INTRODUCTION

The word senescence derives from two Latin words: senex and senescere. Senex means ‘old’; Senescere means ‘to grow old’. Senescence is a universal phenomenon in living organisms, the concept senescence was divided into mitotic and post mitotic processes. A mitotic cell is not dead it may undergo degenerative process leading to death. When the degenerative process is solely, they act as post mitotic senescence. The post mitotic senescence occurs in plant somatic tissues such as leaves, flowers and fruits.

Leaf senescence is a highly regulated and genetically controlled degenerative process that involves a series of concerted biochemical reactions chloroplast degradation and concomitant attenuation of anabolic activates such as photosynthesis and overall protein synthesis begin as a leaf surpasses its peak of assimilatory capacity. The slow degeneration of cell during leaf senescence is in part to ensure efficient remobilization of nutrients that are generated by macromolecular hydrolysis during senescence. According, the temporal control of leaf senescence, result in fitness and survival plant.

Environmental senescence may be biotic, resulting from the interaction with other organisms, or a biotic, resulting from change in physical or chemical components in their environment compared to optimal growth conditions. It is clear that global changes endangering our future environment, such as increasing temperatures, changes in precipitation, and altered atmospheric gas compassion and radiation, will have a profound effect on plant growth and development. These environmental factors have distinct effect on the initiation and progress of plant senescence.

Environmental stress is frequently diverting the program of plant development from a normal and relatively slow program into a process with greater metabolic activity and accelerated progress of senescence. As a response to these stresses was suggested to have an adaptive significance, enabling the plant to complete its life cycle. The available information regarding environmental control of senescence may regulate senescence via common internal factors such as oxidative state and sugars.

Effect of Light Irradiance on Regulation of Leaf Senescence
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ABSTRACT

Leaf senescence can be described as the dismantling of cellular component during the terminal stage in development of plant organs and tissues. Changing in physical or chemical components, they result in an environmental senescence. The light irradiance have controllable role in plant growth and development. It is required for photosynthesis and the vital biological process in plants, due to the diverse effect s of different light intensities and qualities on plant development. Also the photoreceptors are important for photosynthetic acclimation. The low light enhancement the senescence due to the shortage in energy which effect on sugar metabolism and reduced oxidative damage. Darkness enhance the rate of senescence mainly in senescence associated gene(SAGs). Although high light induces chlorophyll loss and degrease in photosynthesis efficiency causing senescence. However, prolong accumulated light intensities during the long day photoperiod result in elevated stress, which in turn enhanced senescence. Also the wavelength is available regarding the dependency of senescence. The effect due to the key photoreceptors such as red/far red responsive phytochrome, the blue light UV-A responsive crypto chrome pigments and on photosynthesis. Different, the UV-B light accelerated senescence. The low R: F values cause mobilization of resources leading to accelerated senescence..

Received: 16/10/2017
Accepted: 19/10/2017
Published: 24/10/2017

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Keywords: Environmental factors, Leaf senescence, Light irradiance
Light Irradiance

Light has a vital and central role in plant growth and development. It is required for photosynthesis as a signal for normal development and interaction with the environment. This interaction is mediated through specific pigments such as the phytochrome and cryptochrome or the phototropins light receptor and its signal transduction pathways. In addition to light quantity and intensity, plants can measure incident light quality, direction, and periodicity, and use that information to optimize growth and development in accordance with the changing environment. The effect of irradiance, or lack thereof, on senescence induction is complex due to the diverse effects of different light intensities and qualities on plant development. These effects may be direct or indirect, depending on the developmental stage of the plant and on the stress response it elicits. Various studies have demonstrated a direct effect of light on the initiation or advancement of senescence. The influence of light on plants, and subsequently on senescence, could be mediated via several routes including the efficiency of photosynthesis, the generation of damages due to oxidative stress, signaling via interaction with light receptors, photoperiod, and the affected circadian clock. The actual effect of each of these stimuli is determined by the specific characteristics of light irradiation, which include wavelength quality and amount, determined by light intensity and duration of exposure.

