The role of sugars in integrating environmental signals during the regulation of leaf senescence

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Abstract

Although leaf senescence results in a loss of photosynthetic carbon fixation, the senescence-dependent release of nutrients, especially of nitrogen, is important for the growth of young leaves and for reproduction. Environmental regulation of senescence is therefore a vital factor in the carbon and nitrogen economy of plants. Leaf senescence is a highly plastic trait that is affected by a range of different environmental factors including light, nutrient supply, CO2 concentration, and abiotic and biotic stress. In this review, the focus is on the impact of environmental conditions on sugar accumulation and sugar signalling during senescence. By signalling a high availability of carbon relative to nitrogen in the old leaves, sugar accumulation can trigger leaf senescence. Sugar-induced senescence is therefore particularly important under low nitrogen availability and may also play a role in light signalling. Whether or not sugars are involved in regulating the senescence response of plants to elevated CO2 remains unresolved. Senescence can be delayed or accelerated in elevated CO2 and no clear relationship between sugar accumulation and senescence has been found. Senescence can be delayed or accelerated in elevated CO2 and no clear relationship between sugar accumulation and senescence has been found. Plasticity in the response to environmental factors, such as daylength and sugar accumulation, varies between different Arabidopsis accessions. This natural variation can be exploited to analyse the genetic basis of the regulation of senescence and the consequences for growth and fecundity. Different evolutionary strategies, i.e. early senescence combined with a high reproductive effort or late senescence combined with a low reproductive effort, may be an important adaptation of Arabidopsis accessions to their natural habitat.

Key words: Carbon/nitrogen interaction, elevated CO2, leaf senescence, life history trait, light signalling, natural variation, phenotypic plasticity, sugar signalling.

Leaf senescence: a plastic trait

Leaf senescence is characterized by a decline in chlorophyll content and in photosynthetic activity. As senescence ultimately results in the death of a leaf, it could be regarded as a form of programmed cell death (van Doom and Woltering, 2004). However, leaf senescence is not only a degenerative process, but it also plays a vital role in nutrient recycling, especially in the remobilization of nitrogen (Himelblau and Amasino, 2001). During early senescence, recycling of nitrogen from the photosynthetic apparatus requires that cellular membranes remain intact (Hörtensteiner and Feller, 2002) and, in contrast to programmed cell death, cellular compartmentalization is maintained until the final stages (Lee and Chen, 2002).

The onset of leaf senescence has to be tightly regulated and is dependent on the growth environment. Too early senescence would reduce a plant’s overall capacity to assimilate CO2, whereas too late senescence would interfere with nutrient remobilization, thereby compromising photosynthetic activity in the young leaves and reproductive success. An early start of senescence can be expected to be favourable when the availability of photoassimilates is high or when the supply of inorganic nutrients, such as nitrogen, is low. Indeed, leaf senescence can be induced by low nutrient supply (Ono et al., 1996; Thomas and de Villiers, 1996; Crafts-Brandner et al., 1998). Leaf senescence can therefore be regarded as a plastic trait. Plasticity in the timing of senescence may allow acclimation to the growth conditions, thereby maintaining the overall carbon balance of a plant.

Compared with other important life-history traits, such as flowering, the mechanisms that control senescence are still

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not very well understood. This may be because senescence is a gradual process and therefore difficult to quantify. Furthermore, senescence can be induced by a large number of different conditions, and several signalling pathways are involved in the regulation of senescence (Buchanan-Wollaston et al., 2003). As some of the environmental conditions that affect leaf senescence, such as light conditions, CO₂ concentration, nitrogen supply, stress and pathogen infection, also have major effects on leaf sugar contents, it is possible that environmental signals are integrated by sugar signalling.

