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Review

Molecular Biochemistry and Physiology of Postharvest Chilling Injury in Fruits: Mechanisms and Mitigation

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Abstract

Postharvest chilling injury (PCI) is a significant limitation in the storage of temperature-sensitive fruits, leading to quality deterioration and reduced marketability. However, low temperatures delay senescence—consistent with the Q10 principle, where metabolic reaction rates change 2–3-fold per 10 °C—and chilling-sensitive fruits experience membrane destabilization, oxidative imbalances, and structural degradation under cold stress. Physiological assessments consistently report elevated electrolyte leakage, increased malondialdehyde accumulation, and reduced membrane fluidity, coupled with disruptions in respiration and cellular energy metabolism. Biochemically, PCI is characterized by enhanced ROS production and a 20–50% decline in key antioxidant enzymes, along with disturbances in calcium signaling and hormone regulation. At the molecular level, chilling-responsive transcription factors such as *CBF*, *CAM*, *HSF*, and *WRKY* show strong induction, while lipid remodeling and epigenetic modifications further shape cold adaptation responses. Advances in multi-omics, including transcriptomics, proteomics, metabolomics, lipidomics, and volatilomics, have revealed chilling-associated metabolic shifts and regulatory cascades, enabling the identification of potential biomarkers of tolerance. Emerging mitigation strategies, including physical and chemical treatments, as well as CRISPR-based interventions, have shown a 30–60% reduction in PCI in controlled studies. This review synthesizes recent progress in physiology, molecular biochemistry, and postharvest technology to support future research and practical PCI management.

Keywords: cold stress; low temperature; oxidation; postharvest management; storage

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1. Introduction

Postharvest chilling injury (PCI) is a physiological disorder that affects a wide range of fruits, particularly those originating from tropical and subtropical regions when exposed to low, non-freezing temperatures during cold storage and transportation [1,2]. These situations induce a cascade of metabolic, biochemical, and structural disruptions, resulting in peel pitting, flesh browning, off-flavor development, increased susceptibility to decay, and premature senescence [3,4]. PCI primarily results from loss of membrane integrity, oxidative stress, hormonal imbalances, and disruptions in primary metabolism, particularly in carbohydrate and energy-associated pathways, which consequently disrupt cell wall

metabolism and lead to chilling-induced softening or abnormal firmness changes [5]. Fruits including mango [6–8], banana [9], avocado [10], papaya [11], kiwifruit [12], apple [13], and citrus [14] exhibit varying degrees of chilling sensitivity with PCI severity being affected by cultivars, genotype, preharvest environmental conditions, and postharvest handling practices. While cold storage is necessary to maintain the shelf-life of horticultural produce, chilling-sensitive fruits undergo physiological and biochemical disturbances that compromise their commercial quality and consumer acceptability [8,15].

The occurrence of PCI seriously challenges the global fresh fruit industry, leading to substantial economic losses due to quality deterioration and postharvest losses [3,16]. The financial burden of PCI is particularly pronounced in fruits intended for long-distance export markets, where inadequate cold storage conditions can lead to shipment rejection or increased reliance on chemical treatments [16]. Additionally, PCI also influences the nutritional composition, textural attributes, and bioactive compound retention, thereby lowering the functional characteristics of cold-stored fruit [17]. The increasing demand for sustainable strategies to mitigate PCI has urged extensive research into understanding its molecular basis and developing novel mitigation strategies. This necessitates an integrative approach that encompasses biochemical, physiological, and molecular perspectives to unravel the complex mechanisms underlying PCI and formulate targeted postharvest interventions.

Recent advances in postharvest biology have highlighted that PCI is closely linked to membrane lipid peroxidation, impaired energy homeostasis, and metabolic imbalances triggered by chilling stress [15,18]. An important factor contributing to PCI is the disintegration of cellular membranes due to phospholipid degradation and the phase transition of membrane lipids, resulting in increased permeability and ion leakage [19]. This structural disintegration further exacerbates oxidative stress, characterized by the accumulation of uncontrolled reactive oxygen species (ROS), which exacerbates cellular damage and initiates cell death [15,20]. The interplay between ROS and antioxidant defense mechanisms, including enzymatic and non-enzymatic antioxidants, plays an essential role in governing the extent of chilling-associated damage in fruits [21,22].

On a molecular level, PCI disrupts gene expression, stress signaling, and homeostasis, with ethylene, abscisic acid (ABA), jasmonic acid (JA), and salicylic acid (SA) regulating chilling stress response [16]. Hormonal crosstalk affects sugar metabolism and energy balance, resulting in reduced sweetness, altered acidity, and textural changes [23]. Omics technologies have recognized key molecular markers for chilling tolerance, assisting postharvest optimization. Mitigation strategies, including calcium (Ca) application, melatonin (MT) treatment, modified atmospheric packaging (MAP), and Clustered Regularly Interspaced Short Palindromic Repeats (CRISPR)/CRISPR-associated protein (Cas) gene editing, show promise.

Several reviews have previously summarized different aspects of PCI, including its physiological features [5,24], oxidative and hormonal regulation [21,22,25], and postharvest interventions [26]. However, these reviews primarily focus on individual mechanisms and do not integrate recent multi-omics advances or connect molecular responses with translational postharvest practices. Despite substantial progress, critical knowledge gaps remain—particularly regarding how membrane lipid remodeling, epigenetic regulation, and metabolome-wide shifts collectively drive PCI; how genotype-specific regulatory networks modulate chilling sensitivity; and which molecular targets are most suitable for breeding, gene editing, or commercial-scale interventions.

This review addresses these gaps by synthesizing current findings across physiology, lipidomics, transcriptomics, metabolomics, and hormonal signaling to generate a unified mechanism of PCI. Additionally, we highlight emerging candidate genes, lipid

metabolic biomarkers, and promising mitigation treatments with potential for breeding, CRISPR-based improvement, and industry-level scaling. In doing so, this review provides a comprehensive and future-focused perspective that extends beyond existing literature.

2. Physiology of PCI in Fruits

Fruits exposed to low temperatures undergo biochemical, molecular, and structural alterations, including in the physiology of PCI. Chilling compromises the integrity of membranes by changing the lipid composition and increasing permeability and ion leakage [27]. This leads to metabolic abnormalities, such as oxidative stress brought on by an overabundance of ROS, which results in protein oxidation, lipid peroxidation, and enzymatic failure. Cellular function is further compromised by impaired mitochondrial respiration and decreased adenosine triphosphate (ATP) generation, which impacts signal transduction pathways and energy metabolism [5]. Furthermore, chilling modifies hormone regulation, interfering with Ca homeostasis, ABA signaling, and ethylene biosynthesis—essential for preserving fruit quality.

2.1. Structural and Membrane Integrity Loss

PCI in fruits is often characterized by increased ion leakage due to membrane destabilization [28]. At low temperatures, membrane lipids transition from a fluid to a gel-like state, leading to pore formation and increased permeability [29]. This disruption hampers ion homeostasis, triggering modifications to the cytoskeleton, impaired intracellular transport, and osmotic imbalances. Consequently, the swelling and ultrastructural damage to mitochondria and chloroplasts further aggravate cellular dysfunction [30]. The degree of membrane stability is affected by lipid composition. A higher proportion of unsaturated fatty acids enhances membrane fluidity and reduces electrolyte leakage (EL). Beyond general fatty acid saturation levels, specific membrane lipid classes—phospholipids, sphingolipids, and glycerolipids—play targeted regulatory roles in chilling responses. Chilling stress typically reduces saturated lipid species and promotes the accumulation of unsaturated lipid forms, a shift that maintains membrane fluidity under cold conditions. Enzymes such as fatty acid desaturases (FADs) and acyl-lipid desaturases catalyze this desaturation process, and mutants with impaired FAD activity exhibit increased chilling sensitivity due to membrane rigidification [31]. Phospholipases, particularly PLDα1 and PLDδ, remodel phospholipids during low-temperature stress by generating lysophospholipids and phosphatidic acid (PA).

In contrast, the phospholipase C (PLC)–diacylglycerol kinase (DGK) pathway further contributes to PA production from phosphatidylinositol derivatives [32]. PA functions as both a structural lipid and a signaling molecule, linking membrane stress to Ca^{2+}-mediated cold signaling. Glycerolipids such as monogalactosyldiacylglycerol (MGDG) and digalactosyldiacylglycerol (DGDG) also undergo restructuring, mediated by galactolipid galactosyltransferase (GGGT/SFR2), which forms oligogalactolipids that help maintain bilayer stability and prevent phase transition into non-lamellar structures. Additionally, sphingolipid-related enzymes such as ceramide glucosyltransferase contribute to chilling tolerance by modulating glycosphingolipid biosynthesis and maintaining membrane integrity. Collectively, these lipid modifications represent a coordinated cold-responsive mechanism that preserves membrane structural integrity under chilling stress [30].

Exogenous application of polyamines has been shown to reduce the rate of EL by increasing the unsaturated fatty acid content, thereby preserving membrane integrity in chilling-sensitive fruits, such as grapefruit [33]. Several studies on horticultural crops further demonstrate that chilling tolerance is closely linked to the expression of genes involved in lipid metabolism. In chilling-tolerant grapefruit, up-regulation of fatty acid

desaturase (FAD) transcripts and ceramide glucosyltransferase enhances membrane fluidity. In contrast, chilling-sensitive cultivars exhibit down-regulation of genes involved in lipid and sterol metabolism [34]. Enzymes such as lipid transfer proteins (LTPs), diacylglycerol acyltransferase (DGAT), DGK, and acyl carrier proteins (ACPs) also participate in lipid remodeling during low-temperature exposure. Cold-responsive transcription factors, including CBF, MYB, ERF, bZIP, and NAC, modulate these lipid metabolic pathways [35]. Examples include OsDREB1A-mediated regulation of *PLDα1*, which enhances cold tolerance in rice, MaMYB4-dependent control of omega-3 desaturases in banana [36], and CaNAC1-induced *PLDα4* expression promoting lipid degradation and chilling injury in bell pepper [30]. These findings highlight the central role of transcriptional regulation in orchestrating membrane lipid adjustments under chilling conditions.

Chilling stress also hampers cellular metabolism by impairing the activities of mitochondria and chloroplasts. Mitochondrial respiration is inhibited, and ATP synthesis declines due to lower activity of membrane-bound ATPase and electron transport chain components [37,38]. Similarly, photosynthetic activity reduces as enzyme activity declines and thylakoid membranes stiffen, exacerbating energy deficits. This metabolic impairment leads to a surge in ROS as standard cellular energy generation fails to meet antioxidant demands [39]. ROS accumulation, driven by mitochondrial dysfunction and respiratory chain uncoupling, severely damages cellular macromolecules, including proteins, lipids, and nucleic acids. The loss of antioxidant defense exacerbates oxidative stress, enhancing membrane lipid peroxidation and cellular collapse. The gel-solid membrane readily disrupts the ROS equilibrium. Singlet oxygen, hydrogen peroxide (H_2O_2), superoxide radicals ($O_2^{\bullet -}$), and hydroxyl radicals (•OH) are examples of ROS, which are by-products of regular cell metabolism that the antioxidant system can scavenge to preserve cellular stability. Low-temperature storage, however, has the potential to decouple the respiratory chain and cause a significant amount of ROS to be produced [21]. Furthermore, cold stress reduces the function of the antioxidant system, making it unable to handle the rising concentration of ROS. Rising ROS levels began to attack proteins, deoxyribonucleic acid (DNA), and membrane lipids, resulting in the breakdown of the membrane system, the accumulation of hazardous metabolites, metabolic diseases, and ultimately, cell death [30,40].

A comprehensive understanding of the chilling damage cascade, from membrane instability to oxidative collapse, should be the top priority of research. Investigating synergistic mitigation techniques that enhance cellular energy metabolism and antioxidant defense, while also stabilizing membrane structures, is crucial. Additionally, comparing several fruit cultivars with different chilling sensitivity could aid in identifying critical biochemical markers for breeding and screening initiatives.

2.2. PCI-Induced Metabolic Activities

The PCI development alters oxidative balance, energy production, and the breakdown of carbohydrates, which upsets fruit metabolism. The ROS build-up during cooling stress causes oxidative damage, membrane lipid peroxidation, and increased EL [41]. Chilling stress inhibits essential enzymes, such as sucrose synthase, amylase, and phosphorylase, which reduces sugar conversion and energy availability, ultimately negatively impacting carbohydrate metabolism [42]. While bananas' reduced starch breakdown results in peel browning and meat hardening [9], mangoes experience reduced sucrose buildup, which causes surface pitting and unpleasant tastes [25]. Cucumbers suffer water-soaked lesions and increased softness due to cell wall disintegration.

