Review

The membrane lipid metabolism in horticultural products suffering chilling injury

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Abstract

Horticultural commodities suffer chilling injury following exposure to extremely low temperatures, which results in visible symptoms and considerable quality loss. Therefore, it is of significance to understand the mechanism of this physiological disorder and to develop effective strategies to control it. Chilling stress causes alteration in structure and function of the plasma membrane, which is assumed to be the primary event in response to cold stress. During this process, the membrane lipid metabolism plays a pivotal role in membrane fluidity and stability. In this review, we summarized the possible roles of membrane lipid metabolism in the development of chilling injury, having the potential for developing effective strategies to alleviate chilling injury in horticultural products under refrigerated storage in practice.

Key words: cell membrane; chilling injury; fruits and vegetables; lipid metabolism.

Introduction

Fruits and vegetables comprise considerable amounts of nutritional compounds such as carbohydrates, minerals, vitamins, dietary fibre, antioxidants, and other health-promoting phytonutrients, and hence they constitute important components in the human diet (Bellavia et al., 2013). Fresh fruits and vegetables have a very short postharvest life and are highly perishable, and as a result, severe postharvest loss occurs if they are handled improperly. Refrigeration is an effective storage approach commonly used to extend the postharvest life and to reduce postharvest deterioration of fruits and vegetables during transportation to distant markets, which ensures the availability of good-quality products to consumers for an extended period. However, horticultural products of tropical or subtropical origin are susceptible to chilling injury (CI) when exposed to chilling temperatures (Wang, 1994). CI is a physiological disorder, the symptoms of which involve browning, pitting, or abnormal ripening, thereby affecting negatively the horticultural products’ commercial quality and storage life (Chen et al., 2008). For example, when exposed to temperatures less than 13°C, banana fruits are subjected to CI, displaying pitting on peel surface, disappearance of aroma, and abnormal ripening (Guo et al., 2018). In the case of papaya, improper low temperatures can lead to water-soaked flesh and scalding skin (Zou et al., 2014). Cucumber is highly susceptible to CI, mainly developing into dehydration and large sunken areas on the surface (Wang et al., 2018a). Therefore, chilling damage has a negative effect
on the quality of horticultural products and thus minimizing their commercial profits.

It is generally accepted that damage to the membrane is the primary event in CI. The cell membrane acts as a barrier to protect the cell from injury and perturbation, providing a constant environment for intracellular bioprocesses. Once the membranes disintegrate under cold stress, multiple metabolic processes are affected, which then causes ion leakage, insufficient energy, and overproduction of reactive oxygen species (ROS), thereby resulting in membrane disruption and cell death (Patel et al., 2016). For example, ultrastructure analysis indicated that membrane integrity and function were destroyed in chilled blueberries, leading to cell death and finally pitting surface (Wang et al., 2019). The composition of the membrane lipids manifests themselves in different forms in their physical state at low temperature, and high levels of unsaturated fatty acids are beneficial to sustaining normal function of the membrane (Mendoza, 2014).

In this review, we present the recent findings on the physical changes in membrane lipids that lead to further damage in fruits and vegetables under CI, as well as their metabolic response to CI at the molecular level.

**CI Initiates Membrane Lipid Change**

A well-known theory was proposed by Lyons (1973), who explained the membrane lipid phase change in response to CI. When subjected to low temperature (not freezing), cellular membrane in chilling-sensitive plants undergoes a rapid physical change from a flexible liquid-crystalline to a solid gel structure. Because of the altered physical state, the membrane fails to keep a constant milieu for biochemical processes, which leads to cellular metabolism disorders and appearance of CI. Early studies have reported that the lipid changes in mitochondrial membranes isolated from chilling-injured fruits, but none of the mitochondrial membranes showed phase transitions from chilling-resistant plants (Raison et al., 1971). Also, mitochondrial membranes from different varieties of apple were approved to experience lipid phase transitions at 30°C to 10°C (McGlasson and Raison, 1973). However, later studies reported that less than 5% lipid changes to gel-solid state in most plants (Raison and Orr, 1986). Moreover, Martin pointed out that bulk phase transition was unlikely to happen in the biological membrane (Martin, 1986). Since there is a lack of direct observation of the phase change, the lipid phase change may not be convincing. Later, development of phase transition has been proposed, i.e. lateral phase separations (Platt-Aloia and Thomson, 1987). Avocado fruits stored in cold temperatures display particle-depleted regions in chilling-injured fruits, but no change occurs when they are kept at 20°C. However, particle-depleted regions disappeared when these injured fruits (but not damaged by low temperature) were transferred to room temperature. This strongly indicates that the membrane undergoes a series of changes owing to temperature-dependent physical properties of lipid, and that chilling-induced changes in the membrane can be reversed before irreversible destruction of the membrane occurs. Perhaps novel techniques are required for assessing phase transitions in biological membranes of plants (such as fluorescence polarization of 1,6-diphenyl-1,3,5-hexatriene, Fourier-transform infrared spectroscopy, scanning electron microscopy, and transmission electron microscopy) to gain more insights into the membrane changed by temperatures (Los and Murata, 2004; Chong and Deng, 2012; Kong et al., 2019).

