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Invited Expert Review

Regulation of Leaf Senescence and Crop Genetic Improvement[F]

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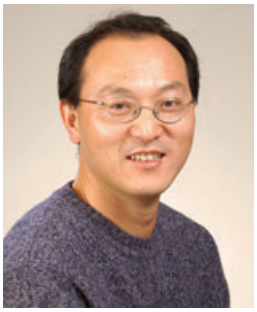

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Abstract

Leaf senescence can impact crop production by either changing photosynthesis duration, or by modifying the nutrient remobilization efficiency and harvest index. The doubling of the grain yield in major cereals in the last 50 years was primarily achieved through the extension of photosynthesis duration and the increase in crop biomass partitioning, two things that are intrinsically coupled with leaf senescence. In this review, we consider the functionality of a leaf as a function of leaf age, and divide a leaf's life into three phases: the functionality increasing phase at the early growth stage, the full functionality phase, and the senescence and functionality decreasing phase. A genetic framework is proposed to describe gene actions at various checkpoints to regulate leaf development and senescence. Four categories of genes contribute to crop production: those which regulate (I) the speed and transition of early leaf growth, (II) photosynthesis rate, (III) the onset and (IV) the progression of leaf senescence. Current advances in isolating and characterizing senescence regulatory genes are discussed in the leaf aging and crop production context. We argue that the breeding of crops with leaf senescence ideotypes should be an essential part of further crop genetic improvement.

Keywords: Leaf Senescence; photosynthesis and photoassimilates; nutrient remobilisation; *senescence-associated genes*; crop ideotypes.

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Introduction

Leaf Senescence is a Genetically Programmed Developmental Process

Aging is defined as the changes associated with the growth and development of an organism, whereas senescence often refers to as the deteriorating effects of aging. These two terms are primarily developed from animal and human health research owing to their obvious intrinsic links with lifespan and longevity, as well as the desire to seek the fountain of youth. Plants display diverse lifespans, ranging from a few weeks to as long as millennia. For instance, short-life plants, including spring, weedy and desert ephemerals, can finish their life cycle in a few weeks, but a bristlecone pine, currently known as the oldest living organism, is estimated to have a lifespan of over 5,000 years, and some clonal plants which propagate through asexual reproduction, have a recorded lifespan of over 10,000 years (Lanner 2002; Thomas 2002). Plants are also very different in their rate of senescence. The monocarpic plants

undergo abrupt senescence following a single reproductive cycle, but for long lifespan trees, senescence is almost negligible (Lanner and Connor 2001). These observations indicate that both animals and plants possess genetic programs controlling their lifespan and senescence, which might be similar at the cellular and molecular genetics levels (Jing et al. 2003).

However, plants differ from animals in a number of fundamental life features and survival strategies, which imbues the terms of "aging" and particularly "senescence" with different meanings in plant research. Plants are particularly "causal" in designing body plans during embryogenesis, and the meristematic stem cells at the root and shoot tips can proliferate and generate almost all structures and organs throughout post-embryonic life. Furthermore, plants differ from animals in modular growth, and individual organs such as leaves, shoots, and roots are modulable and dispensable for the survival of the whole plant. In animals and human beings, the deterioration, malfunction and death of an organ (for example, the liver, stomach or brain) will ultimately lead to the death of an animal or a person, whereas in plants the dismantling of an organ is meant to support the growth and development of new organs and/or of the whole plant. Another feature distinguishing plants from animals is that the death of a cell and an organ is uncoupled from the death of the whole organism. In this sense, senescence in plants means a genetically well-programmed process that leads to the death of the tissue and organ, rather than just a "wear and tear" of aging.

Leaf senescence is the final phase of leaf development. The phenomenon of leaf senescence can be appreciated by the spectacular color changes in deciduous trees and in the maturation of cereal crops in late summer and autumn, which can occur at a global scale to transform the appearance of the earth from space. In annual plants, leaf senescence is tightly associated with the death of the whole plant (monocarpic senescence), whereas in perennials, leaf senescence occurs multiple times throughout the plant lifespan. Although the importance of leaf senescence has long been recognized, a systematic dissection of genes and regulatory networks controlling leaf senescence is still lacking in the context of crop genetic improvement.

In this review, we first describe the processes and events during leaf senescence, and analyze its relevance to crop production. Much of the manuscript is devoted to the current understanding of leaf senescence, with special attention paid to the key regulatory genes which can shift the onset and progression of leaf senescence. We propose to view the functionality of a leaf as a function of leaf age, and discuss how to explore the genes controlling the onset and progression of leaf senescence for crop genetic improvement.

The Senescence Syndrome: Leaf Senescence Requires Massive Changes in the Expression of Senescence-Associated Genes

As the final phase of the development of a leaf, senescence activates a self-destructive program to degenerate the cellular structures, and allows a leaf to make its final contribution to the plant by remobilizing the nutrients accumulated in the senescing leaf. During leaf senescence, the sum of morphological, physiological and molecular changes is generally referred to as the senescence syndrome, which includes several hallmarks such as visible color changes, a reduction in photosynthesis, the dismantling of chloroplasts, the degradation of RNA, proteins and DNA, and the translocation of macro-/micromolecules to other parts of the plant, leading to the death of the senescing leaf (Bleecker and Patterson 1997; Ougham et al. 2008). Hence, nutrient remobilization imparts an evolutionary meaning to the senescence program in plants (Bleecker 1998; Jing et al. 2003), and differs from animal senescence, which is generally considered to occur in the absence of natural selection and evolutionary driving forces (Kenyon 2010; Barzilai et al. 2012).