Photoreceptors

Phytochrome are involved in the perception of spectral canopy shade, but they can also be involved in the perception of other aspects of the light environment. Phototropins and cryptochrome are involved in responses to irradiance of blue light. Hence, photoreceptors are important candidates for the perception of canopy light gradients and the regulation of the response. Working with a limited number of photoreceptor mutants in Arabidopsis thaliana, Walters et al, found little evidence for an important role of photoreceptors in photosynthetic acclimation when whole plants were exposed to spectrally neutral shading. Weaver and Asamino simulated the canopy light gradient by darkening individual Arabidopsis leaves, which caused induction of senescence as with shading. The phytochrome and crypto chrome mutants that they used all showed essentially the wild type response with respect to this trait. Arabidopsis photoreceptor mutants were also used by Boonman et al. They included also phototropin mutants for the spectrally neutral shade effect on single leaves. Photosynthetic capacity was down-regulated in all mutants as in the wild type. There was one exception, the mutant of Arabidopsis deficient in phytochrome D (phyD) and the phyA phyB phyD triple mutant lacked the decrease in Chl a/b that is normally associated with shade. PhyD is thus important in this species for the acclimation of pigment composition in the canopy light gradient. Contrary to wild type tobacco plants (Nicotiana tabacum), senescence was not induced in low R:FR light when phyA was over expressed. However, phyA mutants showed accelerated loss of chlorophyll in shaded leaves irrespective of spectral composition (Brouwer et al, indicating that phyA is involved in the maintenance of chlorophyll in shade, counteracting the effect of phyB. The maintenance of chlorophyll is achieved by up-regulation of chlorophyll synthesis rather than the down-regulation of its degradation.

Light Intensity

Low light

The low light is known to cause the enhancement of senescence. The fraction of photo synthetically active radiation in the light perceived by the plant is an important factor in determining the initiation and advancement of senescence. For example, the shading of fully expanded cassava leaves results in accelerated senescence. Senescence of sunflower basal leaves was found to be enhanced as they received reduced photo synthetically active light. Accordingly, increased levels of photosynthetic active light reaching the basal leaves of maize canopy can delay senescence. In order for photo synthetically active light to retard senescence it is required to be above the photosynthetic compensation point. The specific mechanism mediating low-light-induced senescence is generally unknown. Shortage in energy or an effect on sugar metabolism may have an important role. The possible involvement of oxidative stress was suggested based on the observation that plant shading increases lipid per oxidation in wheat. This may result from the weakening of antioxidant protection, although low light was expected to result in reduced oxidative damage.

Darkness

Also the darkness is known to extensively enhance the rate of senescence and is frequently used for senescence studies. Senescence was demonstrated to be also induced by darkness in individually attached Arabidopsis leaves, but darkness inhibited the process in whole darkened plants. This result implies that the light status of the entire plant affects the senescence of individual leaves. One possibility is that a decrease in source strength was created in complete darkness, which led to a delay in senescence. In tobacco rbcS antisense mutants, which have reduced Rubisco levels, a prolongation of the senescence phase was observed which may be due to the impact of reduced source strength. Still, dark-induced senescence in individual leaves is highly localized and is possibly cell autonomous.

When various senescence associated genes (SAGs) were examined in Arabidopsis for their responsiveness to different hormonal and environmental treatments known to be associated with senescence, darkness was found to be most effective. Incubation of detached leaves in the light also had some senescence inducing effect, but was reduced when compared to darkness. Senescence processes, induced either naturally in attached leaves or by darkness in detached
leaves, share physiological and biochemical characteristics. However, molecular analyses of the two processes show differences in the sets of induced genes [26]. In addition, some genes have been shown to be expressed during leaf senescence regardless of whether it was induced naturally or by darkness, suggesting that the senescence processes under different conditions share common features [27,28]. For example, in Arabidopsis, the AtPaO gene, encoding for pheophorbide an oxygenase, involved in senescence-associated chlorophyll degradation, was shown to be induced in both natural and darkness-induced senescence [29].

