAC3*	NAC transcription factor	[147]
	RhNAP	NAC transcription factor	[148]
	RhCKX6	Cytokinin oxidase/dehydrogenase	
	RhbHLH92	Basic helix-loop-helix (bHLH) proteins	[149]
	RhMYB123	R2R3-type transcription factor	
	RhNAC2	NAC transcription factor	[146]
	RhEXPA4	Expansin protein	
	RhCASPL1D1	Scaffold protein	[150]
	RhPIP2	Aquaporins	
	RhMPK6	Mitogen-activated protein kinase	[151]
	RhACS1	ACC synthase	
	RhMKK9	Mitogen-activated protein kinase kinase	[152]

4.8. Prickle

Prickles, commonly named thorns, play crucial roles in defense against herbivory and environmental stress adaptation. Prickles in rose originate from the ground meristem and are not modified trichomes [153]. For cut roses, thorns on the branches can affect the workers' operation efficiency and increase the process and cost of thorn removal. For garden roses, the thorns on the branches can easily cause accidental injuries to people. Functional proteins play a crucial role in regulating rose prickle development, with LONELY GUY 1 (RcLOG1) emerging as a key player in this process. A single nucleotide polymorphism (SNP) in CTK-activating enzyme RcLOG1 has been linked to prickle number, suggesting its regulatory role in this trait. Two protein variants, RcLOG1-A and RcLOG1-B, were identified, differing by a non-synonymous mutation at position 152 (N to D), which suppresses prickle formation in roses [154]. In addition to *RcLOG1*, other genes have also been implicated in prickle development. For instance, overexpression of vacuolar sodium/proton (Na^+/H^+) *ANTIPORTERS OR EXCHANGERS* (*RhNHX*) in transgenic plants led to a significant reduction in prickle number [155]. Furthermore, silencing the expression of *PIP2;1* was found to markedly attenuate prickle production along rose stems [156]. Using a segregating diploid rose F1 population, Zhou et al. identified a complex genetic determinism with a major locus on linkage group 3 (LG3) that controls the presence of prickles and a few QTLs that control prickle density [157]. Further studies are necessary to develop markers for breeding selection and to identify the molecular bases. These findings provide valuable genetic targets for breeding prickle-reduced ornamental roses without compromising stress resilience (Table 8).

Table 8. Examples of Genes Controlling Prickle Traits.

Trait	Gene	Encoding Product	References
Prickle	*RcLOG1*	Cytokinin riboside 5′-monophosphate phosphoribohydrolases	[154]
	RhNHX	Vacuolar sodium/proton antiporters	[155]
	PIP2;1	Aquaporin	[156]

Taken together, the functional genomic studies summarized in Sections 4.1–4.8 reveal a recurring theme: complex ornamental traits in roses are governed by multi-layered regulatory networks involving transcriptional cascades, hormonal crosstalk, post-translational modifications, and epigenetic regulation. While many core components are conserved with model annual plants (e.g., MYB–bHLH–WD40 for color, CO/FT for flowering, and ethylene signaling for senescence), roses have evolved unique features—such as the absence of autocatalytic ethylene feedback in petals, the non-canonical monoterpene synthase RhNUDX1, and low-temperature-induced homeotic transformations—that reflect their perennial, polyploid, and highly heterozygous nature. These insights provide a rich inventory of target genes and regulatory modules for precision breeding. However, as discussed in the following section, translating this knowledge into practical applications faces substantial bottlenecks that require coordinated technological and strategic solutions.

5. Challenges and Perspectives of Rose Genomics Research for Biobreeding

Although remarkable progress has been made in rose genomics research, several critical bottlenecks remain that hinder the seamless translation of genomic discoveries into practical biobreeding applications.

5.1. Current Challenges

Firstly, the intrinsic complexity of rose genomes poses a persistent challenge. Most cultivated roses (*Rosa hybrida*) are highly heterozygous and polyploid (predominantly allotetraploid), with a high proportion of transposable elements [5,14,17]. While diploid model roses (e.g., *Rosa chinensis* ‘Old Blush’) have been well-sequenced, dissecting allele-specific expression, structural variations (SVs), and epigenetic regulations in complex tetraploid cultivars remains methodologically demanding. Secondly, the recalcitrance to genetic transformation is a vital limiting factor for functional validation, genome editing, and molecular breeding. Current *Agrobacterium*-mediated transformation systems in roses are highly genotype-dependent, inefficient, and time-consuming [23,24]. This severe bottleneck restricts the high-throughput application of advanced CRISPR/Cas systems. Finally, a significant genotype-phenotype gap persists, reflecting the challenge of translating genetic information into observable traits. While massive amounts of transcriptomic and metabolomic data have been generated, integrating these diverse datasets to uncover the precise regulatory networks of complex quantitative traits (such as continuous flowering, scent emission, and stress resilience) is still in its infancy.

Taken together, these three challenges, genomic complexity, transformation recalcitrance, and genotype-phenotype gap, are not isolated but deeply interconnected. Overcoming them requires a coordinated, multi-pronged strategy that leverages recent advances in pangenomics, gene editing, and AI-driven data integration. The following perspectives are therefore structured to directly address each challenge in turn, building a logical pathway from gene discovery to field application.

5.2. Perspectives

The rapid evolution of rose genomics research has laid a robust foundation for transitioning from traditional empirical selection to trait-based biobreeding. To fully realize the potential of next-generation rose genetic improvement, future research should focus on the following strategic directions, which are

arranged in a logical cascade: (i) expanding genetic and omics resources to identify target genes (Section 5.2.1); (ii) developing efficient tools to validate and edit these genes (Section 5.2.2); (iii) translating knowledge into practical breeding via molecular markers (Section 5.2.3); and (iv) integrating BT, IT, and AI to create an intelligent breeding system (Section 5.2.4).

5.2.1. Pangenomics and High-Resolution Multi-Omics Integration for Gene Discovery

A primary objective is the systematic identification of genes in the complex regulatory networks, which can be utilized in biobreeding. Future efforts may benefit from leveraging haplotype-resolved pangenomic resources to capture the full landscape of genetic diversity, especially presence/absence variations (PAVs) and structural variants (SVs) lost during domestication. By integrating cutting-edge technologies—such as single-cell RNA sequencing (scRNA-seq), spatial transcriptomics, metabolomics, proteomics, epigenomics, and phenomics—researchers can achieve unprecedented cellular-level insights into developmental processes, such as the spatial regulation of scent biosynthesis and the ROS-mediated orchestration of petal senescence and abscission. Such multi-layered approaches will transform rose research from single-gene functional studies to the elucidation of systematic gene-to-trait networks, thereby bridging the gap between genotype and phenotype.

Addressing the first challenge of genomic complexity, this pangenomic and multi-omics foundation provides the essential catalog of targets. However, identifying candidate genes is only the initial step; their functional validation and subsequent manipulation demand advanced genetic engineering platforms.

5.2.2. Advancing Precision Genome Editing and High-Efficiency Transformation

Breaking the technical bottleneck of recalcitrant regeneration is essential for functional validation and molecular breeding. Future efforts should explore the use of morphogenic regulators (e.g., *BBM*/*WUS* or *GRF–GIF* chimeras) to bypass genotype-dependent barriers in somatic embryogenesis. Concurrently, upgrading from standard CRISPR/Cas9 to precision tools—such as base editing, prime editing, and multiplex gene editing—will enable the surgical modification of key alleles. This will allow for the simultaneous "stacking" of favorable traits, such as thornlessness, broad-spectrum disease resistance, and enhanced postharvest longevity, without introducing transgenic footprints. In light of recent progress *in planta* transformation, virus-induced gene editing (VIGE) technologies should become a critical tool that overcomes the obstacle of rose transformation [158].

