Current Topics in Plant Biology
Vol. 10, 2009

Review

Plant senescence: A self-induced process

Victorio S. Trippi*, H. Ramiro Lascano and Mariana Melchiorre
Instituto de Fitopatología y Fisiología Vegetal IFFIVE-INTA, Camino 60 Cuadras km 5 ½ (X5020ICA), Córdoba, Argentina

ABSTRACT

This review suggests that plant senescence is a self-induced process. This proposition is based on changing relations among cells and organs in a growing system, originated by growth itself, and differentiation due to adaptation to internal and external environments conditions, which are also changing conditions during development. This succession of events has been called stress-functions or emerged features. Among them are mentioned: growth in height restricting water supply to the most distal parts; the seed germination, modifying the relations with oxygen during the change from anaerobic metabolisms to an aerobic one; leaf growth and differentiation generating toxic hyperoxic conditions in autotrophic cells; the source-sink relations, inducing senescence in source organs by deficiency; sugar accumulation and growth regulators or hormones inducing oxidative process. The reactive oxygen species (ROS), generated under different stress condition, should be major modulators of growth and differentiation processes that became in senescence of both autotrophic organs, which are more sensitive to oxidative process and heterotrophic ones, which being more tolerant to oxidations assure specie perpetuation.

KEYWORDS: senescence, auto-organization, stress

INTRODUCTION

Although the cell has been recognized as the basic component of living organisms that makes up their structural and functional unit, the study of senescence has been focused considering multicellular organisms as the main conceptual object.

The study of senescence in multicellular organisms has been addressed from different perspectives: the cell, the organs and the organisms; however, the convenient approach to the problem is still to be elucidated.

A study of senescence focusing on an organism has not been conducted yet probably because the rules of cell organization (multicellular organisms) and the changing nature of cells during development of an organism are unknown. Thus, we believe that growing, reproducing and dying are basic stages in the life of multicellular organisms. We also believe that growth detention-a consequence of the sigmoid function-simply means the end of the growth stage.

A cell-oriented analysis of senescence based on self-organization of cells leading to the development of multicellular organisms allows us to understand development and senescence from a new perspective, even if we overlook molecular relationships and physicochemical components of the organization. Indeed, growth detention in a multicellular organism means that the cell-environment interaction is no longer suitable for division and growth to continue. It has been long acknowledged that cells that have stopped growing in an organism's tissue recover their capacity to divide when they are provided with favourable conditions (i.e. vegetative propagation) and that they are even capable of developing a new organism [1, 2].

*Corresponding author
vtrippi@correo.inta.gov.ar

Our analysis considers that senescence at any organization level is basically related to the cell and an environment (internal or external) that tends to become less suitable for life and cell persistence during development. Accordingly, we will attempt to elucidate changes in multicellular organisms induced both by growth and by modified relationships between organs (Fig. 1).

1. Stress-inducing functions during development

During the life of organisms, reduction and oxidation reactions take place. Prevalence of oxidation determines degradation of cell functions, senescence and cell death. Some of the stress factors induced by the functions that take place in the organisms as well as the stress factors exerted by the environment are listed below.

Growth as a senescence-inducing factor

Growth as a senescence-inducing factor has not been much documented possibly because senescence is considered inherent to the organism due to genetically determined internal causes. Likewise, the sigmoid growth function does not seem relevant to the organism, since it only serves as an indicator that development of an individual has been completed. Growth is considered as a stage in the life of organisms. However, the observation of plant organisms reveals that growth is a senescence-inducing factor. We will briefly explore some interesting cases.

Gravity as a water stress-inducing factor during growth

The development of xeromorphic structures in plants from the lower to the upper canopy and along the shoots has been frequently described approximately since 1900. Wangerman (1961) [3] indicates that the upper part of shoots show more xeromorphic traits than the lower part: cell size decreases upwards, the cuticle becomes less waxy, the number of stomata increases and the palisade tissue becomes thicker, vein reticulum is denser, tissues become more compact. A gradient in leaf shape can be frequently observed, which led Goebel (1908) [4] to define these plants as having heteroblastic growth.

When fodder cabbage plants are subjected to water stress conditions, the lower leaves develop the greatest Water Saturation Deficit values, showing a decreasing gradient towards the apex [5]. The same was observed in plants that have been completely removed during wilting [6] (Figs. 2 and 3).

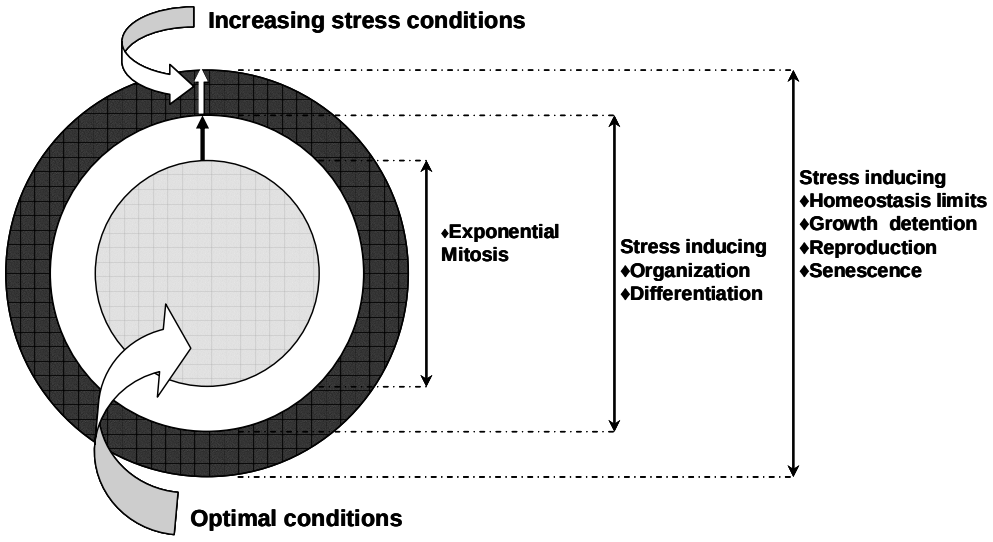

Figure 1. Conceptual scheme of cell behaviour under different (internal+external) environmental conditions that may lead to indefinite division when environmental conditions are optimal; organization and differentiation; up to cell division precluded by the environment, leading to growth detention, reproductive growth and senescence.

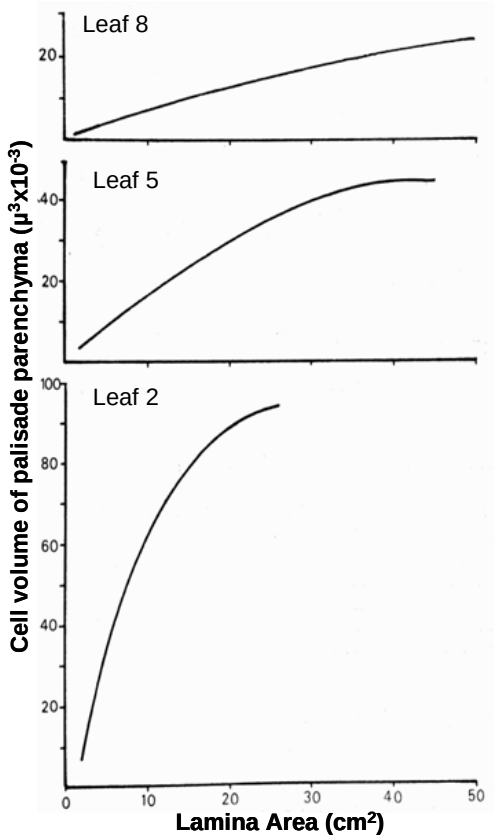

Figure 2. Changes in cell volume of the palisade parenchyma in leaves of *Capsicum frutescens* L. with increasing area of laminas number 2, 5 and 8 [7].