Whether the temperature causes the membrane to undergo a phase change or phase separation is still unclear, and any significant observations of the temperature-induced membrane physical state are yet shorted. Nonetheless, numerous studies implicated that the plasma membrane is the primary site of CI (Lyons, 1973; Platt-Aloia and Thomson, 1987; Parkin et al., 1989; Marangoni et al., 1996).

**Membrane Lipid Composition Determines its Structure and Stability**

The membrane consists of lipids (mainly phospholipids) and proteins (Larson et al., 1990). The composition of the membrane, especially the type of lipids, plays a determining effect on its physical characteristics and stability. Meanwhile, the proportion of unsaturated fatty acids is closely correlated with the membrane fluidity, as well as cold tolerance. Many studies have found that compared with the cold-sensitive plants, the percentage of unsaturated fatty acids in cold-resistant plants is higher. For example, ‘ Qingzhong’ fruits (a cold-resistant loquat cultivar) have a higher content of unsaturated fatty acids than those in ‘ Fuyang’ fruit (a cold-sensitive loquat cultivar) (Pareek et al., 2014). Intermittent warming attenuated the CI of peppers largely because of efficiently increasing the unsaturated fatty acids’ content (Liu et al., 2015a). This might be attributed to the easier solidification of saturated fatty acids than of the unsaturated fatty acids at low temperature, which can enhance membrane fluidity at a critical temperature. It seems possible that the more the contents of unsaturated fatty acids the membrane contains, the more flexible the membrane is exposed to cold stress. More recently, reduction of the levels of phosphatic acid (PA) and diacylglycerol (DAG) may contribute to the enhanced CI tolerance of the mutant plants (Chen et al., 2015; Tan et al., 2018). Likewise, monogalactosyldiacylglycerol (MGDG) and phosphatidylcholine were sharply decreased during the CI development in pepper (Kong et al., 2018). Together, these results suggest the high sensitivity of lipids in response to cold temperature. In addition to lipids, other membrane compositions such as membrane proteins are also thought to make an effect on its physical inflection (Yoshida et al., 1986). Overall, alterations of plasma membrane composition play a decisive role in its flexibility and function and hence chilling tolerance.

**Mechanism of CI in Terms of the Changes in Membrane Lipid**

Membrane fluidity can influence the activity of membrane-bound proteins (including ion channels, receptor-related protein kinases, sensor proteins, transfer proteins) and thus affect various metabolic processes and signal transduction.

**Ion Leakage**

An increase in ion leakage has been found in many fruits and vegetables suffering from CI (Antunes and Sfakiotakis, 2008; Zhao et al., 2009; Ruan et al., 2015). As the membrane lipids become a gel structure due to the chilling temperatures, a contraction is expected to appear in the membrane and this could cause cracks or channels, leading to ion leakage and elevated permeability (Lyons, 1973). This immediate effect on increased ion leakage may lead to a change in cytoskeleton or a perturbation of the transport and an altered environment, which is responsible for swelling and ultrastructural disorganization of chloroplasts and mitochondria (Palta, 1990). On the contrary, increasing the unsaturated fatty acids’ level can impede electrolyte leakage. For example, exogenous treatment of polyamines on grapefruits can slow down the rate of electrolyte leakage, presumably due to maintaining the membrane integrity via accumulation of unsaturated fatty acids (Champa et al., 2014).
Energy Imbalance