The role of light in the regulation of leaf senescence

Senescence can be induced by darkening of individual Arabidopsis leaves (Weaver and Amasino, 2001). This treatment probably simulates the effects of shading by other leaves. With respect to the nitrogen economy of a plant, induction of senescence by shading of the bottom leaves can be beneficial as the nitrogen released can be used to increase photosynthetic capacity of the sun-exposed, upper leaves. As a result, whole-plant photosynthetic capacity can be optimized by distributing nitrogen parallel to the light gradient (Schieving and Poorter, 1999). Photoreceptor responses play an important role in this regulation. In sunflower plants grown in dense stands, senescence at the bottom of the canopy is accelerated due to a decreased ratio of red to far-red light (Rousseaux et al., 1996). Illumination with red light increases the lifespan of sunflower leaves, indicating an involvement of phytochromes in this process (Rousseaux et al., 2000). In addition, ectopic overexpression of an oat phytochrome A gene in tobacco suppresses far-red-induced leaf senescence, thereby reducing phenotypic plasticity (Rousseaux et al., 1997). However, Weaver and Amasino (2001) did not find any effect of mutations in phytochrome or cryptochrome genes on senescence induced by darkening of Arabidopsis leaves.

In contrast to darkening of individual leaves, dark incubation of whole Arabidopsis plants delays rather than accelerates leaf senescence (Weaver and Amasino, 2001). Shading (36% of full light intensity) of whole sunflower plants also delayed senescence (Ono et al., 2001). In addition, Noodén et al. (1996) have shown that growth at an increased light intensity accelerates Arabidopsis leaf senescence under long-day conditions. Experiments to investigate the effect of light conditions on photosynthetic development confirm that leaf senescence in Arabidopsis thaliana is delayed under low light (100 μmol m⁻² s⁻¹) compared with moderate light (200 μmol m⁻² s⁻¹) (Fig. 1A, B). The senescence-dependent decline in chlorophyll content and maximum photosynthetic efficiency (F₅₀/F₇₁⁰) was further delayed under short days. Since even the higher light intensity in this experiment was low compared with natural conditions, it is unlikely that the induction of senescence by light was caused by photo-oxidative stress. Instead, illumination-dependent changes in carbohydrate contents may have influenced senescence. Ono et al. (2001) have shown that shading of the young leaves of sunflower and bean plants leads to a decrease in sugars in the unshaded, old leaves and a delay in leaf senescence. These results indicate that senescence may be induced by carbohydrate accumulation and not by starvation, however, this might depend on a combination of environmental factors.

Sugars as regulators of leaf senescence

Comparing Arabidopsis transcriptome data, Buchanan-Wollaston et al. (2005) found significant differences between developmental and dark/starvation-induced leaf senescence. For example, the pathways of nitrogen remobilization differed in developmental and dark-induced senescence. While glutamate dehydrogenase and asparagine synthetase genes were induced in the dark, glutamate decarboxylase and cytosolic glutamine synthetase genes were induced during developmental senescence. Differences between changes in gene expression caused by sugar starvation and senescence were also reported by Contento et al. (2004) and Lee et al. (2004). Experiments on sugar-regulated senescence indicate that leaf senescence can be induced by growing Arabidopsis plants in the presence of 2% glucose in combination with low nitrogen supply (Pourtau et al., 2004; Wingler et al., 2004). Affymetrix GeneChip data confirmed that changes in gene expression during glucose-induced senescence are characteristic of developmental senescence (N Pourtau et al., unpublished results). For example, the senescence-associated gene (SAG) SAG12, whose expression is highly senescence-specific, was induced several hundred-fold by growth on glucose in combination with low nitrogen supply.

SAG12 is expressed late during the senescence process and it has been argued that late SAGs are sugar-repressible, whereas early SAGs are sugar-inducible (Paul and Pellny, 2003). This hypothesis was based on the finding that SAG12 is repressed when sugars are fed to cut leaves (Noh and Amasino, 1999) and the idea that sugar contents would fall during late senescence, as, for example, shown for tobacco (Masclaux et al., 2000). However, glucose and fructose accumulate in Arabidopsis leaves until late senescence (Fig. 1C, D). Similarly, Quirino et al. (2001) and Stessman et al. (2002) found that hexoses accumulate in senescing Arabidopsis leaves. In combination with the induction of SAG12 by glucose in intact Arabidopsis plants, these findings clearly demonstrate that late SAGs are not necessarily repressed by sugars.

