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CURRENT PROBLEMS IN RESEARCH

# Senescence in Plant Development

## The death of plants or plant parts may be of positive ecological or physiological value.

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Of the steps in biological development, senescence is one of the least well defined. In the development of plants the stages of germination, flower initiation, fruit set, and maturation are relatively precise and are subject to relatively simple and concise measurement. Senescence, on the other hand, is a relatively gross change or series of changes, leading finally to the death of the plant. Alex Comfort (*1*), the animal physiologist, has described senescence as a decrease in viability with an increase in vulnerability. In plants we recognize these changes as decreases in growth rates and vigor and increases in susceptibility to challenge by the environment (through shortages of water, nutrients, or other physical comforts) or to challenges by pathogens or physical disturbance.

A characteristic consequence of senescence is the occurrence of death. The patterns of death curves—or, more euphemistically, of survivorship curves—for various biological populations can tell us a good deal about the senescence characteristics of the population. If a population of creatures dies off with a random mortality it will decline at a constant rate. For example, if 50 percent die within one unit of time, 50 percent of the remainder within the next similar period, and so on, this population would not be exhibiting senescence, and the survivorship curve would have the form of curve *A* in Fig. 1. Many populations of animals and some plant populations exhibit such a survivorship curve, or one that is closely similar, either because the members of the population are removed by predatory activities or environmental forces faster than they are removed by the processes of senescence or because the population just does not have the physiological characteristic of senescence. A homely example of a population which shows essentially no senescence would be glass tumblers in a cafeteria (*2*). Any given tumbler is just as likely to be broken today as tomorrow, and the curve of survivorship for such a population may follow exactly curve *A* in Fig. 1.

The survivorship curve for an organism such as man takes the shape of curve *B* in Fig. 1, with a relatively low mortality rate until nearly the age of retirement and then a rapid decline in survivorship. Such a flexing curve, then, identifies a senescing population. This type of survivorship pattern is common to many animals, and again probably to many perennial plants. We can safely assume that there are some internal biological mechanisms which bring about decline in viability and increase in vulnerability in such populations.

The extreme case of senescence would be one in which all the members of a population die at once and in unison as in the abrupt curve *C* in Fig. 1. This situation is essentially realized in nearly all annual species of plants. The sudden and synchronous death of literally millions of individuals of the same population is an ordinary sight in the wheat fields of the Plains states or the cornfields of the Midwest. The signal for mass hari-kari, so to speak, for all members of these enormous populations is such a fantastically dramatic physiological event that it seems most singular that plant physiologists have not given more attention to the matter. Surprisingly enough, this event in the life cycle of plants is not discussed in any of the current plant physiology texts.

## Manifestations of Senescence

In animal species which display senescence there is a reasonable amount of evidence that, with aging, there may be a running out of the metabolic and developmental functions associated with youth. In fact, just recently Strehler and Mildvan (*3*) have presented an interesting analysis of statistical aspects of aging in animals to suggest that senescence may have such a physiological basis, with a progressive decline in many hormonal, enzymatic, and physiological functions leading to decreased viability and increased vulnerability of the organism. In the plant there are several expressions of senescence, including a variety of patterns of morphological deterioration and a characteristic subsidence of growth rates. Instead of a general running-out of the organism, there may be distinctive changes in the morphology, pigmentation, and internal nutrition pattern in the plant.

It may be helpful to try to recognize

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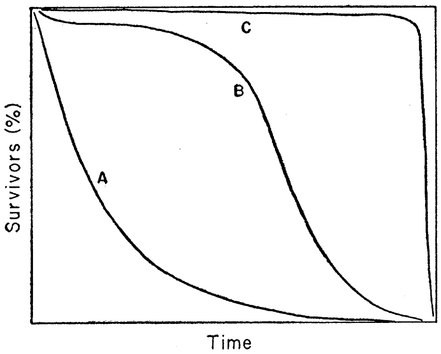

Fig. 1. Generalized survivorship curves for (*A*) populations which do not show senescence; (*B*) populations, such as man, which are more likely to die in the later years of their life-expectancy period; and (*C*) populations, such as annual plants, which die en masse at a rather specific time. The maximum life expectancy is plotted as the extreme right limit of the graph.

the morphological patterns of senescence in plants. There are obvious parallels between senescence of the entire plant and of the organs of the plant. Thus, for example, Fig. 2 illustrates in a diagrammatic way how some species of plants senesce as organisms, the entire individual dying in an abrupt, over-all senescence, whereas in other species only individual organs senesce. The process in the latter species may involve the senescence of leaves and stems (top senescence, as in the case of perennial forbs), or it may involve the annual senescence of leaves only (deciduous senescence, as in the woody perennials), or it may involve only the progressive senescence of leaves from the base of the plant slowly up the stem. The ripening and deterioration of fruits is, of course, a generally occurring organ senescence. The point I want to make is that senescence may involve the entire organism or it may be limited to separate organs. The intergradations between organism senescence and organ senescence suggest that the same physiological signals may be involved, perhaps in varying degrees of intensity. There are, of course, many instances of single gene differences between species which are annuals and those which are perennial forbs or woody, deciduous plants, and the enticing possibility exists that these differences may be genetic differences in the intensities of the senescence signals.