The induction of SAGs by darkness can occur within 3-24 h as demonstrated by the din genes in Arabidopsis, but is highly dependent on the developmental stage of the leaf examined. Not much is known about the molecular regulation of darkness-induced SAGs. Studies involving the photosynthesis inhibitor DCMU and sucrose reveal that expression of some darkness-induced genes is related to sugar starvation in the dark, and involves different protein phosphatases and Ca²⁺/calmodulin signaling [30,31]. In Arabidopsis, the transcript level of the erd1, a clpA protease homolog, is induced during senescence due to dehydration stress. The promoter region of erd1 contains cis-acting elements that confer specifically darkness-induced expression in intact Arabidopsis plants [32]. Other darkness-induced SAGs encode for various products including proteins that may have a regulatory role [33,34]. Mutations that alter natural and darkness-induced senescence support overlapping components shared by both these processes. In Arabidopsis, the delay of natural and dark-induced senescence was observed in the ore1, ore3, ore9, and dis1 mutants, while acceleration of both senescence types was observed due to a mutation in the HYS1/CPR5 gene [35-37].

Analysis of the dependency of genes induced during dark-induced senescence in signaling pathways involving SA, JA, and ethylene have demonstrated that the SA pathway is not expressed in dark-induced senescence, while ethylene and JA signals are active as in natural developmental senescence [38]. While developmental senescence is delayed in plants defective in SA signaling, dark-induced senescence progresses normally in these plants. The differences in regulation of dark-induced senescence is also manifested by the differential effect of the senescence-retarding mutation ore-4 for both processes in Arabidopsis. In this mutant, the plastid-encoded ribosomal small subunit protein level is dramatically reduced, which has a retarding effect specifically on the age-dependent leaf senescence pathway but not on dark- or phytochrome-induced senescence. Furthermore, this mutation does not affect senescence induced by other factors such as abscisic acid (ABA), JA and ethylene, which are associated with other environmental stress factors.

**High Light**

Prolonged exposure to high-light irradiance induces chlorophyll loss and a decrease in photosynthesis efficiency, which was sometimes referred to as senescence [39]. However, in early studies only chlorophyll loss and changes in photosynthetic parameters were examined and not other senescence characteristics. It is possible that in continuously highly illuminated plants, senescence-like symptoms result from photo-oxidative damage due to an excessive amount of light, resulting in chlorophyll breakdown [40]. In general, the oxidative stress status of the leaf is intensified during senescence as levels of reactive oxygen species (ROS) are enhanced and antioxidant enzyme activity is reduced [41,42]. Thus, the metabolic changes that occur during senescence may further increase susceptibility to high-light-induced oxidative damage of the tissue. Senescing leaves are more sensitive to light irradiation also due to the significant decline in the photosynthetically active system. It was suggested that optical masking of the remaining chlorophyll by anthocyanins reduces risk of photo-oxidative damage to leaf cells as they senesce, which otherwise may lower the efficiency of nutrient retrieval from senescing autumn leaves. This hypothesis is supported by a study conducted in anthocyanin-deficient mutants of deciduous woody species. Interestingly, analysis of the reduction in photosynthetic efficiency and capacity during different stages of senescence of cotton leaves indicated no difference in the decline of photosynthesis under various light levels ranging from 15% to 100% full sun light [43]. When high-light stress is accompanied by an additional environmental stress, the senescence process is even further accelerated. In field conditions, high irradiance is often associated with water deficit. Examination of the combined effect of high light and water stress conditions suggests additive and possible synergistic action of both, causing an accelerated loss of pigments and proteins, compared to samples exposed to either of these stresses individually [44].