This addresses the second challenge, transformation recalcitrance, by providing the technical means to edit genes directly in elite germplasm. Once key alleles are validated and edited, the next imperative is to translate these molecular insights into scalable breeding pipelines that can be applied across diverse rose varieties without requiring laborious transformation for each cross.

5.2.3. Molecular Marker Development and Multi-Trait Pyramid Breeding

To bridge the gap between genomic discovery and field application, the development of high-density molecular markers tailored for polyploid roses is imperative. Utilizing the extensive genetic variations derived from diverse *Rosa* accessions, researchers can develop cost-effective SNP arrays or KASP markers. These tools will empower high-throughput marker-assisted selection (MAS) and genomic selection (GS), facilitating the efficient pyramiding of multiple agronomic and ornamental traits. Such precision trait-based breeding strategies will significantly shorten breeding cycles and improve the selection intensity for complex quantitative traits. Especially, rose genomics research will increasingly transition from generalized trait studies to precision breeding tailored to specific market segments. By dissecting the distinct molecular modules governing various traits, breeding objectives can be refined based on end-uses. For cut roses, the priority is to extend postharvest longevity and enhance environmental resilience. For landscape roses,

aesthetic sustainability and low-maintenance resilience are paramount. For industrial processing of roses, high biomass and biochemical efficiency are the core targets.

Marker-assisted and genomic selection directly confront the third challenge, the genotype-phenotype gap, by enabling breeders to predict trait outcomes from DNA without fully understanding the underlying regulatory networks. However, these approaches still rely on phenotypic data from field trials, which are time-consuming and environment-dependent. To truly accelerate breeding, it would be advantageous to integrate these marker systems with advanced computational models that can learn from massive datasets.

5.2.4. Integration of BT, IT, and AI for Intelligent Biobreeding

The next frontier in rose genetic improvement lies in the deep convergence of biotechnology (BT), information technology (IT), and artificial intelligence (AI). Such an "Intelligent Biobreeding" paradigm utilizes machine learning and AI algorithms to process massive multi-omics datasets and high-throughput phenotyping data, enabling the accurate prediction of complex breeding values and the optimized design of hybrid crosses. Furthermore, synthetic biology approaches, such as the *in silico* reconstruction of metabolic pathways, could be utilized to create "bespoke" rose varieties with engineered fragrances, enhanced bioactivity, or superior environmental resilience. By merging AI-driven genomic prediction with precise CRISPR editing, we can enter into an era of "designer roses", where customized cultivars are developed with surgical precision to meet evolving global market demands.

In conclusion, the integration of pangenomics, efficient genome editing, and AI-assisted genomic selection will bridge the gap between genomics research and practical breeding. This multidisciplinary approach will ultimately usher in a new era of design breeding, cementing the rose's status not only as the "queen of flowers" but also as a premier model for woody ornamental research.

Statement of the Use of Generative AI and AI-Assisted Technologies in the Writing Process

During the preparation of this manuscript, the authors used DeepSeek V4 to correct grammar errors. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the content of the published article.

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References

1. Hibrand Saint-Oyant L, Ruttink T, Hamama L, Kirov I, Lakhwani D, Zhou NN, et al. A high-quality genome sequence of *Rosa chinensis* to elucidate ornamental traits. *Nat. Plants* **2018**, *4*, 473–484. DOI:10.1038/s41477-018-0166-1
2. de Vries DP, Dubois LAM. Rose breeding: Past, present, prospects. *Acta Hortic.* **1996**, *424*, 241–248. DOI:10.17660/ActaHortic.1996.424.43
3. Bendahmane M, Dubois A, Raymond O, Bris ML. Genetics and genomics of flower initiation and development in roses. *J. Exp. Bot.* **2013**, *64*, 847–857. DOI:10.1093/jxb/ers387
4. Roberts AV. *Encyclopedia of Rose Science*; Academic Press: Cambridge, MA, USA, 2003.
5. Esselink G, Smulders M, Vosman B. Identification of cut rose (*Rosa hybrida*) and rootstock varieties using robust sequence tagged microsatellite site markers. *Theor. Appl. Genet.* **2003**, *106*, 277–286. DOI:10.1007/s00122-002-1122-y
6. Fougère-Danezan M, Joly S, Bruneau A, Gao XF, Zhang LB. Phylogeny and biogeography of wild roses with specific attention to polyploids. *Ann. Bot.* **2015**, *115*, 275–291. DOI:10.1093/aob/mcu245
7. Zhang Z, Yang T, Liu Y, Wu S, Sun H, Wu J, et al. Haplotype-resolved genome assembly and resequencing provide insights into the origin and breeding of modern rose. *Nat. Plants* **2024**, *10*, 1659–1671. DOI:10.1038/s41477-024-01820-x
8. Zhou L, Wu S, Chen Y, Huang R, Cheng B, Mao Q, et al. Multi-omics analyzes of *Rosa gigantea* illuminate tea scent biosynthesis and release mechanisms. *Nat. Commun.* **2024**, *15*, 8469. DOI:10.1038/s41467-024-52782-9
9. Cheng B, Zhao K, Zhou M, Bourke PM, Zhou L, Wu S, et al. Phenotypic and genomic signatures across wild Rosa species open new horizons for modern rose breeding. *Nat. Plants* **2025**, *11*, 775–789. DOI:10.1038/s41477-025-01955-5
10. Liu D, Liu K, Tong B, Guo H, Qu K, Xu T, et al. Telomere-to-telomere, gap-free assembly of the *Rosa rugosa* reference genome. *Hortic. Plant J.* **2024**, *12*, 1450–1464. DOI:10.1016/j.hpj.2024.06.005
11. Kim CK, Chung JD, Park SH, Burrell AM, Kamo KK, Byrne DH. *Agrobacterium tumefaciens*-mediated transformation of *Rosa hybrida* using the *green fluorescent protein* (*gfp*) gene. *Plant Cell Tissue Organ Cult.* **2004**, *78*, 107–111. DOI:10.1023/B:TICU.0000022529.16697.90
12. Liu G, Yuan Y, Jiang H, Bao Y, Ning G, Zhao L, et al. *Agrobacterium tumefaciens*-mediated transformation of modern rose (*Rosa hybrida*) using leaf-derived embryogenic callus. *Hortic. Plant J.* **2021**, *7*, 359–366. DOI:10.1016/j.hpj.2021.02.001
13. Wang C, Li Y, Wang N, Yu Q, Li Y, Gao J, et al. An efficient CRISPR/Cas9 platform for targeted genome editing in rose (*Rosa hybrida*). *J. Integr. Plant Biol.* **2023**, *65*, 895–899. DOI:10.1111/jipb.13421
14. Bourke PM, Arens P, Voorrips RE, Esselink GD, Koning-Boucoiran CFS, van't Westende WPC, et al. Partial preferential chromosome pairing is genotype dependent in tetraploid rose. *Plant J.* **2017**, *90*, 330–343. DOI:10.1111/tpj.13496
15. Raymond O, Gouzy J, Just J, Badouin H, Verdenaud M, Lemainque A, et al. The *Rosa* genome provides new insights into the domestication of modern roses. *Nat. Genet.* **2018**, *50*, 772–777. DOI:10.1038/s41588-018-0110-3
16. Zhang X, Wu Q, Lan L, Peng D, Guan H, Luo K, et al. Haplotype-resolved genome assembly of the diploid *Rosa chinensis* provides insight into the mechanisms underlying key ornamental traits. *Mol. Hortic.* **2024**, *4*, 14. DOI:10.1186/s43897-024-00088-1
17. Zhang X, Lan L, Yang Y, Guan H, Peng D, Jiang H, et al. Pangenomic analyses of rose uncover widespread structure variation and empower genomics-directed breeding. *Nat. Genet.* **2026**, *58*, 1164–1175. DOI:10.1038/s41588-026-02569-z
18. Gahlaut V, Kumari P, Jaiswal V, Kumar S. Genetics, genomics and breeding in *Rosa* species. *J. Hortic. Sci. Biotechnol.* **2021**, *96*, 545–559. DOI:10.1080/14620316.2021.1894078
19. Firoozabady E, Moy Y, Courtney-Gutterson N, Robinson K. Regeneration of transgenic rose (*Rosa hybrida*) plants from embryogenic tissue. *Nat. Biotechnol.* **1994**, *12*, 609–613. DOI:10.1038/nbt0694-609
20. Marchant R, Power JB, Lucas JA, Davey MR. Biolistic transformation of rose (*Rosa hybrida* L.). *Ann. Bot.* **1998**, *81*, 109–114. DOI:10.1006/anbo.1997.0538
21. Wang P, Si H, Li C, Xu Z, Guo H, Jin S, et al. Plant genetic transformation: Achievements, current status and future prospects. *Plant Biotechnol. J.* **2025**, *23*, 2034–2058. DOI:10.1111/pbi.70028
22. Kaur N, Pati PK, Sharma M, Ahuja PS. Somatic embryogenesis from immature zygotic embryos of *Rosa bourboniana* desp. *In Vitro Cell. Dev. Biol.-Plant* **2006**, *42*, 124–127. DOI:10.1079/IVP2005737
23. Lu J, Huang Y, Guo Y, Fan C, Yuan G, Zhou R, et al. *Agrobacterium rhizogenes*-Mediated Hairy Root Transformation in Rosa. *Horticulturae* **2025**, *11*, 49. DOI:10.3390/horticulturae11010049