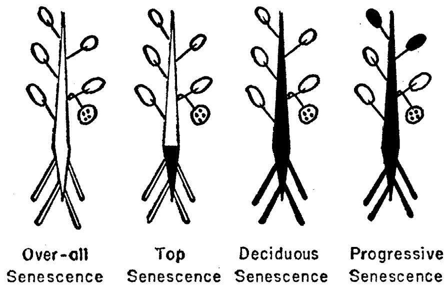

Fig. 2. Schematic representation of some types of senescence found in plants. Open areas, senescing parts; black areas (of leaves, stems, fruits, and roots), nonsenescing parts.

The decrease in viability with senescence is evident not only in the occurrence of death but also in the subsidence of growth rate. Senescence in higher animals is not likely to be associated with a decline in growth rate, for the obvious reason that the animal essentially terminates its growth with adulthood. But the plant, having the essential form of a linear axis with meristems at each end, may grow continually and so is much more likely to reflect senescence in a changed growth rate. The sigmoid growth curve for organisms, for organs of the plant, or for individual cells is relevant to the observation of senescence. The growth curve for an oat plant, for example, follows this pattern, and the onset of the reproductive stages of flowering and fruiting are associated with a decline in and then termination of growth, followed by death of the whole plant. A somewhat analogous pattern is seen for each organ of the plant—for example, the sigmoid growth curve for each leaf up the stem, or the approximately sigmoid growth curve for each fruit, though the correlation of the growth curve with organ senescence is less impressive than the correlation with senescence of the whole plant, since leaves and fruits do not have the terminal meristem type of growth which the whole plant has.

Some further expressions of senescence are found in morphological and pigmentational changes in the plant or plant organs. As plants or their organs senesce there is frequently an associated increase in abscission, and leaves or fruits are shed from the plant. There is generally a major change in pigmentation, with loss of green color and development of the red or yellow colors of ripening fruits or falling leaves. These visible pigment changes are associated frequently with a respiratory peak or climacteric, both in fruits and in leaves (*4*), and with an exhaustive export of nutrients from the ripening leaves. The association of these catabolic functions with the senescence of plants and plant organs suggests, again, the similarity of plant senescence and organ senescence. The frequent association of dormant or rest periods with most types of plant senescence (overall, top, or deciduous) is discussed in a later section.

## Functions of Senescence

In zoological circles senescence is considered to have only negative values —those of sloughing off the older components of populations, limiting the size of the population, and removing, as in the case of man, the individuals with the most learning. The zoologist tells us that natural selection can probably serve only to restrict senescence to a section of the life cycle later than the reproductive phase, hence the frequent correlation of senescence with animal age groups past the reproductive period (*1*). Analogy between the botanical and zoological systems is dangerous here, however, for a little contemplation permits one to see that in many plants senescence has distinct and positive values, in terms of ecological adaptation, natural selection, and efficiency of internal physiology.

Discussion of the occurrence of senescence in various species as a seasonal function helps to illustrate its usefulness in ecological adaptation (Fig. 3). The annual march of the seasons is a timepiece to which the senescence of various species is attuned. The adaptation of many early spring species (such as tulips) to the earliest part of the growing season permits this species to grow freely, without the intense competition which occurs later in the season, and some species limit themselves to this season through the process of senescence—top senescence in the case of tulips. Other species are adapted to somewhat later parts of the growing season. Spring wheat, for example, is so adapted and terminates its growth through over-all senescence on about 1 July in the Midwest. Many species, such as soybean, straddle the middle of the growing season and senesce about 1 September; other species, such as corn, grow best in the warm temperatures of late summer and complete their life cycle just before frost in October. Illustrations could be as easily taken from wild species as from cultivated ones. The point to be made is that senescence may limit the growth of a plant species to a certain part of the growing season. Far from having a negative function, here senescence may enable the species to adapt to environmental conditions such as seasons of cold, drought, or plant competition.

One can argue that the seasonal limitation is an adaptation of the species for a specific time of completion of the reproductive phases of growth instead of an adaptation for the time of senescence. But the reproductive phases and plant senescence appear to be causally related, and the net effect is the advent of plant senescence at a time when environmental features may become limiting—an effect which may be of positive value to the species.

Another effect of senescence of positive value may be its impact on natural selection and hence on evolutionary change. With the long life span of perennial plant species, there is a buffering against rapid evolutionary change. If a species of plant were to exist which did not senesce at all and which was subject to limited mortality, the original population would linger on and on, continuing to reproduce its original genome, and the evolutionary ability of this species to adapt to new environmental changes would be minimal. I assert, then, that in plants senescence is a catalyst for evolutionary adaptability. It is interesting to note in this connection that the most aggressive species of plants, those which adapt most readily to new environmental niches—the weeds—are predominantly annuals, in which over-all senescence imposes a rapid turnover of individuals in the population. If an advantageous genetic change occurs in an annual weed species, it is spread through the population at maximal rate because of the complete turnover of the breeding population each year.