The maximum glucose content reached in compost-grown Arabidopsis plants (Fig. 1C) was the same as in plants grown on glucose-supplemented agar medium (about 250 μmol g⁻¹ dry weight; Pourtau et al., 2004), indicating that
the required sugar threshold may be reached in naturally senescing leaves. Sucrose did not however, accumulate during developmental senescence (Fig. 1E). The question remains as to what causes the strong accumulation of hexoses despite the decline in photosynthetic carbon assimilation in senescing leaves. A possible source of hexoses is the breakdown of starch. In addition, Jongebloed et al. (2004) have shown that phloem blockage by callose deposition could lead to an age-dependent sugar accumulation. However, it is not clear how the export of amino acids from the senescing leaves, which is essential for nitrogen mobilization, would be achieved under these conditions.

Recently, first steps have been taken to unravel the signalling pathways that are involved in sugar-regulated senescence. Moore et al. (2003) demonstrated delayed senescence in the Arabidopsis hexokinase-1 mutant, gin2-1, indicating that the sugar sensor hexokinase-1 is involved in sugar signalling during senescence. It has been shown that this mutant does not accumulate hexoses in senescing leaves (N Pourtau et al., unpublished results). Furthermore, the induction of senescence by externally supplied glucose was delayed in this mutant, indicating that hexokinase-1 is involved in sugar metabolism as well as in the response to sugars during senescence. Several interactions have been described between phytohormones and sugar signalling. For example, Arabidopsis mutants in abscisic acid synthesis (aba mutants) or signalling (abi mutants) show decreased sugar sensitivity during seedling development. However, it has been shown that abscisic acid is not required for sugar signalling during senescence (Pourtau et al., 2004), although the transcription factor ABI5 may be involved. Further interactions between cytokinin or ethylene with sugar signalling have been described (Wingler et al., 1998; Moore et al., 2003). Balibrea Lara et al. (2004) have provided a mechanistic explanation for the interactions between cytokinins and sugars in the regulation of senescence: By inducing extracellular invertase,
cytokinin increases sugar utilization, thereby, surprisingly, decreasing glucose accumulation and delaying senescence.

**Interactions of sugars and light in the regulation of leaf senescence**

Since treatment with externally supplied glucose can induce leaf senescence, it was investigated if light conditions affect leaf senescence through their effect on sugar accumulation. Under long days, moderate light led to an early accumulation of hexoses compared with low light conditions (Fig. 1C, D) and the start of hexose accumulation coincided with the decline in chlorophyll (Fig. 1A). This indicates that increased light intensity triggered senescence due to an earlier accumulation of hexoses. Ono et al. (1996) also found that sunflower plants grown at low nitrogen accumulated more sugar and senesced earlier in high than in low light. However, despite delaying leaf senescence, growth under short days led to a slightly earlier accumulation of hexoses compared with growth under long days (Fig. 1). Thus, it is unlikely that photoperiod effects on leaf senescence are mediated by sugar accumulation. However, sugar sensitivity appears to be affected by daylength (Fig. 2A). Plants grown under long days reacted more strongly to externally supplied glucose, as indicated by an earlier decline in maximum photosynthetic efficiency ($F_v/F_m$). Sugar-induced leaf senescence was further accelerated under moderate compared with low light, suggesting that sugars and light act synergistically. Another factor that may affect the impact of sugars under different light conditions is light quality. For example, sugar can potentiate the effects of far-red light and attenuate the effects of white, blue, or red light on gene expression (Thum et al., 2003). Such interactions of sugar signalling with light quality may determine the sugar responsiveness of leaves at the bottom of the canopy that experience a decreased red/far red ratio.

As floral initiation and leaf senescence of *Arabidopsis* accessions are linked (Levey and Wingler, 2005), it is possible that photoperiod controls leaf senescence through its effect on floral initiation. *Arabidopsis* plants grown under short days and low light on Petri dishes did not bolt throughout the course of the experiment (Fig. 2B). Under long days, moderate light accelerated flowering compared with low light and plants bolted earlier in the presence than in the absence of glucose. However, the nature of the relationship between floral initiation and leaf senescence remains unresolved.