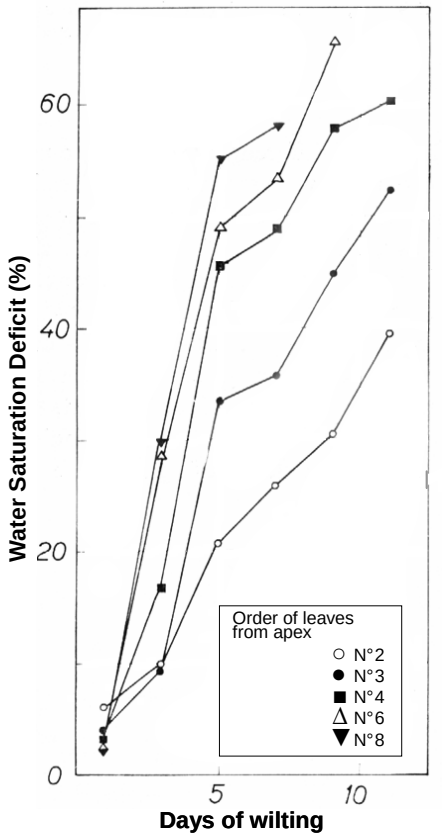

Figure 3. Development of Water Saturation Deficit induced by water stress in cabbage fodder [5].

Gravity determines and influences orthotropic shoot growth. It seems reasonable to assume that water and solute supply to the upper shoot becomes more difficult as height growth occurs. For water to be transported to the most apical zones, cells develop low water potentials, generating a root-upper shoot gradient. This gradient is formed during growth by the influence of gravity and water column weight in the xylem. Cells adapt to water availability by modifying their structure and functions, depending on their genetically determined capacity. Thus, the shoot will continue growing until the homeostatic capacity allows cells to maintain their life functions. When their homeostatic capacity has declined, division and growth stops. Hence, the formation of xeromorphic traits in cells and organs is the result of water stress conditions that limits growth, which in turn are generated by growth. It is also easily assumed that in a growing system, water requirements increase as well; in turn, water depends on roots that are in a soil whose capacity to supply water is often unpredictable, and where water supply tends to be a limiting or stress factor.

2. Growth and differentiation modify cell-oxygen interaction

a. Germination changes cell-oxygen interaction

It is easily perceived that as plants and their organs grow, they change their interaction with the external environment, since in an open system meristems may grow indeterminately if the surrounding environment allows their cells to preserve their functionality.

Observation of seed germination shows that seed cells (embryo) are in a hypoxic environment. Only few species, such as rice, can germinate in anoxia. Dicotyledonous seeds with lipid reserves germinate at 2-10 kPa oxygen, and those with

starch reserves germinate at 0.01-0.1 kPa oxygen [8] (Fig. 4).

It has also been demonstrated that the earliest germination stages of *Phaseolus vulgaris* occur in the presence of the fermentative enzymes Alcohol-DH, Lactic-DH and Formic-DH, which possess isoenzymes that disappear on the fourth day. An increase in isoforms and total peroxidase activity is also observed [9]. These facts suggest that species persistence (Weissman's germ line) is based on a genetic determinism of cells that allows them to adapt their functioning to very diverse environmental conditions, ranging from very low or anoxic oxygen content to conditions that may be classified as hyperoxia. Therefore, we may assume that embryo germination or growth forces cells to change their relationships with oxygen. Their genetic potential would allow species persistence through resistance forms (seeds), which improve control of cell oxidation because of their heterotrophic tissues.

b. Leaf and petal growth and its relationship with oxygen concentration

Leaf growth consists in the transformation of a foliar primordium into a leaf by differentiation of the primordium's small aggregated cells. Changes involving an increase in the carbon/nitrogen (C/N) or dry weight (DW/N) relationship have been observed during plant and leaf development by several authors [10]. The decrease of both C and N content with respect to dry weight observed was interpreted as an alteration of the proportion of basic elements produced by enriched oxygen compounds, probably determined by the environment (Fig. 5).

The possible incidence of the Nitrate reductase enzyme (NR) was studied. NR activity decreases with increased oxygen pressure [11] (Fig. 7), showing that inhibition by oxygen pressure occurs at translational or post-translational level [12]. The enzyme activity increases up to 7 days of age and then decreases. In an atmosphere of 100% O_2, decrease of NR activity is accelerated, and at 4% O_2 activity decrease is delayed, as when cysteine is employed as antioxidant [13]. The evolution of NR activity through leaf growth suggests that growth might result from a change in leaf oxidation (Fig. 6).

The analysis of the development of senescence parameters-chlorophyll degradation, α amino nitrogen, malondialdehyde (MDA), and conductivity-during oat leaf growth revealed that MDA increased constantly whereas chlorophylls and proteins decreased (Fig. 7).

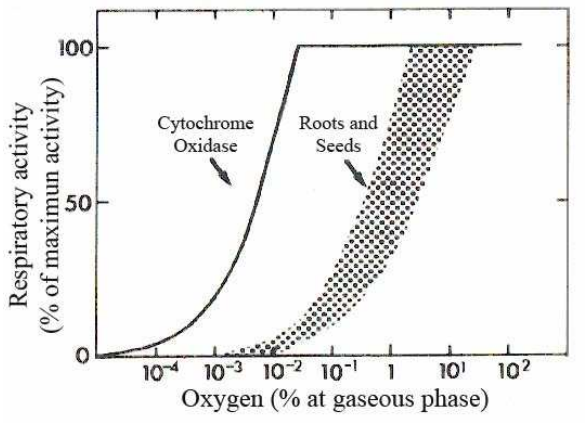

Figure 4. Saturating oxygen concentrations for cytochrome oxidase requirements and for respiration in seeds and roots [8].

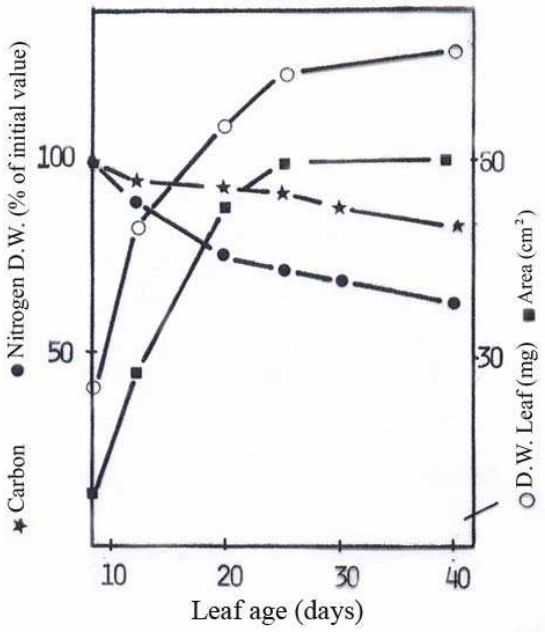

Figure 5. Changes in the relationship between Carbon and Nitrogen content and Dry Weight of *Phaseolus vulgaris* leaves at different ages [10].