The senescence syndrome is best explained from the nutrient remobilization point of view (Bleecker 1998; Masclaux–Daubresse et al. 2008). Indeed, it has been documented that massive biomolecules and micronutrients are transported out of senescing leaves (Himelblau and Amasino 2001; Masclaux–Daubresse et al. 2010). This notion is particularly supported by gene expression profiling during leaf senescence. Being associated with the development of the senescence syndrome, so-called senescence-associated genes (*SAGs*) exhibit up-regulated expression. The identification of genes that exhibit differential expression profiles during leaf senescence has been a central focus in senescence studies. So far, *SAG* expression profiling has been carried out in a number of plants, but genome-wide data are only available in *Arabidopsis* and are therefore used for analysis here (Buchanan-Wollaston et al. 2003; Gepstein et al. 2003; Buchanan-Wollaston et al. 2005; Breeze et al. 2011). It is generally acknowledged that the expression of *SAGs* can serve as reliable molecular markers for monitoring the onset and progression of leaf senescence and the effects of induction conditions. *SAG* profiling has provided a fairly consistent picture of the senescence syndrome at the gene expression level. Here, we briefly summarize what we have learned from these studies.

One of the distinct features of leaf senescence is the clear metabolic shift from primary anabolism to catabolism, and the number of catabolic genes highly expressed in senescing leaves is almost two-fold that of anabolic genes (Guo et al. 2004). Chloroplasts are the major cellular organelles in a photosynthetic cell, and up to 80% of total leaf nitrogen is reserved in

the chloroplasts, while Rubisco (D-ribulose-1,5-bisphosphate carboxylase/oxygenase) represents up to 50% soluble proteins. Hence, efficiently achieving chloroplast breakdown and Rubisco and chlorophyll degradation is crucial for nutrient recycling. Recent findings show that protein degradation is initiated within the chloroplasts, and subsequently proceeds with the vacuolar proteinases (Feller et al. 2008; Kato and Sakamoto 2010). Autophagy machinery targets Rubisco to the vacuole via the formation of Rubisco-containing bodies, and plays an important role in chloroplast breakdown (Ishida et al. 2008; Wada et al. 2009). Interestingly, this function of autophagy does not cause chloroplast lysis, and relies on leaf carbon status (Stettler et al. 2009; Izumi et al. 2010). Chlorophyll degradation is another central theme during senescence, and is executed through the Pheophorbide A Oxygenase (PAO) pathway (Hortensteiner and Krautler 2011; Hortensteiner 2012). These protein degradation genes are at the top list of the expression levels of *SAGs* and of the cellular behaviors of some proteins characterized. For instance, RD21, a senescence-associated protease, remains in the vacuole as inactive aggregates and becomes active during senescence through the cleavage of its C-terminal granulin domain (Yamada et al. 2001). In addition, the formation of senescence-associated vacuoles (SAVs), which contain proteolytic enzymes such as *SAG12,* has been observed (Otegui et al. 2005; Martinez et al. 2008).

Many *SAG*s encoding transporters such as ABC transporters, amino acid permease, and cation exchanges, also exhibit senescence-enhanced expression, which is indicative of active remobilization. In total, genes that are involved in macromolecule degradation and nutrient recycling have been shown to occupy about 9% of the total genes expressed in senescing leaves, and this percentage increases to about 20–30% if only those with enhanced expression are analyzed (Gepstein et al. 2003; Guo et al. 2004; Buchanan-Wollaston et al. 2005). Limited studies have been carried out on the behavior of transporters during leaf senescence (Van der Graaff et al. 2006), although monosaccharide and vacuolar organic cation transporters have been reported (Quirino et al. 2001; Frelet-Barrand et al. 2008).

During leaf senescence, the dysfunction of the cellular redox machinery results in the overproduction of reactive oxygen species (ROS), which are the trigger of the high expression of stress and defense-related genes. Hence, a senescing leaf cell is in a stressful environment. One possible reason for such high expression levels of these genes is to detoxify the oxidated protein intermediates and guarantee the functionality of mitochondria and of the nucleus (Guo and Crawford 2005; Sakuraba et al. 2012b). Alternatively, the stress-related hormones such as ethylene, jasmonate and abscisic acid increase their endogenous levels during senescence, which in turn induces the expression of stress-related genes (Navabpour et al. 2003). Although it has long been observed that the expression of defense-related genes in senescing leaves is up-regulated (which suggests an intrinsic link between the pathogen attack-triggered and senescence-associated cell death), it is yet not clear why it is necessary to do so, since enhanced expression of defense proteins may divert resources and energy away from nutrient recycling. Considering that nutrient acquisition is a driving force for a particular pathogen to attack plants, the shelter provided by the enhanced expression of defense genes may be a necessary cost to pay for nutrient remobilization. Furthermore, one theory concerning the origin of senescence states that senescence evolved as a strategy against pathogen invasion. In this sense, the enhanced expression of defense genes may merely be a consequence of plant defense responses parallel to senescence.