**Interactions of sugars and nitrogen in the regulation of leaf senescence**

Leaf senescence can be induced by low nutrient, especially low nitrogen, supply (Ono et al., 1996; Thomas and de Villiers, 1996; Crafts-Brandner et al., 1998). Interestingly, nitrogen deficiency often results in sugar accumulation (Ono et al., 1996; Ono and Watanabe, 1997; Paul and Driscoll, 1997). Furthermore, an external supply of glucose leads to a much stronger accumulation of sugars during senescence in *Arabidopsis* plants grown at low nitrogen supply than in plants grown at high nitrogen supply (Pourtou et al., 2004). It is therefore likely that the role of nitrogen supply in regulating leaf senescence is at least partly due to effects on sugar metabolism. Nitrogen deficiency could, for example, lead to sugar accumulation by decreasing the demand for carbon skeletons for amino acid and protein synthesis.

In addition to these metabolic interactions, interactions between sugar and nitrogen signalling in the regulation of gene expression have been found (Palenchar et al., 2004; Price et al., 2004). With respect to the regulation of leaf senescence, it is interesting that glucose has a stronger effect on the regulation of genes associated with nitrogen metabolism than nitrogen supply (Price et al., 2004). Overall, the regulation of photosynthesis and plant development appears to depend on the carbon/nitrogen ratio instead of carbohydrates alone (Paul and Driscoll, 1997;
Martin et al., 2002). These carbon/nitrogen interactions are likely to play an important role in the regulation of leaf senescence. Sugar and nitrogen contents show distinct changes during leaf senescence, with sugars accumulating while amino acids decline (Masclaux et al., 2000; Diaz et al., 2005). Such metabolic changes affect the expression of genes that are involved in nitrogen remobilization, such as cytosolic glutamine synthetase and glutamate dehydrogenase (Masclaux et al., 2000; Masclaux-Daubresse et al., 2002). Experiments on sugar-induced senescence in Arabidopsis have also shown that sugars induce cytosolic glutamine synthetase genes, which are probably involved in the synthesis of glutamine that is exported from senescing leaves (N Pourtau et al., unpublished results).

Is the effect of elevated CO₂ on senescence mediated by sugars?

Growth in elevated CO₂ typically results in an accumulation of starch and of soluble sugars, while nitrogen and Rubisco contents are reduced (Ainsworth and Long, 2005). Overall, plants grown in elevated CO₂ are nitrogen and not carbon limited. Accelerated senescence under these conditions would increase nitrogen availability, for example, by releasing nitrogen from Rubisco in the old leaves. Thereby, early senescence could form part of the photosynthetic acclimation response of plants to elevated CO₂. The possible role of sugar signalling in photosynthetic acclimation to elevated CO₂ has been discussed by Stitt and Krapp (1999). Based on our results on the regulation of leaf senescence, senescence is expected to be accelerated if hexoses accumulate in elevated CO₂. The effect of elevated CO₂ on hexose accumulation is, however, variable. While hexoses, for example, accumulated in soybean plants (Rogers et al., 2004), glucose and fructose were not increased in Arabidopsis plants grown in elevated compared with ambient CO₂ (Bae and Sicher, 2004).

Reports on the effect of elevated CO₂ on leaf senescence are also inconsistent. Although accelerated leaf senescence in elevated CO₂ was found in several experiments conducted in growth chambers (Miller et al., 1997; Ludewig and Sonnewald, 2000), open-top or closed-top chambers (Fangmeier et al., 2000; Lawson et al., 2001; Sigurdsson, 2001; Bindi et al., 2002) or under free-air CO₂-enrichment (Nie et al., 1995; Miglietta et al., 1998; Bindi et al., 2002), other studies revealed either no effect (Herrick and Thomas, 2003) or even delayed senescence in elevated CO₂ (Li et al., 2000; Tricker et al., 2004; S Long, personal communication). Unfortunately, most of the studies do not provide information on hexose contents. Ludewig and Sonnewald (2000) report that elevated CO₂ accelerated the decline in photosynthetic gene expression and chlorophyll content in tobacco even when sugar contents were not increased compared with ambient CO₂. It was concluded that early senescence is caused by accelerated leaf ontog-

ey, but not by sugar-dependent changes in gene expression. However, the effect of sugars does not depend on the overall content, but the sugar concentrations in specific cellular compartments and on sugar sensitivity of the cell. In this context, it is important to note that old leaves are generally more sensitive to sugars than young, expanding leaves (von Schaewen et al., 1999; Wingler et al., 1998; Araya et al., 2005). Effects of elevated CO₂ on leaf ontogeny could therefore determine whether sugars induce senescence.