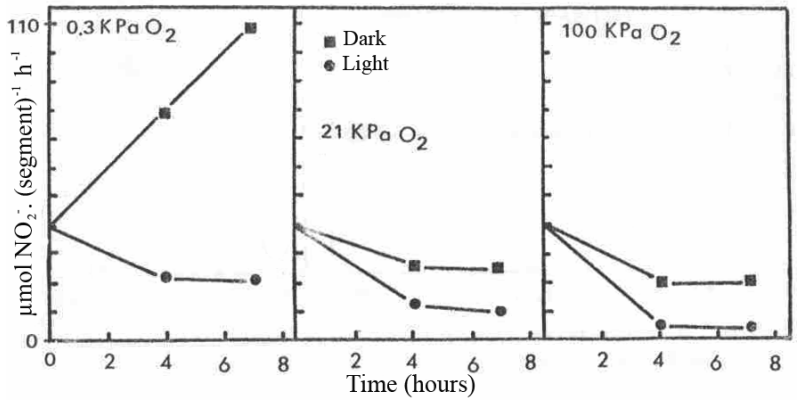

Figure 6. Nitrate Reductase activity in oat leaves at different oxygen content and in light and dark conditions as a function of time [11].

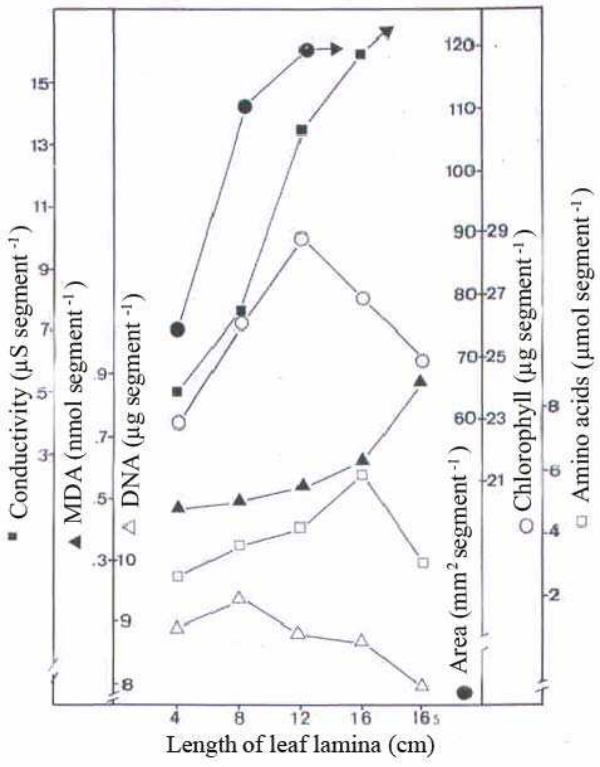

Figure 7. Changes in senescence/aging parameters (conductivity, MDA, DNA, area, chlorophyll, and α-amino nitrogen) during leaf lamina growth in *Triticum aestivum.* The only parameter that increases, including the senile phase (16s), is MDA, which is an indicator of membrane lipid peroxidation.

In vitro aging of oat leaf segments under partial oxygen pressure (pO_2) ranging between 0.5 and 99%, showed that senescence occurred more rapidly at higher O_2 concentrations, and that 0.5% concentrations delayed the senescence process in light conditions [14]. This suggests that O_2 content of 21% of the atmosphere is a true hyperoxia condition for leaves in the light, and that leaf growth might be a senescence factor that supports MDA formation and alteration of membrane permeability.

The effect of oxygen pressure was also confirmed in flowers, in which senescence is accelerated with increased partial oxygen pressure [15]. Flowers met their oxygen requirements at 5-10% O_2 concentrations, showing that 21% of the atmosphere is a hyperoxic concentration, in this case for a heterotrophic tissue.

The analysis of energy metabolism confirms that senescence accelerated by higher oxygen pressure is accompanied by degradation of ATP levels, both in flowers and leaves [15, 16].

If leaf growth accelerates oxidations, then the former is a senescence (internal) factor inherent to the organism, possibly resulting from multicellularity and differentiation. The study of factors modifying leaf growth, like osmotic potential, temperature and kinetin, showed that MDA production based on DNA content was higher with greater growth (Trippi *et al.*, unpublished data). Determination of peroxides using titanium reagent showed that peroxide content of *Phaseolus vulgaris* leaves increased with leaf age (Del Longo & Trippi, unpublished data).

The anatomical analysis suggests that during leaf primordial growth the free intercellular space increases. Differentiation of internal structures, formation of stomata and development of the photosynthetic apparatus produce an increase of partial oxygen pressure in tissues, which is inferred simply by the diffusion of gas into the atmosphere. Only more recently was it determined that growth is directly associated with the presence of superoxide radical in the elongation zone of maize seedlings [17]. Likewise, the importance of NOX proteins in different processes and several organisms was defined. Cell wall loosening, development of reproductive structures in fungi and differentiation of cotton fiber show the need for ROS participation [18].

The effects of oxygen pressure on senescence onset suggest that changes in cell-oxygen relationship may be an oxidative stress condition. Likewise, the effects are different for autotrophic tissues, which work better at about 0.5% of O_2, than for heterotrophic organs, which work very well at 5-10% of O_2 in the atmosphere. For both types of tissues, oxygen content in the atmosphere is a toxic hyperoxic condition for the cell.

3. Growth and plant architecture change the relationship between light quality and plant parts

The structure of organisms is the result of cells' genetic determinism and of the environment in which they develop.

Leaf senescence is influenced by the quality of incident light. It has been indicated and repeatedly confirmed that leaf senescence is induced by far red (FR) radiations (730 nm) and, in contrast, is precluded by red (R) radiations (660 nm) [19, 20].

Cell-genome interaction with the environment may produce different forms: light and gravity determine a growth model; organs or parts of organs that form earlier always remain in the lower part of shoots (which produces the irreversible changes determining the notion of time, age and senescence). The way in which distal parts of branches with their leaves will induce senescence in more basal leaves is inferred on the basis of the fact that mostly FR radiations, which trigger senescence in lower leaves, will reach the lower stratum, through higher and distal leaves that function as a filter absorbing red wave length [21, 22]. The qualitative change of light composition depends on canopy density, which is inherent to the organism and its genetic potential.

4. The source-sink relationship is a senescence-inducing factor

a. During vegetative growth

As from zygote and as cell division generates cell lineages ending in organ formation, modifications of the internal environment take place that involve the source-sink relationship (S-S). A sink is a site with high metabolic activity that supposedly moves nutrients and other organic substances from other sites with lower metabolic activity, or sink.

S-S relationship is easier to analyze in plants, in which a cauline apex generates leaves along the shoot. Under the influence of gravity, the cauline apex grows in an orthotropic fashion; thus, the successive generation of leaves forms an age gradient. The apex and developing leaves are heterotrophic or parasites, at least part of their life, of the lower leaves. Therefore, there is a continuous redistribution of solutes between leaves that have completed their growth (source) and the apex region (sink) that is actively growing.