Enhanced transcription is observed for *SAG*s involved in signaling and transcriptional regulation. Among the components identified are receptor-like kinase (Lee et al. 2011; Xu et al. 2011), MAP kinase cascade (Zhou et al. 2009), and those in protein-protein interactions (Vainonen et al. 2012). Many transcriptional factors exhibit a senescence-associated pattern, including those containing NAC (Guo and Gan 2006; Uauy et al. 2006; Kim et al. 2009; Yang et al. 2011; Lee et al. 2012a; Zhang and Gan 2012), WRKY (Miao and Zentgraf 2007; Zentgraf et al. 2010) and MYB (Warner et al. 2007; Guo and Gan 2011; Zhang et al. 2011) domains, indicating the importance of transcriptional regulation for senescence. Since many of these regulatory genes belong to large gene families, they have been shown to be involved in diverse growth and developmental processes and to have complex interactions amongst them, and it will be necessary to identify their specific downstream effectors which are responsible for regulated cellular and biochemical events during senescence.

Clearly, internal and external factors could initiate leaf senescence through different signaling pathways (Lim et al. 2007). However, a recent bioinformatic comparison of the gene expression profiles of 27 different senescence-induction treatments with that of developmental leaf senescence indicates that common pathways exist for the execution of leaf senescence following its onset (Guo and Gan 2012). This suggests that a common execution machinery is preserved to complete the senescence process, regardless of the initial induction signals in *Arabidopsis*. It would be interesting to examine whether this core set of execution *SAGs* is conserved amongst different species.

Taken together, genome-wide transcriptional profiling has provided a holistic picture on the molecular events during leaf senescence, further supporting the long-standing notion that leaf senescence is a complex processes involving many catabolic pathways. Unfortunately, high resolution transcriptome data for other species are still lacking, particularly for major crops. With the development of the Next Generation Sequencing Technology and bioinformatic tools, it is feasible to

carry out large-scale profiling for crops with complex genomes. In the coming year, we expect a rapid expansion of *SAG* profiling data for crops, which will enable cross-species comparison and identification of divergence and convergence of *SAGs*.

Leaf Senescence is Tightly Linked to Crop Yield, Fruit Ripening and Biomass Production

Crop yield is achieved through grain filling in cereals, which depends on two carbon and nitrogen sources: the photoassimilates formed and transported directly to the grain from a photosynthetic active leaf, and those remobilized from the vegetative tissues (Yang and Zhang 2006). In small-grained cereals such as hexaploid wheat (*Triticum aestivum*) and rice (*Oryza sativa*), pre-anthesis photoassimilates contribute 10%–40% of the final grain weight (Gebbing and Schnyder 1999; Yang and Zhang 2006). Leaf senescence can influence the final grain weight negatively and positively. Often, an early occurrence of leaf senescence caused by intrinsic genetic factors or by adverse environmental changes results in a drop in photosynthesis and precocious cell death, and curtails the supply of the pre-anthesis photoassimilates (Gregersen et al. 2008). In reality, premature leaf senescence and the subsequent total loss of crops induced by harsh conditions such as drought have frequently been in news headlines (for example, the severe drought in the Corn Belt in the United States this year, http://www.washingtonpost.com/business/economy/). Recent modeling work using nine years of satellite measurements of wheat growth in northern India to monitor the rates of wheat senescence following exposure to temperatures greater than 34 °C, shows a statistically significant acceleration of senescence from extreme heat (Lobell et al. 2012). Hence, breeding crops with enhanced tolerance to heat-induced leaf senescence is a prerequisite for future crop success. On the other hand, as demonstrated by the breeding efforts of the past 50 years, delaying leaf senescence and extending the duration of active photosynthesis could substantially increase the instant photoassimilate source, and hence increase the grain yield (Richards 2000; Long et al. 2006). Many stay-green varieties displaying delayed leaf senescence have been shown to possess multiple beneficial effects, including promoting more root growth, providing extra carbon, and shortening the intervals between anthesis and silking, as reviewed by Davies et al. (2011). Thus, the timing of the onset of leaf senescence is important for crop yield.

The rate or the progression of leaf senescence is also important for crop yield via controlling the remobilization of post-anthesis photoassimilates (Thomas and Howarth 2000; Himelblau and Amasino 2001). This is best exemplified in nitrogen use efficiency, which involves nitrogen uptake, assimilation, translocation and remobilization (Hirel et al. 2001; Hortensteiner and Feller 2002; Hirel et al. 2007). As the availability of nitrogen almost always limits plant growth, the efficient use of nitrogen is essential for the plant life-cycle. Crop grain yield relies on pre-anthesis nitrogen uptake and post-anthesis remobilization during seed maturation (Masclaux-Daubresse et al. 2008). In barley, wheat, and rice, up to 90% of the nitrogen is remobilized from the vegetative plant parts to the grain, while in maize 35%–55% of the grain nitrogen is derived from soil uptake after anthesis (Hirel et al. 2007; Gregersen et al. 2008). A complex relationship exists between the onset of leaf senescence and nitrogen use efficiency (Chardon et al. 2010; Masclaux-Daubresse et al. 2010; Masclaux-Daubresse and Chardon 2011). The stay-green trait can increase crop yield; however, unfavourably prolonged delayed leaf senescence results in a low grain filling rate, a low nitrogen use efficiency and a low grain protein content, creating a dilemma for the breeding of the stay-green trait (Mi et al. 2002; Gong et al. 2005). The effect of delaying leaf senescence on grain yield and grain protein concentration relies on nitrogen availability during the post-anthesis period (Bogard et al. 2011). Hence, post-anthesis leaf senescence should be under tight genetic and management control.

