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Review Article

Delaying Floral Senescence: Molecular Mechanisms and Ethylene Regulation

Abayneh Melke Woldegebriel

Debre Markos University, College of Agriculture and Natural Resources Management, Debre Markos, Ethiopia
Correspondence: abaynehmelke@gmail.com / Abayneh_melke@dmu.edu.et

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ABSTRACT

Floral senescence the natural process leading to the decline and eventual death of flowers is a major factor influencing flower shelf-life and aesthetic value, especially within the floriculture sector where postharvest longevity holds economic importance. This review highlights current progress in deciphering the molecular pathways and hormonal signals that regulate this aging process, with a specific focus on ethylene. Ethylene, a central plant hormone, serves as a key modulator of floral aging by activating intricate signaling cascades and gene expression networks. It influences numerous transcription factors, notably the Ethylene Response Factors (ERFs), and modulates the activity of senescence-associated genes (*SAGs*), thereby accelerating the senescence process in flowers that are responsive to ethylene. This review provides a detailed examination of the physiological and molecular changes occurring during floral senescence, focusing on ethylene's effects on gene regulation, transcription factors such as *RhWRKY33*, and other key components of senescence pathways. The review further explores the genetic regulation of floral senescence, highlighting ethylene's interaction with other plant hormones like *auxin*, *cytokinins*, *polyamines*, and *abscisic acid*. Recent research on gene regulation mechanisms and signaling pathways is discussed, alongside technological advancements like ethylene inhibitors and genetic engineering approaches aimed at mitigating floral senescence. The implications of ethylene's role in gene regulation and its interaction with other phytohormones are examined in depth. We also analyze the feasibility of applying ethylene-related technologies from both an economic and practical perspective, including a comparison of hormone treatment costs and their impact on flower pricing and longevity in the floriculture market. Despite the focus on ethylene, other environmental and hormonal factors such as climate conditions, macronutrients, and ecosystem influences are also considered in relation to their simultaneous impact on floral senescence. This review concludes by offering future research directions to address existing gaps in our understanding and provide potential solutions for enhancing flower quality, longevity, and productivity.

Keywords: Floral senescence, ethylene regulation, gene expression, RhWRKY33, transcription factors, genetic engineering

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INTRODUCTION

The global floriculture industry has seen substantial expansion in recent years, largely fueled by rising consumer interest in ornamental flowers and plants. With an estimated market value exceeding *USD* 50 billion and growing annually, this sector plays a pivotal economic role by generating employment and supporting allied industries such as transportation, packaging, and retail (International Trade Centre, 2023; United Nations, 2023). Cut flowers are especially valued for their aesthetic appeal, fragrance, and variety, making them integral to events such as weddings, cultural celebrations, and festivals, thereby sustaining high market demand (Jones et al., 2024). Countries like the Netherlands, Colombia, and Kenya are among the leading exporters, while major importers include the United States and Japan (European Union, 2023; *USDA*, 2023). Colombia's flower industry stands out as a major contributor to its national economy and a key source of employment (Gómez et al., 2023).

Despite the sector's growth, floral senescence, the biological aging of flower remains a persistent issue that affects product quality and shelf life. Characterized by programmed cell death (*PCD*), floral senescence involves a series of physiological and biochemical shifts that negatively impact attributes such as petal color, texture, and scent (Van Doorn & Woltering, 2022). The pace of this deterioration is influenced by a combination of genetic traits, environmental conditions, and handling techniques, often resulting in wilted petals, discoloration, and reduced market value (Morris et al., 2023). These quality losses translate into economic drawbacks, including diminished sales and higher costs related to waste disposal (Lee et al., 2022).

To mitigate these challenges, extensive research has been directed toward improving flower longevity through post-harvest treatments and technological interventions. A deep understanding of the molecular frameworks that control senescence is essential for developing such preservation strategies (Pérez et al., 2023; Davis et al., 2023). Ethylene, a crucial plant hormone, plays a dominant role in regulating the senescence process. Recent advances have shed light on ethylene biosynthesis, its signaling networks, and the molecular pathways through which it influences flower aging. Ethylene triggers the expression of senescence-associated genes (*SAGs*) and modulates key transcription factors, accelerating aging in ethylene-sensitive species (Khan et al., 2017; Liu et al., 2024). Moreover, innovative approaches such as ethylene inhibitors and gene-silencing techniques targeting transcription factors like *RhWRKY33* offer promising avenues for enhancing floral longevity (Zhang et al., 2023).

This review presents a detailed exploration of floral senescence, with an emphasis on the hormonal and molecular mechanisms involved, particularly the role of ethylene in regulating gene activity and transcriptional control. Additionally, it discusses emerging technologies in genetic engineering and post-harvest management that aim to extend flower shelf life. By integrating current biological insights with technological progress, the review highlights pathways to enhance flower quality and durability. Finally, it underscores the importance of collaborative efforts between scientific researchers and industry stakeholders to address the ongoing challenges of floral senescence and to drive innovation in floriculture.

METHODOLOGY

This review consolidates current research on the molecular basis of floral senescence, with a particular emphasis on ethylene's regulatory functions, associated signaling pathways, key regulatory genes, and interactions with other hormones. A systematic literature search was conducted across established academic databases, including PubMed, Scopus, Web of Science, and Google Scholar. Studies were selected based on their methodological relevance, particularly those employing genetic analyses, transcriptome sequencing, and hormonal profiling techniques.

The search was conducted with the following keywords: "floral senescence," "molecular mechanisms," "ethylene regulation," "gene expression," and "floriculture," focusing on studies published in the last fifteen years. The inclusion criteria encompassed peer-reviewed journal articles, original research, and comprehensive reviews that addressed the molecular mechanisms, gene regulation, and the role of ethylene in floral senescence. Articles not directly related to the topic and non-English publications were excluded from the review. The search process, including the terms and databases used, was systematically documented.

The results were organized thematically, with a focus on ethylene-sensitive versus ethylene-insensitive flowers, gene regulation, key transcription factors like RhWRKY33, and the effects of ethylene on flower senescence. Comparative analysis was employed to summarize findings, identify common themes, and highlight any discrepancies or gaps in current research. Finally, the review provides recommendations for future research

Effects of Ethylene on post-harvest quality Flowers

Ethylene, an endogenous phytohormone, serves as a key regulator of floral senescence, profoundly influencing both the lifespan and overall quality of flowers. When flowers are cut from the plant, they initiate ethylene production as part of their natural life cycle. This internal ethylene production is further amplified by external sources such as tobacco smoke, exhaust fumes, fruits, and microorganisms, which contaminate the surrounding atmosphere. These external sources of ethylene, when absorbed by flowers, accelerate their senescence and reduce their resistance to environmental stress (Lee et al., 2022).

The influence of ethylene on flowers is largely adverse, causing various unwanted changes in appearance and function. These include bent necks, abnormal opening, petal wilting, petal shattering, discoloration, and flower popping, and yellowing or shedding of foliage (Liu et al., 2023). Such changes not only diminish the aesthetic quality of the flowers but also reduce their market value by affecting flower development, color, and consistency. As a result, ethylene exposure can lead to dissatisfaction among consumers, who expect fresh, vibrant flowers with long vase lives For the floriculture industry, managing ethylene levels throughout the distribution chain from storage to sale is crucial for ensuring flowers reach consumers in optimal condition (Pérez et al., 2023; Smith et al., 2024). Without effective control of ethylene exposure, the flower industry faces financial losses, decreased shelf life, and challenges to sustainability efforts.

Although ethylene is a naturally occurring hormone, its effects on cut flowers are particularly pronounced. Ethylene is colorless, odorless, and gaseous, making it difficult to detect without specialized equipment. All plants produce ethylene, and while some species or varieties are more sensitive to its effects (Table 1), the damage often goes unnoticed until the flowers are removed from cold storage, reach the point of sale, or are in the consumer's home (Morris et al., 2023). The combined impact of internally produced ethylene and external sources accelerates the flower's life cycle, leading to premature senescence and reduced flower quality (Fig. 1). Ethylene’s detrimental effects on flowers underscore the importance of monitoring and managing its presence to preserve flower quality, extend their vase life, and meet consumer expectations. Effective mitigation strategies, including the use of ethylene inhibitors and optimal handling conditions, are essential for minimizing the adverse effects of this hormone on cut flowers (Lee et al., 2023)

Table 1: How cut flower species are affected by ethylene damage (Felix Instruments, 2024).