24. Zhang T, Liu K, Chen J, Zhao S, Zhou W, Chen S, et al. An efficient hairy root transformation system for protein interaction and gene editing analysis in roses. *Hortic. Plant J.* **2025**, *in press*. DOI:10.1016/j.hpj.2025.03.013
25. Nelson A, Ranney T, Liu W, Kelliher T, Duan H, Da K. Overcoming recalcitrance: A review of regeneration methods and challenges in roses. *Plants* **2025**, *14*, 3797. DOI:10.3390/plants14243797
26. Rout GR, Debata BK, Das P. Somatic embryogenesis in callus cultures of *Rosa hybrida* L. cv. Landora. *Plant Cell Tissue Organ Cult.* **1991**, *27*, 65–69. DOI:10.1007/BF00048208
27. Yokoya K, Walker S, Sarasan V. Regeneration of rose plants from cell and tissue cultures. *Acta Hortic.* **1996**, *424*, 333–338. DOI:10.17660/ActaHortic.1996.424.61
28. Doench JG, Fusi N, Sullender M, Hegde M, Vaimberg EW, Donovan KF, et al. Optimized sgRNA design to maximize activity and minimize off-target effects of CRISPR-Cas9. *Nat. Biotechnol.* **2016**, *34*, 184–191. DOI:10.1038/nbt.3437
29. Mao Y, Botella JR, Liu Y, Zhu JK. Gene editing in plants: Progress and challenges. *Natl. Sci. Rev.* **2019**, *6*, 421–437. DOI:10.1093/nsr/nwz005
30. Gao C. Genome engineering for crop improvement and future agriculture. *Cell* **2021**, *184*, 1621–1635. DOI:10.1016/j.cell.2021.01.005
31. Zhao Y, Liang Y, Ni Z, Sun Q, Zong Y, Wang Y. Advances and prospects of large DNA fragment editing in plants. *Nat. Plants* **2025**, *11*, 2461–2475. DOI:10.1038/s41477-025-02160-0
32. Su N, Wang M, Zhang R, Xiao Y, Zhang D, Song Y. Precision editing without footprints: Advancing transgene-free systems in plants. *Curr. Plant Biol.* **2026**, *46*, 100588. DOI:10.1016/j.cpb.2026.100588
33. Sun J, Lu J, Bai M, Chen Y, Wang W, Fan C, et al. Phytochrome-interacting factors interact with transcription factor CONSTANS to suppress flowering in rose. *Plant Physiol.* **2021**, *186*, 1186–1201. DOI:10.1093/plphys/kiab109
34. Yu R, Xiong Z, Zhu X, Feng P, Hu Z, Fang R, et al. RcSPL1–RcTAF15b regulates the flowering time of rose (*Rosa chinensis*). *Hortic. Res.* **2023**, *10*, uhad083. DOI:10.1093/hr/uhad083
35. Iwata H, Gaston A, Remay A, Thouroude T, Jeauffre J, Kawamura K, et al. The *TFL1* homologue *KSN* is a regulator of continuous flowering in rose and strawberry. *Plant. J.* **2012**, *69*, 116–125. DOI:10.1111/j.1365-313X.2011.04776.x
36. Randoux M, Jeauffre J, Thouroude T, Vasseur F, Hamama L, Juchaux M, et al. Gibberellins regulate the transcription of the continuous flowering regulator, RoKSN, a rose TFL1 homologue. *J. Exp. Bot.* **2012**, *63*, 6543–6554. DOI:10.1093/jxb/ers310
37. Soufflet-Freslon V, Araou E, Jeauffre J, Thouroude T, Chastellier A, Michel G, et al. Diversity and selection of the continuous-flowering gene, RoKSN, in rose. *Hortic. Res.* **2021**, *8*, 76. DOI:10.1038/s41438-021-00512-3
38. Hamama L, Bosselut J, Voisine L, Thouroude T, Ogé L, Chameau J, et al. Rose *FT* homologous gene overexpression affects flowering and vegetative development behavior in two different rose genotypes. *Plant Cell Tissue Organ Cult.* **2024**, *156*, 87. DOI:10.1007/s11240-024-02695-8
39. Sun J, Liu H, Wang W, Fan C, Yuan G, Zhou R, et al. RcOST1L phosphorylates RcPIF4 for proteasomal degradation to promote flowering in rose. *New Phytol.* **2024**, *243*, 1387–1405. DOI:10.1111/nph.19885
40. Wang W, Sun J, Fan C, Yuan G, Zhou R, Lu J, et al. RcSRR1 interferes with the RcCSN5B-mediated deneddylation of RcCRL4 to modulate RcCO proteolysis and prevent rose flowering under red light. *Hortic. Res.* **2025**, *12*, uhaf025. DOI:10.1093/hr/uhaf025
41. Franklin KA, Praekelt U, Stoddart WM, Billingham OE, Halliday KJ, Whitelam GC. Phytochromes B, D, and E act redundantly to control multiple physiological responses in *Arabidopsis*. *Plant Physiol.* **2003**, *131*, 1340–1346. DOI:10.1104/pp.102.015487
42. Galvão VC, Fiorucci AS, Trevisan M, Franco-Zorilla JM, Goyal A, Schmid-Siegert E, et al. PIF transcription factors link a neighbor threat cue to accelerated reproduction in *Arabidopsis*. *Nat. Commun.* **2019**, *10*, 4005. DOI:10.1038/s41467-019-11882-7
43. Wang LN, Liu YF, Zhang YM, Fang RX, Liu QL. The expression level of *Rosa Terminal Flower 1* (*RTFL1*) is related with recurrent flowering in roses. *Mol. Biol. Rep.* **2012**, *39*, 3737–3746. DOI:10.1007/s11033-011-1149-8
44. Randoux M, Davière J, Jeauffre J, Thouroude T, Pierre S, Toualbia Y, et al. RoKSN, a floral repressor, forms protein complexes with RoFD and RoFT to regulate vegetative and reproductive development in rose. *New Phytol.* **2014**, *202*, 161–173. DOI:10.1111/nph.12625
45. Bai M, Liu J, Fan C, Chen Y, Chen H, Lu J, et al. KSN heterozygosity is associated with continuous flowering of *Rosa rugosa* purple branch. *Hortic. Res.* **2021**, *8*, 26. DOI:10.1038/s41438-021-00464-8
46. Peng Y, Li Q, Gong Y, Yang Q, Dong Q, Han Y. RcPLATZ8 as a novel negative regulator of flowering in *Rosa chinensis*. *Plant Cell Rep.* **2025**, *44*, 125. DOI:10.1007/s00299-025-03513-x
47. Jing W, Gong F, Liu G, Deng Y, Liu J, Yang W, et al. Petal size is controlled by the MYB73/TPL/HDA19-miR159-CKX6 module regulating cytokinin catabolism in *Rosa hybrida*. *Nat. Commun.* **2023**, *14*, 7106. DOI:10.1038/s41467-023-42914-y