**Photoperiod**

The role of photoperiod in the control of leaf senescence was suggested mainly demonstrating a delay of leaf senescence in short days and its acceleration in long days, as is the case with temperate seasonal changes [45,48]. However, when the photoperiod effect was examined in terms of light period and dosage it was concluded that the enhancing effect was mainly a result of light dosage rather than of photoperiod. Prolonged accumulated light intensities during the long-day photoperiod apparently resulted in elevated stress, which in turn enhanced senescence. Natural variation in the effect of day length on leaf senescence was measured for different Arabidopsis ecotypes [49]. For most ecotypes, senescence occurred earlier in long days, and for two ecotypes it was either less pronounced or absent.

**Wavelength**

Almost no information is available regarding the dependency of senescence on wavelength. Any effects are likely related to the effect of light on key photoreceptors such as the red/far red responsive phytochrome, the blue/UV-A responsive crypto chrome pigment, and on phototropism. An early study reported the senescence-retarding action of non-photosynthetic light on excised
wheat leaf segments using a crude action spectrum analysis. In recent years, additional physiological and molecular studies demonstrated the importance of non-photo synthetically active light as a signal that affects senescence in plants.

The gradient in the 400–700 nm wavelength band (photosynthetically active radiation; PAR) is strong as a result of absorption by chlorophyll. Short wave infrared (700–3,000 nm), which comprises about half of the energy in the daylight spectrum, is partly absorbed by leaves which generates heat and thus forms an important component of the energy balance of leaves together with absorbed PAR. This contributes to elevated temperatures in the upper leaves and thus to a gradient in leaf temperatures. The 700 nm to about 1,000 nm region of the short-wave infrared is much less absorbed by leaves. The differential absorption between this spectral region and PAR can be perceived by plants using the phytochrome system. The spectral composition of the light with respect to phytochrome action is characterized by the red: far-red ratio (R:FR), the quantum flux ratio between the 660 nm (R) and 730 nm (FR) wavelength bands. This ratio decreases with intensity of canopy shade as the absorption of R increases stronger compared to FR. Canopy gradients in R:FR are thus similar to PPFD gradients.

Red/Far red

Phytochrome photoreceptors enable plants to sense a reduction in the ratio of red (R) to Far-Red (FR) light in their environment and change their growth or development accordingly. For example, light that has passed through the canopy of leaves has a lower ratio of R/FR due to absorbance of the red by chlorophyll of the upper leaves. The ability to sense modified R/FR light when shaded by their neighbors allows plants to avoid shading by increasing their internodes extension rate. It has been long recognized that the light environment determined, by the density of a plant population may regulate photosynthetic characteristics as well as the timing of senescence. Reduced photosynthetically active radiation and decreased R/FR ratio are the prime senescence-triggering signals in shaded leaves of sunflower and in the leaves of soybean grown under field conditions. Senescence and abscission of leaves positioned in the shaded regions of the canopy were delayed by more than 4 weeks in plots where plant population densities were reduced. This low-density plant population, a higher ratio of R/FR light was measured in the shaded regions of the canopy. Enrichment of far-red light in field-grown sunflower accelerated senescence of individual leaves and was indicated by enhanced chlorophyll loss. The hypothesis that increasing the R/FR ratio perceived by basal leaves within canopies delays senescence was further confirmed in field-grown sunflower plants, in which enriched red light significantly delayed lower leaf senescence. The ability of red light to retard senescence was also demonstrated in the leaves of cut A. exaltata, while low R/FR is able to promote senescence in soybean. The importance of the phytochrome in control of senescence was demonstrated for primitive plants also. In the moss Marchantia polymorpha, the senescence-delaying effect of white light could be reverted by FR, while red light could reverse the FR effect. In the fern N. exaltata, senescence-accelerating effect of red light was observed which could be nullified with FR pulses.