In addition to leaf age, interactions between CO₂ enrichment and nitrogen supply may determine the effect on senescence. As acclimation to elevated CO₂ depends on nitrogen availability (Makino and Mae, 1999; Geiger et al., 1999; Stitt and Krapp, 1999), it is not surprising that CO₂ enrichment induced senescence to a larger extent at low than at high nitrogen supply in black cottonwood (Sigurdsson, 2001). Furthermore, changes in phytohormone concentrations that are dependent on nitrogen supply and CO₂ concentration could influence sugar sensitivity. For example, Yong et al. (2000) report that growth in elevated CO₂ increases cytokinin delivery to the leaves, especially at low nitrogen supply. As described above, increased cytokinin concentrations could delay senescence by inducing extra-cellular invertase and thereby increasing sugar utilization (Balibrea Lara et al., 2004). Interactions with other environmental factors could also explain why senescence was delayed in some experiments and accelerated in others. For example, Cavender-Bares et al. (2000) have shown that elevated CO₂ extended leaf lifespan in shade-grown red oak plants while it accelerated leaf senescence in sun-grown plants. This is in agreement with the finding that sugar sensitivity increases with light intensity (Fig. 2).

Moreover, growth in elevated CO₂ affects other leaf parameters, mainly due to a reduction in stomatal conductance (Long et al., 2004) and stomatal density (Lake et al., 2002). As a consequence, transpiration decreases, which can result in improved water relations and in an increased leaf temperature (Long et al., 2004). Lower stomatal conductance probably also reduces the uptake of ozone (Morgan et al., 2003), thereby protecting plants against ozone-induced senescence (Mulholland et al., 1998; Ommen et al., 1999). It is therefore possible that delayed senescence in elevated CO₂ was mainly found in experiments where ozone concentrations were high. A further factor that could potentially complicate the effect of CO₂ treatments on senescence is contamination of CO₂ sources with ethylene (Gifford, 2004), which itself could induce senescence or interact with sugar signals.

To summarize, the role of sugars in regulating senescence in elevated CO₂ is still unresolved. Too many confounding factors make it difficult to assess to what extent the changes in senescence that have been observed can be linked to sugars. To unravel the interactions between sugar-regulated senescence and growth in elevated CO₂, it
would be necessary to determine hexose concentrations and hexose sensitivity while also excluding effects on water relations and the uptake of pollutants.

**Natural variation in the regulation of leaf senescence**

In addition to species-dependent differences in the regulation of senescence, as indicated by differences in the response to elevated CO2, significant differences can be found between ecotypes (or accessions) of the same species. Natural variation in *Arabidopsis* can, for example, be exploited to investigate the molecular and genetic mechanisms that underlie evolutionary and ecological processes (Pigliucci, 1998; Tonsor *et al.*, 2005) and, in particular, phenotypic plasticity and associated costs (Callahan *et al.*, 2005).

The suitability of *Arabidopsis* as a model for leaf senescence in other species has been discussed by Buchanan-Wollaston *et al.* (2003). *Arabidopsis* leaves are short-lived and senescence is rapidly induced by stress conditions. It has therefore been questioned whether efficient nutrient recycling occurs during *Arabidopsis* leaf senescence. However, Himelblau and Amasino (2001) have shown that nutrients are mobilized out of senescing *Arabidopsis* leaves (e.g. leaf nitrogen and potassium decline by over 80%). The strongest evidence that *Arabidopsis* is a suitable model for leaf senescence in other species is probably the finding that the promoter of the *Arabidopsis* SAG12 gene can be used to delay leaf senescence in tobacco and other plant species through the auto-regulated production of cytokinin (Gan and Amasino, 1995). What makes *Arabidopsis* an ideal model for studies on the regulation of senescence is not only its suitability for molecular and genetic analysis, but also the availability of a large range of *Arabidopsis* accessions and of recombinant inbred line (RIL) populations for quantitative trait locus (QTL) analysis. While natural variation has, for example, revealed the mechanisms that control flowering in *Arabidopsis* (Alonso-Blanco *et al.*, 1998; Johanson *et al.*, 2000), little information was available on natural variation in leaf senescence.