48. Wang Y, Qin M, Zhang G, Lu J, Zhang C, Ma N, et al. Transcription factor RhRAP2.4L orchestrates cell proliferation and expansion to control petal size in rose. *Plant Physiol.* **2024**, *194*, 2338–2353. DOI:10.1093/plphys/kiad657
49. Jin W, Gong F, Zhang Y, Wang R, Liu H, Wei Y, et al. Cytokinin-responsive RhRR1-RhSCL28 transcription factor module positively regulates petal size by promoting cell division in rose. *J. Exp. Bot.* **2025**, *76*, 381–392. DOI:10.1093/jxb/erae331
50. Pei H, Ma N, Tian J, Luo J, Chen J, Li J, et al. An NAC transcription factor controls ethylene-regulated cell expansion in flower petals. *Plant Physiol.* **2013**, *163*, 775–791. DOI:10.1104/pp.113.223388
51. Luo J, Ma N, Pei H, Chen J, Li J, Gao J. A DELLA gene, RhGAI1, is a direct target of EIN3 and mediates ethylene-regulated rose petal cell expansion via repressing the expression of *RhCesA2*. *J. Exp. Bot.* **2013**, *64*, 5075–5084. DOI:10.1093/jxb/ert296
52. Ma N, Xue J, Li Y, Liu X, Dai F, Jia W, et al. *Rh-PIP2;1*, a rose aquaporin gene, is involved in ethylene-regulated petal expansion. *Plant Physiol.* **2008**, *148*, 894–907. DOI:10.1104/pp.108.120154
53. Wei Y, Zhang Y, Li J, Gong F, Hua W, Liu H, et al. Unravelling the role of phytosulfokine (PSK) in regulating rose petal growth through cell proliferation. *Plant J.* **2025**, *123*, e70424. DOI:10.1111/tpj.70424
54. Chen C, Hussain N, Ma Y, Zuo L, Jiang Y, Sun X, et al. The ARF2–MYB6 module mediates auxin-regulated petal expansion in rose. *J. Exp. Bot.* **2023**, *74*, 4489–4502. DOI:10.1093/jxb/erad173
55. Gong F, Jing W, Jin W, Liu H, Zhang Y, Wang R, et al. RhMYC2 controls petal size through synergistic regulation of jasmonic acid and cytokinin signaling in rose. *Plant J.* **2024**, *120*, 459–472. DOI:10.1111/tpj.16993
56. Gong F, Wang X, Zhao Q, Wang D, Yan H, Wang Q, et al. The GAs-RhMYB70 feedback loop fine-tunes cell expansion and petal size by modulating cellulose content in rose. *Hortic. Res.* **2025**, *12*, uhaf134. DOI:10.1093/hr/uhaf134
57. Wang R, Guo Y, Qiu Y, Liu Y, Gong F, Li P, et al. Ethylene restrains brassinosteroid-driven petal growth through RhMPK7-mediated phosphorylation of RhBZR1 in rose. *New Phytol.* **2026**. DOI:10.1111/nph.71247
58. Ji S, Yang Y, Yang S, Jiang S, Cheng H, Liu T, et al. RcAP2L-RcAS1 complex modulates petal number in roses by targeting *RcAGL80* promoter. *Plant J.* **2025**, *124*, e70549. DOI:10.1111/tpj.70549
59. Chen W, Ding Z, Shen Y, Zhou Y, Luo P, Cui Y. Dioxygenase RhDAO1 regulates floral organ identity by controlling auxin homeostasis in miniature rose (*Rosa hybrida*). *Hortic. Plant J.* **2026**, *12*, 1185–1203. DOI:10.1016/j.hpj.2025.10.011
60. Chen J, Li Y, Li Y, Li Y, Wang Y, Jiang C, et al. AUXIN RESPONSE FACTOR 18–HISTONE DEACETYLASE 6 module regulates floral organ identity in rose (*Rosa hybrida*). *Plant Physiol.* **2021**, *186*, 1074–1087. DOI:10.1093/plphys/kiab130
61. Cheng C, Yu Q, Wang Y, Wang H, Dong Y, Ji Y, et al. Ethylene-regulated asymmetric growth of the petal base promotes flower opening in rose (*Rosa hybrida*). *Plant Cell* **2021**, *33*, 1229–1251. DOI:10.1093/plcell/koab031
62. Ma N, Ma C, Liu Y, Shahid MO, Wang C, Gao J. Petal senescence: A hormone view. *J. Exp. Bot.* **2018**, *69*, 719–732. DOI:10.1093/jxb/ery009
63. Ma N, Cai L, Lu W, Tan H, Gao J. Exogenous ethylene influences flower opening of cut roses (*Rosa hybrida*) by regulating the genes encoding ethylene biosynthesis enzymes. *Sci. China Ser. C Life Sci.* **2005**, *48*, 434–444. DOI:10.1360/062004-37
64. Lu J, Zhang G, Ma C, Li Y, Jiang C, Wang Y, et al. The F-box protein RhSAF destabilizes the gibberellic acid receptor RhGID1 to mediate ethylene-induced petal senescence in rose. *Plant Cell* **2024**, *36*, 1736–1754. DOI:10.1093/plcell/koae035
65. Ma N, Tan H, Liu X, Xue J, Li Y, Gao J. Transcriptional regulation of ethylene receptor and *CTR* genes involved in ethylene-induced flower opening in cut rose (*Rosa hybrida*) cv. Samantha. *J. Exp. Bot.* **2006**, *57*, 2763–2773. DOI:10.1093/jxb/erl033
66. Tan H, Liu X, Ma N, Xue J, Lu W, Bai J, et al. Ethylene-influenced flower opening and expression of genes encoding ETRs, CTRs, and EIN3s in two cut rose cultivars. *Postharvest Biol. Technol.* **2006**, *40*, 97–105. DOI:10.1016/j.postharvbio.2006.01.007
67. Wu Y, Zuo L, Ma Y, Jiang Y, Gao J, Tao J, et al. Protein kinase rhcipk6 promotes petal senescence in response to ethylene in rose (*Rosa Hybrida*). *Genes* **2022**, *13*, 1989. DOI:10.3390/genes13111989
68. Lü P, Zhang C, Liu J, Liu X, Jiang G, Jiang X, et al. RhHB1 mediates the antagonism of gibberellins to ABA and ethylene during rose (*Rosa hybrida*) petal senescence. *Plant J.* **2014**, *78*, 578–590. DOI:10.1111/tpj.12494
69. Luo J, Chen S, Cao S, Zhang T, Li R, Chan ZL, et al. Rose (*Rosa hybrida*) ethylene responsive factor 3 promotes rose flower senescence via direct activation of the abscisic acid synthesis-related *9-CIS-EPOXYCAROTENOID DIOXYGENASE* Gene. *Plant Cell Physiol.* **2021**, *62*, 1030–1043. DOI:10.1093/pcp/pcab085
70. Wu L, Ma N, Jia Y, Zhang Y, Feng M, Jiang CZ, et al. An ethylene-induced regulatory module delays flower senescence by regulating cytokinin content. *Plant Physiol.* **2017**, *173*, 853–862. DOI:10.1104/pp.16.01064