In contrast, tomatoes exhibit delayed ripening due to abnormalities in glycolysis and the tricarboxylic acid (TCA) cycle, thereby altering the sugar-acid balance [15]. Chilling lowers ATP synthesis by reducing mitochondrial respiration and glycolytic efficiency,

affecting energy metabolism. Membrane breakdown is accelerated by ATP depletion, which also impairs membrane integrity and interferes with active ion transport, particularly Ca^{2+} homeostasis. This, in turn, triggers phospholipase D (PLD) and LOX [2,5]. Chilling symptoms worsen by changing the expression of genes that respond to stress and hormonal imbalances, such as decreased gibberellic acid (GA) and cytokinin.

The reviewed research emphasizes that, in addition to oxidative stress, effective mitigation of PCI should target disturbances in energy production and carbohydrate metabolism. Maintaining ATP levels and enzyme activity may support metabolic balance and membrane integrity. Responses particular to a given fruit, such as the loss of sugar in mangoes or the retention of starch in bananas, suggest that customized approaches are required. Reducing metabolic abnormalities brought on by cold stress also heavily relies on hormonal modulation. Despite advances, the physiological sequence from early membrane destabilization to visible chilling symptoms remains incompletely mapped. Future studies should combine in vivo imaging and membrane lipidomics to identify early predictive physiological markers of PCI.

2.3. Cell Wall Metabolism and Softening Under PCI

Beyond membrane destabilization and metabolic disruption, PCI profoundly affects cell wall structure and enzyme-mediated remodeling. Chilling stress alters the synthesis, solubilization, and degradation of pectin, hemicellulose, and cellulose—three major components defining fruit firmness [43]. Low temperatures suppress the activity of ripening-associated enzymes such as polygalacturonase (PG), pectin methylesterase (PME), β-galactosidase, and cellulase, leading to incomplete pectin depolymerization and abnormal cell adhesion [44]. This incomplete softening is a hallmark of chilling-induced textural disorders such as woolliness in peaches and mealy texture in nectarines.

Conversely, in fruits like cucumber and zucchini, chilling accelerates cell wall breakdown and water-soaking due to enhanced pectin solubilization and structural weakening. PME-mediated pectin demethylation increases Ca^{2+}-pectate cross-linking, making cell walls more rigid, while suppressed PG activity prevents the expected pectin degradation—resulting in dry, mealy, or leathery textures [45,46]. In mangoes, chilling impairs hemicellulose degradation, leading to surface pitting and internal breakdown, which is closely linked to altered expression of expansins and xyloglucan endotransglycosylases/hydrolases [47].

PCI also disrupts the expression of cell wall-modifying genes regulated by ethylene, ABA, and calcium signaling. Down-regulation of PG, β-galactosidase, and expansin transcripts is frequently reported in chilling-sensitive cultivars, whereas tolerant genotypes maintain higher levels of cell wall hydrolase activity during storage [48]. The integration of cell wall proteomics and transcriptomics has further revealed that cold stress alters polysaccharide–protein interactions, contributing to the irreversible stiffening or collapse of tissues. Thus, impaired cell wall metabolism is a central determinant of chilling-induced textural failure and must be considered in conjunction with membrane lipid remodeling and oxidative damage to understand PCI physiology fully.

3. Biochemical and Molecular Mechanisms of PCI

Fruit PCI results from a series of molecular and metabolic alterations brought on by low temperatures. Excessive ROS induces oxidative stress, which at the biochemical level results in metabolic imbalances, membrane damage, and lipid peroxidation. Fruit texture and quality are impacted by marked changes in enzymatic activity, particularly those related to cell wall metabolism and antioxidant defense. At the molecular level, cold stress alters hormone pathways and triggers alterations in gene expression, activating

stress-responsive transcription factors, including *CBF/DREB*. Auxin, ethylene, ABA, JA, and brassinosteroids interact intricately to shape the fruit's resistance to chilling temperatures by controlling ripening delays, oxidative stress responses, and cold tolerance.

3.1. Oxidative Stress and Antioxidant Defense

Low fruit temperatures cause lipid structural changes and an increase in ROS, which compromise membrane integrity [49,50]. Excessive ROS generation causes oxidative stress, leading to unchecked oxidation and cell death when cellular homeostasis is disrupted [21,51]. Lipid peroxidation, protein oxidation, enzymatic inhibition, and damage to DNA/ribonucleic acid (RNA) are all signs of oxidative damage that occur when ROS levels exceed antioxidant defense [52,53]. Since the chloroplast is involved in high-energy photosynthesis, it is a significant source of ROS. Additionally, mitochondria contribute to the process by decoupling the electron transport chain, which further increases ROS levels [54]. These changes accelerate fruit degradation by intensifying oxidative stress in tissues susceptible to PCI. $O_2^{\bullet -}$ causes lipid peroxidation produced by membrane-bound nicotinamide adenine dinucleotide phosphate (NADPH) oxidases [55].

Furthermore, lipoxygenase (LOX) initiates lipid peroxidation in the plasma membrane, thereby decreasing fluidity and desaturation, and further destabilizing cellular structures [56]. Fruits with PCI exhibit reduced antioxidant enzyme activity when oxidative stress intensifies, thereby exacerbating ROS accumulation [57]. Peroxidase (POD), catalase (CAT), and superoxide dismutase (SOD) are essential antioxidant enzymes that contribute to cold tolerance [58,59].

Unchecked ROS accumulation speeds up postharvest fruit deterioration by interacting with membrane lipids and triggering peroxidation and membrane breakdown [21,60]. Fruits use both enzymatic and non-enzymatic antioxidant systems to counteract these effects. After $O_2^{\bullet -}$ are converted to H_2O_2 by SOD, glutathione peroxidase, thioredoxin peroxidase, CAT, and POD detoxify the H_2O_2 [61]. While non-enzymatic antioxidants such as glutathione, ascorbate, phenolics, and carotenoids also scavenge ROS, the ascorbate-glutathione (AsA-GSH) cycle is essential for ROS detoxification. Furthermore, under stress, metabolic pathways such as the γ-aminobutyric acid (GABA) shunt help preserve redox equilibrium. Increased EL, malondialdehyde (MDA) buildup, and ROS accumulation are signs of severe fruit stress [38]. Ultimately, preventing postharvest PCI and retaining fruit quality depend on having robust antioxidant defenses.

3.2. Hormonal Interplay

Hormonal interplay also plays an essential role in PCI in fruits. It is controlled by intricate hormonal interactions between GA, ethylene, ABA, JA, SA, cytokinin (CK), and MT [62]. Numerous studies have demonstrated how various hormones can work in harmony or in opposition to one another to regulate the cold tolerance of fruit. According to the species, BRs and ethylene perform different roles in cold-stored fruit [63]. Exogenous treatment of 24-epibrassinolide (EBR) in pepper reduced indications of PCI, including pitting and browning of the flesh, by dramatically inhibiting ethylene production. EBR treatment delayed ethylene-induced ripening and enhanced cold resistance by downregulating key ethylene biosynthesis genes, such as *ACS* and *ACO*, as well as signaling components, including ETR and EIN3 [64].

Research on cucumber fruit has shown that BRs promote stress-responsive pathways by increasing ethylene production. Brassinolide (BL) was used to mitigate chilling damage, which increased alternative oxidase (AOX) activity [65]. Inhibiting ethylene production reduced BR-induced chilling tolerance, indicating that BR lowers ET to postpone ripening and related chilling damage in some fruits. BR and ethylene work together to combat

chilling stress in others. This divergent reaction suggests that the BR-ethylene interaction varies by species, and further investigation is required to determine whether BR increases or decreases ethylene in various fruit varieties when cold-stored after harvest. JA, frequently in conjunction with BR, is essential for mitigating PCI. Cold storage caused JA to build up in apple fruit, which triggered the inducer of *CBF* expression (ICE)-C-repeat binding factor (*CBF*) pathway—a vital cold-responsive mechanism [66]. Interestingly, BR signaling also regulates *CBF* genes, as the BR-responsive transcription factor *BIM1* directly increases the expression of *CBF1* and *CBF2*. However, JAZ repressors (JAZ1 and JAZ2) limited the activation of BIM1 by competing with it for binding to *CBF2*, so modulating the integration of JA and BR signals. Despite this regulatory check, the interaction between BR and JA guaranteed a quick and effective cooling reaction, reducing apple fruit damage. This work identifies JA as a critical modulator of fruit's responses to cold stress during storage and emphasizes the significance of the BR-JA-*CBF* pathway in PCI resistance [66].

A well-known stress hormone, ABA, helps fruit survive storage at low temperatures. However, depending on the storage conditions, it can have a cooperative or antagonistic relationship with BR. By boosting antioxidant enzyme activity, ABA levels in tomato fruit increased dramatically during cold storage, thereby enhancing chilling tolerance [67]. An even higher rise in ABA was seen when EBR was administered to tomato fruit that had been refrigerated, indicating that BRs can encourage ABA production in the presence of chilling stress. Mechanistically, BR-induced cold tolerance was associated with the repression of BIN2 kinase, which in turn activated *BZR1*, a crucial regulator of genes involved in ABA biosynthesis [67]. This result suggests that the degree of stress influences BR-ABA crosstalk in fruit; ABA enhances chilling tolerance, but excessive accumulation can lead to delayed ripening and reduced fruit quality. Therefore, optimal fruit storage after harvest requires fine-tuning the balance of BR-ABA.

MT has been shown in recent studies to help prevent chilling damage to fruit. Pre-treatment with 1.5 μM MT significantly increased auxin levels in cold-stored watermelon, enhancing the fruit's ability to withstand chilling stress [68]. Auxin-responsive gene overexpression facilitated this benefit by preventing chilling-induced oxidative damage and stabilizing cellular homeostasis. Furthermore, MT increased the expression of *CBF*, strengthening the plant's innate defenses against cold. Notably, by boosting ATP synthesis, this hormone also enhanced photosynthetic efficiency, guaranteeing that fruit maintained metabolic activity despite low temperatures.

Although key biochemical pathways and stress-related genes have been identified, their temporal coordination during cold stress is poorly understood. Integrating time-series gene expression, enzyme activity profiling, and metabolite flux analysis will help clarify causal relationships in PCI progression.

4. Omics Technologies in PCI Mitigation

The onset of PCI triggers a cascade of molecular and metabolic changes in fruits, which have been thoroughly studied using various omic approaches. These evolved strategies, including transcriptomics, proteomics, metabolomics, and epigenomics, have enabled researchers to unravel the underlying genetic and biochemical responses associated with chilling stress in various fruit species. These technologies provide significant potential for developing targeted mitigation strategies to combat chilling stress in fruits by recognizing key molecular markers, stress-responsive pathways, and metabolic shifts (Figure 1).

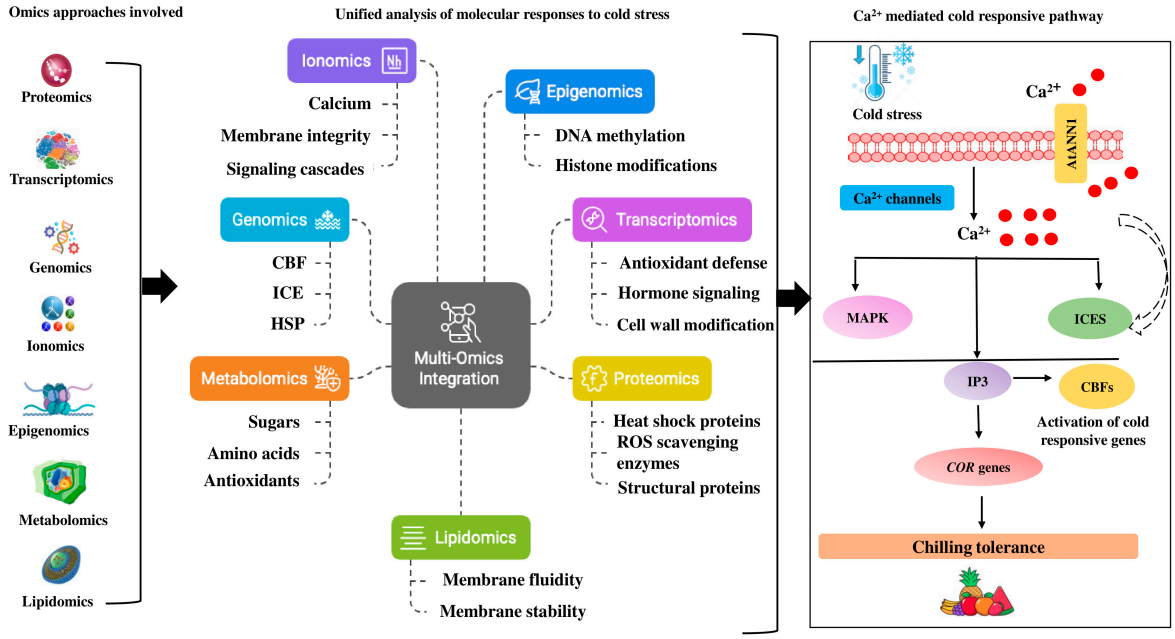


Figure 1. This schematic illustrates the role of multi-omics approaches—proteomics, transcriptomics, genomics, ionomics, epigenomics, metabolomics, and lipidomics—in deciphering the molecular responses to postharvest chilling injury. Each omics platform contributes distinct insights: ionomics highlights calcium's role in membrane integrity and signaling cascades; genomics identifies cold-responsive genes such as CBF, ICE, and HSP; epigenomics involves DNA methylation and histone modifications; transcriptomics elucidates antioxidant defense, hormone signaling, and cell wall modifications; proteomics focuses on heat shock proteins, ROS scavenging enzymes, and structural proteins; metabolomics captures shifts in sugars, amino acids, and antioxidants; and lipidomics investigates membrane fluidity and stability. These diverse data streams integrate to provide a comprehensive understanding of chilling stress responses. The right panel depicts a calcium-dependent signaling cascade initiated by cold stress. Calcium influx through Ca^{2+} channels triggers signaling via MAPK, IP3, and ICES, ultimately activating CBFs and downstream COR genes that enhance chilling tolerance. Abbreviations: CBF: C-repeat binding factor; ICE: Inducer of CBF expression; HSP: Heat shock proteins; ROS: Reactive oxygen species; MAPK: Mitogen-activated protein kinase; IP3: Inositol trisphosphate; COR: Cold-responsive; ICES: Inducers of CBF expression and signaling.