Scientific Name	Common Names	Symptoms of Ethylene Damage
Alstroemeria spp.	Alstroemeria Peruvian Lily	Leaf-yellowing; petal transparency
Anemone spp.	Anemone	Bloom abscission; shattering
Antirrhinum majus	Snapdragon	Bloom abscission; petal transparency
Bouvardia spp.	Bouvardia	Bloom shattering; wilting
Campanula spp.	Bellflower	Premature flower; wilting
Chamelaucium uncinatum	Wax Flower	Bloom abscission; leaf abscission
Consolida spp.	Larkspur	Bloom abscission; shattering
Delphinium spp.	Delphinium	Bloom abscission; shattering
Dianthus spp.	Carnation	In-rolling of petals; "sleepiness"
Euphorbia pulcherrima	Poinsettia	Leaf and bract epinasty (downward curl)
Eustoma grandiflorum	Lisianthus	Flower wilting and reduced vase life
Freesia spp.	Freesia	Young buds fail to open; flower wilting
Gypsophila paniculata	Baby's Breath	Excessive brown flowers

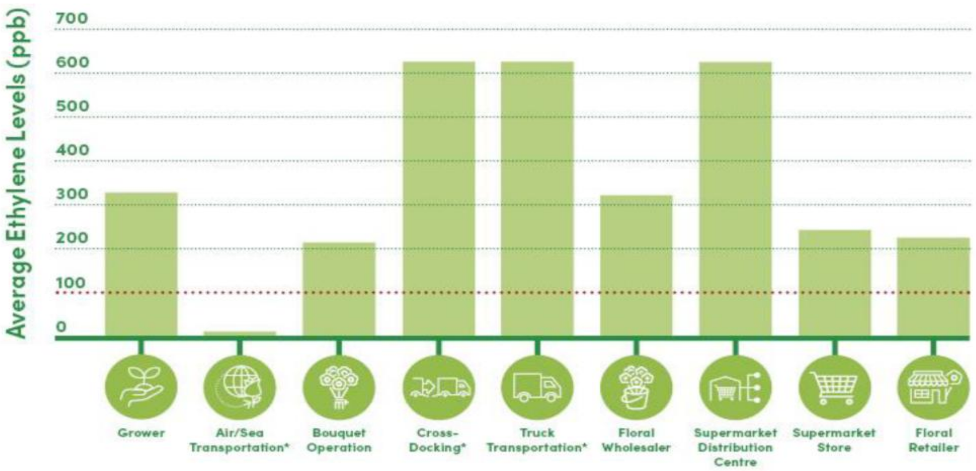

Figure 1: Sources of ethylene in the supply chain (Felix Instruments, 2024). The graph shows the average amount of ethylene exposure encountered at each stage of the supply chain. Ethylene damage starts at 100 ppb

As indicated in Figure 1, the negative effects of ethylene on the flower industry include several critical challenges that can result in significant financial losses. Flowers may not withstand longer shipment periods, such as sea freight, leading to products that do not reach retailers in sellable condition (Fig. 1). This quality reduction extends to retail displays, where flowers exhibit a shorter shelf life, and to consumers, who experience a diminished vase life in their homes, leading to dissatisfaction. These issues also negatively impact sustainability efforts, culminating in financial losses, customer dissatisfaction, and damage to brand reputation (Felix Instruments, 2024; van Doorn et al., 2011). To mitigate these detrimental effects, several preventative measures can be implemented. While the developmental stages of flowers are irreversible, ethylene production can be managed effectively. Since ethylene is always present, often undetectable, it is recommended to treat flowers with action inhibitors such as EthylBloc™ or FloraLife® EthylGuard. These compounds function by attaching to ethylene receptors within plant cells, thereby preventing the hormone from triggering its usual responses. Once applied, the inhibitors remain effective even after flowers are unpacked, with their protective effects lasting for approximately 10 to 14 days. For optimal results, it is crucial to apply these treatments consistently throughout the entire supply chain; ensuring floral quality is maintained from production to the end consumer (Wang et al., 2018). Additional recommendations include maintaining temperature control and following proper care and handling procedures throughout the distribution chain. These practices will enhance vase life, increase resilience to external conditions, reduce waste, and positively impact sustainability efforts, ultimately boosting profits (Felix Instruments, 2024; Schlereth et al., 2010).

Floral Senescence: Effects on Market Demand and Industry Constraints

Floral senescence, referring to the post-harvest aging of cut flowers, involves a complex series of physiological and biochemical changes that manifest as visible deterioration, including wilting, petal abscission, and loss of color vibrancy. These aging symptoms result from processes such as enzymatic breakdown of cell wall components, reduced cellular turgor, and pigment degradation (Lee et al., 2023). The onset of senescence severely impacts the visual appeal and commercial viability of flowers. For the floriculture sector, shorter flower lifespan necessitates frequent restocking, increasing operational expenses for producers, wholesalers, and retailers alike. Rapid flower deterioration also contributes to heightened waste levels, especially when blooms fail to meet retail or consumer quality expectations (Zhao et al., 2022). Therefore, effectively managing and delaying senescence is vital for enhancing profitability and ensuring market competitiveness (Smith & Reynolds, 2023).

Consumer buying behavior is closely tied to flower freshness and longevity. Shoppers tend to prefer blooms that retain their appearance for extended durations, which positively influences customer satisfaction, repeat purchases, and overall demand (Liu et al., 2023). However, managing senescence across the supply chain presents several challenges, including the need for improved post-harvest handling, the development of more effective floral preservatives, and a deeper understanding of the biological mechanisms responsible for flower aging. Meeting these challenges requires an integrated approach combining innovations in molecular genetics, hormone regulation, and environmental management to enhance flower longevity and reduce waste (Dai et al., 2021).

To address evolving consumer expectations for longer-lasting flowers, the floriculture industry has embraced technological advancements aimed at preserving floral freshness. New preservative formulations and hormone-based treatments have been introduced to delay senescence and maintain bloom quality (Seymour et al., 2019). Moreover, improvements in storage technologies, such as controlled atmosphere systems and advanced refrigeration methods, are being used to maintain flower viability during shipping and retail display (Zhang et al., 2021). A central focus of these efforts is ethylene regulation, as this hormone plays a dominant role in floral aging. The use of ethylene inhibitors has shown promise in extending flower shelf life and maintaining quality throughout distribution (Zhao et al., 2022). These strategies are crucial for aligning with consumer demands and addressing long-standing industry hurdles, ultimately improving product performance and profitability.

The process of floral senescence progresses through distinct phases. **Early-stage senescence** is marked by subtle signs such as initial wilting and pigment fading. In the **mid-stage,** more severe symptoms like petal shedding and elevated ethylene production become apparent. **Late-stage senescence** is characterized by extensive tissue breakdown, complete color loss, and structural collapse, rendering flowers commercially unmarketable (Seymour et al., 2019). Ethylene accelerates this sequence by modulating physiological processes such as protein degradation, lipid peroxidation, loss of water content, and cell wall weakening, all of which contribute to visible flower decay (Zhang et al., 2020). Additionally, pigment breakdown, particularly of anthocyanins and carotenoids, further contributes to the loss of flower coloration (Kumar et al., 2023).

Economically, floral senescence poses significant challenges across the value chain. Flowers that retain their aesthetic appeal longer command higher prices, reduce product losses, and enhance consumer satisfaction. In contrast, rapid senescence diminishes flower quality and shelf life, increasing waste and reducing returns for growers and sellers (Dai et al., 2019). These challenges are further compounded by rising costs associated with more frequent harvesting, labor, and product handling (Wang et al., 2021). Fast-aging flowers may also require specialized logistics such as temperature and humidity control which adds to distribution costs. At the retail level, short shelf life contributes to unsold inventory and product spoilage, adversely affecting profit margins (Smith & Reynolds, 2019; Williams & Morgan, 2022).

Progress in Mitigating Floral Aging: Mechanisms, Regulatory Networks, and the Central Role of Ethylene

Floral senescence is governed by complex regulatory systems involving a network of hormones, transcription factors, and signaling molecules (see Fig. 2). Ethylene acts as a dominant hormone driving the progression of flower aging, markedly accelerating the deterioration of floral quality. Alongside ethylene, other hormones like abscisic acid (*ABA*) and jasmonic acid (*JA*) also play significant roles by influencing senescence through distinct signaling routes (Zhao et al., 2022). Key transcription factors including members of the *WRKY, MYB*, and *NAC* families are instrumental in controlling the expression of senescence-associated genes, orchestrating the genetic programs that underlie the aging process (Havaux et al., 2019). Beyond hormonal regulation, signaling cascades such as the Mitogen-Activated Protein Kinase (*MAPK*) and cyclic Guanosine Monophosphate (*cGMP*) pathways serve as integrators of hormonal, environmental, and developmental cues to finely tune the senescence process (Wang et al., 2021).

The senescence phase involves substantial metabolic and cellular reprogramming. Enzymatic remodeling of the cell wall, driven by cellulases and pectinases, is intensified to promote cell death and tissue breakdown, leading to visible wilting and floral degradation (Zhang et al., 2020). Concurrently, there is a metabolic shift from biosynthesis to catabolism, which fuels the energy requirements for senescence, characterized by increased respiration and altered carbohydrate metabolism (Li et al., 2021). Additionally, the accumulation of secondary metabolites such as antioxidants and flavonoids provides protection against oxidative damage during this phase.

Ethylene biosynthesis in flowers proceeds via the methionine-dependent pathway, where methionine is first converted into S-adenosylmethionine (*SAM*) by *SAM* synthetase. *SAM* is then transformed into 1-aminocyclopropane-1-carboxylic acid (*ACC*) by *ACC* synthase (*ACS*), followed by oxidation of ACC to ethylene by ACC oxidase (Wang et al., 2019; Yoshida et al., 2023). This hormone promotes key processes like cell wall disassembly and pigment degradation, which collectively reduce flower vase life and hasten wilting (Bleecker & Kende, 2023).

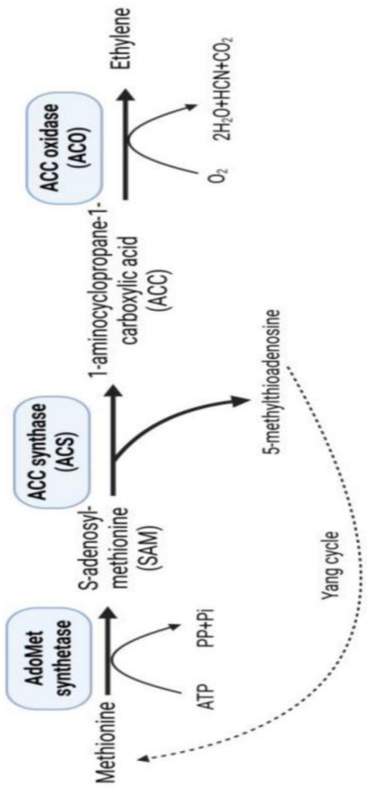

Figure 2: Schematic representation of the ethylene biosynthesis pathway highlighting the main enzymatic steps. Methionine is first converted to S-adenosylmethionine (SAM), which is then transformed into 1-aminocyclopropane-1-carboxylic acid (ACC) by ACC synthase (ACS). Finally, ACC oxidase (ACO) catalyzes the formation of ethylene from ACC. (Adapted from Yang & Hoffman, 2022).