71. Khaskheli AJ, Ahmed W, Ma C, Zhang S, Liu Y, Li Y, et al. RhERF113 functions in ethylene-induced petal senescence by modulating cytokinin content in rose. *Plant Cell Physiol.* **2018**, *59*, 2442–2451. DOI:10.1093/pcp/pcy162
72. Chen C, Ma Y, Zuo L, Xiao Y, Jiang Y, Gao J. The CALCINEURIN B-LIKE 4/CBL-INTERACTING PROTEIN 3 module degrades repressor JAZ5 during rose petal senescence. *Plant Physiol.* **2023**, *193*, 1605–1620. DOI:10.1093/plphys/kiad365
73. Zhang S, Zhao Q, Zeng D, Xu J, Zhou H, Wang F, et al. RhMYB108, an R2R3-MYB transcription factor, is involved in ethylene- and JA-induced petal senescence in rose plants. *Hortic. Res.* **2019**, *6*, 131. DOI:10.1038/s41438-019-0221-8
74. Qin M, Wu Z, Zhang C, Jiang Y, Jiang CZ, Sun X, et al. The histone deacetylase RhHDA15 represses petal senescence by epigenetically regulating reactive oxygen species homeostasis in rose. *Plant Physiol.* **2024**, *197*, kiae612. DOI:10.1093/plphys/kiae612
75. Jiang L, Liu K, Zhang T, Chen J, Zhao S, Cui Y, et al. The RhWRKY33a-RhPLATZ9 regulatory module delays petal senescence by suppressing rapid reactive oxygen species accumulation in rose flowers. *Plant J.* **2023**, *114*, 1425–1442. DOI:10.1111/tpj.16202
76. Zhang Y, Wu Z, Feng M, Chen J, Qin M, Wang W, et al. The circadian-controlled PIF8-BBX28 module regulates petal senescence in rose flowers by governing mitochondrial ROS homeostasis at night. *Plant Cell* **2021**, *33*, 2716–2735. DOI:10.1093/plcell/koab152
77. Li P, Qiu Y, Wang R, Zhang B, Ma Y, Sun X, et al. The homeodomain leucine zipper protein RhHB22 promotes petal senescence by repressing ascorbic acid biosynthesis in rose. *J. Exp. Bot.* **2025**, *76*, 1704–1717. DOI:10.1093/jxb/erae503
78. Wang W, Chen C, Zhao Y, Zhang B, Wu Z, Sun X, et al. Transcription factors RhPIF4/8 and RhHY5 regulate autophagy-mediated petal senescence in rose (*Rosa hybrida*). *Hortic. Adv.* **2023**, *1*, 17. DOI:10.1007/s44281-023-00021-4
79. Gao Y, Liu Y, Liang Y, Lu J, Jiang C, Fei Z, et al. *Rosa hybrida* RhERF1 and RhERF4 mediate ethylene- and auxin-regulated petal abscission by influencing pectin degradation. *Plant. J.* **2019**, *99*, 1159–1171. DOI:10.1111/tpj.14412
80. Zhao Q, Zhou X, Chen Q, Yang R, Li Y, Zhao J, et al. ER-related E2-E3 ubiquitin enzyme pair regulates ethylene response by modulating the turnover of ethylene receptors. *Nat. Commun.* **2025**, *16*, 5937. DOI:10.1038/s41467-025-61066-9
81. Zhang B, Xiao Y, Liu Y, Wang W, Li P, Wang R, et al. The RhMPK3-RhLOB41-RhWRKY9 module orchestrates ethylene-induced petal abscission via dual control of ROS homeostasis in rose. *Sci. Adv.* **2026**, *12*, eadu6821. DOI: 10.1126/sciadv.adu6821
82. Liang Y, Jiang C, Liu Y, Gao Y, Lu J, Aiwaili P, et al. Auxin regulates sucrose transport to repress petal abscission in rose (*Rosa hybrida*). *Plant Cell* **2020**, *32*, 3485–3499. DOI:10.1105/tpc.19.00695
83. Gao Y, Liu C, Li X, Xu H, Liang Y, Ma N, et al. Transcriptome profiling of petal abscission zone and functional analysis of an Aux/IAA family gene RhIAA16 involved in petal shedding in rose. *Front. Plant Sci.* **2016**, *7*, 1375. DOI:10.3389/fpls.2016.01375
84. Eugster CH, Märki-Fischer E. The chemistry of rose pigments. *Angew. Chem. Int. Ed. Engl.* **1991**, *30*, 654–672. DOI:10.1002/anie.199106541
85. Grotewold E. The genetics and biochemistry of floral pigments. *Annu. Rev. Plant Biol.* **2006**, *57*, 761–780. DOI:10.1146/annurev.arplant.57.032905.105248
86. He G, Zhang R, Jiang S, Wang H, Ming F. The MYB transcription factor RcMYB1 plays a central role in rose anthocyanin biosynthesis. *Hortic. Res.* **2023**, *10*, uhad080. DOI:10.1093/hr/uhad080
87. Yan Y, Zhao J, Lin S, Li M, Liu J, Raymond O, et al. Light-mediated anthocyanin biosynthesis in rose petals involves a balanced regulatory module comprising transcription factors RhHY5, RhMYB114a, and RhMYB3b. *J. Exp. Bot.* **2023**, *74*, 5783–5804. DOI:10.1093/jxb/erad253
88. Ma M, Li R, Li Y, Dai W, Shang J, He Y, et al. Anthocyanin biosynthesis and transport synergistically modulated by RcMYB75 and RcGSTFL11 play a pivotal role in the feedforward loop in response to drought stress. *Plant J.* **2025**, *121*, e17240. DOI:10.1111/tpj.17240
89. Wang H, Yang Y, Kang Y, Li M, Luo J, Liu X, et al. Key transcription regulators in the formation of yellow traits in rose petals. *Plant Physiol. Biochem.* **2025**, *227*, 110174. DOI:10.1016/j.plaphy.2025.110174
90. Ma X, Li D, Cai J, Yang S, Wang D, Zhang Q, et al. Auxin response factor, RhARF8 contributes to rose flower color fading via regulating anthocyanin biosynthesis by directly activating *RhCHSa*/*c* promoter activity. *Ornam. Plant Res.* **2025**, *5*, e021. DOI:10.48130/opr-0025-0019
91. Castillejo C, Waurich V, Wagner H, Ramos R, Oiza N, Muñoz P, et al. Allelic variation of MYB10 is the major force controlling natural variation in skin and flesh color in strawberry (*Fragaria* spp.) Fruit. *Plant Cell* **2020**, *32*, 3723–3749. DOI:10.1105/tpc.20.00474
92. Jiu S, Guan L, Leng X, Zhang K, Haider MS, Yu X, et al. The role of VvMYBA2r and VvMYBA2w alleles of the MYBA2 locus in the regulation of anthocyanin biosynthesis for molecular breeding of grape (*Vitis* spp.) skin coloration. *Plant Biotechnol. J.* **2021**, *19*, 1216–1239. DOI:10.1111/pbi.13543

