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REVIEW

Loss of photosynthesis signals a metabolic reprogramming to sustain sugar homeostasis during senescence of green leaves: Role of cell wall hydrolases

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Abstract

Leaf senescence is always associated with decline in photosynthesis, consequently a loss of cellular sugar. On the other hand, execution of senescence program needs energy and leaves, therefore, tend to collect sugars from other sources to sustain energy homeostasis. This sugar reprogramming induced by loss of sugar involves operation of a complex catabolic network. The exact molecular mechanism of induction and regulation of the network, however, is not fully resolved but the current literature available suggests sugar starvation as a signal for induction of several senescence-associated genes including the genes coding for the enzymes for degradation of cellular constituents and their conversion to respiratory sugars. The late expression of genes coding for the cell wall hydrolases and enhancement in the activity of these enzymes late during senescence are indicative of the cell wall polysaccharides as the last source of sugars to sustain energy homeostasis for execution of the senescence program.

Additional key words: cell wall hydrolases; photosynthesis; senescence; sugar homeostasis.

Introduction

Senescence in plants did not draw serious attention for quite a long time because it was believed to be a deteriorative process without much physiological significance or any benefit to plant life. This is the primary reason of historical negligence of the subject. The process, in the recent past, however, is recognized as a terminal part of plant development that plays a crucial role in nutrient recycling and therefore, significantly determines plant growth and productivity (Gregersen *et al.* 2014, Havé *et al.* 2017, Sarwat 2017). Although the nature of senescence induction and regulation is not fully understood, it is known to be regulated by several intrinsic and extrinsic factors (Quirino *et al*. 2000, Biswal *et al*. 2003, Lim *et al.* 2007, Fischer 2012, Sabater and Martín 2013, Kim *et al*. 2016). The process involves association of several genes (Gepstein *et al.* 2003, Liu *et al.* 2010, Li *et al.* 2013). Some of these genes have been identified, cloned, and characterized (Biswal and Biswal 1999, Buchanan-Wollaston *et al.* 2005, Breeze *et al.* 2011, Guo and Gan 2012). The exact initiation point of the process during plant development remains unidentified because of the lack of reliable senescence markers. Therefore, the initiation points and timelines of its program are not properly defined. This developmental process is now explained with tools of molecular biology, but the precise molecular markers for examination of its induction are yet to be identified.

Loss of photosynthesis has been extensively used as a parameter to determine senescence initiation and progress. Very often, leaf yellowing has been considered as a major syndrome of senescence and the loss of chlorophyll during yellowing is always accompanied by ultrastructural changes of thylakoid and decline in photosynthetic efficiency (Biswal *et al.* 2003, Lichtenthaler 2013, Biswal

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Abbreviations: HXK1 – hexokinase 1; NPQ – nonphotochemical quenching; *PAGs* – photosynthesis-associated genes; *SAGs* – senescence-associated genes; SnRK1 – sucrose non-fermenting-1-related protein kinase 1.
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and Pandey 2016). Loss of photosynthesis, consequently loss of production of sugar in green leaves, either during dark-induced or natural senescence has been demonstrated to induce expression of several genes associated with senescence (Fujiki *et al.* 2001, 2005; Chrost *et al.* 2004, Lee *et al.* 2004, 2007; Buchanan-Wollaston *et al.* 2005, Avila-Ospina *et al.* 2014). In recent years, sugars are recognized as signaling molecules that regulate growth and development including senescence in plants in complex regulatory networks (Rolland *et al.* 2006, Wingler *et al*. 2006, Li and Sheen 2016). But the loss of sugars as a signal to initiate senescence process is a difficult proposition because the question whether loss of photosynthesis is a cause or consequence of senescence remains, so far, unaddressed. Second, the exact nature of sugar regulation of the process remains controversial. The findings from different laboratories provide rather a confused and contradictory picture (van Doorn 2008, Biswal *et al.* 2012, Biswal and Pandey 2016). Sugar starvation has been reported to induce leaf senescence (Fujiki *et al.* 2001, 2005; Chrost *et al.* 2004, Lee *et al.* 2004, 2007) that contradicts the opposite view of induction of the process by accumulation of excess sugars (Pourtau *et al.* 2004, 2006; Wingler and Roitsch 2008). The controversy arises primarily due to lack of knowledge on sugar-sensing mechanisms. What are the sugar-sensing signals? How do the signals sense excess or limited sugars that transmit the signals to senescence induction? In our reviews published in the recent past, we have tried to rationalize and integrate the data to explain the controversy (Biswal *et al.* 2012, Biswal and Pandey 2016). It appears, the signal cascades generated by both excess and limited sugars participate in induction of the process. In nature, the rate of photosynthesis in green leaves gradually increases to a maximum when the leaves are fully expanded and matured. The peak of the sugar content in mature leaves is a critical phase during leaf development. High concentration of sugars is likely to be sensed by hexokinase 1, a well-studied sugar sensor (Li and Sheen 2016, Aguilera-Alvarado and Sánchez-Nieto 2017). Reports are available on hexokinase 1-mediated downregulation of photosynthesis genes during excess sugar environment through a feedback mechanism (Aguilera-Alvarado and Sánchez-Nieto 2017). Literature on downregulation of photosynthesis-associated genes (*PAG*s) with concomitant upregulation of senescence-associated genes (*SAGs*) is rich (Quirino *et al*. 2000, Biswal *et al*. 2003, Buchanan-Wollaston *et al.* 2005, Biswal and Pandey 2016). These findings suggest photosynthetic modulation of senescence induction and regulation. The loss of photosynthetic production of sugars is sensed by SnRK1, another sugar sensor, well examined by Sheen's group in Harvard Medical School in Boston (Baena-González *et al*. 2007). SnRK1 cascade seems to activate an extensive catabolic network for degradation of cellular constituents including macromolecules and convert them to respiratory sugars in order to execute and complete energy dependant senescence process. This explanation is summarized in a model shown in Fig. 1. The figure summarizes the participation of two major sugar sensors, hexokinase 1 (high-sugar sensor) and SnRK1 (low-sugar sensor) and their sequential action for completion of senescence process.