Although no exhaustive studies have been conducted on the incidence of a sink on senescence of a source, several facts suggest that source senescence is determined, at least in part, by the sink. First, it is evident that leaf growth and synthesis capacity is ruled by the organism. When a tobacco leaf is detached from the influence of the plant, it can increase as much as 10 times its weight, 100 times its nicotine content, with a considerable increase in longevity [23]. Other facts, like compensatory growth of remnant leaves in defoliated plants, also suggest that plant's internal balance limits leaf growth. Both examples show the possible influence of S-S relationship in leaf growth detention.

Leaf shape evolution and reproductive growth are delayed by the suppression of sinks, as it was demonstrated by cutting vegetative apexes and leaves [24, 25]; increased longevity has also been observed [26].

An important contribution has been made with the application of cytokinine (6-furfuril-aminopurine) in leaves of *Xhantium*, which demonstrated that the hormone has the capacity to delay senescence and stimulate protein synthesis [27], to generate sinks in the application zone [23, 28], and to induce senescence in zones adjacent to the area treated with the hormone [23]. These results were confirmed by Leolpold and Kawase (1961) [29] in *Phaseolus vulgaris*, showing that the application of Bencil-Adenine (BA) regulated growth of surrounding leaves and produced senescence in leaves adjacent to the treated ones, since the zones treated with the hormone acted as sink.

The mobilizing effect of nutrients by cytokinines has been repeatedly documented [30 31, 32, 33, among others]. Although other experiments yielded negative results, possibly due to differences in experimental design, there is consensus on the capacity of cytokinine to promote a high metabolic activity, with organic substance synthesis and fixation capacity.

Antisenescence and rejuvenating action of kinetin has been described associated with the activation of protein synthesis, nucleic acids, decrease in proteases and nucleases [34], decrease of lipoxygenase [35] among others; reorganization of subcellular fractions [36], including membranes [37], has also been mentioned.

The influence of actively growing centers on the evolution of the sigmoid function of organisms and senescence seems reasonable because it includes participation of an aqueous medium and of organic solutes and minerals, which are basic requirements for the continuation of growth and cell functioning in different organs. It has been recently demonstrated that fruits of *Lupinus* can synthesize cytokinines and also receive cytokinines coming from movement in conducting tissues [38].

b. During reproductive growth

When reproductive structures of plants are formed, changes in source-sink occur. Nature, location, number, and size of plants will determine competition for water and nutrients, particularly at the expense of the parts that have stopped growing. Water and nutrients will mostly move from the older parts towards reproductive structures: flowers, fruits, and seeds.

Flower and fruit removal delay senescence in the different species studied [39, 40, 41 29, 42 34, 43]. Development of plants with determinate growth supposes that stem cell or zygote loses at least part of its potential as a result of differentiation. Therefore, those sinks which, due to their size and number produce mobilization of diffusible metabolites, may produce rapid senescence phenomena in particular species.

One of the most remarkable cases of the source-sink effect is probably flower pollination. In the long-living flowers of *Phalaenopsis amabilis*, pollination accelerates senescence of petals (source) by inducing ovary growth (sink). Petals exhibit a reduction in dehydrogenase enzymes and an increase in the number and activity of isoperoxidases [44] showing the onset of oxidative processes (Fig. 8).

Source-sink relationship therefore appears as an important element of the factors which, in association with differentiation and organogenesis, may produce senescence not only in leaves but also in the entire organism. The fact that isolated cells and organs under favourable conditions as well as vegetative propagation by cuttings increase longevity with respect to the mother plant supports this idea.

5. Morphogenesis and senescence can be modified by internal conditions

a. Sugars

Sugars are ordinary components of the plant's internal environment and their functions are not restricted to primary metabolism. Besides providing carbon skeletons in different biosynthetic paths, sugars participate in physiological actions related to differentiation of reproductive structures and senescence.

During germination, plants use the energy obtained from organic molecules; at this heterotrophic stage sugars are essential for the supply of chemical energy to cells. At autotrophic stages, sugars generally appear associated with ontogenic morphogenesis. An important number of works [45, 46, 47, 48] have demonstrated that sucrose concentration is a determining factor in

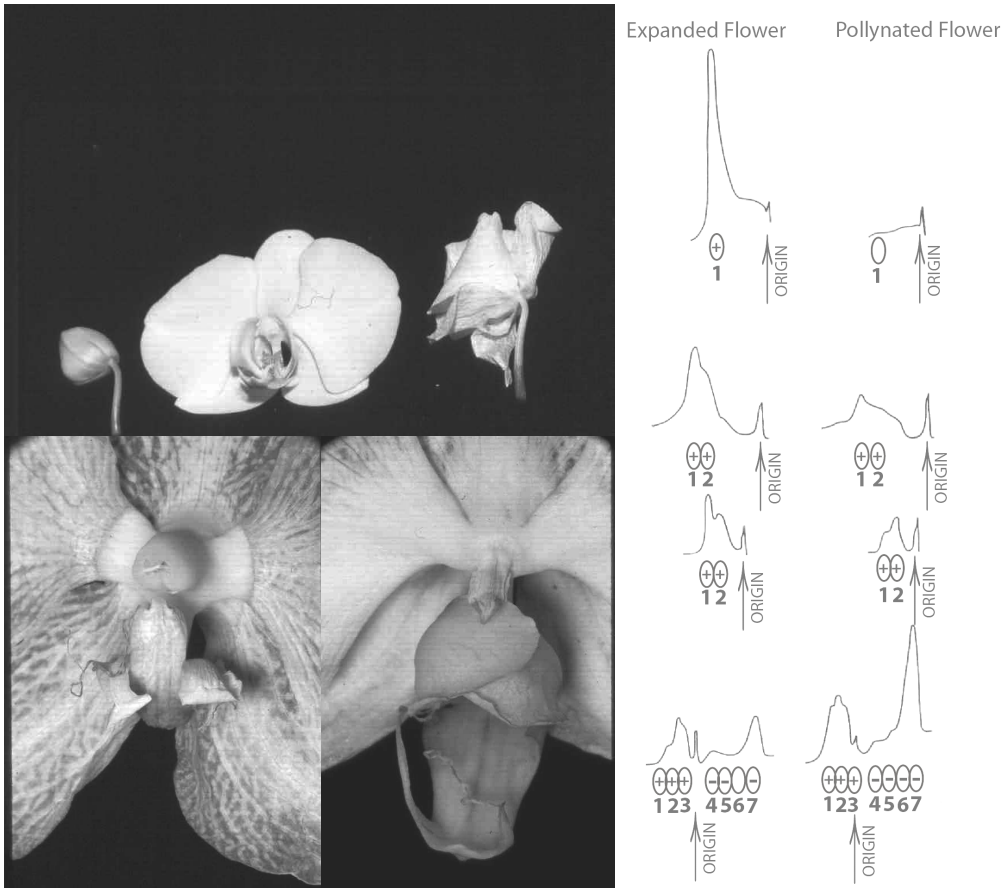

Figure 8. Effects of pollination of *Phalaenopsis amabilis* on senescence of petals (top), on changes in dehydrogenase composition (from top to bottom), Glucose-6-PDH, Malate-DH, Glutamate-DH, and Peroxidases at pollination and 5 days after pollination (adapted from 44). Photos (bottom): pollinated control (left) and ovary treated with cycloheximide.

the step from vegetative to reproductive growth. The results obtained in *Anagallis arvensis* cultivated at 0, 3, 6, and 9% sucrose concentrations show that a shortening of the internodes and a reduction in leaf area occurs with the increase of sugar concentration under light conditions below the compensation point. Flower formation was promoted at 6% of sucrose, ending with fruit formation at 9%, a concentration at which leaf senescence signs were also observed [49] (Fig. 9). The low light intensity that inhibited plant growth was, however, essential for growth and ontogenic morphogenesis generated by sucrose.