93. Gao Y, Wang F, Xia P, Zhang J, Peng Z, Liu Z, et al. Light-induced m6A RNA modification regulates anthocyanin accumulation during rose petal coloration. *Plant Physiol.* **2026**, *201*, kiag082. DOI:10.1093/plphys/kiag082
94. Muhlemann JK, Klempien A, Dudareva N. Floral volatiles: From biosynthesis to function. *Plant Cell Environ.* **2014**, *37*, 1936–1949. DOI:10.1111/pce.12314
95. Baudino S, Caissard JC, Bergougnoux V, Jullien F, Magnard JL, Scalliet G, et al. Production and emission of volatile compounds by petal cells. *Plant Signal. Behav.* **2007**, *2*, 525–526. DOI:10.4161/psb.2.6.4659
96. Bergougnoux V, Caissard JC, Jullien F, Magnard JL, Scalliet G, Cock JM, et al. Both the adaxial and abaxial epidermal layers of the rose petal emit volatile scent compounds. *Planta* **2007**, *226*, 853–866. DOI:10.1007/s00425-007-0531-1
97. Guterman I, Shalit M, Menda N, Piestun D, Dafny-Yelin M, Shalev G, et al. Rose scent: Genomics approach to discovering novel floral fragrance-related genes. *Plant Cell* **2002**, *14*, 2325–2338. DOI:10.1105/tpc.005207
98. Shalit M, Shafir S, Larkov O, Bar E, Kaslassi D, Adam Z, et al. Volatile compounds emitted by rose cultivars: Fragrance perception by man and honeybees. *Isr. J. Plant Sci.* **2004**, *52*, 245–255. DOI:10.1560/P7G3-FT41-XJCP-1XFM
99. Feng L, Chen C, Li T, Wang M, Tao J, Zhao D, et al. Flowery odor formation revealed by differential expression of monoterpene biosynthetic genes and monoterpene accumulation in rose (*Rosa rugosa* Thunb.). *Plant Physiol. Biochem.* **2014**, *75*, 80–88. DOI:10.1016/j.plaphy.2013.12.006
100. Farhi M, Lavie O, Masci T, Hendel-Rahmanim K, Weiss D, Abeliovich H, et al. Identification of rose phenylacetaldehyde synthase by functional complementation in yeast. *Plant Mol. Biol.* **2010**, *72*, 235–245. DOI:10.1007/s11103-009-9564-0
101. Chen X, Baldermann S, Cao S, Lu Y, Liu C, Hirata H, et al. Developmental patterns of emission of scent compounds and related gene expression in roses of the cultivar *Rosa* × *hybrida* cv. 'Yves Piaget'. *Plant Physiol. Biochem.* **2015**, *87*, 109–114. DOI:10.1016/j.plaphy.2014.12.016
102. Helsper JPFG, Davies JA, Bouwmeester HJ, Krol AF, van Kampen MH. Circadian rhythmicity in emission of volatile compounds by flowers of *Rosa hybrida* L. cv. Honesty. *Planta* **1998**, *207*, 88–95. DOI:10.1007/s004250050459
103. Hendel-Rahmanim K, Masci T, Vainstein A, Weiss D. Diurnal regulation of scent emission in rose flowers. *Planta* **2007**, *226*, 1491–1499. DOI:10.1007/s00425-007-0582-3
104. Picone J, Clery R, Watanabe N, MacTavish H, Turnbull CN. Rhythmic emission of floral volatiles from *Rosa damascena semperflorens* cv. 'Quatre Saisons'. *Planta* **2004**, *219*, 468–478. DOI:10.1007/s00425-004-1250-5
105. Magnard JL, Roccia A, Caissard JC, Vergne P, Sun P, Hecquet R, et al. Biosynthesis of monoterpene scent compounds in roses. *Science* **2015**, *349*, 81–83. DOI:10.1126/science.aab0696
106. Bai M, Wang M, Kou X, Xu X, Wei G, Feng L. The RrMYB4-RrTPS31 module regulates the synthesis of sesquiterpenoids in *Rosa rugosa*. *Plant Physiol. Biochem.* **2025**, *229*, 110708. DOI:10.1016/j.plaphy.2025.110708
107. Bao M, Xu Y, Wei G, Bai M, Wang J, Feng L. The MYC gene RrbHLH105 contributes to salt stress-induced geraniol in rose by regulating trehalose-6-phosphate signalling. *Plant Cell Environ.* **2025**, *48*, 1947–1962. DOI:10.1111/pce.15266
108. Roccia A, Hibrand-Saint Oyant L, Cavel E, Caissard JC, Machenaud J, Thouroude T, et al. Biosynthesis of 2-phenylethanol in rose petals is linked to the expression of one allele of RhPAAS. *Plant Physiol.* **2019**, *179*, 1064–1079. DOI:10.1104/pp.18.01468
109. Li Y, Li R, Shang J, Zhao K, Sui Y, Liu Z, et al. A *de novo*-originated gene drives rose scent diversification. *Cell* **2025**, *188*, 6121–6137.e24. DOI:10.1016/j.cell.2025.08.011
110. Wu S, Watanabe N, Mita S, Dohra H, Ueda Y, Shibuya M, et al. The key role of phloroglucinol O-methyltransferase in the biosynthesis of *Rosa chinensis* volatile 1,3,5-trimethoxybenzene. *Plant Physiol.* **2004**, *135*, 95–102. DOI:10.1104/pp.103.037051
111. Liu X, Li D, Zhang S, Xu Y, Zhang Z. Genome-wide characterization of the rose (*Rosa chinensis*) WRKY family and role of RcWRKY41 in gray mold resistance. *BMC Plant Biol.* **2019**, *19*, 522. DOI:10.1186/s12870-019-2139-6
112. Fang P, Arens P, Liu X, Zhang X, Lakwani D, Foucher F, et al. Analysis of allelic variants of *RhMLO* genes in rose and functional studies on susceptibility to powdery mildew related to clade V homologs. *Theor. Appl. Genet.* **2021**, *134*, 2495–2515. DOI:10.1007/s00122-021-03838-7
113. Terefe-Ayana D, Yasmin A, Le TL, Kaufmann H, Biber A, Kühr A, et al. Mining disease-resistance genes in roses: Functional and molecular characterization of the *rdr1* locus. *Front. Plant Sci.* **2011**, *2*, 35. DOI:10.3389/fpls.2011.00035
114. Liu X, Zhou X, Li D, Hong B, Gao J, Zhang Z. Rose WRKY13 promotes disease protection to *Botrytis* by enhancing cytokinin content and reducing abscisic acid signaling. *Plant Physiol.* **2023**, *191*, 679–693. DOI:10.1093/plphys/kiac495
115. Li D, Li X, Wang Z, Wang H, Gao J, Liu X, et al. Transcription factors RhbZIP17 and RhWRKY30 enhance resistance to *Botrytis cinerea* by increasing lignin content in rose petals. *J. Exp. Bot.* **2024**, *75*, 1633–1646. DOI:10.1093/jxb/erad473
116. Li D, Liu X, Shu L, Zhang H, Zhang S, Song Y, et al. Global analysis of the AP2/ERF gene family in rose (*Rosa chinensis*) genome unveils the role of RcERF099 in Botrytis resistance. *BMC Plant Biol.* **2020**, *20*, 533. DOI:10.1186/s12870-020-02740-6