Leaf senescence responses to FR were found to be localized, and sensitivity to FR was also inversely correlated with the local PHYA phytochrome gene expression level. The localized FR response in the leaf is consistent with the localized senescence response to dark observed in Arabidopsis and with the frequent observation that senescence can be induced locally in parts of the leaves shaded by upper leaves. Few different molecular genetic studies further support the role of the phytochrome system in senescence control. Ectopic overexpression of an oat PHYA (phytochrome A gene) cDNA in tobacco under the CaMV 35S promoter resulted in a delay in leaf senescence. Also in tobacco, over expression of the oat PHYA gene reduced morphological responses to FR radiation and resulted in suppressed leaf-senescence responses. Transgenic potato plants constitutively expressing the Arabidopsis PHYB were found to have a delay in the onset of senescence under white-light irradiation. An earlier study of these plants had claimed that the initiation of senescence in the PHYB over expressing plants occurred at approximately the same time as in the wild-type, but the lifetime of the photo synthetically active transgenic plants was extended by 3–4 weeks. In pea a dominant mutation in PHYA, resulting in reduced sensitivity to FR light, had a pleiotropic effect including delayed flowering and senescence. In some cases, the mutant pea plants grew for more than 6 months before senescence was initiated as compared to wild-type plants which grew for about 3 months. Phytochrome A levels are low in green plants at high irradiance and high R:FR ratios as a result of degradation of PHYA in the Pfr form. An Arabidopsis phyA mutant showed a similar reduction of A_680 and Chl_a in response to supplemental FR at moderately high irradiance compared to wild type Arabidopsis, whereas a phyB mutant showed no response (unpublished results). This is consistent with the notion that phyA is not active at high PPFD, whereas phyB appears to be the main player in the regulation of resource reallocation in response to the R:FR ratio in these conditions.

Blue light

The role of blue light in regulation of senescence is much less clear compared to that of the R/FR light and only few early studies using non-natural senescence systems suggest such a role. Monochromatic blue light (450 nm) was suggested to retard the decline of photosynthetic activity in detached leaves induced to senescence in the dark. Dark-stimulated chlorophyll loss was shown to be retarded by blue light pulses in senescing papaya leaf discs. Delay of senescence by blue light was also demonstrated in Hosta Tratt. cut flowers. It was suggested that an interaction between red, FR and blue light exists to affect senescence. Such interactions and integration between the different light signaling in plants were suggested to occur throughout the development.
Ultraviolet

Possible increase in the level of UV radiation as a result of depletion of the stratospheric ozone layer is a major environmental concern in recent years. The three UV radiation bands are UV-A (320–400 nm), UV-B (280–320 nm), and UV-C (200–320 nm). UV-C is the most damaging radiation to biological systems followed by UV-B. However, as sunlight passes through the atmosphere, all UV-C and approximately 90% of UV-B radiation are absorbed by ozone, water vapors, oxygen and carbon dioxide. UV-A radiation is less affected by the atmosphere. Therefore, the UV radiation reaching the earth’s surface is largely composed of UV-A with a small UV-B fraction. Since UV-B is much more damaging than UV-A, more research investigating its effects on plants was performed. Before UV-B radiation can give rise to a cellular response, it has to be perceived, which is thought to occur via a UV-B photoreceptor followed by several different signaling pathways. These pathways include second messengers such as calcium, kinases and the signaling ROS. High levels of UV-B probably cause cellular damage and major oxidative stress; thus activating a general stress and signal transduction pathway which leads to a response similar to that which occurs after pathogen attack or other stresses. The biological consequence of UV-B is very active when it is applied artificially at a high dose in controlled experiments. However, realistic levels of UV-B in field experiments were also shown to have physiological effect on plant growth and development as well as affect gene expression.

Enhanced UV-B radiation produces deleterious effects on physiological and morphological traits of plant and thus, poising a server threat to the existence and survival of organisms.

The senescence-inducing effect of UV was demonstrated in various plant systems. Senescence-induced loss in pigments and proteins of detached maize leaves was significantly enhanced by UV. In both Arabidopsis and pea, it was found that older leaves become damaged by UV faster and to a greater extent than do younger leaves and an initial phase of chlorophyll loss was followed by desiccation of the tissue. At the biochemical level, the rate of photosynthesis is greatly reduced in the UV-treated leaves, primarily as a result of a decline in RUBISCO protein levels and disruption to the chloroplast membranes. Some of these changes can be attributed partially to the effects of UV-B on expression of genes encoding key photosynthetic proteins. Thus, in leaves at a certain stage of development, expose can induce changes at the physiological, biochemical and molecular levels that resemble symptoms identified in plants undergoing senescence, including induction of SAGs as demonstrated in Arabidopsis.