The ethylene signaling pathway comprises several critical elements (Fig. 2). Ethylene is perceived by membrane-bound receptors such as *ETR1, ERS1*, and *ETR2*, which function as negative regulators of the signaling cascade. In the absence of ethylene, the protein kinase *CTR1* suppresses signaling by phosphorylating *EIN2,* preventing downstream activation. Upon ethylene binding, *EIN2* undergoes dephosphorylation and translocates to the nucleus, where it activates transcription factors including *EIN3* and *EIN3-Like* (*EIL*). These transcription factors then induce the expression of ethylene-responsive genes (Alonso et al., 1999; Chao et al., 1997). A key downstream regulator, *ERF1* (Ethylene Response Factor 1), modulates the expression of genes involved in senescence and stress response pathways.

The influence of ethylene on flower longevity is well documented in species such as roses and lilies, where it accelerates senescence by regulating genes responsible for cell wall degradation and pigment loss, thus shortening vase life (Li et al., 2011; Zhang et al., 2012). The biosynthetic enzymes *ACC* synthase and *ACC* oxidase are crucial for ethylene production, with the signaling cascade mediated by receptors and transcription factors like EIN3 controlling the activation of senescence-related genes (Yang & Hoffman, 2022; Bleecker & Kende, 2023). To counteract ethylene's senescence-promoting effects and enhance flower longevity, interventions such as ethylene inhibitors, controlled atmosphere storage, and genetic modification techniques have shown promising results (Yoon et al., 2023; Yang & Hoffman, 2022).

Through extensive research, the pathway of ethylene biosynthesis has been clearly elucidated (Fig. 2) (Yang & Hoffman, 2022). The primary precursors involved in this process are S-adenosyl-l-methionine (*SAM*) and 1-aminocyclopropane-1-carboxylic acid (*ACC*) (Bleecker & Kende, 2023). Ethylene production proceeds via three enzyme-driven steps: initially, methionine is converted into *SAM* by methionine adenylyl transferase; subsequently, *SAM* is transformed into *ACC* through the action of *ACC* synthase (*ACS*), during which 5′-methylthioadenosine (*MTA*) is also generated and recycled back to methionine via the Yang cycle, a crucial regulatory loop in plant ethylene metabolism (Schaller & Bleecker, 1995). Lastly, *ACC* oxidase (*ACO*) catalyzes the oxidation of *ACC* to produce ethylene (Figures 2 & 3) (Li et al., 2021). Both *ACS* and *ACO* serve as key rate-limiting enzymes controlling the pace of ethylene biosynthesis (Bleecker & Kende, 2023).

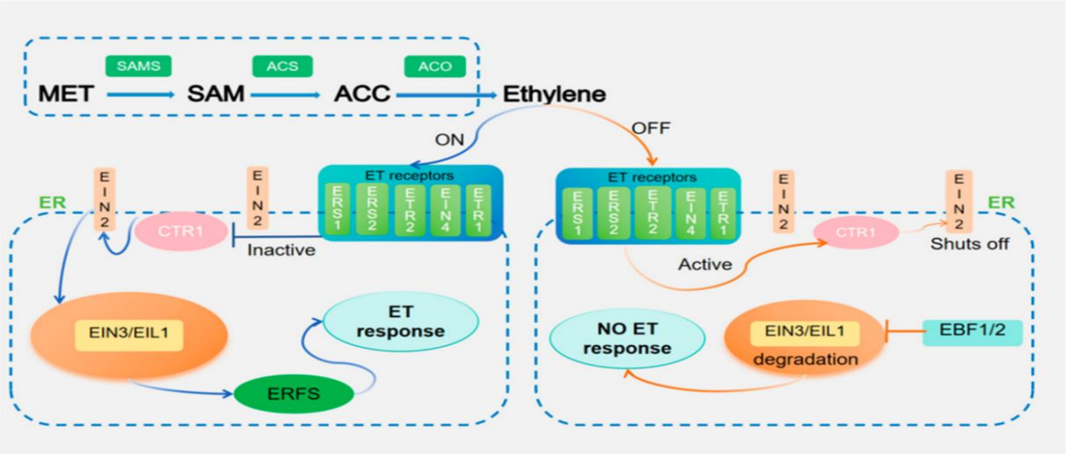

Figure 3: Overview of ethylene biosynthesis and its signal transduction pathway in plants, illustrating key enzymatic steps and signaling components involved in the regulation of ethylene production and response. (Adapted from Yang & Hoffman, 2022)

Ethylene perception and signal transduction involve a tightly regulated mechanism centered on membrane-bound receptors located on the endoplasmic reticulum (*ER*). In the absence of ethylene, these receptors activate the Raf-like kinase Constitutive Triple Response 1 (*CTR1*), which phosphorylates *ETHYLENE INSENSITIVE2 (EIN2*), a key *ER* membrane protein. This phosphorylation targets *EIN2* for degradation, leading to the destabilization and breakdown of transcription factors *ETHYLENE INSENSITIVE3 (EIN3)* and *EIN3-LIKE1 (EIL1)*, thereby preventing activation of ethylene-responsive genes (Fig. 4) (Schaller & Bleecker, 1995; Alonso et al., 1999).

When ethylene is present, it binds to these receptors—such as *ETHYLENE RESPONSE1/2 (ETR1/2), ETHYLENE RESPONSE SENSOR1/2 (ERS1/2)*, and *ETHYLENE INSENSITIVE4 (EIN4)* in Arabidopsis—resulting in the inactivation of *CTR1*. This allows *EIN2* to evade degradation and translocate to the nucleus, where it stabilizes *EIN3* and *EIL1*. Stabilized *EIN3/EIL1* then promote the transcription of ethylene-responsive factors (*ERFs*), which drive downstream gene expression in response to ethylene signaling (Bleecker & Kende, 2023).

Further regulation involves the F-box proteins *EIN2*-Targeting Proteins 1 and 2 (*ETP1* and *ETP2*), which mediate degradation of phosphorylated *EIN2* in the absence of ethylene, thus blocking signal transmission (Chao et al., 1997). Additionally, EIN3 and EIL1 are regulated by *EIN3*-Binding *F-BOX* proteins 1 and 2 (*EBF1* and *EBF2*), which facilitate their degradation via the *26S* proteasome, effectively switching off the ethylene response when necessary (Bleecker & Kende, 2023).

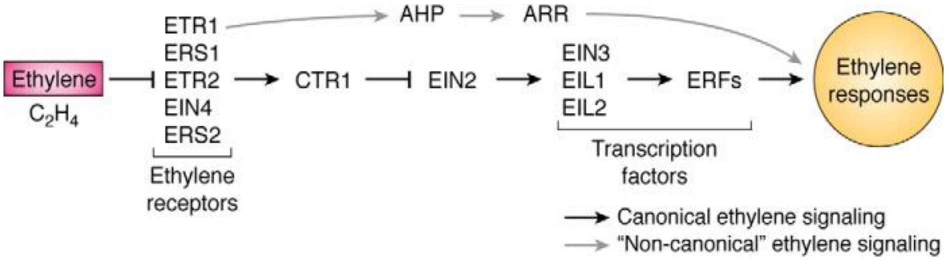

Figure 4: Simplified Ethylene Signaling Model (Bleecker & Kende, 2023)

This model, derived from genetic studies in *Arabidopsis thaliana*, illustrates the ethylene signaling pathway, which involves several ethylene receptors—including *ETHYLENE RESPONSE1 (ETR1), ETHYLENE RESPONSE SENSOR1 (ERS1), ETHYLENE RESPONSE2 (ETR2), ETHYLENE INSENSITIVE4 (EIN4)*, and *ETHYLENE RESPONSE SENSOR2 (ERS2)* along with the protein kinase Constitutive Triple Response 1 (*CTR1*) and the central transducer *ETHYLENE INSENSITIVE2 (EIN2).* Upon ethylene perception, EIN2 activates key transcription factors such as *ETHYLENE INSENSITIVE3 (EIN3), EIN3-LIKE1 (EIL1*), and *EIL2*. These factors, in turn, induce downstream transcription factors, notably the Ethylene Response Factors (*ERFs*), which regulate diverse ethylene-mediated responses including stress adaptation and developmental processes (Fig. 4).

CTR1 functions as a negative regulator of the pathway; in the absence of ethylene, *CTR1* phosphorylates EIN2, maintaining it in an inactive state. Ethylene binding to its receptors inhibits both the receptor and *CTR1* kinase activity, thereby preventing *EIN2* phosphorylation. As a result, *EIN2* escapes degradation mediated by *EIN2*-Targeting Proteins 1 and 2 *(ETP1/2*). The unphosphorylated *EIN2* undergoes cleavage, and its *C*-terminal fragment *(EIN2-C)* translocate to the nucleus to activate *EIN3* and *EIL1* transcription factors, thus initiating the ethylene response cascade (Schaller & Bleecker, 1995; Alonso et al., 1999). EIN3 directly binds promoter regions of *ERF* genes, facilitating their transcription. *ERFs,* a plant-specific family of transcription factors, modulate gene expression in response to ethylene and various environmental stresses, orchestrating downstream physiological changes (Bleecker & Kende, 2023).