4.1. Transcriptomics: Gene Expression Changes in PCI Response

Transcriptomic studies offer insights into differential gene expression patterns in chilling-affected fruits. RNA sequencing (RNA-Seq) and microarray analyses have been universally employed to recognize chilling-responsive genes involved in oxidative stress, hormone regulation, and membrane integrity [69]. In mango, transcriptomic studies have found the upregulation of *CBF*, a cold-responsive gene [70]. Likewise, in tomatoes, chilling stress upregulated the *SlCBF1*, a key regulator of cold acclimation, along with genes encoding dehydrins and late embryogenesis abundant (LEA) proteins, which help in membrane stabilization and osmotic balance [71]. In citrus fruits, transcriptomic analysis of oranges under chilling stress found upregulation of *WRKY* and *AP2/ERF* transcription factors, which regulate stress-responsive pathways. At the same time, the downregulation of genes associated with auxin biosynthesis indicated hormonal disruptions as a key factor in PCI susceptibility [72]. In apples, transcriptomic and metabolomic profiling under long-term low-temperature conditioning revealed extensive transcriptional reprogramming associated with chilling injury. Key cold-responsive transcription factors, including *MYB*, *bHLH*, *ERF*, *NAC*, and *bZIP*, were differentially expressed, indicating activation of cold-acclimation pathways. At the same time, genes involved in starch biosynthesis, such as *BE*, *SBE*, and *GBSS*, were strongly downregulated, aligning with the observed decline in starch levels

during chilling. These molecular changes reflect a coordinated regulation of sugar, lipid, hormone, and stress-response pathways contributing to chilling-induced deterioration in apple fruit [73]. Transcriptomic profiling in peach fruit revealed downregulation of genes involved in polyamine biosynthesis, leading to increased membrane permeability and loss of fruit firmness [74]. The chilling stress in grapes leads to changes in flavonoid biosynthesis gene expression, resulting in decreased anthocyanin accumulation and skin browning [75].

4.2. Proteomics: Cold-Stress-Responsive Proteins

The proteomic analysis provides profound insights into the post-transcriptional modifications and protein-level alterations in PCI-affected fruits. Cold stress frequently triggers modifications in energy metabolism-associated proteins, ROS scavenging, and membrane stability [76]. Proteomic profiling of cold-stored bananas found increased expression of heat shock proteins (HSPs) and peroxidases, which aid in combating oxidative stress and lowering lipid peroxidation [77]. In peaches, chilling stress results in the upregulation of aquaporins and LTPs, indicating their importance in maintaining cell membrane integrity and preventing water loss during cold storage [78]. In cucumber, differential proteomic analysis found cold stress upregulated proteins associated with polyamine biosynthesis, conferring better chilling tolerance, whereas energy-associated enzymes were significantly reduced [79]. In hardy kiwifruit, chilling conditions altered the expression of mitochondrial electron transport proteins, resulting in metabolic imbalance and increased ethylene production [80].

4.3. Metabolomics: Metabolic Shifts in Response to PCI

Metabolic studies provide a comprehensive understanding of PCI-induced biochemical alterations, particularly in carbohydrate metabolism, lipid composition, and secondary metabolites [81]. The metabolic analysis revealed an increased accumulation of raffinose and trehalose in papaya, which serves as an osmoprotectant against chilling-associated oxidative damage [82]. In contrast, cold-stored kiwifruit exhibited a decrease in ascorbic and phenolic compounds, resulting in lower antioxidant activity and increased susceptibility to oxidative degradation [83]. In cold-stored pomegranates, chilling stress resulted in a surge in GABA and sucrose, which act as protective metabolites against chilling-induced energy metabolism and membrane destabilization [29].

4.3.1. Lipidomics

Lipidomics has emerged as a critical component of metabolomics for understanding PCI, as membrane lipids are among the earliest and most severely affected cellular components under low-temperature stress [84]. Chilling stress disrupts membrane fluidity, lipid composition, and the balance between saturated and unsaturated fatty acids, ultimately leading to loss of membrane integrity and enhanced physiological damage [30]. In bananas, comprehensive lipidomic and transcriptomic analyses have revealed that low-temperature storage markedly accelerates membrane deterioration through coordinated enzymatic and genetic responses. Cold stress significantly elevates the activities of membrane lipid–degrading enzymes, including LOX, PLD, PLC, phospholipase A (PLA), and lipase, while reducing the activity of FAD [85]. This metabolic shift results in reduced levels of unsaturated fatty acids such as γ-linolenic and linoleic acids and a concurrent increase in saturated fatty acids, phosphatidic acid, lysophosphatidic acid, diacylglycerol, and free fatty acids. Collectively, these changes lower the USFA/SFA ratio and promote severe membrane rigidity and lipid peroxidation, directly contributing to CI development. Supporting this, untargeted lipidomics profiling showed that bananas stored at chilling temperatures accumulate PA and digalactosyldiacylglycerol (DGDG), while exhibiting

declines in phosphatidylcholine (PC), phosphatidylethanolamine (PE), and monogalactosyldiacylglycerol (MGDG). Upregulation of *MaPLD1* and *MaLOX2*, together with elevated MDA levels, further demonstrated that membrane lipid degradation and peroxidation are central drivers of banana PCI [86].

Similar lipid remodeling processes have been documented in mango fruit, where MT treatment significantly mitigates CI by stabilizing membrane lipid metabolism. The MT application suppresses the activities of PLD, PLC, and LOX, while enhancing PLA2 activity, thereby collectively maintaining membrane fluidity and reducing oxidative deterioration. Lipidomic profiling confirmed that MT-treated mangoes retain more stable levels of phospholipids, lysophospholipids, sphingolipids, and glycerides, indicating active remodeling of lipid molecular species to preserve membrane structure [87]. These modifications are linked to improved photochemical efficiency (Fv/Fm), reduced electrolyte leakage, and lower MDA accumulation, emphasizing the protective role of MT in maintaining membrane homeostasis during cold storage.

Together, these studies demonstrate that chilling injury is tightly governed by lipid metabolism-related processes, including phospholipid degradation, fatty acid desaturation, lipid peroxidation, and glycerolipid turnover. Lipidomics provides a high-resolution understanding of these pathways, offering key biomarkers and mechanistic insights for developing CI-mitigation strategies in tropical and subtropical fruits.

4.3.2. Volatilomics

Volatilomics, a specialized branch of metabolomics that focuses on the comprehensive profiling of volatile organic compounds (VOCs), has become an effective tool for elucidating metabolic alterations associated with PCI in fruits [88]. Unlike transcriptomic studies, which reveal gene-level regulation, volatilomics provides insight into the downstream biochemical consequences of stress-induced modifications to enzymatic and metabolic pathways. Chilling stress commonly inhibits the biosynthesis of aroma-related esters, terpenoids, and lactones while promoting the accumulation of lipid-derived aldehydes and alcohols, which serve as biochemical indicators of membrane lipid peroxidation and oxidative damage. In peaches (*Prunus persica* L. Batsch), [89] demonstrated that VOCs such as hexanal, (E)-2-hexenal, and γ-decalactone were closely correlated with PCI development during cold storage, suggesting their potential as early biomarkers of chilling-induced damage. Similarly, [90] investigated volatilomic changes in fresh-cut nectarines stored at 4 °C and 8 °C, showing that distinct volatile trajectories were temperature-dependent, with 17 key VOCs significantly affected by storage conditions; notably, no PCI symptoms were detected at 4 °C, indicating that optimized temperature management can prevent volatile disruption. These findings underscore the importance of volatilomic profiling as a sensitive and non-destructive approach to characterizing metabolic disturbances under chilling stress and evaluating the effectiveness of postharvest interventions aimed at preserving volatile biosynthesis, membrane stability, and overall sensory quality during cold storage.

4.4. Epigenomics

Epigenomic studies found that DNA methylation and histone alterations are essential in regulating chilling-responsive genes and stress adaptation in fruits [91]. Low–temperature-mediated dynamic methylation has a significant impact on the regulation of CI, oxidative metabolism, flowering, dormancy, ripening, flavor, and nutrient accumulation in horticultural crops. Cold conditions typically induce an overall rise in DNA methylation, which often exacerbates CI symptoms. For example, postharvest peach fruit exposed to a gradient of low temperatures (0, 5, 8, 12, and 16 °C) showed markedly higher genome-wide

methylation levels, with CHH methylation strongly correlating with the loss of softening ability and internal browning [92].

Specific CI manifestations, such as the powdery texture in peach flesh, occur because cold treatment suppresses DNA glycosylase expression, leading to hypermethylation of key genes, including *CYP82A3* and *UDP-arabinose 4-epimerase 1* [93]. In tomatoes and peaches, CI-associated loss of characteristic flavor results from the inhibition of demeter-like DNA demethylases at low temperatures, causing hypermethylation of the promoters or gene bodies of significant volatile synthesis genes. In tomato, these include *RIPENING INHIBITOR (RIN)*, *NONRIPENING (NOR)*, and *COLORLESS NONRIPENING (CNR)*, while in peach, genes such as *ACC synthase 1*, *alcohol acyltransferase 1*, and *terpene synthase 3* are silenced, suppressing volatile formation and impairing flavor development [94,95]. These findings highlight how elevated methylation represses crucial genes associated with cold tolerance and ripening. In orange, changes in DNA methylation and histone modifications were observed in genes involved in the phenylpropanoid pathway, influencing flavonoid biosynthesis and antioxidant defense under cold storage [96]. In tomatoes, histone acetylation patterns of *CBF* genes altered their transcriptional role, affecting the fruit's capability to combat chilling temperatures [97]. Research on bananas has demonstrated that the differential methylation of genes associated with ethylene biosynthesis and starch degradation influences fruit ripening and chilling sensitivity, underscoring the role of epigenetic regulation in postharvest fruit quality [98]. Omics technologies have significantly advanced the understanding of PCI mechanisms by offering deeper molecular insights.

Histone modifications involve post-translational modifications (PTMs), such as methylation, acetylation, phosphorylation, and ubiquitination, that occur at the histone core regions or the N-terminal tails extending from nucleosomes. These PTMs are strongly influenced by low temperatures and modulate histone–DNA interactions, thereby altering chromatin structure and transcriptional activation or repression. In citrus fruit, 37 histone acetyltransferases have been identified, and most of them contain low-temperature response elements (LTRs) in their promoters, indicating their active involvement in cold stress signaling [99]. In banana, the transcription factor *MaMYB4* physically interacts with histone deacetylase HDA2 and recruits it to the promoters of ω-3 fatty acid desaturase (*FAD*) genes to suppress histone acetylation; however, low temperatures inhibit MaMYB4 activity, thereby relieving repression and activating FAD expression, which helps suppress browning by modulating the saturated-to-unsaturated fatty acid ratio [36]. In apple, the transcription factor MdTCP15 normally activates the cold-stress negative regulator ABI1. Still, under low temperatures, it recruits histone deacetylase MdHDA6 to the ABI1 promoter, leading to histone deacetylation, transcriptional inhibition, and reduced low-temperature-induced flower drop and fruit set failure [100].

Furthermore, histone acetylation in horticultural crops generally neutralizes the positive charge on histone tails, weakens histone–DNA affinity, increases chromatin accessibility, and thereby activates transcription. In contrast, deacetylation typically represses gene expression [101]. During cold stress in banana fruit, low temperatures induce elevated acetylation levels of histones H3 and H4 (H3ac/H4ac) on the promoters of FAD-related genes (MaFAD3-1/3-3/3-4/3-7), promoting their expression and enhancing the conversion of linoleic acid to α-linolenic acid. This key mechanism maintains membrane integrity under chilling conditions.