Execution of senescence program needs energy

Senescence in plants is an extensive process with several sequential events that involve expression of several *SAGs* and synthesis of many enzymes associated with the operation of catabolic network (Biswal *et al.* 2003, Buchanan-Wollaston *et al.* 2005, Lim *et al.* 2007, Schippers 2015). In addition, the transport of nutrients and their relocation in plants needs energy (Hörtensteiner and Feller 2002, Avila-Ospina *et al*. 2014). Energy is also required to build defence systems in senescing leaves, prone to pathogen attack and abiotic stress. In green plants, photosynthetic production of sugars is the major source of energy. But senescence causes decline in photosynthesis, consequently loss in production of sugars. This is likely to result in a change in cellular sugar metabolism in order to maintain energy homeostasis. However, the nature of the metabolic reprogramming that provides sugars from alternative sources to execute energy dependent senescence process is not fully understood (Biswal *et al.* 2003, Buchanan-Wollaston *et al.* 2005, Biswal and Pandey 2016).

Sugar reprogramming during senescence

Senescence-induced loss in photosynthesis signals a metabolic reschedule that is likely to activate a catabolic network responsible for breakdown of starch, lipids, proteins, and other cellular constituents stored in order to provide respiratory sugars (Hörtensteiner and Feller 2002, Biswal *et al.* 2003, Avila-Ospina *et al*. 2014, Biswal and Pandey 2016). Literature on senescence-induced degradation of these cellular constituents, which are finally converted to sugars through several metabolic pathways to compensate for loss in photosynthetic production of sugars, is rich (*see* the book by Biswal *et al.* 2003).

The mechanism of the degradation of cellular macromolecules to sugars for sustaining energy homeostasis and nutrient recycling is not fully understood so far. However, the participation of two cellular organelles, namely chloroplasts and vacuoles, is reported to play a crucial role in the degradation process during senescence (Costa *et al.* 2013, Avila-Ospina *et al.* 2014, Carrión *et al.*