Besides this canonical signaling pathway, an alternative "non-canonical" route (often depicted in gray) has been identified, where *ETR1* interacts with Arabidopsis Histidine Phosphotransfer proteins (*AHPs*), which then signal through Arabidopsis Response Regulators (*ARRs*), adding an additional layer of modulation to ethylene responses. Together, ethylene biosynthesis and signaling pathways work in concert to regulate the broad spectrum of ethylene's biological functions. The ethylene signaling cascade in *Arabidopsis* serves as a well-established model, from ethylene perception by the receptor family to gene regulation via *ERFs* (Chao et al., 1997).

Epigenetic Modifications in Ethylene Signal Transduction and DNA Methylation

Epigenetic modifications of heritable changes in gene function without alterations to the underlying *DNA* sequence play a significant role in regulating gene expression. These modifications can influence numerous aspects of gene regulation, including *RNA* splicing, transcript stability, translation efficiency, nucleosome positioning, and chromatin organization (Zhang et al., 2020). By modulating these processes, epigenetic changes impact both normal cellular physiology and pathological states and can even affect traits passed on to subsequent generations (Li & Zhang, 2021).

Ethylene signaling pathways are also regulated by epigenetic mechanisms, with *DNA* methylation serving as a key modification in this regulation (Yang et al., 2019). *DNA* methylation alters the expression of genes involved in ethylene biosynthesis and signal transduction, thereby influencing the hormone's activity. Environmental cues, such as stress conditions, can trigger dynamic changes in *DNA* methylation patterns, which subsequently modulate ethylene signaling pathways and their downstream responses (Liu et al., 2022).

DNA methylation, the most prevalent type of epigenetic modification, is crucial for controlling various aspects of plant growth and development (Liu et al., 2020). This modification can either promote or suppress the expression of genes involved in ethylene biosynthesis and signaling pathways. For instance, increased methylation (hypermethylation) in the promoter regions of certain genes can lead to reduced gene activity, thereby impacting ethylene signaling (Zhang et al., 2021). Studies conducted on tomatoes have revealed that exposure to low-temperature stress induces changes in *DNA* methylation within the promoters of ethylene-related genes such as *SlEIN3*, *SlERF-A1*, and *SlERT10* (Chen et al., 2022). These methylation alterations caused a decrease in the expression of these genes, while the expression of *SlCTR1* was elevated. This shift in gene expression resulted in delayed fruit ripening, illustrating how epigenetic modifications regulate ethylene signaling and contribute to plant adaptation under environmental stress (Wang et al., 2021).

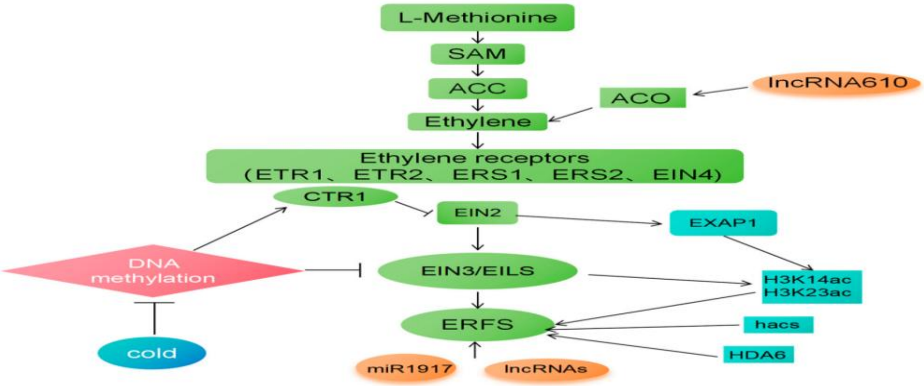

Figure 5: Schematic representation of the interaction between ethylene signaling and epigenetic modifications, highlighting the role of DNA methylation in regulating ethylene biosynthesis and signal transduction pathways. Environmental factors, such as stress, can alter methylation patterns, thereby influencing the activity of ethylene-responsive genes (adapted from Liu et al., 2022).

Ethylene Signaling Pathways and Hormonal Interactions in Flower/Petal Senescence

Flower senescence is a multifaceted process regulated by various plant hormones, with ethylene playing a pivotal role. Ethylene, a gaseous hormone produced naturally by plants, promotes flower aging by triggering physiological changes post-pollination. For instance, in *Petunia*, ethylene production surges after pollination, linked to increased expression of the *ACO1* gene in senescing petals (Wang et al., 2021). Similarly, ethylene receptors and downstream genes such as *DC-ERS2* and *DC-ETR1* in carnation are crucial for senescence regulation (Chen et al., 2020). Expression of ethylene receptor genes like *ETR3* in roses and *ERS1* and *CTR1* in *Delphinium* during senescence highlights ethylene perception's importance in this process (Li et al., 2022). Mutations in ethylene biosynthesis and signaling genes, such as *ACO* and *etr1-1*, have been shown to delay senescence and extend vase life in transgenic flowers (Yang et al., 2019).

During senescence, nutrients are reallocated from petals to other tissues like developing ovaries or young leaves (Rogers, 2013; Rogers and Munné-Bosch, 2016). Alongside ethylene, hormones including jasmonic acid (*JA*), salicylic acid (*SA*), abscisic acid (*ABA*), and brassinosteroids promote senescence, while cytokinins, gibberellins (*GA*), and auxins generally act as inhibitors (Reid and Chen, 2008). Ethylene is particularly influential in triggering petal wilting and abscission following pollination (Woltering and Van Doorn, 1988). Exogenous ethylene or its precursor *ACC* accelerates corolla senescence, whereas ethylene biosynthesis inhibitors such as *AOA* and *1-MCP* delay the process (Trivellini et al., 2011a; Ferrante et al., 2015).

ABA plays a distinctive role, especially in ethylene-insensitive flowers like daylilies, where it primarily accelerates senescence (Rogers, 2013). In ethylene-sensitive species like carnations, *ABA* promotes senescence by boosting endogenous ethylene synthesis (Reid & Chen, 2008). Cytokinins delay senescence by reducing ethylene sensitivity, as observed in transgenic plants overexpressing *IPT* genes that elevate cytokinin levels and prolong floral longevity (Woltering & Van Doorn, 1988). The effects of auxins and *GA* on senescence are less well-characterized; however, *GA* is known to delay senescence in some flowers by antagonizing ethylene effects (Trivellini et al., 2011). Jasmonic acid can stimulate senescence through enhanced ethylene production, although its role varies across species, such as orchids where its function remains unclear.

Ethylene also interacts extensively with other hormones, influencing growth and development. It modulates auxin distribution, often enhancing auxin-mediated processes like root development and fruit ripening (Vanneste & Friml, 2009). In contrast, ethylene and gibberellins generally act antagonistically, with *GA* promoting stem elongation and ethylene inhibiting it, although these interactions depend on developmental context and environment (Achard et al., 2003). Ethylene and *ABA* cooperate or antagonize each other in stress responses, such as drought tolerance, depending on conditions (Zhang et al., 2006). Ethylene commonly inhibits cytokinin signaling pathways, affecting cell division and differentiation critical for leaf senescence and root growth (Moubayidin et al., 2009). Meanwhile, ethylene and jasmonic acid often synergize to enhance defense responses against wounding and pathogens, with each hormone influencing the other's signaling pathway (Schilmiller et al., 2007).

Overall, the intricate hormonal crosstalk governs flower and petal senescence, with ethylene acting as a primary trigger in many species post-pollination. Ethylene biosynthetic genes like *ACO* and receptor genes such as *DC-ERS2*, *DC-ETR1*, and *ETR3* regulate this process in species including carnations, roses, and petunias (Shibuya et al., 2002). Reducing ethylene sensitivity through mutations or genetic modification delays senescence, extending flower vase life (Hunter et al., 2004). Meanwhile, hormones such as *ABA* and cytokinins regulate senescence in complementary or antagonistic manners depending on the species and ethylene sensitivity. ABA promotes senescence by enhancing ethylene production or independently in ethylene-insensitive flowers, whereas cytokinins delay aging by maintaining tissue health and lowering ethylene responsiveness (Chang et al., 2003). Auxins and *GA* influence senescence variably, with *GA* sometimes antagonizing ethylene’s effects (Saks et al., 1992), and jasmonic acid typically promoting senescence via ethylene induction (Setyadjit et al., 2006).

Enzymatic Pathways and Programmed Cell Death in Floral Senescence

Floral senescence is characterized by programmed cell death (*PCD*), accompanied by critical enzymatic activities and structural alterations within floral tissues. PCD in flowers resembles processes such as apoptosis and autophagy, where cells undergo tightly regulated self-destruction (Fig. 6). Key enzymes including proteases, lipoxygenases, and cell wall-degrading enzymes—play vital roles in breaking down cellular components during senescence (Zhu et al., 2016; Li et al., 2021). These enzymes facilitate the degradation of proteins, lipids, and polysaccharides, driving the senescence process forward. Structural changes during floral senescence include degradation of the cell wall, chlorophyll breakdown, and cellular collapse, all of which contribute to visible symptoms such as petal wilting and loss of coloration (Yuan et al., 2020; Li et al., 2023). Understanding these molecular and structural changes is essential for developing approaches to delay senescence, thereby extending flower longevity and improving post-harvest quality (Kim et al., 2022).