Small RNA-mediated regulation has emerged as an additional epigenetic layer influencing postharvest chilling responses in fruits. MicroRNAs (miRNAs) and their associated competing endogenous RNA (ceRNA) networks modulate the expression of cold-responsive transcription factors, enzymes involved in membrane stability, and antioxidant defense [102]. In cucumber and citrus, cold-induced lncRNAs act as endogenous miRNA

sponges, altering the activity of miRNA families that target stress-regulated genes such as desaturases, ROS-scavenging enzymes, and CBF-associated transcription factors [103]. Similar small RNA-mediated interactions have also been predicted in several fruits, where miRNA–mRNA modules participate in regulating membrane fluidity, osmotic balance, and flavonoid biosynthesis—key processes that directly influence chilling injury sensitivity and fruit quality during low-temperature storage.

Multi-omics studies in fruits such as kiwifruit, mango, and tomato further reveal that small RNAs coordinate complex regulatory networks during cold storage [102]. In kiwifruit, chilling activates lncRNA–miRNA–mRNA modules associated with starch and sucrose metabolism, expansins, PME/PG enzymes, and sugar transporters, all of which help maintain firmness and reduce cold-induced texture loss [104]. Mango [105] and tomato [106] cold-responsive transcriptomes exhibit extensive interactions between lncRNAs, miRNAs, and target genes associated with ROS detoxification, lipid remodeling, hormonal balance, and cell wall stability. These integrated networks demonstrate that small RNAs serve as crucial epigenetic regulators, fine-tuning postharvest chilling tolerance, and supporting their potential application in identifying biomarkers and breeding cold-resilient fruit cultivars.

Future work must shift from isolated omics studies to integrated multi-omics modeling to develop robust biomarkers of chilling tolerance. Machine-learning tools are increasingly needed to merge transcriptomic, lipidomic, and metabolomic datasets into predictive PCI susceptibility models.

5. Emerging Strategies for PCI Mitigation in Fruits

Recent advancements in molecular biology and biochemical interventions have led to the development of novel exogenous treatments and genetic modifications to combat PCI. These strategies target key pathways, including ROS scavenging, Ca^{2+}-Driven membrane stability, hormonal signaling, and transcriptional regulation of cold-responsive genes. This section examines these emerging strategies, their molecular mechanisms, and their effectiveness across different fruit species.

5.1. Physical Treatments

Physical therapies are essential for reducing PCI because they improve stress tolerance and alter fruit physiology. Fruits may adapt to low temperatures by employing temperature control techniques, such as preconditioning and occasional warming, which help mitigate oxidative stress and membrane damage. By inducing heat shock proteins and activating antioxidant defenses, heat therapies such as hot water treatment (HWT), low-temperature condition (LTC), ultraviolet (UV) treatment, intermittent warming (IW), cold shock treatment (CST), and gaseous modifications enhance chilling tolerance (Table 1).

Table 1. Physical treatments for postharvest chilling injury and their effects.

Treatment	Fruit	Treatment Conditions	Molecular/Physiological Effects	PCI Mitigation Mechanism	References
Hot Water Treatment (HWT)	Zucchini	HWFC (40 °C, 2 m/s, 20 min) & HWD (40 °C, 25 min)	EL ↓, MDA ↓, weight loss ↓, firmness ↑, TSS ↑, ascorbic acid ↑	Heat diffusion enhances stress tolerance, reduces lipid peroxidation	[107]
	Banana	52 °C for 3 min, 7 °C storage	ROS ↓, lipid peroxidation ↓, EL ↓, proline ↑, HSPs ↑	Activates stress-response genes, maintains cell structure	[108]

Table 1. *Cont.*

Treatment	Fruit	Treatment Conditions	Molecular/Physiological Effects	PCI Mitigation Mechanism	References
	Sweet Pepper	45 °C for 15 min, 6 °C storage	APX ↑, GR ↑, DHAR ↑, MDHAR ↑, ROS ↓	Enhances antioxidant defenses, reduces oxidative stress	[109]
	Cherry Tomato	40 °C for 15 min, 5 °C storage	Chloroplast-to-chromoplast transition ↑, mitochondrial integrity ↑	Protects cellular structures, minimizes oxidative damage	[110]
	Bell Pepper	53 °C for 4 min, 8 °C storage	PCI symptoms ↓, polyamines ↑	Polyamines stabilize membranes	[111]
Low-Temperature Conditioning (LTC)	Loquat	5 °C for 6 d, then 0 °C for 54 d	Internal browning ↓, juice content ↑	Regulates lignin biosynthesis, oxidative stress	[24]
	Avocado	6–8 °C for 3–5 d, then 0 °C for 3–4 weeks	Skin pitting ↓, discoloration ↓	Enhances membrane stability, stress-response genes	[112]
	Mango	12 °C for 24 h, then 5 °C for 25 d	Soluble solids ↑, proline ↑, ROS ↓, EL ↓	Upregulates MiCBF1, activates cold-response genes	[70]
	Grapefruit	16 °C for 7 d, then 2 °C storage	Ascorbic acid ↑, flavonoids ↑, carotenoids ↑	Enhances nutritional and sensory quality	[113]
UV Treatment (UV-C/UV-B)	Bell Pepper	UV-C (7 kJ/m^2), 10 °C for 18 d	Firmness ↑, decay ↓, EL ↓	Induces ROS, boosts antioxidant defenses	[114]
	Tomato	UV-C/UV-B, 2 °C for 20 d, then 10 d at 20 °C	Ethylene ↓, firmness ↑, TSS ↑	Activates HSPs, antioxidant enzymes	[115]
	Peach	UV-C (1.5 kJ/m^2), 1 °C for 35 d	Sugar ↓, acid ↓, fruit quality ↑	Regulates sugar and acid metabolism	[116]
Intermittent Warming (IW)	Bell Pepper	4 °C storage, IW at 20 °C	Firmness ↑, membrane integrity ↑, UFAs ↑	Delays lipid peroxidation, stabilizes membranes	[117]
	Tomato	2.5 °C or 6 °C storage, IW at 20 °C	PCI symptoms ↓, deterioration ↓	Enhances antioxidants, ethylene pathway	[118]
	Peach	IW at 2 °C	ACO ↑, ACS ↑, woolliness ↓	Activates ethylene, cell-wall enzymes	[119]
	Pomegranate	2 °C storage, IW at 20 °C	UFAs/SFAs ↑, polyamines ↑	Stabilizes membranes, reduces stress	[120]

Abbreviations: ACO: 1-Aminocyclopropane-1-Carboxylate Oxidase; ACS: 1-Aminocyclopropane-1-Carboxylate Synthase; APX: Ascorbate Peroxidase; DHAR: Dehydroascorbate Reductase; EL: Electrolyte Leakage; GR: Glutathione Reductase; HSPs: Heat Shock Proteins; HWFC: Hot Water Forced Convection; HWD: Hot Water Dipping; IW: Intermittent Warming; LTC: Low-Temperature Conditioning; MDA: Malondialdehyde; MDHAR: Monodehydroascorbate Reductase; PCI: Postharvest Chilling Injury; ROS: Reactive Oxygen Species; SFAs: Saturated Fatty Acids; TSS: Total Soluble Solids; UFAs: Unsaturated Fatty Acids; UV-C: Ultraviolet-C Radiation. ↑: increase; ↓: decrease.

5.1.1. Hot Water Treatment (HWT)

The HWT has been thoroughly investigated as a successful postharvest method to reduce PCI in various fruits by affecting molecular and biochemical pathways linked to antioxidant defense mechanisms, membrane stability, and stress response. Hot water forced convection (HWFC) at 40 °C with a water flow rate of 2 m/s for 20 min and hot water dipping (HWD) at 40 °C for 25 min both significantly decreased PCI symptoms in zucchini when stored for 15 d at 4 ± 0.5 °C with 85–90% relative humidity [107]. While lowering relative EL, MDA buildup, and weight loss, HWFC outperformed HWD in preserving fruit firmness, total soluble solids (TSS), and ascorbic acid content. HWFC's exceptional performance was ascribed to its effective heat transfer mechanism, which promoted intracellular heat diffusion and inhibited oxidative stress-induced lipid peroxidation and membrane breakdown. In bananas, HWT at 52 °C for 3 min and then storage at 7 °C reduced PCI by inhibiting oxidative stress ($O_2^{\bullet -}$ buildup), lipid peroxidation, and EL while boosting proline production, a crucial osmoprotectant implicated in cellular stress response. In addition to the activation of HSPs, which are essential for maintaining protein homeostasis under cold stress, transcriptomic analysis showed that genes related to photosynthesis, chlorophyll metabolism, lipid metabolism, glutathione metabolism, brassinosteroids biosynthesis, and carotenoid biosynthesis were markedly upregulated [108].

Similarly, via modifying the AsA-GSH cycle, a crucial antioxidant defense system, a 45 °C HWT for 15 min in sweet pepper produced tolerance to chilling stress at 6 °C storage [109]. Ascorbate peroxidase (APX), GR, dehydroascorbate reductase (DHAR), and monodehydroascorbate reductase (MDHAR) all showed increased activity in treated fruit, which improved ROS scavenging and decreased oxidative damage. This enzymatic improvement prevented membrane lipid peroxidation and maintained fruit integrity by reducing MDA and H_2O_2 buildup and alleviating PCI [109].

The HWT at 40 °C for 15 min, followed by storage at 5 °C for 19 d and ripening at 20 °C, significantly reduced PCI in cherry tomatoes by maintaining the chloroplast-to-chromoplast transition and promoting typical red coloration. According to ultrastructural examination, heat-treated fruits retained their mitochondrial integrity, whereas untreated fruits showed significant cellular disarray, damaged chloroplasts, and widespread thylakoid degradation [110].

In bell peppers, PCI-related degradation was prevented more effectively by 53 °C HWT for 4 min before storage at 8 °C than by 45 °C for 15 min [111]. Because HWT reduces weight loss and modifies internal gas composition, it prevents ethylene accumulation and further improves CI tolerance when combined with low-density polyethylene (LDPE) packing. Polyamines, particularly putrescine and spermine, which stabilize membranes and scavenge free radicals, are associated with cold stress tolerance. Notably, fruits that were heat-treated and packaged retained higher quantities of these compounds.

5.1.2. Low Temperature Conditioning

LTC has become a viable method for reducing postharvest PCI in various fruits by causing molecular and physiological changes that improve resistance to cold. When kept below 5 °C, PCI in the loquat cv. Luoyangqing causes lignification, tissue browning, and juice loss, a serious postharvest problem. These PCI symptoms were successfully decreased, storage life was increased, and fruit quality was preserved by LTC at 5 °C for 6 d before moving to 0 °C storage for 54 d. With a 3 d shelf life at 20 °C, control fruits that were kept directly at 0 °C showed significant internal browning (IB index > 0.4), tissue hardness (flesh stiffness > 6.0 N), and decreased juice content (<60%), limiting their storage potential to 40 d [24]. On the other hand, fruits treated with LTC could be kept at 0 °C for 60 d without losing their quality. LTC-mediated PCI mitigation in loquat most likely involves

modulating lignin biosynthesis and reducing oxidative stress to maintain cellular integrity and postpone senescence.

When kept below 3 °C, avocado cv. Hass suffers from external chilling stress, which shows up as pitting and blackening of the skin. LTC was tested at 15 °C after being tested at 4–15 °C for 1–5 d and then stored at 0 °C for 3–4 weeks. External damage was significantly reduced by conditioning at 6–8 °C for 3–5 d, whereas untreated fruits experienced severe pitting and discoloration [112]. Interestingly, the decrease in PCI was unrelated to the production of ethylene or CO_2, indicating that LTC directly affects membrane stability and stress-responsive pathways, rather than ripening-induced metabolic alterations. Although the precise molecular mechanism remains unknown, LTC may enable avocados to resist cold stress by altering the composition of cuticular wax, the activity of antioxidant enzymes, or the expression of genes that respond to stress.