Fruit ripening and postharvest storage are important aspects of plant senescence, and regulating the timing of ripening and extending the shelf-life of postharvest vegetables could be achieved through the control of key regulatory genes (Causier et al. 2002; Klee 2010). Recent concerns regarding breeding for dedicated biofuel crops have stimulated research on biomass production. In maize, it has been shown that delaying leaf senescence is a key component for increasing the overall biomass in new hybrids (Richards 2000), and biomass production for biofuels can be maximized in wood plants if senescence is synchronized with seasonal growth (Jackson 2009). Sorghum and many other grasses are considered future biofuel crops with high potential (Byrt et al. 2011; Calvino and Messing 2012), and leaf senescence management is crucial for achieving high biomass (Robson et al. 2012). In sorghum, the stay-green trait is tightly coupled with post-flowering drought tolerance to achieve high biomass and high stem sugars (Harris et al. 2007).

Societal demands require that the roles of crop species be expanded to applications beyond their conventional use as food sources; for example, developing plants for biofuel sources, and turning plants into a factory to produce pharmaceutical ingredients and vaccines (Lossl and Waheed 2011). Hence, the understanding of leaf senescence is a necessary step toward manipulating senescence in the plant life cycle to help secure the world's food and energy supply in a changing global climate and parallel population growth. The following section is devoted to the discussion of a genetic framework to show the gene action in the regulation of leaf senescence.

The Onset and Progression of Leaf Senescence: A Genetic Framework to Identify Genes of Interest for Crop Improvement

As hinted at in the previous section, the contribution of a leaf to the life-cycle of a plant is two-fold. First of all, as the primary physical platform, a photosynthetic active leaf provides essential photoassimilates to support the growth and development of other parts of the plant, including the reproductive structures. Secondly, the accumulated nutrients in a dying leaf are remobilized via the senescence program. Clearly, maximizing the contribution of a leaf is achievable either by increasing the net photosynthetic photoassimilates or by improving the efficiency of the nutrient remobilization machinery. Leaf age or leaf developmental stages are the primary determinants of these two functions. As a developmental program, leaf senescence is controlled by leaf age and can influence both functions. Hence, it is essential to dissect the developmental controls of leaf senescence to identify genes of interest for crop genetic improvement.

We propose to view the functionality of a leaf as a function of leaf age. Here, the functionality of a leaf is defined as its ability to perform photosynthesis and provide net photoassimilates to the reproductive structures, new growth points, and storage organs. As outlined in **Figure 1A**, the functionality of a leaf changes with leaf aging, and the life-cycle of a leaf is divided into three different phases: the functionality increasing phase at the early growth stage, the full functionality phase, and the senescence and functionality decreasing phase. In the first phase, starting with the formation of leaf primordia, rapid cell division and expansion occur to reach full maturation. During this initial growth stage, photosynthetic activities are gradually acquired, and most of the photoassimilates (either formed through the photosynthetic activity of the leaf or imported from other leaves) are used to build the leaf body itself, and hence the functionality of the leaf, i.e., the net photoassimilates, gradually increases. The second phase is the stage when a leaf is photosynthetically fully functional and steadily provides the net photoassimilates to reproduction growth. In the later stage of the third phase, the functionality of a leaf decreases following the onset of leaf senescence.

The implication of this diagram (**Figure 1A**) is that there are a number of checkpoints to maximize the contribution of a leaf to reproduction growth, or crop production. At least four categories of genes can possibly exist in a plant genome and work in a developmentally-controlled manner to contribute to crop production: those which regulate (I) the speed of early leaf growth and the transition to the full functionality phase, (II) the photosynthesis rate, (III) the onset and (IV) the progression of leaf senescence (**Figure 1B**).

Category I genes regulate various aspects of the early growth of a leaf and the transition to reach full expansion and functionality. These genes probably control traits such as leaf size, shape and number, and have been analyzed through mutational analysis (Fleming 2006; Walter et al. 2009). Improvements in photosynthesis rate have only played a minor role in the increase of the yield potential of major cereals in the past 50 years (Richards 2000; Long et al. 2006). However, it has been argued that further increases in crop yield potential will largely rely on identifying Category II genes to improve the rate of photosynthesis (Zhu et al. 2010; Parry et al. 2011), particularly in those environments with foreseeable globally-elevated CO_2 levels associated with climate change. However, the discussion of these two categories of genes is beyond the scope of this review. Here, we focus primarily on analyzing the regulatory genes of leaf senescence.

Category III and IV genes control the onset and rate of leaf senescence. Leaf senescence is eventually initiated and progresses in an age-dependent manner in plants grown under optimal conditions, with sufficient nutrition, and no pathogen attacks and abiotic stresses (Gan and Amasino 1997; Quirino et al. 2000). This is a clear indication that leaf senescence is a developmentally-programmed process. In *Arabidopsis,* individual leaves live for an identical life-span (Hensel and Bleecker 1992; Nooden and Penney 2001), which allows for the use of such a model plant in hunting for genes of age factors. A genetic approach has been actively used to dissect the genes regulating the onset of leaf senescence, and to address how temporally the action of these genes is integrated into the developmental program. To this end, the promoting effect of the phytohormone ethylene on leaf senescence was explored to analyze the developmental control of leaf senescence (Jing et al. 2002). A senescence window concept was proposed to illustrate that ethylene can only promote leaf senescence in a specific age window, and that multiple genetic loci (e.g. the *OLD* (*ONSET OF LEAF DEATH*) genes) tightly control the effects of ethylene in leaf senescence (**Figure 2**; Jing et al. 2003; Jing et al. 2005). Clearly, the senescence window can be applied to explain the action of multiple genes and pathways in leaf senescence. In a plant genome, numerous loci are involved in leaf senescence, and there are genes which act as negative regulators, such as the *OLD* genes, as well as genes which act as positive regulators, as demonstrated through the isolation of *oresara* (*ore*) mutants by Nam and co-workers (Oh et al. 1997).