PCD is a defining feature of floral senescence, involving hallmark changes like chromatin condensation, cell shrinkage, and collapse (Fig. 6). This process is carefully orchestrated by senescence-associated proteins and enzymes that direct the orderly dismantling and death of cells within aging tissues (Moulin et al., 2013). Proper regulation of PCD ensures that cellular resources are recycled efficiently, supporting the plant's overall adaptation to aging and stress conditions

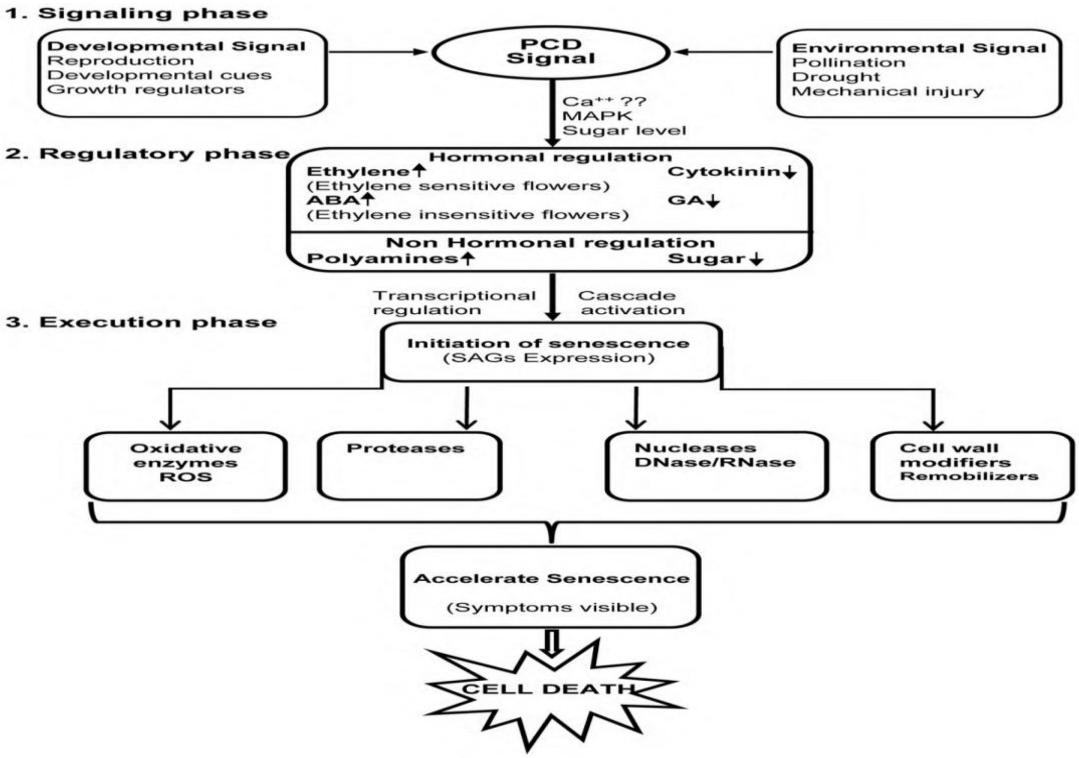

Figure 6: Model of regulatory pathways in flower senescence (Li et al., 2023). Programmed cell death (*PCD*) signals, triggered by both internal and external stimuli, cause hormonal imbalances within the cell. These hormonal changes activate signaling cascades and transcriptional regulators that induce the expression of senescence-associated genes *(SAGs*), including those encoding proteases, nucleases, cell wall-degrading enzymes, and oxidative enzymes. The combined action of these enzymes accelerates the senescence process, making it irreversible. Visible symptoms develop in the later stages, ultimately leading to flower cell death.

Senescence in plants involves a range of physiological and biochemical changes, including water loss from tissues, ion leakage, and metabolite transport. It is also characterized by the generation of reactive oxygen species (*ROS*), increased membrane fluidity, and the hydrolysis of lipids, proteins, nucleic acids, and carbohydrates. One of the earliest and most notable events during senescence is membrane degradation, which leads to structural changes such as vacuolar vesiculation and loss of membrane permeability. These changes have been documented in species like carnations and daylilies (Stead and van Doorn, 1994). This membrane degradation correlates with a decrease in membrane phospholipids and an increase in neutral lipids, primarily due to the activation of phospholipases and acyl hydrolases (Hempel et al., 2001).

Lipid peroxidation, catalyzed by lipoxygenases, plays a significant role in membrane damage during senescence in flowers such as carnations (Hong et al., 2000), daylilies (Kende and Zeevaart, 1982), and roses (Lennon et al., 1994). In ethylene-sensitive plants, lipoxygenase activity contributes to oxidative damage, thereby accelerating the senescence process (Pichersky et al., 1983). However, this is not a universal mechanism; for example, lipoxygenase activity does not appear to contribute to senescence in ethylene-insensitive flowers like Alstroemeria (Leverentz et al., 2002) or in orchids such as Phalaenopsis (Lennon et al., 1994). These differences underscore the complex, species-specific nature of senescence and its variable dependence on ethylene signaling and lipid peroxidation.

At the molecular level, transcription factors have emerged as key regulators of senescence pathways. The *ORE1* transcription factor integrates ethylene signaling with the activation of senescence-associated genes in Arabidopsis (Balazadeh et al., 2010). NAC family transcription factors, such as *ANAC*092, influence programmed cell death and stress responses, further linking ethylene signaling with plant aging (Wu et al., 2009). Other transcription factors like *WRKY53* and members of the *MYB* family interact with multiple signaling pathways to modulate senescence progression (Miao et al., 2004; Sablowski & Meyerowitz, 1998). Additionally, Ethylene Response Factors (*ERFs*), especially *ERF1*, play a central role in mediating ethylene's effects on stress responses and senescence (Solano et al., 1998). Understanding these transcriptional regulators is essential for developing approaches to control floral senescence and enhance plant longevity.

Advances in Ethylene Signaling and Senescence Research: Inhibitors, Genes, Markers, and Transcription Factors

Recent advances in ethylene signaling research have significantly enhanced our understanding of its central role in regulating plant senescence and longevity, particularly through complex signaling pathways (Fig. 7). Ethylene receptors such as ***ETR1 (ETHYLENE RESPONSE 1)*** and ***ERS1 (ETHYLENE RESPONSE SENSOR 1*)** serve as key components in ethylene perception and are instrumental in initiating the signaling cascade that regulates floral senescence. Studies have elucidated how these receptors interact with downstream signaling elements to modulate the aging process of flowers (Schaller et al., 2015; Zhang et al., 2023).

One of the most critical regulators in the ethylene signaling pathway is ***EIN2 (ETHYLENE INSENSITIVE 2),*** which acts as a central hub integrating ethylene signals with developmental and stress-related pathways. Post-translational modifications of EIN2 have been shown to influence its activity and its interaction with ***EIN3/EIL1 (ETHYLENE INSENSITIVE 3 / EIN3-Like 1)***, both of which function as transcriptional activators of ethylene-responsive genes (Qiao et al., 2012; Liu et al., 2020). These insights provide a foundational basis for the development of targeted inhibitors that could modulate ethylene responses, particularly in post-harvest floral management.

Advances in ethylene biosynthesis research have led to the discovery of several compounds capable of inhibiting ethylene production or signaling. For example, ***Aminoethoxyvinylglycine (AVG)*** and ***CoA* inhibitors** effectively target ethylene biosynthetic enzymes such as **ACC synthase (*ACS*)** and ***ACC* oxidase (*ACO*)**, thereby reducing ethylene production and delaying the onset of senescence (Liu et al., 2019). Similarly, **1-Methylcyclopropene (*1-MCP*)**, a well-characterized ethylene antagonist, binds irreversibly to ethylene receptors, preventing ethylene from initiating downstream signaling and thus preserving flower freshness (Sisler & Serek, 1997).

In addition to biosynthesis inhibitors and receptor antagonists, current research is also investigating molecular modulators that affect signaling proteins and transcriptional regulators. For example, targeting ***EIN3*** and ***ERF1*** **(*ETHYLENE RESPONSE FACTOR 1*)** with genetic tools or chemical inhibitors offers a promising approach to fine-tuning ethylene responses for improved crop quality and stress resilience (Huang et al., 2016; Klee & Tieman, 2020). These approaches are particularly relevant for floricultural and horticultural industries aiming to extend post-harvest life and reduce waste.

Transcription factors also play a crucial role in ethylene-mediated senescence. ***WRKY53, ERF1,*** and other transcriptional regulators are known to control the expression of **senescence-associated genes (*SAGs*)** that orchestrate the breakdown of cellular components during the aging process. *WRKY53,* in particular, is implicated in the regulation of leaf senescence, whereas ERF1 mediates responses to both developmental cues and environmental stresses (Miao et al., 2004; Fujimoto et al., 2000). These transcription factors represent valuable molecular markers and potential targets for genetic manipulation to modulate the senescence process across various plant species.

Regulation of Leaf Senescence by Ethylene and Transcription Factors

Ethylene is a key regulator of leaf senescence, influencing this process through its complex signaling network (Fig. 7). Increased ethylene production is often correlated with the onset of leaf senescence, while the ethylene receptor ***ETHYLENE RESISTANT 1 (ETR1)*** generally acts as a negative regulator in this pathway. Mutations in ethylene receptors, such as the ***etr1*** mutation in *Arabidopsis thaliana* and petunia, have been shown to delay leaf senescence, underscoring the repressive role of these receptors in ethylene signaling (Grbić and Bleecker, 1995; Wang et al., 2013). Conversely, *Arabidopsis* mutants lacking multiple ethylene receptor genes display constitutive ethylene responses and exhibit accelerated senescence, indicating that receptor-mediated repression is essential for proper timing of leaf aging (Qu et al., 2007).