The development of PCI was dramatically inhibited in mango by LTC at 12 °C for 24 h, followed by refrigeration at 5 °C for 25 d. This also facilitated softening and enhanced the accumulation of soluble solids and proline, a crucial osmoprotectant implicated in stress adaptation and membrane stability. Additionally, LTC preserved membrane integrity by lowering ROS production ($O_2^{\bullet -}$ and H_2O_2), EL, and MDA buildup [70]. Molecularly, LTC increased the expression of the *MiCBF1* gene, which is essential for cold stress signaling because it activates COR genes, stabilizes cellular structures, and strengthens antioxidant defenses. This implies that LTC sets up a series of physiological and gene-regulatory reactions in mango fruit, preparing it for cold tolerance. When grapefruits cv. Star Ruby was stored at 2 °C, and PCI was at its worst; however, chilling-related damage was avoided when fruits were stored at 11 °C. Ascorbic acid, carotenoids, and flavonoids were among the bioactive components that were preserved, while PCI was effectively reduced by a conditioning treatment at 16 °C for 7 d before cold storage at 2 °C [113]. Fruits stored with CD at 2 °C had higher amounts of ascorbic acid for 12 weeks, while those stored at 11 °C accumulated more flavonoids and carotenoids after 16 weeks. Fruits treated with conditioning also showed reduced decay rates and higher taste ratings, indicating that long-term storage improves both nutritional and sensory quality, in addition to alleviating PCI [113].

5.1.3. Ultraviolet (UV) Treatment

After exposure to UV-C at 7 kJ/m^2, peppers were kept at 10 °C for 18 d. In untreated fruit, the low storage temperature caused severe PCI, which was manifested by rotting, weakening, and damage to the fruit's tissue. However, UV-C treatment reduced PCI by preserving fruit firmness, minimizing decay, and reducing EL, indicating improved cellular membrane stability [114]. The ROS are generated during UV-C-induced stress reactions, which trigger antioxidant defenses that protect cellular structures from oxidative damage. This is the molecular mechanism underlying this. To mitigate PCI symptoms and maintain fruit quality during cold storage, UV-C alters the levels of phenolic compounds involved in defense responses.

The tomatoes were kept for 20 days at 2 °C and then for 10 d at 20 °C. Chilling damage affected fruit quality, which was a significant concern during cold storage. By postponing ethylene generation, slowing ripening, and maintaining firmness and soluble solid content, preconditioning with UV-C or UV-B irradiation has been shown to lessen CI symptoms. The biological process entails the activation of heat shock proteins and antioxidant enzymes by UV light, which reduces oxidative stress and stabilizes cell membranes [115]. Reduced chilling damage and improved postharvest quality were significant benefits of the UV treatment, which also affected sugar and acid metabolism by postponing the breakdown of sucrose and organic acids.

A study by [84] stated that peaches, when exposed to UV-C at 1.5 kJ/m^2 and kept at 1 °C for 35 d, experienced reduced PCI. By slowing down the decomposition of sugars and organic acids, UV-C therapy reduced PCI during storage while preserving fruit quality. UV-C regulates the enzymes responsible for sugar metabolism (invertase) and organic acid metabolism (citrate synthase, malate dehydrogenase) as part of the biochemical mechanism behind PCI mitigation. Fruit stability under cold storage conditions is maintained, and energy reserves are preserved by UV-C treatment, which inhibits invertase activity and downregulates the enzymes involved in the breakdown of citric and malic acids. By reducing oxidative damage, this control helps to improve freezing tolerance.

5.1.4. Intermittent Warming (IW)

Peppers kept at 4 °C with IW cycles at 20 °C demonstrated firmness, membrane integrity, and decreased PCI. IW prevented lipid peroxidation and maintained cell membrane integrity by delaying the decrease in unsaturated fatty acid concentration. This process lessened deterioration and structural damage while preserving the texture and freshness of the fruit [117]. Another study by [86] showed that while cultivar-specific efficacy varied, tomatoes stored at 2.5 °C or 6 °C with IW cycles at 20 °C showed fewer PCI symptoms and less deterioration. IW improved fruit resistance to cold stress by increasing the activity of antioxidant enzymes and ethylene production. Furthermore, in specific cultivars, IW encouraged the development of a red hue, which may help preserve regular ripening processes during postharvest storage.

IW treatment at 2 °C increased ethylene production by upregulating 1-aminocyclopropane-1-carboxylic acid oxidase (ACO) and synthase (ACS); however, peaches held at 0 °C developed woolliness, a CI disease. This ethylene pathway activation halted the ripening inhibition caused by CI. To maintain fruit texture and ripening potential, IW also stimulated the development of enzymes that change cell walls, such as PG and endo-1,4-glucanase (EGase) [119]. When exposed to IW at 20 °C, pomegranates kept at 2 °C with 90% relative humidity had the best PCI mitigation. IW prevented membrane damage and lipid peroxidation by maintaining the ratio of unsaturated to saturated fatty acids (UFAs/SFAs). Furthermore, IW increased levels of polyamines, particularly putrescine and spermine, which stabilized cellular structures and functioned as antioxidants, thereby reducing symptoms of PCI and preserving fruit quality after harvest [120].

5.1.5. Cold Shock Treatment (CST)

The PCI development in papaya held at 5 °C for 30 d and then at 25 °C for 3 d was successfully alleviated by CST [121]. After an hour of treatment with ice water (4 °C), papayas exhibited decreased respiration rates, ethylene production, and PCI incidence. While lowering levels of ROS, such as $O_2^{\bullet -}$ and H_2O_2, MDA, and cell membrane permeability, CST preserved higher amounts of ascorbic acid and exhibited a greater 2,2-diphenyl-1-picrylhydrazyl (DPPH) radical scavenging capacity. Oxidative damage was reduced in part by the increased activity of antioxidant enzymes, including glutathione reductase (GR), APX, CAT, and SOD. Significantly increased *CpSOD2*, *CpCAT1*, *CpAPX2*, and *CpGR2* gene expression indicates that CST triggers genetic reactions that enhance oxidative defense systems and preserve membrane integrity, ultimately maintaining postharvest papaya quality [121].

Following CST at 0 °C for 4 h, mangoes stored at 2 °C with 85–95% RH showed a 59.7% lower PCI index than untreated fruit. The treatment considerably decreased ion leakage; cold-shocked mangoes exhibited 16% and 10% less leakage at storage intervals than controls. Furthermore, the MDA content decreased by 70% and 50% at various storage times compared to the control fruit, suggesting enhanced membrane stability. The activities

of CAT and APX increased, along with higher glutathione and phenolic compound content, while SOD, GR, and ascorbic acid levels were minimally affected [122]. These biochemical and antioxidant responses underscore the role of CST in mitigating oxidative stress and maintaining fruit integrity during cold storage, rendering it a promising approach for postharvest mango management.

Compared to other temperatures, guavas kept at 6 °C displayed the lowest PCI and the highest overall fruit quality. Guavas exposed to CST for 6, 9, or 12 h before storage at 4 °C showed less weight loss, increased firmness, and higher nutritional quality [123]. With the lowest MDA concentration and higher levels of proline and soluble protein—important biomarkers of cold tolerance—a 12 h CST produced the best outcomes. By increasing the activities of essential antioxidant enzymes, such as SOD, POD, CAT, and APX, while maintaining a higher ascorbic acid concentration, CST regulated the metabolism of ROS. Longer CST improves oxidative defense mechanisms, prolonging guava's shelf life and maintaining its postharvest quality under cold storage. The study also found a direct association between CST duration and antioxidant capability.

Cold shock-treated peaches (0 °C for 10 min) kept at 5 °C for 28 d showed lower MDA levels, decreased EL, and noticeably delayed PCI symptoms. Peaches treated with CST exhibited increased antioxidant enzyme activity (SOD, APX, CAT, and POD) and decreased ROS accumulation [124]. Additionally, CST reduced the activity and gene expression of PLD, lipase, and LOX—essential enzymes involved in membrane lipid metabolism. This control helped to stabilize membrane lipids, lower the buildup of phosphatidic acid, and maintain a higher ratio of unsaturated to saturated fatty acids. These results support the potential use of CST in preserving peach quality during cold storage by reducing PCI, strengthening enzymatic antioxidant defenses, and maintaining membrane integrity.

5.1.6. Gaseous Modifications

Gaseous modifications involving MAP and controlled atmosphere (CA) conditions (approximately 5% CO_2 and 10% O_2) significantly reduced PCI symptoms, including lenticel spotting, in mangoes stored at 12 °C. The most efficient PCI reduction was achieved by packaging using Xtend® film (XF), most likely due to its capacity to control the gas composition and humidity levels within the packaging. By slowing metabolic processes and limiting oxidative stress, this altered environment reduced damage caused by cooling [125]. Similarly, in ethylene-suppressed cantaloupe melons stored at 2 °C, MAP helped mitigate PCI symptoms by reducing ethylene production, limiting water loss, and minimizing ion leakage. The controlled gas environment similarly limited the accumulation of ethanol and acetaldehyde, which are linked to chilling stress [126]. According to these findings, it is possible to prolong storage life and avoid chilling-induced physiological diseases by inhibiting ethylene production and preserving a low-O_2, high-CO_2 environment.

For Japanese plums, CA conditions (1% O_2 + 3% CO_2) were superior to MAP in terms of preserving fruit firmness, lowering PCI, and titratable acidity (TA) during cold storage (0–1 °C) [127]. CA preservation enhanced the coordinated activity of antioxidant enzymes, such as peroxidase and SOD, while reducing ROS generation and lipid peroxidation. The AsA-GSH cycle's role in controlling oxidative stress was also demonstrated, indicating that CA enhances cellular defenses against cold stress and slows fruit ripening.

5.2. Chemical Treatments and Their Molecular Impact

Exogenous treatments play a crucial role in combating PCI by modulating cold-stress signaling, enhancing membrane stability, and activating key genes involved in combating PCI. These treatments act via multiple mechanisms: (a) lowering oxidative damage by accelerating antioxidant enzyme activities, (b) stabilizing cell membranes via lipid remodeling

and Ca regulation, and (c) modulating hormonal interplay to activate PCI-responsive transcription factors. Each treatment affects fruit physiology exclusively, resulting in distinct molecular actions (Figure 2; Table 2).

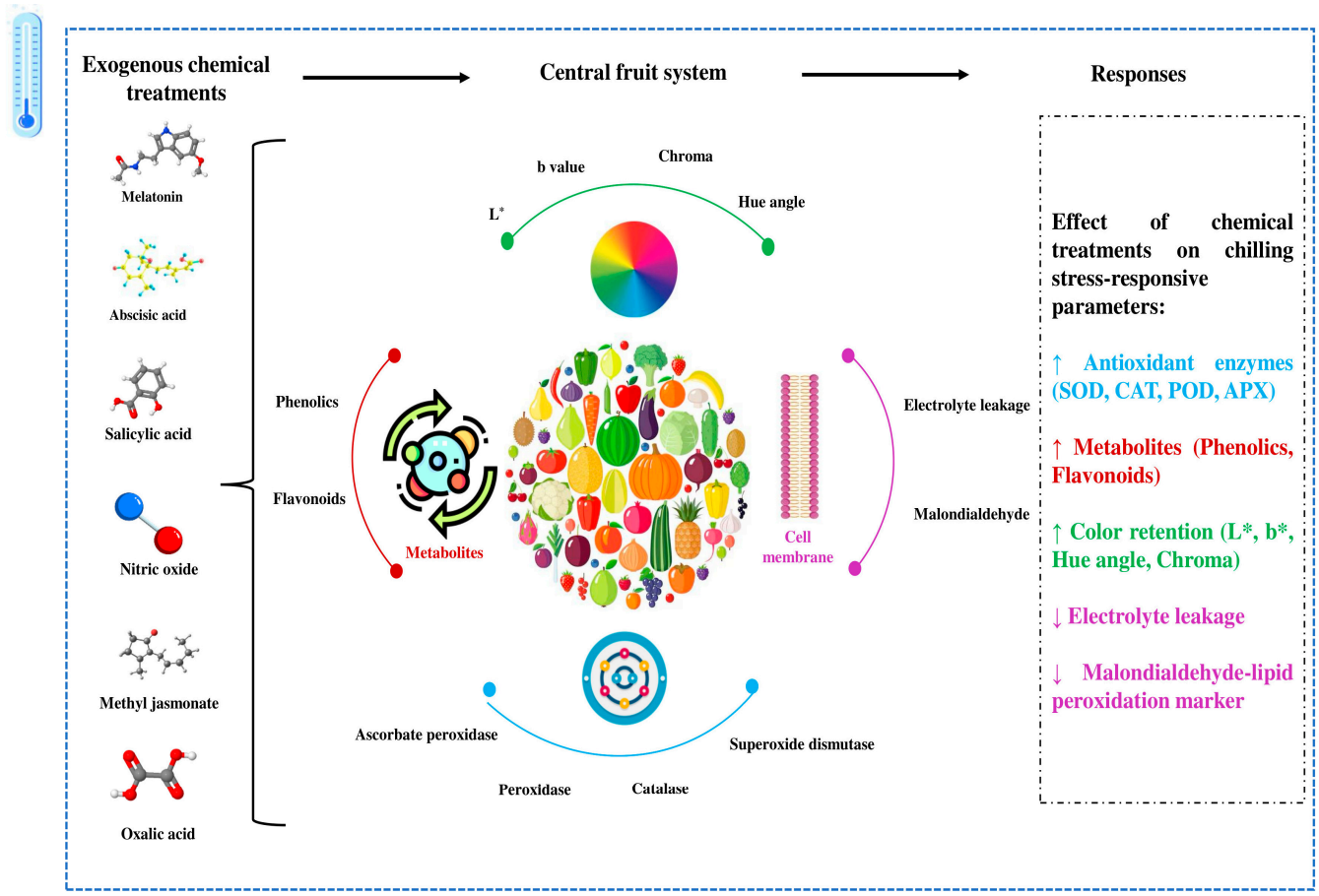


Figure 2. Exogenous chemical elicitors enhance antioxidant capacity, metabolite accumulation, and membrane stability, thereby alleviating postharvest chilling injury in fruits. Various signaling molecules and plant growth regulators, including melatonin, abscisic acid, salicylic acid, nitric oxide, methyl jasmonate, and oxalic acid, induce multiple protective responses. Chemical treatments stimulate antioxidant enzymes (SOD, CAT, POD, APX), promote the biosynthesis of metabolites such as phenolics and flavonoids, and preserve color attributes (L*, b*, hue angle, chroma). Collectively, these changes restrict electrolyte leakage and reduce the accumulation of the lipid peroxidation marker MDA, thereby maintaining cell membrane integrity and improving chilling tolerance. Abbreviations: SOD: Superoxide dismutase; CAT: Catalase; POD: peroxidase; APX: Ascorbate peroxidase; L*: lightness; b*: yellowness; MDA: Malondialdehyde.