So far, little is known about the Category IV genes controlling the progression of leaf senescence. The fact that some ephemerals finish their life-cycle in a couple of weeks, monocarpic senescence completes in one season, and long-lived woody plants hardly show any signs of senescence, indicates that plants differ in their rate of leaf senescence. The efficiency of nitrogen remobilization is perhaps a good parameter to study this topic (Ono et al. 2001; Masclaux-Daubresse et al.

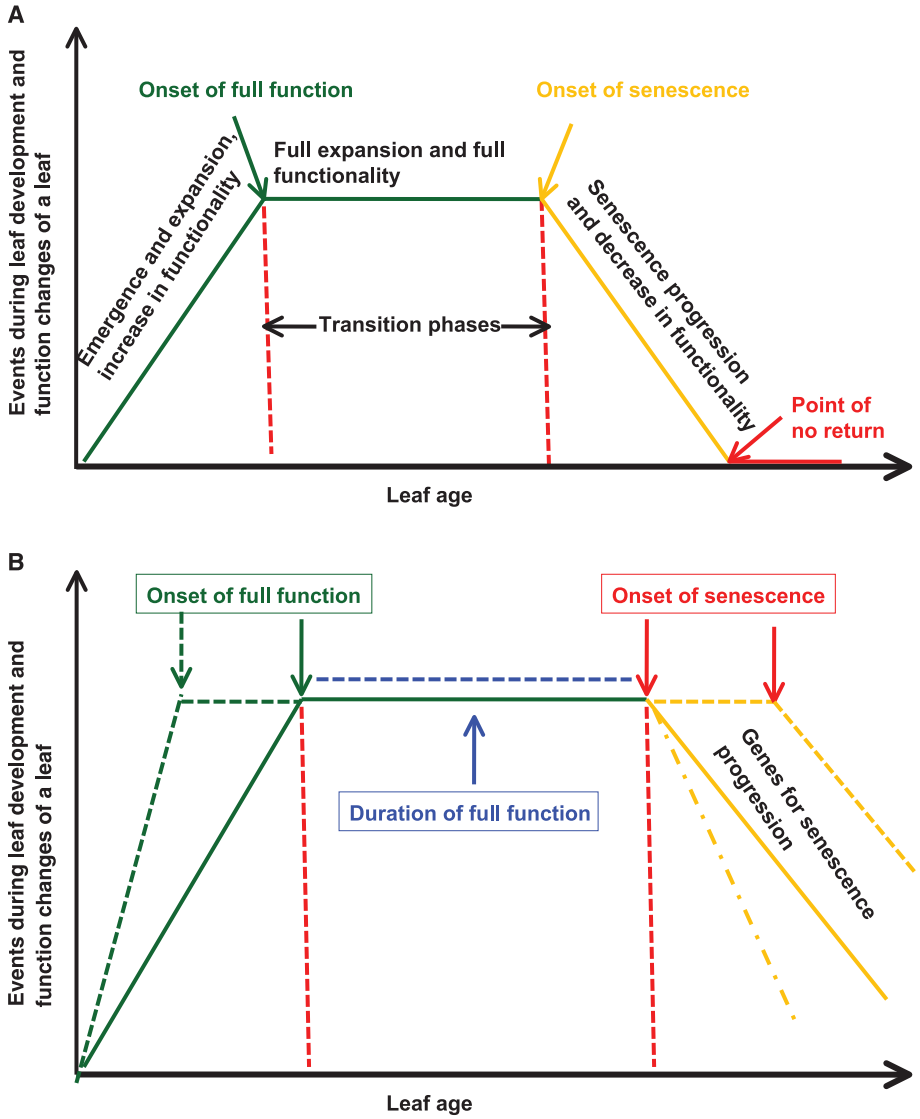

Figure 1. A diagram illustrating the relationships between leaf age and the changes in leaf functionality and associated events and gene actions during the life of a leaf.

(A) Events during leaf development and the function changes of a leaf during development. The functionality of a leaf is defined as its ability to perform photosynthesis and provide net photoassimilates to reproductive growth. See text for details.
(B) Genes controlling phase transition and leaf senescence. Four categories of genes are important targets for crop genetic improvement. See text for details.

2010). Autophagy has long been recognized as the key machinery for nutrient recycling under both ambient and limited nutrition and during senescence (Bassham 2007; Guiboileau et al. 2012). Agronomic practices such as imposing post-anthesis soil-drying can induce leaf senescence and enhance nitrogen remobilization, and it would be interesting to dissect the signaling components involved (Yang and Zhang 2006).

Taken together, we believe that many distinctive events occur throughout the life of a leaf, and that it is necessary to dissect the functionalities of a leaf and the associated regulatory processes in a developmental and aging context. The proposed genetic framework, albeit rudimentary, might help dissect specific categories of genes for leaf full functionality and crop improvement.