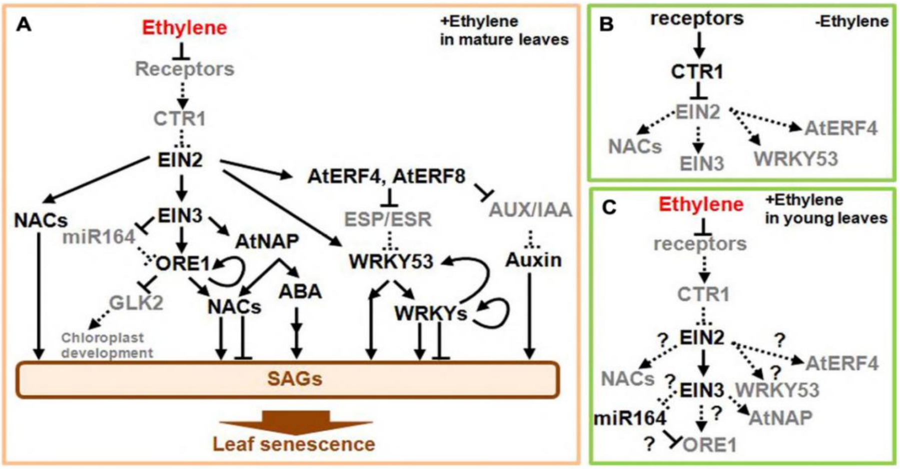

Figure 7: Ethylene signaling pathway regulating leaf senescence (Zhang et al., 2014). (A) In mature leaves, ethylene detection activates downstream signaling, inducing senescence-associated genes (SAGs) and triggering leaf senescence. (B) In both young and mature leaves, ethylene receptors constitutively repress downstream signaling when ethylene is absent, preventing premature senescence. (C) In young leaves, although ethylene detection activates downstream signaling, it does not directly induce leaf senescence, indicating age-dependent regulation.

The signaling component ***EIN2*** acts as a positive regulator of leaf senescence. This is supported by studies where ***EIN3*** mutations delay senescence, while overexpression of EIN3 accelerates it, highlighting the transcriptional control exerted downstream of ethylene perception

(Li et al., 2013; Kim et al., 2014). However, the role of ***CTR1 (CONSTITUTIVE TRIPLE RESPONSE* 1)** in leaf senescence remains ambiguous, as mutants in CTR1 do not display premature leaf aging, suggesting that its regulatory function may differ between tissues or developmental contexts (Jing et al., 2005).

EIN2 and *EIN3* are active components in ethylene signaling and induce several ethylene responses but do not directly trigger leaf senescence through an as-yet-uncharacterized mechanism likely involving leaf development regulators. In Figure 7, arrows and bars indicate positive and negative regulation, respectively; solid lines and black gene names represent active forms, while dotted lines and gray gene names denote inactive forms. Due to space limitations, some transcription factors (*TFs*) and signals like jasmonic acid (*JA*) are not shown.

NAC transcription factors play a pivotal role in ethylene-mediated leaf senescence. Notably, *NAC* genes such as ORESARA1 (ORE1) and ANAC019 are activated by ethylene via the *EIN2* pathway (Kim et al., 2009, 2014). *ORE1* is a key senescence regulator, promoting its own expression and that of other senescence-associated genes. Its activity is modulated by micro *RNA* miR164, which declines with leaf age, allowing *ORE1* accumulation (Kim et al., 2009). *EIN3* directly activates *ORE1*, reinforcing ethylene's role in senescence regulation (Li et al., 2013). Other *NAC* genes, such as AtNAP, function in the abscisic acid (*ABA*) pathway, promoting leaf senescence and stress responses (Guo and Gan, 2006). Downstream *NAC* factors influenced by *ORE1* and *AtNAP* contribute to senescence via hormone signaling and stress response pathways (Kim et al., 2014).

Ethylene-responsive transcription factors (*ERFs*) are also crucial regulators of leaf senescence. Some *ERFs*, including AtERF4 and **AtERF8,** act positively by repressing negative regulators like EPITHIOSPECIFIER PROTEIN (ESP/**ESR*),*** which inhibits senescence (Miao and Zentgraf, 2007). ERF expression increases with leaf age and ethylene exposure (Koyama et al., 2013). Conversely, *ERFs* such as **RAV1** negatively regulate senescence, with overexpression delaying the process (Woo et al., 2010). *WRKY* transcription factors interact with ethylene signaling and JA pathways, forming a complex regulatory network; for example, **WRKY53 is** modulated by both ethylene and JA, affecting leaf senescence timing (Miao and Zentgraf, 2007; Breeze et al., 2011). The interplay of these TFs with environmental and developmental cues fine-tunes leaf senescence, ensuring a tightly controlled ethylene response.

Ethylene-Mediated Gene Regulation and Senescence: Signaling, Hormonal Interactions, and Mechanisms

Ethylene is a pivotal plant hormone regulating diverse physiological processes, including fruit ripening, leaf and flower senescence, and stress responses through complex signaling pathways (Jiang et al., 2023). Ethylene perception initiates when the hormone binds to receptors such as ***ETR1, ETR2, EIN4*, and *ERS1*** located in the endoplasmic reticulum (Schaller et al., 2015). This binding relieves the inhibition by the *CTR1* kinase, enabling **EIN2** to accumulate and translocate to the nucleus. There, transcription factors **EIN3** and ***EIL1*** activate ethylene-responsive genes by binding ethylene response elements (*EREs*) in their promoters (Alon et al., 2003; Chao et al., 1997).

During floral senescence, ethylene accelerates aging by upregulating senescence-associated genes (***SAGs***), such as ***SAG12***, which encodes a cysteine protease critical for protein degradation (Gepstein et al., 2003), and by modulating programmed cell death (Buchanan et al., 2000). Ethylene also regulates Ethylene Response Factors (***ERFs***), which mediate stress responses and developmental processes (Solano et al., 1998). In fruit ripening, ethylene influences softening and flavor by regulating genes involved in cell wall degradation (Brummell, 2020; Klee and Giovannoni, 2021).

Ethylene interacts extensively with other hormones, including **abscisic acid (*ABA*)** and **jasmonic acid (*JA*)**, to coordinate stress responses and defense pathways (Fujita et al., 2023; Sakuma et al., 2022). This hormonal cross-talk is essential for integrating environmental signals with developmental programs. Ethylene-mediated gene regulation encompasses activation, repression, and post-transcriptional modifications. Activation occurs via transcription factors like EIN3 and ERFs binding to target promoters (Alon et al., 2003), while repression involves downregulating genes such as those encoding **Jasmonate *ZIM*-Domain (*JAZ*)** proteins, which modulate defense (Chini et al., 2007). Furthermore, ethylene influences microRNAs (***miRNAs***) controlling *mRNA* stability and translation, and alternative splicing events, generating protein isoforms key to stress and senescence regulation (Zhao et al., 2022; Morris and Kramer, 1998).

Post-translational modifications modulate the stability and activity of transcription factors and enzymes critical for senescence. For example, ***WRKY* transcription factors**, implicated in defense and senescence gene regulation, are modulated by ethylene, which fine-tunes their activity (Rushton et al., 2010; Zhao et al., 2022). Other *TF* families, such as ***NAC*** and ***MYB***, regulate genes involved in programmed cell death (PCD) and stress responses, driving senescence progression (Kim et al., 2009; Solfanelli et al., 2006). These *TFs* orchestrate the dismantling of cellular components, essential for tissue degradation during senescence.

Genetic manipulation studies have demonstrated the importance of ethylene biosynthesis genes in senescence. Transgenic plants with suppressed ***ACS*** or ***ACO*** genes exhibit delayed senescence and prolonged floral display, as seen in carnations and other ornamentals (Savin et al., 1995; Kiss et al., 2000; Huang et al., 2007). Senescing flowers often show increased ethylene production and upregulated expression of ethylene-related genes, including *ERFs* that regulate *SAG* expression (Liu et al., 2011). Modulating ethylene receptor expression and signaling pathways in petunia and Arabidopsis has similarly delayed flower senescence and enhanced longevity (Gubrium et al., 2000; Shibuya et al., 2004).

Ethylene inhibitors are valuable tools in managing floral senescence and improving flower quality. Antagonists such as **1-Methylcyclopropene (*1-MCP*)** competitively block ethylene binding to its receptors, thereby extending vase life (Sisler and Serek, 2021). Biosynthesis inhibitors like **Silver Thiosulfate (*STS*)** inhibit *ACC* oxidase, preventing ethylene production (Lester and Dunlap, 1985). Other inhibitors, including cyclohexylamine and natural extracts from ginger and garlic, disrupt ethylene signaling pathways (Buchanan et al., 2000; Bai et al., 2015).

Technological advancements, such as controlled atmosphere storage and slow-release formulations, have improved the efficiency of ethylene inhibitors in commercial applications (Paull, 2022; Serek et al., 2023). These approaches are widely used in floriculture to extend vase life, regulate flowering times, and reduce post-harvest waste, thereby enhancing commercial flower production (De Meyer et al., 2012; Serek et al., 2023). By targeting key elements of the ethylene signaling network—including transcription factors and receptor antagonists—floriculture can effectively mitigate flower aging and improve shelf life. Continued development of novel inhibitors promises to further optimize senescence management and flower quality in commercial settings.

Dynamics of DNA Methylation in Leaf Senescence

DNA methylation plays a pivotal role in regulating leaf senescence, with notable changes in methylation patterns observed as leaves age (Fig. 8). In *Arabidopsis thaliana*, a global decline in *DNA* methylation occurs during senescence, which is correlated with the downregulation of *DNA* methyltransferases such as ***CMT3*** and ***MET1**,* alongside an upregulation of DNA demethylases including ***ROS1**, **DME**, **DML2***, and ***DML3*** (Ogneva et al., 2016). This dynamic modulation of *DNA* methylation affects the expression of numerous senescence-associated genes.