Table 2. Chemical treatments for postharvest chilling injury and their effects.

Treatment	Fruit	Gene Expression/Enzymatic Activity	Result	Reference
Melatonin	Plum	↓ *ETR*, *EBF*, *ERF*, *EIN*	↓ Ethylene production, ↓ Ripening rate	[81]
	Eggplant	↑ *SOD*, *CAT1/2*, ↓ *PME*, *PG*	↑ Antioxidant activity, ↓ Senescence	[128]
	Tomato	↑ H-ATPase, Ca-ATPase ↓ PLD, LOX	↑ Chilling tolerance, ↓ Membrane damage	[129]

Table 2. *Cont.*

Treatment	Fruit	Gene Expression/Enzymatic Activity	Result	Reference
	Mango	↑ P5CS, P5CR, OAT; ↓ PDH3	↑ Proline accumulation, ↑ Stress resistance	[130]
	Banana	↑ *MaSOD*, *MaCAT* ↓ *MaRBOH*	↑ Antioxidant defense, ↓ ROS damage	[131]
Salicylic acid	Kiwifruit	↑ *PAL*, *C4H*, *4CL*, *DREB*	↑ Aroma biosynthesis	[132]
	Peach	↑ *DREB*	↑ Sugar accumulation, ↑ Cold tolerance	[133]
	Winter jujube	↑ *SOD1/3*	↑ Antioxidant response, ↑ Fruit firmness	[134]
Oxalic acid	Tomato	↑ SDH, Ca^{2+}-ATPase, H^{+}-ATPase	↑ Chilling resistance, ↑ Carotenoid biosynthesis	[135]
	Hami melon	↑ *CmGR*, *CmAPX*, *CmPOD*	↑ Antioxidant enzyme activity, ↓ Senescence	[136]
Methyl jasmonate	Peach	↑ *PpNAC1* ↓ *PpACS1*, *PpExp1*	↓ Softening, ↓ Ethylene biosynthesis	[137]
	Kiwifruit	↓ LOX, *ACC*, *ACS* ↑ *MYB*, *WRKY*, *bZIP*	↓ Ripening rate, ↑ Storage quality	[138]
	Tomato	↑ Starch breakdown, ↑ Sucrose accumulation; ↓ Glucose, Fructose	↑ Sugar metabolism, ↑ Taste retention	[139]
Abscisic acid	Peach	↑ AsA-GSH cycle; ↓ *PpACO1*, *PpEIN2*	↑ Antioxidant activity, ↓ Ethylene response	[140]
Polyamines	Apricot	↑ POX, CAT, SOD	↑ Postharvest storage, ↓ Decay	[141]

Abbreviations: *ACC*: 1-Aminocyclopropane-1-Carboxylate; *ACO*: ACC Oxidase; *ACS*: ACC Synthase; APX: Ascorbate Peroxidase; AsA-GSH: Ascorbate-Glutathione Cycle; *bZIP*: Basic Leucine Zipper Protein; Ca-ATPase: Calcium-Transporting ATPase; Ca^{2+}: Calcium Ion; CAT: Catalase; *C4H*: Cinnamate 4-Hydroxylase; *CmAPX*: Cucumis melo Ascorbate Peroxidase; *CmGR*: Cucumis melo Glutathione Reductase; *CmPOD*: Cucumis melo Peroxidase; *DREB*: Dehydration-Responsive Element-Binding Protein; *EBF*: EIN3-Binding F-Box Protein; *EIN*: Ethylene-Insensitive Protein; *ERF*: Ethylene Response Factor; *ETR*: Ethylene Receptor; H-ATPase: Proton-Transporting ATPase; LOX: Lipoxygenase; *MaCAT*: *Musa acuminata* Catalase; *MaRBOH*: *Musa acuminata* Respiratory Burst Oxidase Homolog; *MYB*: Myeloblastosis Transcription Factor; *NAC*: NAC Transcription Factor; OAT: Ornithine Aminotransferase; PAL: Phenylalanine Ammonia-Lyase; P5CS: Δ^{1}-Pyrroline-5-Carboxylate Synthase; P5CR: Pyrroline-5-Carboxylate Reductase; PDH3: Pyruvate Dehydrogenase 3; PME: Pectin Methylesterase; PG: Polygalacturonase; SDH: Succinate Dehydrogenase; SOD: Superoxide Dismutase; *WRKY:* WRKY Transcription Factor; ↑: increase; ↓: decrease.

5.2.1. Melatonin (MT)

MT, a potent antioxidant and signaling molecule, plays a crucial role in ROS detoxification, Ca homeostasis, and the transcriptional regulation of PCI-responsive genes. One of its primary safeguarding mechanisms is membrane stabilization via lipid remodeling. A 1 mM dose of MT in plums mitigated PCI by reducing flesh reddening and ethylene accumulation. Transcriptomic profiling revealed that MT downregulated key ethylene-associated genes (*ETR*, *EBF*, *ERF*, *EIN*), suppressing ethylene-related responses that amplify PCI symptoms [81]. Additionally, MT downregulates the expression of anthocyanin biosynthesizing genes (*PAL*, *C4H*, *F3H*, *DFR*, *ANS*, *UFGT*), thereby inhibiting the excessive accumulation of cyanidin-3-O-glucoside, which is associated with flesh reddening under chilling temperatures. The suppression of *PsMYB124*, a transcription factor noted for enhancing anthocyanin biosynthesis, further strengthened the inhibition of color changes in MT-treated

plums [81]. The mechanism indicates MT interferes with ethylene and MYB-mediated anthocyanin pathways, thereby mitigating PCI.

Application of MT at a concentration of 100 µM effectively alleviated PCI in 'Langra' mango fruit during low-temperature storage. The treatment enhanced antioxidant enzyme activities and upregulated stress-responsive genes such as *MiCBF*, *MiASMT*, *MiSNAT*, *MiCML*, and *MiCAM*, underscoring its pivotal role in conferring chilling tolerance at the molecular level. These findings highlight MT's potential as a promising postharvest treatment for mitigating PCI and preserving fruit quality [142]. The MT application at 100 µM in cold-stored eggplant has been studied to improve postharvest quality by preventing PCI symptoms, such as surface pitting and weight loss. Molecular investigation revealed that MT contributed to the upregulation of antioxidant-related genes, including *SOD* and *CAT1/2*, thereby lowering oxidative damage by reducing MDA and H_2O_2 accumulation [128]. Polyamine-related genes, *ADC* and *ODC*, and MT-synthesizing genes, *TDC1*, *T5H*, *SNAT1*, *ASMT*, and *COMT*, were upregulated, indicating MT enhancement reinforced the antioxidant activity. MT also upregulated *CBF*, *COR1*, *ZAT2*, *ZAT6*, and *ZAT12*, the cold-responsive genes, which aided in combating PCI. Also, inhibition of cell wall-disrupting genes, including *PME* and *PG*, and senescence-related genes (*IAA17*, *SAG12*, *SEN4*), delayed fruit softening, thus maintaining the shelf-life of cold-stored eggplant [128].

A 100 µM MT application in tomato mitigated PCI by safeguarding cellular energy homeostasis and membrane integrity. MT-treated tomatoes showed higher ATP-synthesizing activity by enhancing *H-ATPase*, *Ca-ATPase*, *CCO*, and *SDH*, thus ensuring sufficient intracellular ATP levels under chilling conditions. Moreover, exogenous MT was observed to upregulate *FAD3* and *FAD7*, the FADs, and therefore modulated lipid metabolism. Simultaneously, the MT application downregulated *PLD* and *LOX*, thereby inhibiting uncontrolled lipid peroxidation and maintaining membrane stability and postharvest quality [129]. A 0.1 mM MT application on mangoes mitigated PCI symptoms and maintained fruit quality by retaining the enzymatic activities of *P5CS*, *P5CR*, and *OAT*, while the degradation pathway, governed by *PDH3*, was downregulated [130]. Likewise, exogenous MT (500 µM) significantly alleviated PCI in bananas. The transcriptomic study in MT-treated bananas revealed that MT application upregulated *MaSOD*, *MaCAT*, *MaAPX*, and *MaGR*, the key antioxidant genes, thereby enhancing ROS detoxification. Also, MT lowered the expression of *MaRBOH*, a ROS-producing enzyme, thus inhibiting excessive oxidative damage [131].

5.2.2. Salicylic Acid (SA)

The transcriptomic study in SA-treated kiwifruit showed that SA upregulates *PAL*, *C4H*, and *4CL*, the key genes related to the phenylpropanoid pathway, resulting in increased accumulation of phenolic content [132]. By scavenging ROS and enhancing cell wall integrity, these phenolics help fruit tolerate cold temperatures [143]. Furthermore, SA enhanced the fruit's stress adaptation mechanisms by modifying GA, JA, and ABA signaling, which in turn affected phytohormone signaling pathways. The presence of SA response elements in the promoter regions of genes with variable expression further supports the direct regulatory role of SA in reducing PCI through transcriptional regulation.

By altering sugar metabolism, SA treatment (1 µM for 15 min) in peaches considerably decreased browning and postponed damage caused by cooling [133]. Fruit treated with SA showed increased expression of key genes related to sucrose metabolism, including *PpSUS4* (*sucrose synthase*), *PpNINV8* (*neutral invertase*), and *PpTMT2* (*tonoplastic monosaccharide transporter*), which resulted in increased buildup of sucrose and improved resistance to chilling. Furthermore, SA increased the expression of transcription factors that respond to cold, specifically *DREB* (*Dehydration-Responsive Element-Binding Proteins*), essential for triggering

pathways that protect against cold stress. By controlling genes in the ester production pathway, including *PpLOX1* (*lipoxygenase*), *PpHPL1* (*hydroperoxide lyase*), *PpADH1* (*alcohol dehydrogenase*), and *PpAAT1* (*alcohol acyltransferase*), SA also contributed to the preservation of the fruit's volatile profile. These genes helped preserve fruit quality and aroma during cold storage by increasing volatile retention [133].

Cold storage causes oxidative damage and fast senescence in winter jujube (*Ziziphus jujuba*) [134]. SA treatment activated essential antioxidant-related genes, such as *SOD1/3*, *GRXC2* (glutaredoxin), *APX1*, and *CTV5*, to enhance the ROS scavenging ability and mitigate chilling stress. Additionally, transcriptome research has demonstrated SA's role in controlling plant hormone signaling. Differential expression of genes linked to ABA (*PYL2*, *PP2C*, *SnRK2*), ethylene (*ERF*, *EBF2*), and JAZ signaling suggests that SA has a wide-ranging effect on hormonal crosstalk in the chilling stress response. SA also delayed fruit aging and preserved postharvest quality by modulating senescence-associated genes (*SAG21*, *PME15*, *CTL2*, *INVE*, and *ADH2*). Furthermore, activating transcription factors such as *MYB*, *ERF*, *C2H2*, *MTB3*, *WRKY*, and *FAR1* improved the winter jujube's ability to adapt to stress and withstand cold [134].