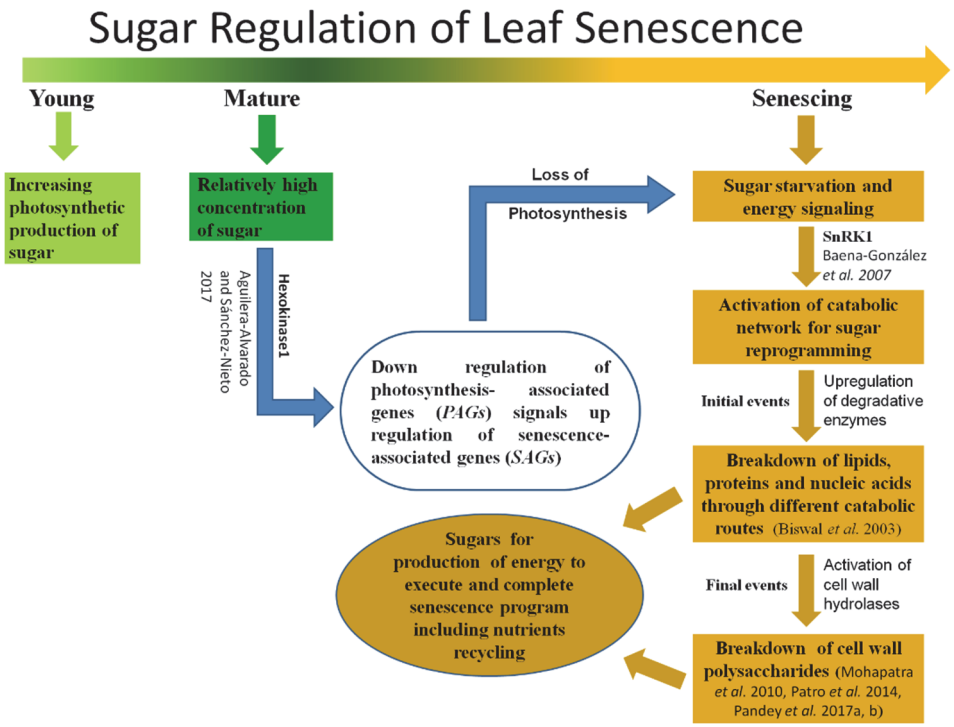

Fig. 1. Senescence-induced sugar reprogramming in green leaves during senescence: The figure shows the changes in the sugar concentration in leaves during development and senescence. It also indicates the possible regulatory mechanisms mediated by sugar sensors for induction of senescence and maintenance of cellular sugar homeostasis. Higher sugar concentration is sensed by the hexokinase 1 (HXK1), which results in down-regulation of photosynthesis-associated genes (*PAGs*). Down-regulation of *PAGs* results in a sugar starvation environment which initiates a senescence signaling cascade and expression of senescence-associated genes (*SAGs*). Another sugar sensor, sucrose nonfermenting-1-related protein kinase 1 (SnRK1), activates a catabolic network that participates in the degradation of starch, lipids, proteins, and nucleic acids to provide sugars for execution and completion of the energy-dependent senescence program, including nutrient recycling. The final event of sugar reprogramming includes the degradation of cell wall polysaccharides to provide sugars to energy-deficient leaves during terminal phase of senescence (modified from Biswal and Pandey 2016).

2014, Xie *et al.* 2015). Chloroplasts are not only the major source of carbohydrates, but also contain most of the cellular proteins. Vacuoles are the organelles that cleave, degrade cellular constituents, and salvage the nutrients for recycling. The degradation pathway, which connects chloroplast and other cellular organelles to vacuoles, is complex. In recent years, autophagy is suggested to participate in the degradation pathway (Ono *et al.* 2013, Ohsumi 2014, Kabbage *et al.* 2017). Autophagosomes, a double membrane bound vesicular structure, carry macromolecules, either cleaved or intact, to vacuoles, where they are degraded to simple molecules for nutrient recycling.

Senescence-induced activity of cell wall hydrolases and sustenance of sugar homeostasis: Last event of senescence program

Cell walls are the richest source of organic carbon in plants (Minic 2008, Moreira *et al.* 2011). The walls are well known as to provide mechanical support to cell inclusions, maintain osmotic balance, and also participate in cell communication and signaling (Wolf *et al.* 2012). In addition, the literature is available on the possibility of contribution of cell wall polysaccharides, the major components of the wall, to maintain sugar homeostasis during plant development including germination and senescence, when the level of photosynthetic activity remains relatively low (Tonini *et al.* 2007, Biswal and Pandey 2016). The cell wall catabolism responsible for breakdown of the polysaccharides to produce sugars for energy starved cells is rather a recent area of senescence research (Biswal *et al.* 2012, Biswal and Pandey 2016). The wall-bound hydrolases degrade polysaccharides of the wall (Brummell *et al.* 2004, Minic 2008, Moreira *et al.* 2011). Excellent reviews are available on the nature and catabolic function of these hydrolases (Minic 2008, Jamar *et al.* 2011, Moreira *et al.* 2011, Franková and Fry 2013). The participation of the polysaccharides to maintain sugar homeostasis during dark-induced or terminal phase of natural senescence is reviewed by Biswal and Pandey (2016). The literature on senescence-induced activation of cell wall hydrolases, however, is not very rich (Chrost *et al.* 2004, Lee *et al.* 2004, 2007; Gunawardena *et al.* 2007,

Mohapatra *et al.* 2010, Moreira *et al.* 2011, Jamar *et al.* 2011, Patro *et al.* 2014, Pandey *et al.* 2017a,b). Some reports are available on upregulation of the genes coding for these enzymes during senescence (Buchanan-Wollaston *et al.* 2005, Fujiki *et al.* 2005, Breeze *et al.* 2011).