In *Gossypium hirsutum* (cotton), senescent tissues exhibit decreased *DNA* methylation specifically in promoter regions and *CpG* islands, which correlates with alterations in metabolic pathways during aging (Dou et al., 2017). Similar observations in bamboo and rice highlight the dynamic changes across different methylation contexts ***CG**, **CHH***, and ***CHG*** which influence the expression of genes involved in senescence and stress responses (Zhang et al., 2021; Moritoh et al., 2012).

The balance between *DNA* methyltransferases and demethylases is central to the regulation of methylation during senescence. In *Arabidopsis*, mutations in methyltransferases such as **MET1**, or loss-of-function of demethylases like ***DML3***, disrupt normal senescence progression. For instance, the absence of ***DML3*** leads to hypermethylation at promoters of senescence-related genes, delaying their expression and consequently slowing the senescence process (Kim et al., 2008; Yuan et al., 2020). Conversely, the demethylase ***ROS1*** promotes senescence by altering methylation patterns at specific loci, thereby modulating the timing of leaf aging. Further evidence from tomato and pak choi supports the critical role of *DNA* methylation in senescence regulation, particularly emphasizing methylation changes in promoter regions of senescence-associated genes as key modulators of senescence onset (Zhu et al., 2015; Li et al., 2022).

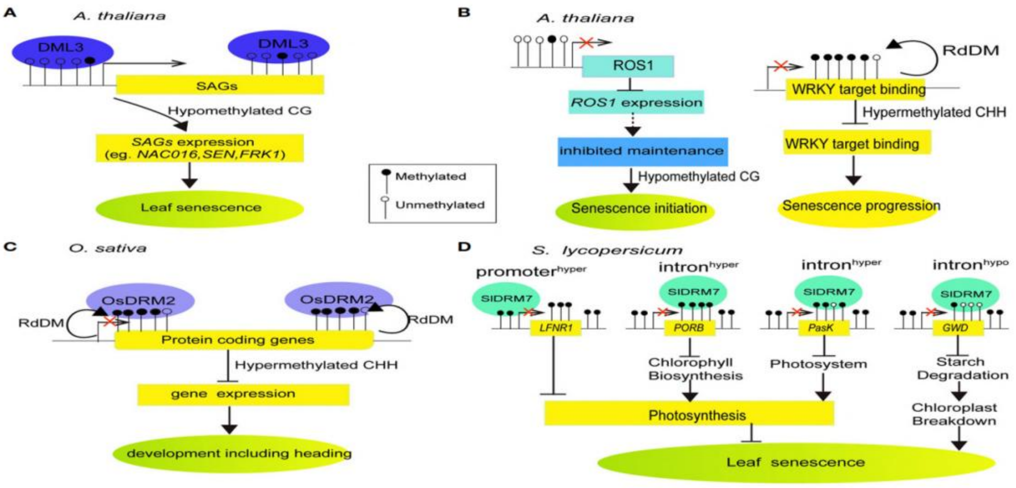

Figure 8: Advances in Senescence Research: Models of *DNA* Methylation-Mediated Gene Regulation in Plant Aging.

(A) In *Arabidopsis thaliana*, the *DNA* demethylase **DML3** reduces *CG* methylation in gene regions, activating senescence-associated genes and thereby promoting leaf senescence (Yuan et al., 2020).(B) **ROS1,** another *DNA* demethylase in *Arabidopsis*, plays a role in leaf senescence. During senescence, reduced expression of ROS1 correlates with decreased demethylation activity; *CG* hypomethylation observed is attributed to inhibited methylation maintenance (Vatov et al., 2022).(C) In rice (*Oryza sativa*), the DNA methyltransferase ***OsDRM2*** regulates development and heading date by controlling *CHH* methylation, especially in small transposable elements (*TEs*) proximal to protein-coding genes (Moritoh et al., 2012; Tan et al., 2016).(D) In tomato (*Solanum lycopersicum*), *DNA* methylase ***SlDRM7*** influences leaf chlorosis and senescence. Silencing ***SlDRM7*** disrupts *DNA* methylation and alters expression of genes involved in chlorophyll biosynthesis, photosynthesis, and starch degradation, resulting in premature senescence and chlorosis (Wen et al., 2022).

Isolating and characterizing senescence-related genes employ a suite of advanced molecular techniques to dissect plant aging mechanisms at the molecular level (Fig. 8). Gene expression profiling methods, such as microarrays and *RNA* sequencing (*RNA-Seq*), have identified key senescence-associated genes (SAGs), including ***SAG12*** and ***SAG21*** (Gepstein et al., 2003; Ding et al., 2012). These approaches enable the quantification of gene expression dynamics across developmental stages, illuminating the molecular networks underlying senescence. Functional analyses utilizing gene knockout and overexpression strategies—via *CRISPR/Cas9*, *RNA* interference (*RNAi*), and transgenic approaches—allow precise manipulation of candidate genes to elucidate their roles in aging processes at both molecular and phenotypic levels (He et al., 2004; Liu et al., 2023). Complementary methods such as yeast two-hybrid screening and co-immunoprecipitation investigate protein–protein interactions and regulatory complexes that control senescence-related gene expression (Choi et al., 2008; Gregersen et al., 2008).

Furthermore, integrating transcriptomics, proteomics, and metabolomics offers a comprehensive systems-level perspective on how senescence genes modulate cellular functions and metabolic pathways during plant aging (Miller et al., 2007; Munne-Bosch and Pallas, 2007). This multi-omics approach enhances understanding of the complex regulatory networks orchestrating senescence.Key genes like ***SAG12,*** encoding a cysteine protease, and ***ORE1,*** a transcription factor, serve as crucial molecular targets for improving crop longevity and quality through genetic and agronomic innovations (Gepstein et al., 2003; He et al., 2004). Advances in techniques such as RT-PCR, high-throughput sequencing, and bioinformatics have accelerated the application of senescence research in plant breeding and crop improvement programs (Saha et al., 2017; Zhao et al., 2017).

Role of Ethylene in Fruit Ripening

Ethylene is a key regulator of ripening in climacteric fruits, orchestrating essential physiological changes such as color development, texture softening, and the production of volatile organic compounds (*VOCs*) responsible for fruit aroma (Fig. 9). It facilitates chlorophyll degradation while enhancing the biosynthesis of carotenoids and anthocyanins, contributing to the characteristic coloration of ripe fruits (Barry & Giovannoni, 2007). Inhibition of ethylene biosynthesis or signaling, through genetic manipulation or chemical inhibitors, reduces VOC production. For example, transgenic apples and melons expressing antisense constructs targeting ethylene biosynthetic enzymes *ACC* synthase (*ACS*) and *ACC* oxidase (*ACO*) exhibit diminished VOC synthesis (Dandekar et al., 2004; Bauchot et al., 1998). Similarly, inhibitors such as amino-ethoxyvinylglycine (*AVG*) and 1-methylcyclopropene (*1-MCP*) modulate *VOC* profiles by blocking ethylene biosynthesis or receptor binding, respectively, with effects dependent on the treatment context (Valdes et al., 2009).

Climacteric fruits display a distinct ethylene production and respiration pattern, dividing ripening into two phases: an initial low ethylene production stage (System 1), during which the fruit remains unripe and largely insensitive to external ethylene; followed by a high ethylene production stage (System 2), where autocatalytic ethylene biosynthesis occurs, and fruits become responsive to ethylene (Lelièvre et al., 1998). Key genes such as ***ACS6*** and ***ACS1*** regulate this transition by controlling ethylene synthesis (Alexander & Grierson, 2002). Post-harvest inhibition of ethylene action remains a widely used strategy to delay ripening and senescence, thereby extending the shelf life of climacteric fruits.

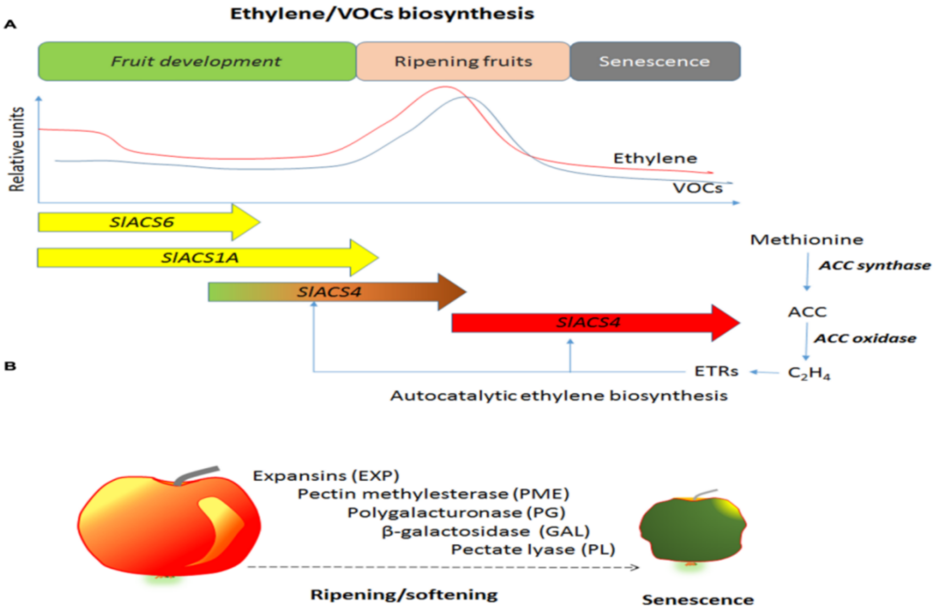

Figure 9: **(A)** Simplified schematic of ethylene and volatile organic compound (*VOC*) biosynthesis during fruit development (adapted from Kevany & Klee, 2007). Members of the *Solanum lycopersicum* ACC synthase (*SlACS*) gene family are differentially expressed throughout fruit development, contributing to ethylene production. *VOC* biosynthesis originates from multiple metabolic pathways, including phenylpropanoid metabolism, fatty acid degradation, and carotenoid breakdown. **(B)** Major enzymes involved in cell wall degradation during fruit ripening and senescence. The coordinated action of these enzymes—such as polygalacturonase, pectin methylesterase, and cellulase—leads to cell wall disassembly, resulting in fruit softening and loss of firmness.