It has been shown that the ATP level in the cells of chilling-injured fruits was lower (Duan et al., 2004; Pan et al., 2017), which gives rise to deficiency in energy. Mitochondria and chloroplasts are related to the production of energy that maintains the metabolism of a living cell. Changes in lipid composition and membrane can affect the functions of mitochondria and chloroplasts during cold stress. It is suggested that chilling temperature can suppress mitochondrial respiration, along with the altered activity of membrane-bound ATPase system, which, in turn, causes a sharp decline in ATP production (Rurek et al., 2015). Similarly, the rate of photosynthesis is depressed by low temperature as a result of rigidified thylakoid membranes and inactive enzymes (Liu et al., 2018). If these two energy-producing pathways are impaired because of the chilling-injured membrane, they are unable to satisfy the need for other metabolic processes. Owing to metabolic disorders, ROS increases markedly, which is considered to be the most harming destroyer of the cell. In this regard, sufficient energy supply may maintain normal bioprocesses (Aghdam et al., 2018). Increasing ATP by different postharvest practices such as low-temperature conditioning (Wang et al., 2018a), H2S (Li et al., 2016a), and OA (Li et al., 2016b) was shown to protect the integrity of membranes, and hence effectively reduce CI and extend the shelf life of fruits and vegetables.

Oxidative Stress

The gel-solid membrane easily breaks down the ROS balance. ROS are the by-products of normal cell metabolism, such as singlet oxygen, hydrogen peroxide, superoxide anions, and hydroxyl radicals, which can be scavenged by the antioxidant system to maintain cellular stability. However, low-temperature storage may uncouple the respiratory chain and trigger the production of ROS in large quantities (Valenzuela et al., 2017). Additionally, the antioxidant system is unable to cope with increasing content of ROS due to its decreased activity by cold stress. Increasing amounts of ROS began to attack membrane lipids, DNA, and proteins, leading to destroyed membrane system, accumulation of toxic metabolites, metabolic disorders, and eventually cell death. In order to mitigate the effect of ROS on plant cells, many chemical treatments such as polyamines (PUT, SPE, and SPD), H2S, and γ-aminobutyrate (GABA) have been applied on kiwifruit (Yang et al., 2016), pears (Li et al., 2019), grape (Maul et al., 2011), and zucchini (Palma et al., 2015). These chemical applications on harvested products alleviate CI of chilling-susceptible horticultural products, via enhancing the antioxidant system to eliminate excess ROS and lessen membrane lipids’ peroxidation, therefore prolong the shelf life of products.

Gel-solid state of membrane lipids at chilling temperatures could negatively affect membrane-bound enzyme activity by altering protein conformation and structure (Wolfe, 1978). Protein dysfunction or reduced enzyme activity has a severe impact on plant cells, resulting in ion leakage, lack of energy, ROS explosion, further damaging membrane normal structure, metabolism, and signal transduction pathways. All of these disorders and damage could cause irreversible deterioration of plant cells, resulting in CI and related visible physiological symptoms.

Molecular Basis of Membrane Lipid Responsive to CI

Considering the important role of the membrane and its lipid composition in cold stress, researchers endeavoured to reveal the molecular mechanisms underlying lipid metabolism responding to CI. When plants are subjected to CI, the saturated lipid components in the membrane are significantly reduced, and the unsaturated lipid components are remarkably accumulated (Kuiper, 1970; Welti et al., 2002). Mutants of the fatty acid desaturases (FADs) (fad2, fad5, and fad6) in Arabidopsis accumulate abundant saturated lipid components, resulting in impairment of plants’ tolerance to low temperatures (Okuley et al., 1994). In addition to FADs, acyl-lipid desaturase 2 in Arabidopsis can also reduce the saturation of cell membrane lipids under low-temperature stress, thereby enhancing