The capacity of sugars to determine organogenesis in plants offers great possibilities for the study of ROS participation in differentiation.

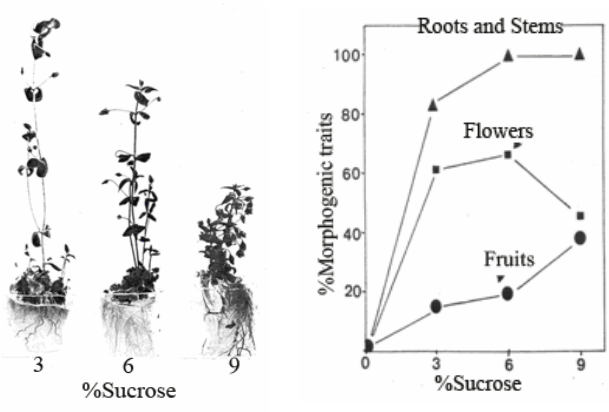

Figure 9. Effects of sucrose concentration on different morphogenic traits, such as internode elongation, formation of roots, shoots, flowers and fruits in *Anagallis arvensis* [49].

Regulation of leaf senescence by sugars shows that sugar effects are different under different light conditions, with heterotrophic behaviour in the dark and autotrophic behaviour in the light. Several authors [50, 51] indicated that sugars delay senescence evolution in dark. The accelerating effect of senescence in the light has been mentioned by Moore *et al.* (1972) [52], as well as its inhibitory effects on root formation. The latter effect may be reverted by applying IAA [53].

Later it was found that sucrose is more efficient than fructose and glucose both in delaying senescence in the dark and in accelerating it in the light. The action of sugars in the alteration of membrane permeability through the formation of hydroperoxides measured as MDA was also confirmed; these phenomena are closely related to activation of lipooxigenase enzyme and loss of catalase activity [54]. More recently, sugars were found to modulate and in some way coordinate the effects of external factors, like light intensity. High sugar concentrations in tissues produce a decrease in photosynthesis and senescence [55]. Sugar signalling at different development stages has been recently evaluated by Rolland *et al.* (2006) [56], and participation in different aspects, including senescence, has been widely documented.

b. Hormones

Hormones are essential elements of the plants' internal environment. Their participation in organogenic processes of development has been widely documented [57], as well as in the control of senescence processes in organs and even organisms [58]. Among the components of the internal environment, cytokinin and ethylne make up the group of hormones that has received more attention in the analysis of senescence processes. Studies on senescence retardation by incorporating genes of the biosynthetic pathway of cytokinins (Isopentenil transferase, *ipt*) and SAG 12 (senescence associated genes, SAG) showed extended longevity of transformants [58, 59, 60]. However, molecular mechanisms involved in the senescence-delaying effects are still unknown [61]. The effects that produce senescence in organs adjacent to the area of cytokinin application have not been evaluated yet.

Studies involving mutants of *Arabidopsis* show that an AHK 3 receptor is required for the senecence-delaying effect of cytokinin to occur, and that phosphorylation of regulator 2 (ARR 2) performed by AHK3 is essential in the control of leaf longevity [62]. Other authors have shown that activity of extracellular invertase is necessary for the cytokinin-mediated retarding effect and that its inhibition also inhibits the cytokinin effect [63].

Other hormones, such as ethylene, ABA, MJ, and SA, are effective senescence inducers in leaves. During leaf senescence, increases in ABA, MJ and SA have been observed and their exogenous application induces SAG gene expression [64, 65, 61]. Noticeably, hydrogen peroxide mediates in the signalling pathways of these hormones and is necessary in ABA-induced senescence [66]. It is also notable that a comparative study of gene expressions produced by environmental stress and by "senescence" demonstrates the presence of numerous common traits, since of 43 transcription factors that are induced during senescence, 28 are also induced by stress factors [61].

Senescence depends on oxidative processes

All the facts mentioned so far-height growth, source-sink relationships or facts occurring in the internal environment by changes in sugars and hormones-suggest that senescence results from stress conditions produced by growth itself. In all cases, whichever the stress condition (drought, oxygen, temperature, heavy metal), it translates into an oxidative stress [67, 68], which has also been observed by other authors and confirmed in all types of biotic and abiotic stress factors [69].

Paramagnetic properties of oxygen are the basis of the formation of active oxygen species [70]. Biological activity and chemistry of formation have been widely documented in different subcellular fractions [71, 72, 73,74] as well as cell control mechanisms that enter into action [75, 76, 77, 78].

ROS participation in cell differentiation and development has been extensively investigated and their participation in senescence processes is practically confirmed.

A genetic control does not seem necessary for senescence and death; instead, genome expression

in such circumstances should be considered an attempt to adapt to the stress conditions the cell is subjected to and as a residual cell function.

The phenomenon of life might be a reaction, whose aim is that of producing more living matter and in which genetic determinism is only involved in species perpetuation.

ROS and their effects - oxidant actions

ROS have different properties. The superoxide anion, the hydroxyl radical and singlet oxygen are all of localized action; their short average life span does not allow their mobilization even inside the same cell where they form and react immediately after formation. As their formation occurs in the membranes, these are usually the most vulnerable targets. Damage might be repaired while the cell conserves a good functioning state (Fig. 10).

Lipid oxidation in membranes is a very sensitive process that may generate other chemical species, such as hydroperoxides, aldhehides, etc., of greater stability and degrading capacity.

The capacity of the superoxide anion to form hydrogen peroxide directly and/or via ROS participation allows the cell to activate other antioxidant resources-the enzyme resources-with the key participation of –SH groups and of the GR enzyme, as well as that of APX that degrades hydrogen peroxide with the intervention of ascorbic acid in chloroplasts and cytosol, whereas its activity is carried out by catalases in peroxisomes [79].

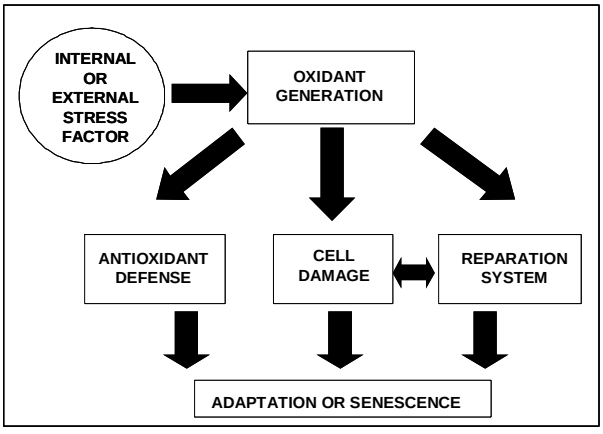

Figure 10. Factors regulating ROS levels in the cells of an organism, leading to adaptation or senescence.