Although the mechanism of initiation and activation of the cell wall hydrolases and the expression pattern of genes coding for the hydrolases during senescence are not clearly understood, sugar starvation as a signal for induction/ activation of the hydrolases is suggested [*see* the review by Biswal and Pandey (2016)]. Late expression of genes coding for cell wall hydrolases and activity of the enzymes late during the process may suggest that cell wall catabolism, which involves activation of the wall bound hydrolases, is the last event of senescence program and cell wall polysaccharides as the last source of organic carbon for maintenance of energy/sugar homeostasis when other sources of cellular sugars are exhausted (Biswal *et al.* 2012, Biswal and Pandey 2016). The proposition is supported by the observations of the activation of cell wall hydrolases, breakdown of cell wall polysaccharides, and release of the sugars during dark-induced leaf senescence (Lee *et al.* 2004, 2007).

This review outlines the major findings of our laboratory on the loss of photosynthesis as a possible signal for activation of cell wall hydrolases during leaf senescence of *Arabidopsis* plants.

A distinct loss of oxygen evolution and net photosynthesis has been observed in first rosette leaves of *Arabidopsis* during senescence and the loss is accompanied by a concomitant increase in the activity of cell wall bound β-glucosidase (Mohapatra *et al.* 2010, Patro *et al.* 2014), the enzyme well known as cell wall hydrolase that participates in the breakdown of the wall polysaccharides (Minic 2008). In earlier report, the gene for β-glucosidase has been demonstrated to express late during dark-induced senescence (Fujiki *et al.* 2005). In addition to β-glucosidase, we have demonstrated similar response of β-glucanase and β-galactosidase, the other important cell wall-bound enzymes to photosynthetic loss. The activity of enzymes increases during progress of senescence (Patro 2012, Pandey *et al*. 2017a,b). Importantly, the activity of these enzymes shows a peak at the terminal phase of senescence, when the level of photosynthesis was marginal (Mohapatra *et al.* 2010, Pandey *et al.* 2017a,b). It appears that these three cell wall-bound enzymes participate in coordinated manner (Jamar *et al.* 2011). β-galactosidase is known as a pectin chain breaker and is likely to bring an initial cell wall modifications followed by the activity of β-glucanase that breaks down polysaccharides to oligosaccharides which are finally degraded by β-glucosidase to monosaccharides in order to provide sugars to energy starved cells (Jamar *et al.* 2011). Abiotic stress, such as drought and nitrogen limitation, enhance senescence-induced loss of photosynthesis with further increase in the activity of the cell wall hydrolases. Not unexpected, withdrawal of the stress causes recovery of photosynthesis and suppression in the activity of hydrolases (Patro *et al.* 2014, Pandey *et al.* 2017a). Both senescence and stress studies in our laboratory support the possibility of loss of photosynthesis activity as a signal for activation of cell wall-bound hydrolases to produce respiratory sugars to sustain cellular sugar homeostasis.

Marginal loss in total soluble sugars in the background of massive loss of photosynthesis during progress of senescence and stress response (Pandey *et al*. 2017b) is suggestive of accumulation of sugars from the sources other than sugars produced by photosynthesis. Induction of sugar production from alternative sources, including cell wall polysaccharides, in absence of photosynthesis, is an effective strategy of green plants to meet the challenges of sugar starvation environment. At the initial stage of senescence, catabolic events are likely to degrade cellular constituents, such as starch, lipids, proteins, and nucleic acids (Biswal *et al.* 2003), and finally the cell wall polysaccharides to produce sugars for energy starved cells (Fig. 1). Senescence involves an extensive program and follows a sequence of development finally resulting in plant´s death. Temporal expressions of *SAGs* are reported to carry out a sequence of catabolic events during progress of the process (Biswal *et al.* 2003, Breeze *et al.* 2011, Guo and Gan 2012). In the sequence, the late expression of genes coding for cell wall hydrolases (Fujiki *et al.* 2005) as discussed earlier, supports the hypothesis that the cell wall polysaccharides are the last target for sugars to provide energy for completion of senescence process.