Recently, the effect of photoperiod on senescence in *Arabidopsis* accessions from different geographic origins was studied (Levey and Wingler, 2005). Plasticity in the response to photoperiod varied significantly between different accessions. For example, two late-senescent accessions from Kashmir (Kas-1-1 and Kas-1-2) did not show the typical response to photoperiod in the regulation of senescence or other life history traits. Accessions that flowered late also senesced late and produced more leaves, but fewer fruits. The finding that fecundity was decreased and not increased in plants with more leaves may be surprising as resource availability would be expected to be higher in larger plants. However, Aarssen and Clauss (1992) also found that *Arabidopsis* accessions reaching large maximum plant sizes had low fecundities, suggesting that these accessions had a K-type selection strategy, compared to the r-type strategy found in other *Arabidopsis* accessions. *Arabidopsis* is a stress-tolerant ruderal (Grime *et al.*, 1986). According to Grime’s C-S-R theory (Grime, 1977), it would be expected that ruderal accessions from disturbed habitats have an r-type selection strategy with a short lifespan and large reproductive effort, while accessions from stressed habitats should have a K-type strategy with a long lifespan and a small reproductive effort. Leaf senescence could be key to this evolutionary adaptation as it provides the nutrients, such as nitrogen, required for reproduction. *Arabidopsis* can therefore be used to analyse the importance of senescence regulation for evolutionary adaptations of plants to their habitat.

As sugars are an important factor in the regulation of leaf senescence, it was interesting to find out if *Arabidopsis* accessions varied in sugar sensitivity. Plants were grown under the conditions that resulted in the strongest sugar response in Col-0 (moderate light and long days). All accessions showed an induction of leaf yellowing on medium with glucose (not shown), suggesting that sugar-regulated senescence is a universal response. Data for the senescence-dependent decline in $F_s/F_m$ suggest that two of the accessions, Sha and Cvi were less responsive to glucose than the other accessions (Fig. 3). Recently, RILs from a cross between Bay-0 and Sha were used to determine the interactions between senescence and metabolism in *Arabidopsis* (Diaz *et al.*, 2005). In one of the lines senescence was slightly delayed rather than accelerated in the presence of glucose. Interestingly, this line did not accumulate hexoses to the same extent as other lines during senescence. In addition, the line showed accelerated senescence of the leaves formed first during development, but continued to produce young, dark-green leaves with late senescence. Overall, sugar metabolism and response appeared severely impaired in this line, resulting not only in unusual senescence characteristics, but also in a severe delay in flowering.

More detailed information about the genetic background of the regulation of senescence can be obtained through QTL analysis. Using the Bay-0×Sha RIL population Diaz *et al.* (unpublished results) identified several QTL for leaf yellowing, some of them overlapping with QTL for nitrogen use efficiency (Loudet *et al.*, 2003). QTL experiments on sugar-regulated senescence are currently being conducted. Ultimately, the aim is to identify the genes that are responsible for the regulation of senescence by sugars.

**Conclusion**

Based on the work described in this review, the following model is proposed for the role of sugars in integrating environmental signals during the regulation of leaf senescence (Fig. 4). Nitrogen deficiency and growth in high light can result in sugar accumulation, thereby inducing...
leaf senescence through hexose-dependent signalling. In addition to affecting sugar accumulation, these environmental conditions may increase sugar sensitivity. However, they may also regulate senescence through pathways that are independent of sugar signalling. For example, high light intensities can trigger senescence through photo-oxidative stress. Growth in elevated CO₂ can either accelerate or delay leaf senescence. Delayed senescence may be due to effects of elevated CO₂ on stomatal conductance and thereby ozone uptake. However, the role of sugar accumulation and sensitivity in senescence induced by growth in elevated CO₂ remains unresolved. Photoperiod effects on leaf senescence are probably independent of sugar signalling and more likely to be controlled by pathways that regulate flowering.

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