Notably, H_2O_2 is relatively stable; this property must be an essential aspect in the transport of oxidative effects to relatively distant sites. Therefore, ROS, as determinants of senescence, would be versatile enough to act directly *in situ*, or through transport to distant sites.

Stress functions and the internal environment increase ROS during development and lead to self-induced senescence

Stress functions, like increase in mass and volume, height, photosynthetic differentiation, source-sink relationship, etc., as well as variations in the internal environment due to changes in sugar and hormone contents, such as ABA, MJ, ethylene, are directly related to ROS production that gradually increases with development, as organs and organisms reach maximum dimensions and growth detention.

When increasing ROS production during development exceeds a critical level, it produces oxidative damages. Indeed, when ROS exceed homeostatic function, they lead to simple and direct degradation of membranes and other molecules that finally translates into total oxidation of cell compounds (Fig. 11). While stress increases without affecting division capacity, the organisms continue growing until the internal environment prevents cell function and growth stops. This point assumes a ROS level that leads cells to the loss of functions, senescence and death. Stress functions would then produce self-induced senescence.

If we assume that growth, differentiation and organogenesis are different developmental stages, and considering ROS participation in caulinar growth [17], cell wall loosening, development of reproductive structures in fungi, fiber differentiation in cotton [18], it seems reasonable to believe that differentiation occurs at an oxidative level, in which homeostatic capacities permit cell adaptation to a new situation, even maintaining their division capacity. Therefore, oxidations seem to underlie any adaptation event and the oxidative degree could determine differentiation at first, and senescence at a higher oxidative level, beyond adaptation. If oxidative damage is even greater, it would lead to necrosis, a damage that rapidly translates into death without the possibility of homeostasis mechanisms to occur (Fig. 12).

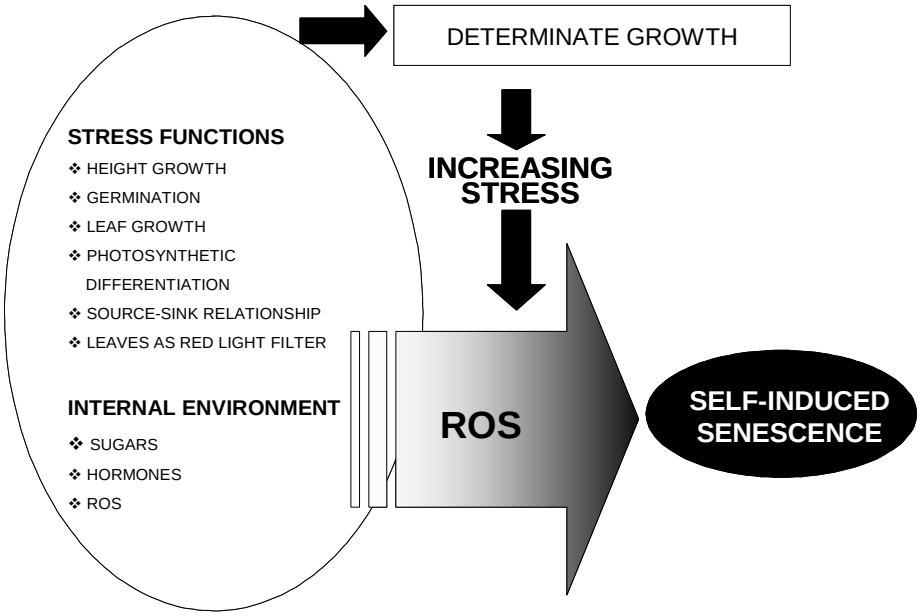

Figure 11. Interaction of genetic determinism and the environment during senescence development. Several stress functions and the internal environment determines increasing stress conditions that produce self-induced senescence.

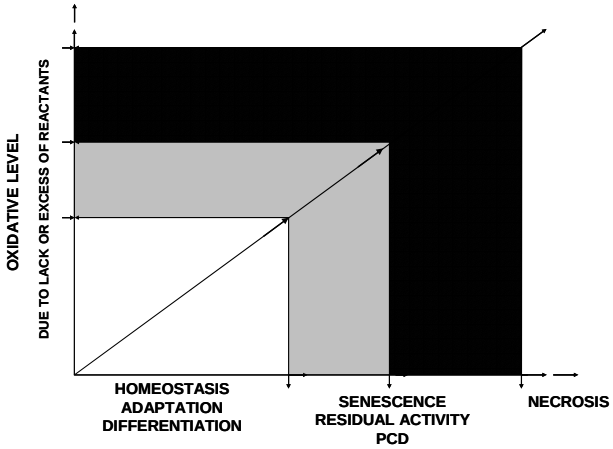

Figure 12. Scheme showing deprivation or excess of reactants (as oxidative level) on the life reaction. During the homeostasis period the cell can adapt its function and differentiation takes place. Later on the cell will show PCD or residual activity or senescence. Necrosis occurs under extreme conditions leading to a fast death due to denaturing effects on the living matter.

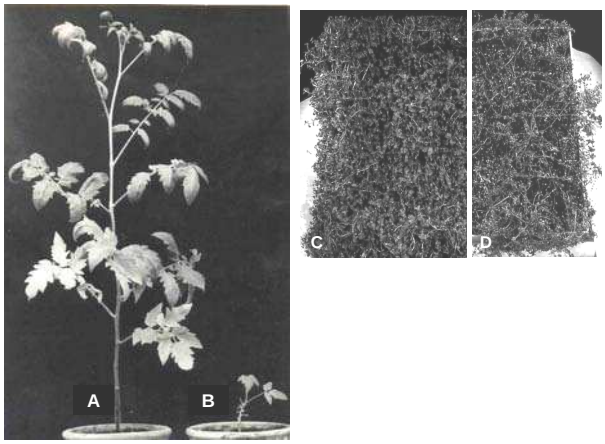

Figure 13. A: Tomato plant (control) and B: plant with regular leaf cutting; notice the delayed heterophilic evolution C: *Anagallis arvensis* in short photoperiod (9h) and D: in long photoperiod (16h), showing senescence symptoms at 120 days.

Why do factors preventing development also delay senescence?

Any growth-delaying action, either on the plant, like flower and fruit removal, or by managing external environment factors, such as light intensity (low) and photoperiod (not inducing flowering) also delays sexual reproduction and senescence of an organism [80, 81, 82, 83] (Fig. 13).

In plants with a strong tendency to evolve towards reproductive growth and senescence, it seems

reasonable to believe that reproductive growth results in a self-induced process, because growth itself (due to stress functions) results in a progressive stress factor. Hence, the different internal (diverse cuttings) and external (low light intensity and non-inducing photoperiod) actions prevent oxidative processes; this, in turn, leads to detention of growth, reproduction and senescence. Accordingly, the development of an organism's architecture becomes a necessary condition to senesce, because full development must show the greatest stress condition to which an organism is subjected.

In plants sensitive to environment action, such as *Anagallis arvensis* and others [80], senescence may be originated also in the external medium by the persistence of light and/or water stress conditions, whose duration and/or intensity turn them into stress factors. It is difficult to elucidate the causes, since both internal and external factors induce oxidative stress and senescence with characteristic progressiveness.