5.2.3. Oxalic Acid (OA)

Tomatoes dipped in 10 mM OA for 10 min exhibited lower PCI symptoms than untreated tomatoes. Exogenous OA maintained membrane integrity and higher ATP and ADP levels, accompanied by a rise in succinic dehydrogenase (SDH), Ca^{2+}-ATPase, and H^{+}-ATPase activity [135]. This increase in activity helps in proper mitochondrial respiration and energy metabolism, indicating OA to increase cellular energy homeostasis, permitting the fruit to perform metabolic activities under cold storage. Another important protective function of OA in tomatoes is its role in lycopene biosynthesis, which is essential for preserving the fruit's color and postharvest quality. The OA application showed higher lycopene accumulation in cold-stored tomatoes, which was associated with the upregulation of phytoene synthase 1 (*PSY1*) and ζ-carotene synthase [135]. The enhanced expression of these genes on OA application indicates their function not only in mitigating PCI-associated pigmentation loss but also in enhancing antioxidant compounds, thus mitigating PCI.

Another study states that exogenous OA (15 mM for 10 min) successfully alleviates PCI symptoms in Hami melons stored at 3 °C for 42 d. An essential mechanism employed by OA was its ability to enhance the melon's antioxidant defense system, thereby reducing PCI-induced oxidative damage. The OA application resulted in the upregulation of *CmGR*, *CmAPX*, and *CmPOD*, the genes responsible for enhancing the activity of GR, APX, and POD [136]. The improved antioxidant activities helped in combating PCI in cold-stored melons.

5.2.4. Methyl Jasmonate (MeJA)

MeJA has emerged as an essential modulator of PCI tolerance in fruits by regulating stress-induced pathways at the molecular level. Exogenous MeJA (1 mM) treatment to peach stored at 0 °C for 28 d delayed PCI symptoms by regulating ethylene synthesis and ripening-linked pathways. Exogenous MeJA upregulated the *PpNAC1* and *PpMYC2.2* genes, which further promoted PCI-resistance, while lowering DNA methylation in the promoter regions of *PpACS1*, *PpExp1*, and *PpAAT1*, aiding in controlled ethylene production and maintaining fruit firmness and volatiles [137]. Another study on cold-stored kiwifruit states an exogenous MeJA dose (10 μM) to mitigate lipid peroxidation by downregulating *LOX* expression. Additionally, by suppressing *PG*, *PL*, and *PE* genes, MeJA treatment delayed cell wall disintegration and regulated ethylene biosynthesis by downregulating

ACC and ACS genes. Moreover, the capability of MeJA to regulate transcription factors, including *MYB*, *WRKY*, *bZIP*, and *C2H2*, helped combat chilling stress [138]. In tomatoes stored at 2 °C, a 0.05 mM MeJA application regulated sugar metabolism by elevating starch deterioration and sucrose accumulation while downregulating the accumulation of glucose and fructose [139].

5.2.5. Abscisic Acid (ABA)

A study states that ABA is essential in alleviating PCI in peaches stored at 2–5 °C. A 100 µM ABA application significantly mitigated PCI development by limiting ethylene biosynthesis and modulating oxidative damage. The application downregulated *PpACO1* and *PpEIN2*, key genes responsible for ethylene synthesis, thus limiting ethylene production. Additionally, ABA treatment stimulates the antioxidant system by activating the AsA-GSH cycle, producing efficient ROS scavenging [140].

5.2.6. Polyamines

Polyamines, such as spermidine (Spd) and putrescine (Put), are essential for reducing PCI in fruits because they alter the fruit's physiological and biochemical reactions to cold stress. Exogenous PA treatments are beneficial in lowering PCI symptoms and preserving fruit quality during cold storage, as shown in studies involving apricots and jujube fruit.

A study by [109] showed that the incidence of PCI in apricot fruit (cv. Bagheri and Asgarabadi) dramatically decreased with 1 mM Put and Spd treatments, with Spd exhibiting a more substantial protective effect than Put. These methods delayed ripening by inhibiting ethylene production while preserving the fruit's firmness and color. Increased activity of essential antioxidant enzymes, such as POX, CAT, SOD, and PPO, along with an increase in total phenolic content (TPC), was associated with a reduction in PCI. This suggests that by strengthening the fruit's antioxidant defenses and lowering oxidative stress, polyamines help to mitigate PCI [141].

Likewise, exogenous administration of 1 mM Put, 1 mM Spd, and their combination (0.5 mM each) successfully reduced PCI symptoms such as spongy browning, mesophyll collapse, and cell structure deterioration in *Zizyphus jujuba* cv. Mill. These treatments reduced the accumulation of MDA, a crucial indicator of membrane lipid peroxidation and oxidative damage, while maintaining higher levels of TSS and TA, and conserving fruit color. By improving PCI tolerance, boosting antioxidant enzyme activity (PPO, POX), and stabilizing cellular integrity under chilling stress conditions, the combined use of Put and Spd demonstrated exceptional efficacy in preserving postharvest quality [144].

5.2.7. Calcium Chloride ($CaCl_2$)

Calcium chloride ($CaCl_2$) can reduce PCI in various fruits by adjusting physiological, biochemical, and molecular reactions, which has been extensively researched. Studies on nectarines, pineapples, and loquats have demonstrated their efficacy in enhancing antioxidant defenses, regulating secondary metabolism, and maintaining fruit quality during cold storage. A study by [113] investigated the use of exogenous $CaCl_2$ to significantly reduce chilling-induced damage in nectarines by upregulating genes related to the synthesis of secondary metabolites, particularly those involved in the phenylpropanoid pathway. Fruit treated with $CaCl_2$ showed an increment in TPC and total flavonoid content (TFC) by 12.5% and 80.6%, respectively. The generation of bioactive chemicals that scavenge ROS and strengthen fruit defense systems was facilitated by the increased expression of phenylalanine ammonia-lyase (*PAL*), 4-coumarate-CoA ligase (*4CL*), and chalcone synthase (*CHS*). By reducing ROS buildup and enhancing antioxidant enzyme activity, the treatment also increased the capacity to scavenge ROS. Furthermore, $CaCl_2$ contributed to PCI tolerance

by modifying genes associated with auxin, GA, and ABA metabolism, which, in turn, regulated hormonal signaling.

In loquat fruit stored at 1 °C for 35 d, $CaCl_2$ treatment reduced PCI symptoms by lowering the browning index and maintaining fruit firmness and extractable juice content [145]. By increasing DPPH and •OH scavenging activity and lowering $O_2^{\bullet -}$ and H_2O_2 levels, the treatment dramatically reduced ROS buildup. The $O_2^{\bullet -}$ and H_2O_2 were successfully neutralized by the increased activity of SOD, CAT, POX, and APX following $CaCl_2$ treatment. *EjSOD*, *EjCAT*, *EjAPX*, *EjGR*, *EjMDHAR*, and *EjDHAR* were significantly upregulated in loquats, as determined by gene expression analysis, suggesting an enhanced ascorbate-glutathione (AsA-GSH) cycle [145]. This cycle is essential for ROS detoxification because it prevents enzymatic browning and membrane damage caused by chilling stress.

$CaCl_2$ treatment (50 μM) preserved fruit quality, reduced ROS levels, and increased antioxidant enzyme activity in cold-stored pineapples, thereby delaying internal browning, a hallmark of PCI [146]. According to transcriptomic analysis, $CaCl_2$ affected several metabolic processes, including the TCA, fatty acid biosynthesis, MAPK signaling, and phenylpropanoid biosynthesis. Postharvest shelf life was maintained through the coordinated control of these pathways, which strengthened the fruit's defense systems.

5.2.8. Nitric Oxide (NO)

Nitric oxide (NO) has been widely recognized as an effective signaling molecule in mitigating PCI in various horticultural crops by modulating antioxidant defense systems, stress-responsive gene expression, and metabolic pathways. It has been demonstrated that brief exposure to NO before cold storage significantly reduces the symptoms of PCI, including oxidative stress, lipid peroxidation, and membrane damage. Pre-treating Hami melon with 60 μL/L NO for 3 h at 25 °C and then storing it for 49 d at 1 ± 0.5 °C and 75–80% relative humidity (RH) significantly inhibited PCI by reducing the generation of ROS, membrane permeability, and MDA accumulation [147]. This reaction was associated with enhanced enzymatic antioxidant defense, characterized by higher levels of APX, CAT, POD, and SOD. On a molecular level, NO treatment markedly increased the expression of two crucial cold-responsive transcription factors involved in cold acclimation: *CmCBF1* and *CmCBF3*. Similarly, exogenous NO (0.02 mM SNP) decreased the PCI incidence in tomatoes by increasing endogenous NO levels, which in turn reduced MDA content and ion leakage, and increased the expression of the gene *LeCBF1*, a gene responsive to cold stress [148].

NO treatment (60 μL/L) decreased lipid peroxidation and EL while upregulating the expression of critical antioxidant-related genes (*MaSOD*, *MaCAT*, *MaPOD*, and *MaAPX*) in banana fruit that had been stored for 15 d at 7 ± 1 °C with 90% RH [149]. Moreover, NO-mediated PCI alleviation in peaches was associated with controlling lipid metabolism and cell wall disintegration. In addition to decreasing the activity of PG, xyloglucan endoglycosyl transferase, cellulase, and β-galactosidase, treatment with NO donor (SNP) downregulated genes encoding xyloglucan *endotransglycosylase/hydrolase* family proteins. Furthermore, NO influenced lipid metabolism by increasing the expression of genes that encode *long-chain acyl-CoA synthetase*, *phosphatidylinositol bisphosphate*, *ketoacyl-ACP synthase*, and *glycerol-3-phosphate acyltransferase*, all of which support membrane stability in cold stress [150].

5.3. Biotechnological Approaches

Biotechnological advancements have offered novel solutions for alleviating PCI in fruits. Genome editing technologies, especially CRISPR/Cas9 and gene silencing, have appeared as potent tools for combating chilling stress. These methods enable the specific alteration of genes associated with the cold stress response, including those regulating

hormone control, oxidative stress, membrane integrity, and cell wall modification—all essential factors in PCI. By allowing the elimination or overexpression of specific genes associated with cold stress, including *CBFs*, antioxidant enzymes, and hormone pathways, CRISPR/Cas9 enables accurate and effective gene editing. Modifying genes involved in ROS generation, lipid peroxidation, and cold-induced gene regulation has created fruits with increased resistance to chilling damage. Furthermore, the expression of genes involved in membrane breakdown, ethylene production, and fruit softening is suppressed at low temperatures by gene silencing methods, such as RNA interference (RNAi). To address a significant issue facing the global fruit industry, these biotechnological techniques have the potential to produce genetically modified fruit varieties with improved postharvest quality, extended shelf life, and enhanced chilling tolerance.

5.3.1. CRISPR/Cas9 Gene Editing for PCI Mitigation in Fruits

CRISPR/Cas genome editing has revolutionized our ability to modify genes involved in cold stress responses in fruits precisely. The functions of particular genes linked to chilling tolerance, particularly those involved in cold acclimation and stress signaling, have been successfully studied using this approach. The *CBF1* is one such gene that is essential for controlling reactions to cold stress. A recent study by [120] utilized CRISPR/Cas9 to generate *slcbf1* mutants in tomato, a fruit that is susceptible to chilling. According to the findings, the *slcbf1* mutants exhibited more severe chilling injury symptoms than wild-type plants, including increased EL, enhanced H_2O_2 content, and significantly higher amounts of MDA.

Furthermore, the mutants exhibited elevated antioxidant enzyme activity, typically associated with oxidative stress, and significantly decreased proline and protein levels. The levels of several plant hormones, including zeatin riboside, MeJA, ABA, and indole acetic acid, were similarly altered by *SlCBF1* deletion, indicating the role of hormonal interactions in the chilling stress response. The lower chilling tolerance in the *slcbf1* mutants was further demonstrated by the suppression of *CBF* genes, which are essential for activating chilling stress-induced pathways [120].

The study found that *SlCBF1* mutagenesis driven by CRISPR–Cas9 markedly reduced tomato chilling tolerance, underscoring the critical function of *SlCBF1* in modulating responses to cold stress. These results highlight the potential of CRISPR/Cas9 technology to modify cold-responsive pathways in fruits for improved postharvest performance and offer important new insights into the genetic basis of PCI. The intricacy of *CBF*-mediated cold acclimation, which is controlled by environmental factors including the circadian clock and photoperiod, was highlighted by the study, which also noted that overexpression of *SlCBF1* in tomatoes did not result in greater chilling tolerance [151]. Nevertheless, the study also stated that overexpression of *SlCBF1* in tomatoes did not increase chilling tolerance, highlighting the intricacy of *CBF*-mediated cold acclimation, which is controlled by environmental factors such as photoperiod and the circadian clock. Future studies examining *SlCBF1's* upstream and downstream effectors and how it interacts with plant hormones may help improve chilling tolerance through CRISPR-based treatments.

5.3.2. Gene Silencing

Gene silencing has emerged as a powerful molecular approach for mitigating PCI in fruits by selectively downregulating genes involved in cold sensitivity. This technique utilizes mechanisms such as short tandem target mimic (STTM) and RNA interference (RNAi), which can efficiently inhibit specific transcription factors or miRNAs that negatively impact chilling tolerance. Fruit resilience after harvest can be significantly increased by inhibiting regulatory genes that control oxidative stress, hormone signaling, and cold-

responsive pathways. Gene silencing has been used extensively to inhibit genes related to cold sensitivity, thereby reducing the accumulation of ROS, lipid peroxidation, and membrane degradation—all of which are major contributors to PCI.