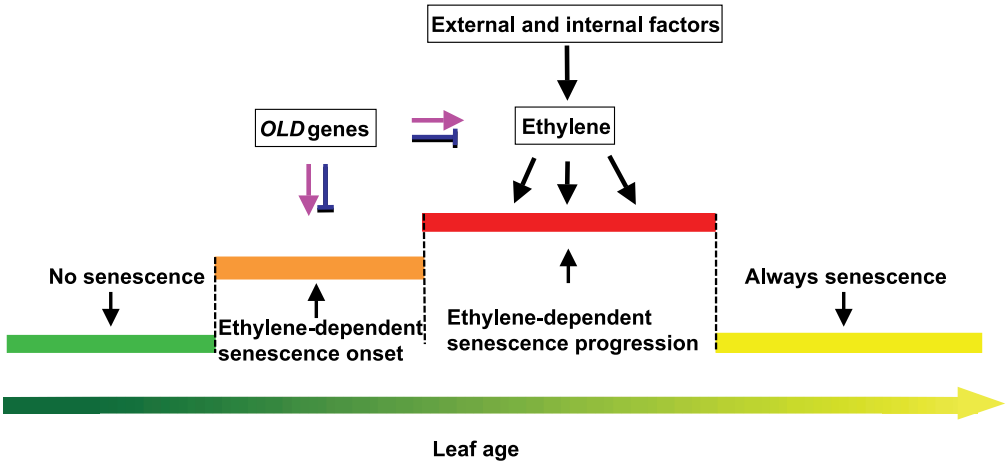

Figure 2. A diagram showing the roles of ethylene and *OLD* (*Onset of Leaf Death*) genes in leaf senescence.

The development of a leaf can be divided into four stages depending on their responses to exposure to exogenously-applied or indigenously-produced ethylene. "No senescence" depicts that at this phase senescence cannot be induced by ethylene, whereas "Always senescence" depicts that senescence initiates even in the absence of ethylene. The roles of *OLD* genes in modulating the onset and progression of ethylene-induced leaf senescence are shown as an indication that many genes are involved in the interaction between ethylene and leaf age factors (Gan 2012).

Leaf Senescence Ideotypes: Delaying the Onset of Leaf Senescence to Extend the Photosynthetically-Functional Phase, and Speeding the Rate of Leaf Senescence to Improve the Efficiency of Nutrient Remobilization

Four categories of genes are proposed to exist in a plant genome as potential exploitation targets for crop improvement. From a leaf senescence regulation point of view, it is desirable to develop crops with leaf senescence ideotypes, in which two important aspects should be optimized. First of all, Category III genes should be intensively explored to delay the onset of leaf senescence so as to extend the photosynthetically functional phase. Secondly, Category IV genes should also be explored to speed up the rate of leaf senescence in order to improve the efficiency of nutrient remobilization. The manipulation of Category III genes is expected to augment the total net photo assimilates for the yield gains, while the manipulation of Category IV genes is expected to increase the harvest index.

By intuition, extending the photosynthetically functional phase is the most straightforward approach to increase total photoassimilates, crop yield, and biomass. In many crops, variations in the functions of genes controlling sensitivity to vernalization and/or photoperiod can substantially alter the duration from crop establishment to anthesis, resulting in huge differences in crop yield and biomass, particularly in maize and sorghum (Rooney and Aydin 1999; Richards 2000). So far, the extension of photosynthetic duration and the resultant yield increase in crops in the past decades have been predominantly achieved through the genetic improvement of foliar disease resistance, the consequence of which is to delay the onset of leaf senescence induced by pathogen attacks. As a matter of fact, many genes and signaling pathways controlling leaf senescence are also involved in resistance to diseases and tolerance to abiotic stresses. Interestingly, it appears to be a rule, rather than an exception, that genes enhancing stress tolerance impact leaf senescence, or vice versa. For instance, transgenic tobacco plants expressing an isopentenyltransferase gene driven by a stress- and maturation-induced promoter displayed delayed whole plant senescence and outstanding drought tolerance (Rivero et al. 2007). In sorghum, genetic loci enhancing post-anthesis drought-induced leaf senescence have been described (Harris et al. 2007). Thus, the manipulation of single genes can substantially modify leaf senescence and hence crop productivity (Gan and Amasino 1995, 1997). In this sense, a number of positive and negative regulatory genes of leaf senescence well-characterized in model plants, including members of the WRKY, NAC and MYB transcriptional factors, should be at the top of the list for interest in exploitation. In theory, knocking-down the positive regulators (for example, *ORE* genes), or over-expressing negative regulators (for example, *OLD* genes), could possibly modulate the onset of leaf senescence and hence extend the full functionality phase of the leaf (**Figure 1B**). However, the fact that over-expressing *OLD1*/*CPR5* does not generate delayed leaf senescence suggests that the relationship is not always straightforward, and a fine-tuning of gene expression might be essential (Gurr and

Table 1. Some of the well-characterized genes with known functions in leaf senescence