Oxidative stress levels that maintain cell division capacity are related to homeostasis, adaptation and differentiation; whereas levels that determine the loss of cell division capacity are related to senescence.

CONCLUDING REMARKS

A rational theory that explains senescence should consider the two principal actors involved in this phenomenon: the cell, because it codes all cell potentials, and the environment where the cell is located, because it modulates its expression and functioning and/or allows its existence or not.

If we agree with this principle, then it is clear that multicellular organization and its cell growth and differentiation components place the cell in a medium that is less suitable for cell division and indeterminate growth to continue. Only in this way can growth sigmoid function be understood in organisms of determinate growth. Considering that life consists in oxidation and reduction phenomena, the prevalence of reductions must lead to growth; and when growth has altered the internal medium, making it unsuitable, senescence will occur along with changes in age.

Analyzing development and senescence as a process in which the cell will persist, keeping its original traits in a changing medium, will lead us to erroneous conclusions and to confusion of cause and effect. The possible increased production of free radicals with age should be interpreted as a gradual alteration of the internal medium where cells live simply due to a change in the organism size, in relationships between organs and nutrient exchange.

Self-induced senescence suggests that senescence and death are gradual processes and are very often the consequence of limitations occurring in an organ or part of the organism. It also allows us to accept and understand that at certain stages the cell may be totipotent and capable of regenerating the entire organism. The cell stages allowing for transmission of development traits, known as topophysis [39], infectivity of the senescent stage in *Podospora anserina* [84, 85, 86] in yeasts, [87] and rotifers [88] certainly mean that genome-environment interaction finds a more stable intermediary in the cytoplasm that keeps functional-morphological traits capable of replicating while cells maintain their multiplication capacity. Finally it is well known at present days, that genome re-programming needs a cytoplasm quality inducing demethylation and other changes in the nucleus [89, 90].

Pluricellular organism development and senescence could be better understood from cell analysis. Genome allows cell adaptation to environmental conditions where growth and differentiation takes place. Under stressful condition, cell will start developing adaptative process using its capacity and it will die when this homeostatic capacity is overhelmed.

The self-induced senescence is also consistent with the Prigogine physical point of view [91]. The "life as a reaction in only one way", is not explicated by Prigogine, but as an irreversible threat, should explain senescence by stress functions because the reaction depends only on reactant levels and the energy flow in the system.

ACKNOWLEDGEMENTS

This work was supported by grants from CONICET (Consejo Nacional de Investigaciones Científicas y Técnicas-Argentina-PID 8170 and 0370). MM, HRL, and VT, are researchers from CONICET.

REFERENCES

1. Steward, F. C. 1958, Amer. J. Bot., 45, 709-713.
2. Steward, F. C., Shantz, M., Pollard, J. K., Mapes, M. I., and Mitra, J. 1961, Growth induction in explanted cell and tissues: metabolic and morphogenetic manifestations. In Molecular and Cellular Synthesis, The Ronald Press Corp. 193-246.
3. Wangerman, E. 1961, Encyclopedia of Plant Physiology , Ruhland, W., (Ed.), XVI 618-633.
4. Goebel, K. 1908, Enleitung in die experimentelle Morphologie der Pflanzen. Leipzig.
5. Catsky, J. 1962, Biol. Plantarum, 4, 306-14.
6. Catsky, J. 1963, The water relation in plants. Edit. F. H. Whitehead and A. J. Butter Blackwel Sci. Pub. Oxford., 101-112.
7. Steer, B. T. 1971, Ann. Bot., 35, 1003-1015.
8. Al-Ani, A., Leblanc, J. M., Raymond, P., and Pradet, A. 1982, C. R. Acad. Sci., Paris, 295, 271-74.
9. Trippi, V. S., and Guzman, C. 1970, Phyton., 27, 113-124.
10. Pereyra de Altamira, S., and Trippi, V. S. 1991, Agriscientia, 8, 3-7.
11. Kenis, J. D., and Trippi, V. S. 1986, Physiol. Plantarum., 68, 387- 390.
12. Kenis, J. D., and Campbell, W. H. 1989, Plant Physiol., 91, 883-888.
13. Kenis, J. D., and Trippi, V. S. 1987, Plant Cell Physiol., 28, 1307-1312.
14. Trippi, V. S., and De Luca d'Oro, G. M. 1985, Plant Cell Physiol., 26, 1303-1311.
15. Trippi, V. S., Paulin, A., and Pradet, A. 1988, Physiol. Plantarum., 73, 374-379.
16. Trippi, V. S., Gidrol, J., and Pradet, A. 1989, Plant Cell Physiol. 30, 157-163.
17. Rodriguez, A. A., Grunberg, K. A., and Taleisnik, E. L. 2002, Plant Physiol., 129, 1627-1632.
18. Gapper, C., and Dollan, L. 2006, Plant Physiol., 141, 341-345.
19. Sugiura, M. 1963, Bot. Mag. Tokio, 76, 174-180.
20. De Greef, J. A., and Fredericq, H. 1971, Planta., 104, 272-274.
21. Rousseaux, M. C., Hall, A. J., and Sanchez R. A. 1996, Physiol. Plantarum, 96, 217-224.
22. Rousseaux, M. C., Hall, A. J., and Sanchez, R. A. 2000, Physiol. Plantarum, 110, 477-482.
23. Mothes, K. 1960, Naturwis, 47, 337-51.
24. Dostal, R. 1960, Pflanzen. Biol. Zbl., 79, 343-346.
25. Trippi, V. S. 1964, Phyton., 22, 113-117.
26. Neville, P. 1969, Ann. Sci. Naturelles, Bot. Paris 12 S, 10, 301-324.
27. Richmond, A., and Lang, A. 1957, Science, 125, 650-651.
28. Engelbrecht, L. 1964, Umchau, 19, 505-509.
29. Leopold, C. A., Niedergan-Kamien, E., and Janick, J. 1959, Plant Physiol., 34, 570-573.
30. Gunning, B. E. S., and Barkley, W. K. 1963, Nature, 199, 262-265.
31. Seth, A. K., and Wareing, P. F. 1967, J. Exp. Bot., 54, 65-77.
32. Adedipe, N. O., and Fletcher, R. A. 1970, J. Exp. Bot., 21, 968-974.
33. Adedipe, N. O., and Fletcher, R. A. 1971, Can. J. Bot., 49, 59-61.
34. Nooden, L. D., and Leopold, A. C. 1978, Phytohormones and Related Compounds: A Comprehemsive Treatise, Letham, D. S, Goodwin, P. B and T. J. V., Higgins (Ed.), Elsevier, North Holland Biomedical Press, Amsterdam, 329-69.
35. Grossman, S., and Leshen, Y. 1978, Physiol. Plantarum, 43, 359-362.
36. Kursanov, A. L., Kulaeva, G. N., Sueshnikova, I. N., Popova, E. A., Bolyakina, Y. P., Klyachko, N. L., and Borobeva, I. P. 1964, Soviet Plant Physiol., 11, 710-719.
37. Gilbert, M. L., Thompson, J. E., and Dumbroff, E. B. 1980, Can. J. Bot., 58, 1797-1803.
38. Neil Emery, R. J., Qifu, M., and Atkins, C. A. 2000, Plant Physiol., 123, 1593-1604.
39. Molisch, H. 1938, The longevity of plants, Fulling, H., Science Press, Lancaster, Pa.
40. Murneek, A. E. 1932, Bot. Gaz., 7, 79-90.
41. Murneek, A. E. 1926, Plant Physiol., 1, 3-56.
42. Lockart, J. A., and Gottschall, V. 1961, Plant Physiol., 36, 389-398.
43. Wareing, P. F., and Phillips, I. D. J. 1973, The control of growth and differentiation in plants, Pergamon Press.
44. Trippi, V. S., and Tran Thanh Van, M. 1971, Plant Physiol., 48, 506-508.
45. Chouard, P., and Aghion-Prat, D. 1961, C. R. Acad. Sci. Paris, 253, 3864-3866.