A study by [121] investigated how Sly-miR171e influences the tomato fruit's ability to withstand cold temperatures after harvest. The results showed that while knockdown of Sly-miR171e (miR171e-STTM) decreased PCI index, decreased H_2O_2 accumulation, and preserved higher fruit firmness during cold storage, overexpression of Sly-miR171e (miR171e-OE) significantly increased chilling symptoms at both the mature red (MR) and mature green (MG) stages. According to a gene expression study, silencing *miR171e* increased GA3 concentration. It upregulated essential genes linked to chilling tolerance, such as *GRAS24*, *CBF1*, *GA2ox1*, and *COR*, while downregulating genes associated with adverse effects on chilling resistance, including *GA20ox1* and *GA3ox1*.

Another study concentrated on the transcription factor *SlHY5*, a *bZIP* protein implicated in tomato light- and cold-stress signaling pathways. By reducing oxidative damage, lipid peroxidation, and membrane stability under chilling conditions, overexpression of *SlHY5* in tomatoes markedly increased cold tolerance [152]. According to a molecular study, *SlHY5* overexpression increased the expression of genes associated with anthocyanin biosynthesis (*CHS*, *CHI*, and *F3H*) and the antioxidant defense system, such as SOD and CAT. To further improve chilling resistance, *SlHY5* also altered the expression of genes triggered by cold, including *PR1*, *CYSb*, *LEA*, *Osmotin*, and *ICE1*. According to these results, *SlHY5* controls several pathways sensitive to cold stress, making it a viable target for genetic engineering to increase tomato fruit's resistance to cold and lower PCI [152].

Although many treatments show promise at the laboratory scale, their commercial scalability, safety, and residue implications remain insufficiently evaluated. Future research should compare treatments under industry-relevant conditions and establish decision-support guidelines for treatment selection based on fruit type and storage duration.

5.4. Practical Decision Making: Guidelines for Choosing Appropriate PCI Mitigation

Selecting an appropriate PCI mitigation approach depends on fruit sensitivity, intended storage duration, market destination, and regulatory limitations. To support practical decision-making, Table 3 provides a structured framework linking PCI susceptibility categories with the most suitable physical, chemical, biological, and combined interventions. The table integrates mechanistic rationale, ideal application scenarios, implementation feasibility, and safety considerations, enabling stakeholders to match treatment strategies with operational requirements.

Table 3. Decision-support framework for selecting PCI mitigation strategies across fruit categories.

PCI Susceptibility Category	Recommended Mitigation Strategy	Underlying Mechanistic Rationale	Ideal Application Scenario	Implementation Considerations	Reference
High PCI sensitivity	Physical	Stabilizes membrane phase transitions; reduces sudden ROS bursts	Long-distance export; fruits requiring prolonged cold storage	Low cost; no chemical residues; requires temperature-controlled facilities	[112,121,153–155]

Table 3. *Cont.*

PCI Susceptibility Category	Recommended Mitigation Strategy	Underlying Mechanistic Rationale	Ideal Application Scenario	Implementation Considera-tions	Reference
	Chemical	Enhances antioxidant capacity; stabilizes membrane lipids	When rapid deployment is needed; domestic markets	Generally low cost; regulatory approval required for some compounds	[3,156–159]
	Edible coatings	Reduces moisture loss; modulates internal atmosphere; delays chilling-triggered senescence	Retail markets; fruits needing appearance retention	GRAS; consumer-friendly; moderate material cost	[19,160,161]
Moderate PCI sensitivity	MAP	Limits oxygen stress; slows metabolic rate and ROS formation	Export shipments; extended storage duration	Requires packaging infrastructure; low per-unit cost	[162,163]
	CA storage	Reduces chilling-related lipid oxidation and respiration	Industrial-scale storage	High infrastructure cost; suitable for large-scale operations	
	GRAS chemical dips	Enhances cell wall stability; lowers membrane leakage	Domestic transport; medium storage periods	Relatively low cost; safe; minimal regulatory issues	[164,165]
Low to moderate PCI sensitivity	Physical cooling + humidity control	Minimizes desiccation-related membrane stress	Short supply chains; local markets	Very low cost; easy adoption	[166]
	Biological coatings/films (aloe/chitosan categories)	Maintains firmness; delays oxidative stress progression	Markets requiring clean-label or organic preference	Consumer-preferred; biodegradable; moderate cost	[167,168]
Fruits requiring long-term export storage	Combination strategies (Physical + Chemical)	Multi-target control on ROS, membranes & metabolism	When a single treatment is insufficient; quality must be export-grade	Requires integration of treatments; moderate cost	[169]
Fruits with regulatory restrictions on chemicals	Physical treatments + Coatings	Avoids restricted chemical inputs	Export to strict regulatory markets	Fully compliant; safe; scalable	[170]

Abbreviations: CA: Controlled atmosphere; GRAS: Generally recognized as safe; MAP: Modified Atmosphere Packaging; PCI: Postharvest chilling injury; ROS: Reactive oxygen species.

Fruits with high PCI sensitivity generally benefit from rapid-response interventions, such as physical conditioning or antioxidant-based chemical treatments, which stabilize membranes and suppress ROS accumulation. For moderate-sensitivity fruits, atmosphere-based technologies (MAP/CA) and generally recognized as safe (GRAS) chemical dips offer effective metabolic suppression with minimal regulatory constraints. Low-sensitivity fruits, particularly those marketed locally, respond well to low-cost options like optimized cooling and humidity management, or biodegradable coatings favored in clean-label supply chains.

For long-duration export markets, combined strategies offer synergistic control over membrane damage, oxidative stress, and senescence. Meanwhile, commodities facing regulatory restrictions on chemical use are better suited to physical approaches paired with edible coatings that ensure compliance without compromising quality.

This framework offers a practical, evidence-based tool that growers, exporters, and storage managers can utilize to select PCI management strategies that strike a balance between efficacy, cost, safety, and market-specific needs.

6. Concluding Remarks and Perspective

Complex disturbances in membrane integrity, redox balance, metabolic coordination, and gene control lead to PCI, a multifactorial physiological condition. The integration of these pathways remains largely unknown, despite significant progress in understanding the biochemical and molecular responses to chilling stress, particularly those involving ROS metabolism, hormone signaling, and cold-responsive gene expression. The relationship between transcriptional regulators and hormone networks, as well as the timing of their activation during cold storage, is still poorly understood. Furthermore, the need for genotype-specific mechanistic research is highlighted by the variation in chilling sensitivity among fruit species and cultivars. A significant gap is the limited identification of universal molecular markers that can reliably predict CI susceptibility across different genotypes, which restricts the development of targeted improvement strategies.

To understand the dynamic regulatory networks driving PCI, future research should focus on high-resolution temporal and geographic investigations using integrated omics platforms. To create genotypes that can withstand cold stress, it will be essential to functionally validate important candidate genes and signaling elements using CRISPR/Cas and RNA interference technologies. Particular emphasis should be placed on key molecular targets such as lipid-modifying enzymes (LOX, PLD, FAD), calcium-signaling components (CAM, CBL–CIPK modules), ROS-scavenging genes (SOD, CAT, APX), and hormone-regulated transcription factors (*CBF*, *NAC*, *MYB*, *ERF*), which hold strong potential for breeding or gene-editing interventions. Additionally, to bridge lab-to-field applicability, translational studies that correlate genetic markers with phenotypic features under actual storage circumstances are crucial. However, most current studies remain laboratory-restricted, and there is a lack of scalable, industry-validated treatment protocols that can consistently reduce CI during large-volume storage and long-distance transport.

Future work should also prioritize evaluating and optimizing promising commercial treatments fortified with antioxidants, modified-atmosphere packaging, and precision cold-chain management to determine their dose–response, cost feasibility, and cross-cultivar efficacy. By facilitating precise postharvest handling, the integration of AI-assisted phenotyping, real-time monitoring, and decision-support systems has the potential to transform PCI management completely. When combined, a multidisciplinary approach that includes physiology, molecular biology, and cognitive technologies has the potential to revolutionize fruit sector PCI prevention measures.

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Abbreviations

The following abbreviations are used in the manuscript.

ACC 1	Aminocyclopropane-1-Carboxylate
ACO 1	Aminocyclopropane-1-Carboxylate Oxidase
ACS 1	Aminocyclopropane-1-Carboxylate Synthase
ACPs	Acyl Carrier Proteins
AOX	Alternative Oxidase
APX	Ascorbate Peroxidase
AsA-GSH	Ascorbate-Glutathione Cycle
ATP	Adenosine Triphosphate
b*	Yellowness
bZIP	Basic Leucine Zipper Protein
BL	Brassinolide
C4H	Cinnamate 4-Hydroxylase
CA	Controlled Atmosphere
Ca	Calcium
Ca^{2+}	Calcium Ion
Ca-ATPase	Calcium-Transporting ATPase
$CaCl_2$	Calcium Chloride
CAT	Catalase
CBF	C-repeat Binding Factor
CHS	Chalcone Synthase
CK	Cytokinin
CmAPX	Cucumis melo Ascorbate Peroxidase
CmGR	Cucumis melo Glutathione Reductase
CmPOD	Cucumis melo Peroxidase
COR	Cold-Responsive
CNR	Colorless Nonripening
CRISPR	Clustered Regularly Interspaced Short Palindromic Repeats
CST	Cold Shock Treatment
DGDG	Digalactosyldiacylglycerol
DGAT	Diacylglycerol Acyltransferase
DGK	Diacylglycerol Kinase
DHAR	Dehydroascorbate Reductase
DNA	Deoxyribonucleic Acid
DPPH	2,2-Diphenyl-1-Picrylhydrazyl
DREB	Dehydration-Responsive Element-Binding Protein
EBR	24-Epibrassinolide
EGase	Endo-1,4-Glucanase

EL	Electrolyte Leakage
EIN	Ethylene-Insensitive Protein
ERF	Ethylene Response Factor
ETR	Ethylene Receptor
FADs	Fatty Acid Desaturases
GABA	γ-Aminobutyric Acid
GA	Gibberellic Acid
GGGT	Galactolipid Galactosyltransferase
GR	Glutathione Reductase
GRAS	Generally recognized as safe
HSP	Heat Shock Proteins
HWFC	Hot Water Forced Convection
HWD	Hot Water Dipping
HWT	Hot Water Treatment
ICE	Inducer of CBF Expression
ICES	Inducers of CBF Expression and Signaling
IP_3	Inositol Trisphosphate
IW	Intermittent Warming
JA	Jasmonic Acid
L*	Lightness
LDPE	Low-Density Polyethylene Packing
LOX	Lipoxygenase
LTC	Low-Temperature Conditioning
LTP	Lipid Transfer Protein
LTPs	Lipid Transfer Proteins
MAP	Modified Atmospheric Packaging
MDA	Malondialdehyde
MDHAR	Monodehydroascorbate Reductase
MG	Mature Green
MGDG	Monogalactosyldiacylglycerol
MR	Mature Red
MT	Melatonin
MYB	Myeloblastosis Transcription Factor
NAC	NAC Transcription Factor
NADPH	Nicotinamide Adenine Dinucleotide Phosphate
NO	Nitric Oxide
NOR	Nonripening
OAT	Ornithine Aminotransferase
P5CS	Δ^1-Pyrroline-5-Carboxylate Synthase
P5CR	Pyrroline-5-Carboxylate Reductase
PA	Phosphatidic Acid
PAL	Phenylalanine Ammonia-Lyase
PCI	Postharvest Chilling Injury
PDH3	Pyruvate Dehydrogenase 3
PG	Polygalacturonase
POD	Peroxidase
PME	Pectin Methylesterase
PSY1	Phytoene Synthase 1
PTMs	Post-Translational Modifications
Put	Putrescine
RNA	Ribonucleic Acid
RNAi	RNA Interference
RNA-Seq	RNA Sequencing
RH	Relative Humidity

RIN	Ripening Inhibitor
ROS	Reactive Oxygen Species
SA	Salicylic Acid
SDH	Succinate Dehydrogenase
SFAs	Saturated Fatty Acids
SOD	Superoxide Dismutase
Spd	Spermidine
STTM	Short Tandem Target Mimic
TA	Titratable Acidity
TCA	Tricarboxylic Acid
TFC	Total Flavonoid Content
TPC	Total Phenolic Content
TSS	Total Soluble Solids
UFAs	Unsaturated Fatty Acids
UV	Ultraviolet Treatment
UV-C	Ultraviolet-C Radiation
VOCs	Volatile Organic Compounds
WRKY	WRKY Transcription Factor
XF	Xtend® Film

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