Figure 1. Model of lipid molecules regulating chilling injury response in a plant cell. CI, chilling injury; MGDG, monogalactosyldiacylglycerol; DGDG, digalactosyldiacylglycerol; ΔAG, diacylglycerol; TAG, triacylglycerol; FA, phosphatidic acid; PC, phosphatidylcholine; PE, phosphatidylethanolamine; PS, phosphatidylserine; PG, phosphatidylycerol; PIP2, phosphatidylinositol 4,5-biphosphate; DGK, diacylglycerol kinase; PLC, phospholipase C; DGAT1, acyl-CoA:diacylglycerol acyltransferase; GGGT, galactolipid galactosyltransferase; IP(3), inositol triphosphate; Pi, phosphatidylinositol; DGDG, digalactosyldiacylglycerol; ROS, reactive oxygen species.
plants’ tolerance to low-temperature stress (Chen and Thelen, 2013). Both Arabidopsis phospholipases, PLD\textsubscript{1} and PLD\textsubscript{8}, are involved in the transformation of lipid components in cell membranes under low-temperature stress by hydrolyzing phospholipid molecules to lysophospholipid and phosphatidic acid. In addition, PA can also be generated under the action of diacylglycerol kinase (DGK) (Figure 1). First, polyphosphoinositide is hydrolyzed to DAG under the action of phospholipase C (PLC), and then DAG is phosphorylated by DGK to produce PA (Arisz et al., 2013). PLC hydrolyzes phosphatidylinositol 4,5-bisphosphate (PIP2) to produce inositol triphosphate (IP3) and DAG. As a second messenger, IP3 rapidly increases Ca\textsuperscript{2+} levels in plant cells and further activates downstream low-temperature signalling (Li et al., 2004). Galactolipid galactosyltransferase (GGGT) encoded by the SENSITIVE TO FREEZING 2 (SFR2) gene transfers galactosyl groups from the monogalactosyldiglyceride to another molecule MGDG to form digalactosyldiglycerides digalactosyldiacylglycerol and DAG under low temperature (van Bessouw and Winternans, 1978). Furthermore, using MGDG as an acyl donor, GGGT generates oligogalactosyl diesters, including trigalactosyl diglycerides and tetragalactosyl diglycerides, whose polar head is large and cannot physically attract other lipid molecules to form a hexagonal II (H\textsubscript{II}) structure in the physical space, thereby maintaining the integrity of the cell membrane structure and exhibiting resistance to cold stress (Figure 1).

Similarly, membrane lipid metabolism and abundant lipid metabolic genes were activated to respond to the cold stress in horticultural products. For example, in chilling-tolerant grapefruit, two fatty acid desaturase transcripts were specifically up-regulated, as well as the expression of ceramide glucosyltransferase involved in glycosphingolipid biosynthesis, which take a positive effect on membrane fluidity and cold tolerance. In contrast, transcripts of the lipid and sterol metabolism were found to be down-regulated in chilling-sensitive grapefruit, thus causing CI (Maul et al., 2008). Studies regarding LTPs, PLDs, FADs, DGAT, DGKs, and ACPs also showed a link with chilling stress (Li et al., 2004; Liu et al., 2015b; Wang et al., 2017; Tan et al., 2018; Wang et al., 2018b; Zhang et al., 2019; Kong et al., 2019). In pepper, CaSAD2 (which is responsible for converting C16:0 to C16:1) was significantly repressed by cold temperature, while CaFAD2/47 showed, at first, increasing and then decreasing tendencies in cold stress (Ge et al., 2019).

Moreover, the regulation of transcriptional factors (TFs) provokes a clear variation in lipid metabolism. Large numbers of TFs were identified and induced by cold treatment in plants and fruits (Wang et al., 2018b), including CBF, MYB, ERF, bHLH, and bZIP. In rice, OsPLD\textsubscript{c1} was regulated by OsDEBR1A, leading to decreased CI and enhanced cold tolerance (Huo et al., 2016). More recently in banana fruit, an MYB TF MaMYB4 interacts with MaHDA2 to modulate the expression of omega-3 fatty acid desaturase encoded by fatty acid biosynthesis (Song et al., 2019). It was found that CaPLD\textsubscript{c4} was positively induced by CaNAC1 in bell pepper, which is responsible for membrane lipid degradation and CI (Kong et al., 2020). These findings indicate that the transcriptional regulation of lipid metabolism has an indispensable role in chilling tolerance/injury, but these transcriptional regulators are largely unknown and need further investigation.

**Conclusions**

We summarize the influence of membrane lipids on inducing CI at biochemical and molecular levels. There is still a lack of comprehensive studies and understandings on Chilling stress sensing and transduction. It is essential to reveal the induction of CI by discriminating the actual initial event induced by low temperature (primary ‘cause’) from following disorders caused by initial event (secondary ‘effect’) (Luengwilai et al., 2012), which would be supportive to clarify the nature of CI.

According to the present studies, it is reasonable to consider membrane change as the cause of CI. Various biochemical changes (including ion leakage, oxidative stress, and energy shortage upon chilling) are accompanied by the membrane change, as explained earlier. Moreover, a common fundamental role of postharvest methods in reducing CI is to avoid membrane from injury, preserving an intact membrane and hence providing pleasant surroundings for cellular metabolism.

It is worth noting that CI can be reversed until the irreversible damages occur. Therefore, chilling-injured fruits and vegetables recovering to normal conditions may be possible, suggesting that cold storage may be feasible. The threshold of reversibility depends on the degree of membrane lipid peroxidation (Parkin et al., 1989). So, measuring the threshold is extremely important to identify the cold storage time, which can be further extended by using advanced postharvest treatments.

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