The senescence-inducing effect of UV-B radiation might be transduced by either its effect on the photosynthesis apparatus or via generation of oxidative stress. UV-B exposure caused increases in JA and ethylene levels, and together with the observed effects on Arabidopsis stress responses and induced genes, the involvement of three distinctive signal transduction associated with ROS, JA, and ethylene was suggested. In the chloroplast, the thylakoid membrane seems to be much more sensitive to UV-B radiation than do the activities of the photosynthetic components within it, and a decrease in mRNA transcripts for the photosynthetic complexes and other chloroplast proteins are considered very early events of UV-B damage. Exposure to UV-B radiation resulted in a loss of chlorophyll and an increase in lipid damage similar to that induced during natural senescence, including decrease in lipids and increased lipid per oxidation indicated by rise in MDA. Difference between UV-B induced, and natural senescence was found in the consequence to the maximum quantum efficiency of PS II photochemistry represented by the fluorescence marker Fv/Fm. Fv/Fm level was found to be more significantly decreased in leaves treated with UV-B.

Some of the genes identified so far as being regulated by UV-B encode proteins involved in the biosynthesis of protective pigments and and oxidative enzymes, DNA repair, photosynthetic genes, cell cycle genes, and stress genes induced by other types of stimuli (i.e., pathogenesis-related proteins). In few studies, the exposure of plants to UV-B resulted in up-regulation of SAGs, including that of the SAG12 protease gene considered to be highly associated with developmentally induced senescence although much reduced level.

The effect of UV-A on senescence is not clear as only few studies were carried out. On one hand UV-A was shown, at low intensities, to be more efficient than white light in inhibiting dark-induced senescence of barley leaf segments and could retard the senescence-inducing effect of UV-B in cluster bean leaves. On the other hand, UV-A was reported to enhance senescence in primary leaves of wheat which could be retarded by a red-light pulse. The effect of UV-A radiation on senescing wheat leaves over a period of days had resulted with negative impact on primary photochemistry of photosystem II (PS II) but did not show any significant effect on the level of photosynthetic pigments. The UV-A induced changes in PS II of chloroplasts from senescing leaves were found to be synergistically accelerated by high-temperature growth. It is possible that the specific effect of UV-A on senescence is dependent on the actual wavelength composition of the radiation used. When more enriched with longer wavelength radiation, there is a senescence-retarding effect, similar to that of blue light; however, with shorter wavelength radiation, the senescence is accelerated as with UV-B.

CONCLUSION

The observed effects of environmental stimuli on plant senescence suggest that almost any environmental stress with a negative consequence to growth conditions may result in the enhancement of plant senescence. However, information is limited about the regulatory pathways involved in mediating the environmental stress signals with the observed enhancement of
senescence. Recent studies of senescence in plants suggest that complex regulatory networks exist in senescence (Guo and Gan, 2005; Lim and Nam 2005). Also not all photoreceptors are not essential for canopy PPFD gradient effect on photosynthetic resource reallocation and senescence, or there is a high degree of redundancy, which means that several photoreceptors are involved that can take over when one or more others are lacking. Different, the UV-B light as an environmental stress accelerates senescence (Kim et al. 1998; van der Krol et al. 1999). Different hypotheses that explain how a leaf can sense its photosynthetic status within the plant were suggested (Ono et al. 2001). Interactions between some of these different systems and factors in relation to the regulation of senescence were demonstrated. The low R:FR values to which shaded leaves are exposed cause mobilization of resources leading to accelerated senescence. Reduced PPFD incident on lower leaves has similar effects as a low R:FR, but when senescence has not been induced yet, the mobilization of resources goes largely at the expense of photosynthetic capacity and not so much chlorophyll.

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