117. Liu X, Cao X, Chen M, Li D, Zhang Z. Two transcription factors, RhERF005 and RhCCCH12, regulate rose resistance to *Botrytis cinerea* by modulating cytokinin levels. *J. Exp. Bot.* **2024**, *75*, 2584–2597. DOI:10.1093/jxb/erae040
118. Ding C, Gao J, Zhang S, Jiang N, Su D, Huang X, et al. The basic/helix-loop-helix transcription factor family gene RcbHLH112 is a susceptibility gene in gray mold resistance of rose (*Rosa Chinensis*). *Int. J. Mol. Sci.* **2023**, *24*, 16305. DOI:10.3390/ijms242216305
119. Li D, Li Z, Wang H, Jian R, Yang Y, Wang X, et al. Genome-wide and transcriptome analyses reveal RcCDPK5 as a positive regulator mediating resistance to Botrytis cinerea in *Rosa chinensis*. *Ind. Crop. Prod.* **2026**, *240*, 122666. DOI:10.1016/j.indcrop.2026.122666
120. Liu X, Wang Z, Tian Y, Zhang S, Li D, Dong W, et al. Characterization of wall-associated kinase/wall-associated kinase-like (WAK/WAKL) family in rose (*Rosa chinensis*) reveals the role of RcWAK4 in Botrytis resistance. *BMC Plant Biol.* **2021**, *21*, 526. DOI:10.1186/s12870-021-03307-9
121. Li X, He G, Jiang S, Yang C, Yang B, Ming F. Function of two splicing variants of RcCPR5 in the resistance of *Rosa chinensis* to powdery mildew. *Plant Sci.* **2023**, *335*, 111678. DOI:10.1016/j.plantsci.2023.111678
122. Whitaker VM, Bradeen JM, Debener T, Biber A, Hokanson SC. Rdr3, a novel locus conferring black spot disease resistance in tetraploid rose: Genetic analysis, LRR profiling, and SCAR marker development. *Theor. Appl. Genet.* **2010**, *120*, 573–585. DOI:10.1007/s00122-009-1177-0
123. Cheng H, Yang Y, Su L, Qi Y, Ji S, Liu T, et al. RcPsaL and RcPsbX, core photosystem subunits targeted by *Marssonina rosae* effector MrSEP20, enhance black spot resistance in rose. *Plant Physiol. Biochem.* **2025**, *229*, 110639. DOI:10.1016/j.plaphy.2025.110639
124. Yang Y, Qi Y, Su L, Yang S, Yi X, Luo L, et al. The *Marssonina rosae* effector MrSEP43 suppresses immunity in rose by targeting the orphan protein RcBROG. *J. Exp. Bot.* **2024**, *75*, 4993–5007. DOI:10.1093/jxb/erae200
125. Han Y, Tang A, Wan H, Zhang T, Cheng T, Wang J, et al. An APETALA2 homolog, RcAP2, regulates the number of rose petals derived from stamens and response to temperature fluctuations. *Front. Plant Sci.* **2018**, *9*, 481. DOI:10.3389/fpls.2018.00481
126. Yang T, Wang Y, Li Y, Liang S, Yang Y, Huang Z, et al. The transcription factor RhMYB17 regulates the homeotic transformation of floral organs in rose (*Rosa hybrida*) under cold stress. *J. Exp. Bot.* **2024**, *75*, 2965–2981. DOI:10.1093/jxb/erae099
127. Lu J, Wang W, Fan C, Sun J, Yuan G, Guo Y, et al. Telo boxes within the AGAMOUS second intron recruit histone 3 lysine 27 methylation to increase petal number in rose (*Rosa chinensis*) in response to low temperatures. *Plant J.* **2024**, *118*, 1486–1499. DOI:10.1111/tpj.16691
128. Zaccai M, Ackerman R, Genis O, Riov J, Zik M. The bent peduncle phenomenon in roses is a developmental process involving auxin. *Plant Sci.* **2009**, *176*, 736–743. DOI:10.1016/j.plantsci.2009.02.014
129. Qi W, Zhang C, Wang W, Cao Z, Li S, Li H, et al. Comparative transcriptome analysis of different heat stress responses between self-root grafting line and heterogeneous grafting line in rose. *Hortic. Plant J.* **2021**, *7*, 243–255. DOI:10.1016/j.hpj.2021.03.004
130. Li ZQ, Xing W, Luo P, Zhang FJ, Jin XL, Zhang MH. Comparative transcriptome analysis of Rosa chinensis 'Slater's crimson China' provides insights into the crucial factors and signaling pathways in heat stress response. *Plant Physiol. Biochem.* **2019**, *142*, 312–331. DOI:10.1016/j.plaphy.2019.07.002
131. Sun Y, Li S, Wu X, Zhu J, Dong F, Pei Z, et al. Characterization and functional analysis of RhHsfA7, a heat stress transcription factor in roses (*Rosa hybrid* 'Samantha'). *Plants* **2025**, *14*, 1155. DOI:10.3390/plants14081155
132. Zhang J, Cao Y, Jin X, Zhang M, Wen Y, Li Z, et al. Integrated ATAC-seq and RNA-seq reveal RcHSF30 regulating sHSP and BAG for thermotolerance in rose. *Ind. Crop. Prod.* **2025**, *233*, 121440. DOI:10.1016/j.indcrop.2025.121440
133. Zhang Y, Yu S, Niu P, Su L, Jiao X, Sui X, et al. RcMYB8 enhances salt and drought tolerance in rose (*Rosa chinensis*) by modulating RcPR5/1 and RcP5CS1. *Mol. Hortic.* **2024**, *4*, 3. DOI:10.1186/s43897-024-00080-9
134. Geng L, Yu S, Zhang Y, Su L, Lu W, Zhu H, et al. Transcription factor RcNAC091 enhances rose drought tolerance through the abscisic acid–dependent pathway. *Plant Physiol.* **2023**, *193*, 1695–1712. DOI:10.1093/plphys/kiad366
135. Chen JR, Chen YB, Ziemiańska M, Liu R, Deng ZN, Niedźwiecka-Filipiak I, et al. Co-expression of *MtDREB1C* and *RcXET* enhances stress tolerance of transgenic china rose (*Rosa chinensis* Jacq.). *J. Plant Growth Regul.* **2016**, *35*, 586–599. DOI:10.1007/s00344-015-9564-z
136. Zang F, Wu Q, Li Z, Li L, Xie X, Tong B, et al. RrWRKY1, a transcription factor, is involved in the regulation of the salt stress response in *rosa rugosa*. *Plants* **2024**, *13*, 2973. DOI:10.3390/plants13212973
137. Yang Z, Lin L, Lu M, Ma W, An H. A bHLH transcription factor RrUNE12 regulates salt tolerance and promotes ascorbate synthesis. *Plant Cell Rep.* **2025**, *44*, 42. DOI:10.1007/s00299-025-03428-7