Classes	Genes	Function description of proteins	Species	References
Regulator				
Transcription factor				
	AtNAP	Binds to the Promoter Region of *SAG113*	*Arabidopsis*	(Guo and Gan 2006; Zhang and Gan 2012)
	ORE1	A NAC family transcription factor	*Arabidopsis*	(Balazadeh et al. 2010)
	VNI2	A NAC family transcription factor, regulates the COR and RD genes	*Arabidopsis*	(Yang et al. 2011)
	NAM-B1	Accelerates senescence and increases nutrient remobilization	Wheat	(Uauy et al. 2006)
	NTL4	Promotes ROS production	*Arabidopsis*	(Lee et al. 2012a)
	JUB1	Modulates cellular H_2O_2 level	*Arabidopsis*	(Wu et al. 2012)
	RAV1	Regulation of transcription	*Arabidopsis*	(Woo et al. 2010)
	GBF1	A DNA-binding protein of the CAT2 promoter and increase the CAT2 expression	*Arabidopsis*	(Smykowski et al. 2010)
	TCP4	Involved in heterchronic regulation of leaf differentiation	*Arabidopsis*	(Sarvepalli and Nath 2011)
	TCPs	Transcription factor genes, control jasmonate biosynthesis and senescence	*Arabidopsis*	(Schommer et al. 2008)
	WRKY 54 and WRKY30	WRKY30 interacts independently with WRKY54, WRKY70, and WRKY53	*Arabidopsis*	(Besseau et al. 2012)
	WRKY53	A positive regulator of leaf senescence	*Arabidopsis*	(Miao et al. 2004)
	WRKY70	A negative regulator of leaf senescence	*Arabidopsis*	(Ülker et al. 2007)
	AD protein	A positive regulator of WRKY53 expression	*Arabidopsis*	(Miao et al. 2008)
	SUVH2	Histone methyltransferase. Regulate WRKY53	*Arabidopsis*	(Ay et al. 2009)
	AtTZF3,AtTZF2	Zinc finger transcription factor	*Arabidopsis*	(Lee et al. 2012b)
Enzyme regulator				
	INVINH1	Reduces cell wall invertase activity	*Arabidopsis*, Tomato	(Jin et al. 2009)
Protein kinase/phosphatase				
	GmSARK	Leucine-rich repeat-receptor-like protein kinase	Soybean	(Xu et al. 2011)
	MKK9,MPK6	Mitogen-activated protein kinase	*Arabidopsis*	(Zhou et al. 2009)
	SAG113	An ABA-regulated and Golgi-localized protein phosphatase	*Arabidopsis*	(Zhang et al. 2012)
	MEKK1	A member of the A1 subgroup of the MEKK family, can bind to the promoter of WRKY53 and also phosphorylate WRKY53 *in vitro*	*Arabidopsis*	(Miao et al. 2007)
Signaling				
	ARF2	A repressor of auxin signaling	*Arabidopsis*	(Lim et al. 2010)

Table 1. Continued

Classes	Genes	Function description of proteins	Species	References
Defense and stress				
	AtATG6	Defense response to fungus	*Arabidopsis*	(Patel and Dinesh-Kumar 2008)
	CPR5/OLD1	Defense response	*Arabidopsis*	(Jing et al. 2007; Jing et al. 2008)
	AAF	Involved in redox homeostasis	*Arabidopsis*	(Chen et al. 2012a; Chen et al. 2012b)
	NOL,NYC1	Act in the form of a complex as a chlorophyll b reductase	Rice	(Sato et al. 2009)
Metabolic process Degradation				
	OsAkαGal	Involved in the degradation of digalactosyldiacylglycerol	Rice	(Lee et al. 2009)
	RLS1	A NB-containing protein with an ARM domain	Rice	(Jiao et al. 2012)
	See2β	A cysteine protease	Maize	(Donnison et al. 2007)
	UPL5	UPL5 is involved in the degradation of WRKY53 and most likely has Ubiquitin ligase activity *in planta*	*Arabidopsis*	(Miao and Zentgraf 2010)
	ORE9	An F-box protein involved in ubiquitin-dependent protein degradation	*Arabidopsis*	(Woo et al. 2001)
	PPH	Pheophytinase, specifically dephytylates pheophytin (phein)	*Arabidopsis*	(Schelbert et al. 2009)
	SAVs	Involved in the degradation of chloroplastic components	Tobacco	(Martinez et al. 2008)
Biosynthesis				
	CAO	Catalyzes Chl b biosynthesis	*Arabidopsis*	(Sakuraba et al. 2012a)
	OLD3/OAS-A1	Catalyzes the final step of cysteine biosynthesis	*Arabidopsis*	(Shirzadian-Khorramabad et al. 2010)
	PES1,PES2	Involved in fatty acid phytyl ester synthesis	*Arabidopsis*	(Lippold et al. 2012)
Transport				
	AtCNGC11,12	Cyclic nucleotide-gated ion channels	*Arabidopsis*	(Urquhart et al. 2011)
	HPR1	A component of the THO/TREX complex that is required for mRNA export	*Arabidopsis*	(Pan et al. 2012)
	IVDH	ATP binding protein, involved in degradation of the branched-chain amino acids	*Arabidopsis*	(Araujo et al. 2010)
	ORE3/ORE2/EBF2	Transporter activity, a key integrator of the signaling pathways that control various plant responses	*Arabidopsis*	(Oh et al. 1997; Kim et al. 2009; Kim et al. 2011a)
	PPDK	Generates the transport amino acid glutamine, accelerate nitrogen remobilization from leaves	*Arabidopsis*	(Taylor et al. 2010)
	SPL28	A clathrin-associated adaptor protein complex 1, medium subunit μ1 (AP1M1),which is involved in the post-Golgi trafficking pathway	Rice	(Qiao et al. 2010)