Defence mechanism and sustenance of cell viability for enzyme activity during terminal phase of senescence

One of the fundamental questions addressed currently in our laboratory is how do the leaves and the leaves experiencing abiotic stress sustain viability even during the terminal phase of senescence when the activity of cell wall hydrolases shows a peak with almost complete loss of photosynthesis [*see* the review by Biswal and Pandey (2016)]. Importantly, the senescing leaves experiencing the stress retain viability and potential for recovery on withdrawal of stress. The stress reversal causes recovery of photosynthesis and suppression of enzyme activity. The defence mechanism during senescence and stress response to sustain cell viability for the activity of cell wall hydrolases appears to be complex. Senescing leaves, mature and developing ones experience abiotic and biotic stresses. No serious studies have been made so far to examine the molecular mechanism of cell viability in senescing leaves experiencing abiotic stress, although literature on stress adaptation mechanism in non-senescing

leaves, such as developing and mature ones experiencing stress, is very rich (Biswal *et al.* 2011, Joshi *et al.* 2013). In *Arabidopsis thaliana*, we have demonstrated an increase in the content of anthocyanin and flavonoids both during senescence and in senescing leaves experiencing stress, that may contribute to the viability to keep the cells active (Patro *et al.* 2014). The findings from the laboratory also indicate an enhancement in nonphotochemical quenching (NPQ), considered as an adaptive mechanism in green plants during progress of senescence (Biswal *et al.* 2011, Patro *et al.* 2014). A remarkable stability of the total cellular proteins compared to rapid loss of chlorophylls and electron transport efficiency as demonstrated during senescence (Pandey *et al.* 2017a) is likely to contribute to cell viability and retention of potential for recovery. Free chlorophylls and/or alteration in the topology of the pigments on distorted thylakoids otherwise would have been photodynamic leading to cellular damage.

Conclusion

Loss of photosynthesis resulting in cellular sugar starvation is a big challenge for yellowing leaves to sustain sugar homeostasis for successful execution and completion of energy-dependent senescence programs. Sugar starvation as discussed in the review leads to induction of several catabolic events that break down cellular constituents to provide sugar to energy starved cells. The activation of this catabolism involves a complex sugar-signaling network. In spite of a large volume of literature available in the area, several knowledge gaps exist in our understanding of nature of sugar sensors, perception of signals followed by signal transduction required for a metabolic reprogramming to sustain sugar homeostasis. The unknowns in the area discussed below, need serious attention and clarification in future.

The mechanism of association of photosynthesis with leaf senescence appears to be complex. However, it is certain that photosynthesis plays a key role in modulating senescence in green leaves. The question whether loss of photosynthesis during leaf senescence is a cause or consequence of the process is yet to be fully resolved without any ambiguity. Although the loss of photosynthesis is proposed as a signal for induction of senescence, the precise nature of interface between two processes largely remains unresolved.

Involvement of sugar signaling in regulating the catabolic events associated with senescence is proposed by several laboratories. In the recent past, some of the sugar sensors have been identified and many more are expected to be identified in future. Sugar signaling is known to regulate several aspects of plant growth and development. It is necessary to examine the exact nature of their participation in signaling network that regulates plant senescence in particular.

As discussed, senescence is an energy dependent process. In the background of loss of photosynthesis, it gets respiratory sugars from other sources including cell wall polysaccharides. Although the cell wall has been proposed to be the richest source of organic carbon, the involvement of cell wall hydrolases to breakdown the wall polysaccharides in order to sustain sugar homeostasis is not fully resolved and needs serious attention in future. The reports available in this area are scattered. So far, the precise actions of different hydrolases on the cell wall catabolism remain largely unclear. The timeline of induction of hydrolases and its relationship with gradual loss of photosynthesis during senescence is yet to be addressed.

The temporal induction of these enzymes is likely to provide clues in the understanding of integrated mechanism of their action during senescence. This approach may also reveal the sequence of degradation of wall polysaccharides and their degradation products, finally leading to formation of monosaccharides including the respiratory sugars to sustain energy homeostasis. The future studies may emphasize on the quantification and identification of the polysaccharides that are the targets of the hydrolases during senescence.

The current data available suggest late expression of genes coding for cell wall hydrolases and activity of these enzymes late during senescence, which raise a fundamental question how the potential and viability of cells are retained even at the late phase of the process for the activity of the enzymes.

Both natural senescence and non-senescing leaves experiencing stress are reported for induction and activation of cell wall hydrolases in green leaves. It seems that the activity of the hydrolases may not be senescence specific. Therefore, the specificity of enzyme activity associated with senescence needs further experimentation in future. It is also desirable to examine the mechanism of involvement of sugar signaling in the background of possibility of its crosstalk with phytohormones and reactive oxygen species-related signaling networks associated with senescence regulation.

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