Ethylene plays a crucial role in controlling the expression of various genes involved in fruit ripening. In tomato plants, ethylene regulates genes such as polygalacturonase *(PG*), pectin methylesterase (*PME*), and phytoene synthase, which are key to processes like cell wall breakdown and the development of fruit color (Smith et al., 1988; Fray & Grierson, 1993). Research focusing on ethylene receptors in tomatoes, including genes like *LeETR4* and *LeETR6*, has demonstrated their participation in both the flowering and ripening stages (Tieman et al., 2000; Kevany et al., 2007). The Never-ripe tomato mutant, characterized by diminished sensitivity to ethylene, exhibits changes in gene expression patterns, fruit shape, and carotenoid production, emphasizing ethylene's fundamental role in controlling fruit growth and maturation (Alba et al., 2005). Additionally, the tomato E8 gene contributes to the regulation of ethylene biosynthesis and signal transduction during ripening, with its expression levels rising as the fruit approaches maturity (Penarrubia et al., 1992).

Fruit softening a key indicator of quality is mainly caused by the breakdown of the cell wall; a process governed by numerous enzymes, including pectin methylesterases (*PMEs*), polygalacturonase (*PG*), cellulases, galactosidases, pectate lyase (*PL*), and expansins. These enzymes are encoded by multi-gene families and are carefully regulated in both time and location throughout fruit ripening and aging. Ethylene is central to controlling the expression of these enzymes, promoting cell wall disassembly and thereby facilitating fruit softening. Expansins, in particular, support the loosening of the cell matrix during ripening and are regulated by both ethylene levels and *pH*. For example, in tomatoes, ethylene stimulates the expression of the *EXP1* gene, while blocking ethylene synthesis results in decreased expression of this gene (Rose et al., 1997). A similar regulatory mechanism by ethylene is observed in other climacteric fruits, such as bananas (Trivedi and Nath, 2004).

Pectin methylesterase (*PME*) is an important enzyme activated before the onset of ripening; it removes methyl groups from pectin, which then becomes vulnerable to degradation by polygalacturonase. PME activity is enhanced by ethylene and plays a significant role in fruit softening during senescence (El-Sharkawy et al., 2016). Likewise, *PG*, which breaks down galacturonic acid linkages in the cell wall, is also induced by ethylene and contributes to cell wall disintegration (Sitrit and Bennett, 1998). *β-galactosidase* participates in fruit softening by cleaving *β-(1,4)-galactans* in the cell wall (Eda et al., 2016). Transgenic tomato plants with suppressed *β-galactosidase* activity retain firmer fruit during ripening, and ethylene exposure elevates *β-galactosidase* activity in avocados (Jeong and Huber, 2004; Karakurt and Huber, 2002). Furthermore, pectate lyase (*PL*), another enzyme critical for cell wall degradation, exhibits increased activity in response to ethylene in fruits like bananas and mangoes (Lohani et al., 2004; Chourasia et al., 2006).

Ethylene's regulation of fruit aging is closely intertwined with other plant hormones (Liu et al., 2023). These hormonal interactions collectively influence growth, senescence, and fruit ripening, affecting qualities such as yield, appearance, and shelf life (Zhang et al., 2022). Insights into how ethylene coordinates with other phytohormones, particularly through post-translational modifications, offer promising avenues for improving crop quality and longevity (Wu et al., 2021). The effect of ethylene on plant development, especially under stress conditions, depends on the balance between its production and perception, which is modulated by other hormonal signals (Chen et al., 2023). Further investigations into these complex hormonal networks across various growth stages and tissues may aid in refining agricultural strategies aimed at prolonging the shelf life of climacteric fruits (Liu et al., 2023).

Progress and Challenges in Ethylene Inhibitors and Technological Methods for Improving Floral Quality and Longevity

Recent advancements in gene expression analysis, functional genomics, and integrative multi-omics techniques have identified crucial genes and transcription factors that govern the process of floral senescence. Ethylene, along with other plant hormones, plays a central role in orchestrating aging at both molecular and physiological scales. Additionally, a deeper understanding of enzyme activities associated with senescence enhances our capability to manipulate flower longevity and overall quality. These combined insights provide valuable strategies for advancing agricultural techniques and producing crops with prolonged shelf life and greater stress tolerance.

Ethylene inhibitors have become indispensable in extending the vase life of cut flowers. One widely used compound, 1-Methylcyclopropene (*1-MCP*), works by binding to ethylene receptors, thereby blocking the hormone's action and delaying the onset of senescence while maintaining flower freshness (Blankenship & Dole, 2023). Other chemical agents, such as silver-based compounds like Silver Thiosulfate and biosynthesis inhibitors like Amino-ethoxyvinyl-glycine (*AVG*), either interfere with ethylene production or act as antagonists at the receptor level, effectively reducing ethylene's influence on floral aging. Beyond chemical treatments, genetic engineering has made significant strides in enhancing floral lifespan. Through the development of transgenic plants with lowered ethylene synthesis or altered receptor sensitivity, innovative approaches have emerged for improving flower preservation (Zhao et al., 2021). These genetic interventions offer promising long-term solutions for maintaining flower quality post-harvest.

Alongside chemical inhibitors, technological advances have played a pivotal role in preserving floral quality and extending longevity. Techniques such as Controlled Atmosphere Storage (*CAS*) and Modified Atmosphere Packaging (*MAP*) regulate environmental factors and gas compositions, which suppress ethylene production and help, retain flower freshness during storage (Lafuente et al., 2011; Ning et al., 2020). Another valuable approach involves ethylene scrubbers, devices that remove ethylene gas from the surrounding air using materials like potassium permanganate, thereby mitigating its senescence-inducing effects (Wiley & Roupas, 1991). Employing a combination of chemical and technological methods offers a comprehensive strategy to manage ethylene-related deterioration and preserve floral quality after harvest.

Recent investigations have shed light on the molecular underpinnings of floral senescence, pinpointing the involvement of key transcription factors such as *WRKY* and *MYB*. These regulators modulate genes linked to cell wall disassembly, chlorophyll degradation, and pigment loss throughout the aging process (Li et al., 2020). In orchid species, ethylene signaling has been implicated in upregulating stress-responsive genes that contribute to accelerated senescence (Liu et al., 2021). Understanding these complex molecular pathways provides novel targets for strategies aimed at extending flower life and controlling senescence.

Looking forward, future research will likely concentrate on refining both ethylene inhibitor applications and genetic engineering approaches. The *CRISPR/Cas9* system holds great promise for precise editing of genes involved in ethylene biosynthesis and signaling, potentially enhancing floral longevity with high specificity (Gepstein et al., 2021). Meanwhile, improvements in smart packaging technologies and controlled atmosphere storage systems will continue to optimize the microenvironment surrounding floral products, further boosting preservation outcomes (Zhao et al., 2021). Sustained collaboration between scientists and the floriculture industry will be essential to translate these technological and genetic advancements into practical solutions, ultimately improving flower quality and shelf life for commercial growers and consumers alike (Kader, 2002; Lafuente et al., 2011).

CONCLUSION, RECOMMENDATIONS, AND FUTURE RESEARCH DIRECTIONS

Significant progress in deciphering the mechanisms of floral senescence and the role of ethylene has profoundly influenced the floriculture sector. These advances have uncovered intricate molecular pathways and introduced innovative methods to improve flower longevity and quality. Important findings highlight ethylene's central function as a key regulator, the discovery of senescence-associated genes such as *SAG12* and *SAG13*, and the deployment of technologies like ethylene inhibitors and controlled atmosphere storage. Together, these developments indicate that targeted genetic engineering and cutting-edge postharvest technologies hold great promise for significantly extending vase life and enhancing the overall quality of flowers. Moving forward, it is recommended to deepen investigations into the molecular networks governing ethylene signaling and senescence, aiming to identify novel targets for intervention. Emphasizing research in genomics, synthetic biology, and the plant microbiome will broaden our capacity to control floral aging. Successfully applying these innovations will depend on interdisciplinary collaboration and robust partnerships between research institutions and industry stakeholders to facilitate the translation of scientific discoveries into practical solutions.

Future research should prioritize closing existing gaps in understanding regulatory networks and post-translational modifications related to senescence. Harnessing synthetic biology to develop precise genetic modules for controlling flower aging represents a promising direction. Supporting these initiatives alongside fostering industry-academic cooperation will be vital to advancing sustainable practices and boosting profitability within the floriculture industry. Improving flower preservation benefits producers and retailers by reducing losses and maintaining product quality, while consumers enjoy longer-lasting, more visually appealing flowers. Key focus areas include: 1) Utilizing advanced tools like *CRISPR/Cas9* gene editing for precise alteration of genes linked to flower quality and lifespan. 2) Breeding or engineering cultivars with customized traits such as increased ethylene tolerance and superior postharvest performance. 3) Incorporating real-time monitoring and environmental control technologies into postharvest handling to optimize flower quality throughout distribution. 4) Combining consumer feedback with artificial intelligence and machine learning techniques to better anticipate and meet customer preferences.

Data Availability Statement

The datasets supporting the conclusions of this study are available from the corresponding author upon reasonable request.

Conflict of Interest

The author declares no conflicts of interest related to this review paper.

Ethical Clearance

The author follows all ethical guidelines necessary to produce the review paper.

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