138. Su L, Zhang Y, Yu S, Geng L, Lin S, Ouyang L, et al. RcbHLH59-RcPRs module enhances salinity stress tolerance by balancing $Na^{(+)}/K^{(+)}$ through callose deposition in rose (*Rosa chinensis*). *Hortic. Res.* **2023**, *10*, uhac291. DOI:10.1093/hr/uhac291
139. Yoshiba Y, Kiyosue T, Katagiri T, Ueda H, Mizoguchi T, Yamaguchi-Shinozaki K, et al. Correlation between the induction of a gene for delta 1-pyrroline-5-carboxylate synthetase and the accumulation of proline in Arabidopsis thaliana under osmotic stress. *Plant. J.* **1995**, *7*, 751–760. DOI:10.1046/j.1365-313X.1995.07050751.x
140. Teng Y, Lv M, Zhang X, Cai M, Chen T. BEAR1, a bHLH transcription factor, controls salt response genes to regulate rice salt response. *J. Plant Biol.* **2022**, *65*, 217–230. DOI:10.1007/s12374-022-09347-4
141. Zhang S, Feng M, Chen W, Zhou X, Lu J, Wang Y, et al. In rose, transcription factor PTM balances growth and drought survival via PIP2;1 aquaporin. *Nat. Plants* **2019**, *5*, 290–299. DOI:10.1038/s41477-019-0376-1
142. Shen Y, Zou J, Zhang Q, Luo P, Shang W, Sun T, et al. Identification of PP2Cs in six rosaceae species highlights RcPP2C24 as a negative regulator in rose drought tolerance. *Plant Physiol. Biochem.* **2024**, *212*, 108782. DOI:10.1016/j.plaphy.2024.108782
143. Shang X, Xie N, Li Y, Zhao Z, Luo P, Cui Y, et al. Mediator subunit RhMED15a regulates drought tolerance in rose. *Horticulturae* **2024**, *10*, 84. DOI:10.3390/horticulturae10010084
144. Xie N, Shi H, Shang X, Zhao Z, Fang Y, Wu H, et al. RhMED15a-like, a subunit of the mediator complex, is involved in the drought stress response in *Rosa hybrida*. *BMC Plant Biol.* **2024**, *24*, 351. DOI:10.1186/s12870-024-05059-8
145. Zhao Q, Jing W, Fu X, Yang R, Zhu C, Zhao J, et al. TSPO-induced degradation of the ethylene receptor RhETR3 promotes salt tolerance in rose (*Rosa hybrida*). *Hortic. Res.* **2024**, *11*, uhae040. DOI:10.1093/hr/uhae040
146. Dai F, Zhang C, Jiang X, Kang M, Yin X, Lü P, et al. RhNAC2 and RhEXPA4 are involved in the regulation of dehydration tolerance during the expansion of rose petals. *Plant Physiol.* **2012**, *160*, 2064–2082. DOI:10.1104/pp.112.207720
147. Jiang X, Zhang C, Lü P, Jiang G, Liu X, Dai F, et al. RhNAC3, a stress-associated NAC transcription factor, has a role in dehydration tolerance through regulating osmotic stress-related genes in rose petals. *Plant Biotechnol. J.* **2014**, *12*, 38–48. DOI:10.1111/pbi.12114
148. Zou J, Lü P, Jiang L, Liu K, Zhang T, Chen J, et al. Regulation of rose petal dehydration tolerance and senescence by RhNAP transcription factor via the modulation of cytokinin catabolism. *Mol. Hortic.* **2021**, *1*, 13. DOI:10.1186/s43897-021-00016-7
149. Luo P, Zhang H, Chen Y, Cui Y, Chen W. RhbHLH92 positively regulates the dehydration tolerance by interacting with RhMYB123 in rose petals (*Rosa hybrida*). *Environ. Exp. Bot.* **2024**, *228*, 106049. DOI:10.1016/j.envexpbot.2024.106049
150. Liu K, Zhang T, Zhao S, Chen J, Zhou W, Chen S, et al. Scaffold protein RhCASPL1D1 stabilizes RhPIP2 aquaporins and promotes flower recovery after dehydration in rose (*Rosa hybrida*). *Hortic. Res.* **2015**, *12*, uhaf119. DOI:10.1093/hr/uhaf119
151. Meng Y, Ma N, Zhang Q, You Q, Li N, Ali Khan M, et al. Precise spatio-temporal modulation of ACC synthase by MPK6 cascade mediates the response of rose flowers to rehydration. *Plant J.* **2014**, *79*, 941–950. DOI:10.1111/tpj.12594
152. Chen J, Zhang Q, Wang Q, Feng M, Li Y, Meng Y, et al. RhMKK9, a rose MAP KINASE KINASE gene, is involved in rehydration-triggered ethylene production in rose gynoecia. *BMC Plant Biol.* **2017**, *17*, 51. DOI:10.1186/s12870-017-0999-1
153. Zhou N, Simonneau F, Thouroude T, Oyant LHS, Foucher F. Morphological studies of rose prickles provide new insights. *Hortic. Res.* **2021**, *8*, 221. DOI:10.1038/s41438-021-00689-7
154. Kumari P, Gangwar H, Gahlaut V, Kumari P, Jaiswal V. Identification of a natural RcLOG1 allele linked to prickle development in Rose (*Rosa* spp.). *Planta* **2025**, *262*, 101. DOI:10.1007/s00425-025-04817-8
155. Lee KY, Lee SY, Kim YJ, Choi YJ, Lim SH, Kang YI. Overexpression of *VtF3'5'H* and *RhNHX* genes alters flower color and plant morphology in transgenic rose 'Red Farm'. *Plants* **2025**, *14*, 3185. DOI:10.3390/plants14203185
156. Zeng H, Zhou SZ, Zhang HL, Zhao BY, Liu YL, Zeng YH, et al. Pan-plantae aquaporin evolution: insights into PIP2 as a potential regulator of rose prickle development. *Hortic. Res.***2016**, uhag143. DOI:10.1093/hr/uhag143
157. Zhou NN, Tang KX, Jeauffre J, Thouroude T, Arias DCL, Foucher F, et al. Genetic determinism of prickles in rose. *Theor. Appl. Genet.* **2020**, *133*, 3017–3035. DOI:10.1007/s00122-020-03652-7
158. Steinberger AR, Voytas DF. Virus-induced gene editing free from tissue culture. *Nat. Plants* **2025**, *11*, 1241–1251. DOI:10.1038/s41477-025-02025-6