Table 1. Continued

Classes	Genes	Function description of proteins	Species	References
Catalytic				
	AtXDH	Catalyze the conversion of the purine catabolic products hypoxanthine and xanthine to uric acid	*Arabidopsis*	(Brychkova et al. 2008)
	DES1	Catalyzes the desulfuration of L-Cys to sulfide plus ammonia and pyruvate	*Arabidopsis*	(Alvarez et al. 2010)
	LAP2	Controls intracellular amino acid turnover	*Arabidopsis*	(Waditee-Sirisattha et al. 2011)
	YUCCA6	Catalyses a rate-limiting step in *de novo* auxin biosynthesis	*Arabidopsis*	(Kim et al. 2011b)
	UGT76B1	Glucosyltransferase activity, involved in SA-JA signaling crosstalk	*Arabidopsis*	(von Saint Paul et al. 2011)
	ATG7	ATP-dependent activating enzyme; ATG8 activating enzyme activity	*Arabidopsis*	(Doelling et al. 2002)
	APG9	Maintain the cellular viability under nutrient-limited conditions	*Arabidopsis*	(Hanaoka et al. 2002)
	KAT2	JA-biosynthetic b-oxidation enzyme	*Arabidopsis*	(Castillo and Leon 2008)
	D2HGDH	Dehydrogenase activity, involved in Lys degradation	*Arabidopsis*	(Araujo et al. 2010)
Binding				
	ACBP3	A phospholipid binding protein, modulates membrane phospholipid metabolism and ATG8 stability	*Arabidopsis*	(Xiao and Chye 2010; Xiao et al. 2010)
	soyBiPD	ER-resident molecular chaperone, overexpression confers resistance to drought and delays leaf senescence	Soybean	(Valente et al. 2009)
	UBA2A, UBA2B, and UBA2C	Heterogeneous nuclear ribonucleoprotein (hnRNP)-type RNA-binding proteins	*Arabidopsis*	(Kim et al. 2008)
Structure				
	ORE4/PRPS17	Plastid ribosomal small subunit protein 17	*Arabidopsis*	(Woo et al. 2002)

Rushton 2005; Jing et al. 2007). We await further evidence to demonstrate the relevance of the modulation of the onset of senescence to crop breeding.

Exploitation of Category IV genes is a complimentary approach to achieve yield gains by modulating the speed of leaf senescence. High mobilization efficiency is desired once the onset of leaf senescence is initiated, especially when crops are faced with post-anthesis-adverse conditions. In crops, breeding lines may have distinctive patterns and rates for senescence progression (Hafsi et al. 2000). *Arabidopsis ore* and *old* mutants displayed different rates of senescence in addition to altered onset of leaf senescence (Oh et al. 1997; Jing et al. 2002; Jing et al. 2005). Thus, the speed of senescence is also genetically controlled. An increase in harvest index is the major contributor to yield increase in wheat cultivars without reducing total above-ground biomass (Richards 2000) through the selection for Green Revolution dwarf genes (Peng et al. 1999). New approaches are now possible to further improve nutrient partitioning, as envisaged by the identification of plant architecture genes in rice (Jiao et al. 2010). However, the nature and the mode of action of Category IV genes are not well known. Recent exciting advances on the role of autophagic machinery in bulk protein degradation and nitrogen remobilization may provide genetic tools to regulate the speed of senescence and nutrient recycling (Wada et al. 2009; Guiboileau et al. 2012). The wheat *GPC-B1* gene was discovered as a key component in increasing grain protein content by accelerating senescence (Uauy et al. 2006) and enhancing nitrogen remobilization (Waters et al. 2009). However, the gene was also found to shorten the grain filling period, and to thus reduce grain dry weight as a consequence of accelerated monocarpic senescence (Brevis et al. 2010). Several studies also indicate that genes involved in amino acid synthesis are important for the rate of leaf senescence and nitrogen remobilization as demonstrated by the *old3* (Shirzadian-Khorramabad et al. 2010), the *ASN2* (Gaufichon et al. 2012) and the *GS* (Martin et al. 2006; Canas et al. 2010) genes. Other possible targets for intervention are proteolytic activities of proteinases (Otegui et al. 2005; Donnison et al. 2007), sink capacity (Rolletschek et al. 2005; Sanders et al. 2009) and the overall regulation of carbon and nitrogen metabolism (e.g. DOF1, Yanagisawa et al. 2004; PPDK, Taylor et al. 2010).

It is important to take into consideration how to synergistically modulate the actions of both the Category III and IV genes. Evidence from crop breeding over the past 50 years indicates that although delaying leaf senescence results in the prolongation of photosynthesis duration and hence gains in yield and biomass, it also reduces nitrogen remobilization and grain protein content, and as a consequence, yield gains sacrifice nitrogen use efficiency and grain protein concentrations (Richards 2000; Yang and Zhang 2006). There is a concern that selecting for the trait of delayed leaf senescence would result in the dilemma of getting an increased yield but a reduced grain protein content. Although there are agronomic management approaches proposed to solve the issue (Yang and Zhang 2006), we believe that fine-tuning the actions of both the Category III and IV genes should be able to shed light on the dilemma, and is thus pave the way forward for plant breeding. **Table 1** lists a number of well-characterized genes, indicating that rich gene resources are now available for exploitation to enhance the control of leaf senescence.

Future Perspectives

The projection of the food supply in the next 50 years indicates that a huge challenge lies ahead due to population growth (Tester and Langridge 2010). Adding even more pressure to agriculture production are the rising frequencies of natural disasters and the adverse alterations of environments caused by climate change, as well as by the search for alternative biofuel sources. It is implied that crop yield potential has reached a plateau (Jaggard et al. 2010) and innovative approaches and new strategies have to be adopted to achieve further yield potential. Due to its importance for crop yield, the senescence of annual crops has been most intensively studied, and a delayed leaf senescence and an increase in harvest index are the two key components for the past yield gains in major crops. We believe that exploitation of the control of leaf senescence, combined with efforts to increase the rate of photosynthesis and the ability to tolerate stresses, is essential for a second wave of crop improvement to either achieve yield potential or to stabilize yield under stress conditions. The breeding of crops with leaf senescence ideotypes would be essential, which may be achievable through fine-tuning both the onset and the progression of leaf senescence.

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