46. Paulet, P. 1965, Etude de la neoformation invitro de bourgeons vegetatifs et floraux. These Fac. Sci. Université Paris pp, 1-116.
47. Nitsch, C. 1968, Ann. Sci. Nat. Bot. 12 S, 9 1-92.
48. Deltour, R. 1970, Gbernier Edit Longman, London pp., 416-429.
49. Silvente, S., and Trippi, V. S. 1986, Plant Cell Physiol., 27, 349-354.
50. Marré, E., Schwendimann, M., and Lado, P. 1966, Ren. Acc. Naz. Lincei Cl. Cs. Fis. Ser., 40, 1089-1094.
51. Tetley, R. M., and Thimann, K. V. 1974, Plant Physiol., 54, 294-303.
52. Moore, K. G., Cobb, A., and Lovell, P. H., 1972. 1972, J. Exp. Bot., 23, 65-74.
53. Larrieu, C., and Trippi, V. S. 1979, Biol. Plantarum, 21, 336-344.
54. Luna, C., and Trippi, V. S. 1986, Plant Cell Physiol., 27, 1051-1061.
55. Wingler, A., Purdy, S., MacLean, J. A., and Pourteau, N. 2006, J. Exp. Bot., 57, 391-99.
56. Rolland, F., Baena-Gonzalez, E., and Sheen, J. 2006, Annu. Rev. Plant. Biol., 57, 675-709.
57. Skoog, F., and Miller, C. O. 1957, Symp. Soc. Exp. Biol., XI, 118–131.
58. Gan, S., and Amasino, R. M. 1995, Science, 279, 1986-1988.
59. Ori, N., Juarez. M. T., Jackson, D., Yamaguchi, J., Banowetz, G. M., and Hake, S. 1999, Plant Cell, 11, 1073-1080.
60. McCabe, M. S., Garratt, L. C., Schepers. L., Jordi, W. J., Stoopen. G. M., Dacelaar, E., van Rhijn, and Power, J. B. 2001, Plant Physiol., 127, 505-16.
61. Lim, P. O., Kim, H. J., and Nam, H. G. 2007, Annu. Rev. Plant Biol., 58, 115-136.
62. Kim, H. J., Ryu, H., Hong, S. H., Woo, H. R., and Lim, P. O. 2006, PNAS. USA, 103, 814-819.
63. Balibrea Lara, M. E., Gonzalez-Garcia, M. C., Fatima, T., Ehne, R., Lee, T. K., Proles, R., Tanner, W., and Roitsch, T. 2004, Plant Cell, 16, 1276-87.
64. Morris, K. Mackerness, A. H., Page, T., John, C. F., Murphy, A. M., Carr, J. P., and Buchanan-Wollaston, V. 2000, Plant J., 23, 677-85.
65. Van der Graaf, E., Schwacke, R., Desimone, M., Flugge, U. I., and Kunze, R. 2006, Plant Physiol., 141, 776-792.
66. Hung, K. T., and Kao, C. H. 2004, J. Plant Physiol., 161, 1347-1357.
67. De Luca d'Oro, G. M. and Trippi, V. S. 1987, Plant Cell Physiol., 28,138-139.
68. Luna, C. M., Casano, L. M., and Trippi, V. S. 1997. 1997, Physiologia. Plantarum, 101, 103-108.
69. Buchanan, B. B., Gruissem, W., and Jones, R. L. 2000, Biochemistry & Molecular Biology of Plants, American Society of Plant Physiologists, USA.
70. Fridovich, I. 1991, Molecular oxygen: Friend and Foe. In EJ Pell KL Steffen (Ed). Active Oxygen/Oxidative Stress and Plant Metabolism, Current Topics in Plant. Physiol., 6, 1-5.
71. Hamilton, G. A. 1991, Chemical and Biochemical Reactivity of Oxygen. In Active Oxygen/Oxidative Stress and Plant Metabolism E. J. Pell, K. L. Steffen (Ed). 6, 6-12.
72. Sagi, M., and Fluhr, R. 2006, Plant Physiol., 141, 336-340.
73. Elstner, E. F. 1991, Current Topics in Plant Physiol., 6, 13-25.
74. Thompson, J. E., Brown, J. H., Paliyath, G., Todd, J. F., and Yao, K. 1991, Membrane phospholipid catabolism primes the production of activated oxygen in senescing tissues. In Active Oxygen/Oxidative Stress and Plant Metabolism, E. J. Pell, K. L. Steffen (Ed.), 6, 6-12.
75. Van Breusegem, F., and Dat, J. F. 2006, Plant Physiol., 141, 384-390.
76. Scandalios, J. G. 1997, Cold Spring Harbor Laboratory Press, Monograph, 34, 890 pp.
77. Shulaev, V. and Oliver, D. J. 2006, Plant Physiol., 141, 357-372.
78. Halliwell, B. 2006, Plant Physiol., 141, 312-322.
79. Del Rio, L. A., Sandalio, L. M., Corpas, F. J., Palma, J. M., and Barroso, J. B. 2006, Plant Physiol., 141, 330-35.
80. Trippi, V. S. 1982, Ontogenia y senilidad en plantas., Dirección General de Publicaciones, Universidad Nacional de Córdoba, República Argentina, 547pp.

81. Trippi, V. S, and Brulfert, J. 1971, Phyton., 30, 53-58.
82. Trippi, V. S., and Brulfert, J. 1973, Amer. J. Bot., 60, 951-955.
83. Trippi, V. S., and Brulfert, J. 1973, Amer. J. Bot., 60, 641-647.
84. Rizet, G. 1953, C. R. Acad. Sc., Paris, 237, 838-840.
85. Marcou, D. 1961, Ann. Sci. Nat., 12 Serie., 2, 653-759.
86. Osiewacz, H. D., and Nuber, U. 1996, Molecular and General Genetics, 252, 115-124.
87. Powell, C. D., Van Zandycke, S. M, Quain, D. E., and Smart, K. A. 2000, Microbiologie., 146, 1023-1034.
88. Lansing, A. 1945, Ann. New Yok Acad. Sci., 57, 455-464.
89. Armstrong, L., Lako, M., Dean, W., and Stojkovic, M. 2006, Stem Cells, 24, 805-14. www.stemcells.com.
90. Rasmussen, A. 2003, Reproduct. Biology and Endocrinology, 1, 100-103 Licence Biomed Central Ltd. http://www.rebej.com. Content /1/1/100.
91. Prigogine, I. 2006, El nacimiento del tiempo Tusquets (Ed.), Bs-As 98 pp.