The zoologists have already rejected the suggestion that nonsenescence would retard evolutionary change (*1*, *5*), on the basis that in a nonsenescing population the older components would constitute an ever-decreasing part of the reproductive source and that their contribution to the over-all progeny would thus be relatively small. In plant populations the situation is quite the reverse, for two reasons: the organisms are immobile and the reproductive potential increases with age. For example, in a stand of mature trees, the great bulk of the population is made up of very old individuals, and since they are immobile and there are only a given number of sites on which the trees can grow, newer individuals occupy a very small part of the space and perhaps are a very small numerical component. Even-aged forests may be constituted almost exclusively of old individuals. Furthermore, the numbers of seeds produced by individual trees may increase enormously with age, again warping the genomes of the progeny toward the genetic types of the older individuals. Therefore, it seems that the botanist can reject the concept of noninvolvement of senescence in evolutionary change and embrace the opposite view, that plant species which experience over-all senescence have a relative advantage in evolutionary adaptation in comparison with other species.

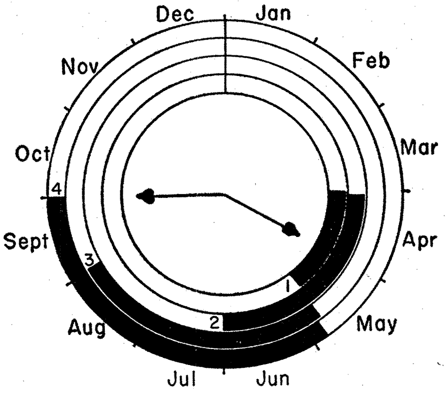

Fig. 3. A schematic clock of annual growing seasons in the Midwest, showing the periods of growth of some sample species and their termination at various points in the total growing season through the imposition of senescence. (1, 2, 3, 4) Approximate date of termination of growing season for tulip, spring wheat, soybean, and corn, respectively. Black areas, growing periods for the respective species.

Organ senescence as well as plant senescence appears to be of positive value to the plant. One can recognize three apparent ways in which organ senescence is beneficial. First, as individual leaves become suppressed and shaded, the onset of leaf senescence permits the recovery of the bulk of inorganic and organic nutrients which have been committed to that leaf. That nitrogenous materials are exported from aging leaves is of course well known. The data of Williams (*6*) illustrates this dramatically in oat plants, for with the accumulation of protein in the fruits there is an immense export of nitrogenous substances from the leaves, as shown in Fig. 4, and even a considerable export to the fruits from the stem and leaves. The retranslocation system is so effective that the oat plant does not need to assimilate any additional nitrogen during the entire last half of its life. So recovery of nutrients from senescing organs constitutes a valuable saving to the rest of the plant. A second benefit derived from organ senescence is the loss of ineffective leaves from the plant body. As leaves age and become shaded and their assimilative properties are imparied with aging, they are not supported as charity cases but, instead, are removed through organ senescence. A third benefit is obtained through the senescence of fruits. With senescence, fruits become more attractive to animals, which assist in the dissemination of seeds; furthermore, the softening of the fruits, or the dehiscence of seams in the fruits, may directly assist in the release of the seeds.

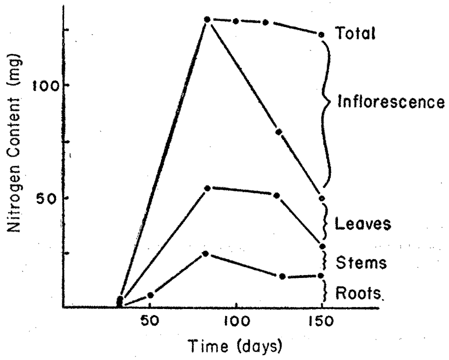

Fig. 4. Changes in the distribution of nitrogen in the oat plant during the growing period, illustrating the mobilization of nitrogenous substances into the inflorescence from the leaves, stems, and roots (*6*).

In cases where senescence is of positive value because it adapts the plant to limited periods of the growing season, there is an associated period of dormancy or rest. For example, the senescence of oats in midsummer serves to adapt the plant to a droughty midsummer season, but the adaptation is contingent upon the ability of the species to have a dormant or rest period. It seems that the ability of a species to have a dormant or rest period provides special opportunities for senescence to be of value to the organism. Perhaps a similar situation exists in some animal species; an example is the presumed senescence of butterflies after the laying of eggs (a dormant stage), which adapts some species to a limited part of the summer season.

In short, senescence in plants appears to serve numerous positive func-

Fig. 5. Deferral of senescence in soybean plants by the removal of flowers and fruits during the growing season (*8*).

tions, in contrast to its role in most animals. Furthermore, senescence has become highly intensified in some species and appears to be a necessary consequence of some positive physiological forces which kill the organ or organism, in contrast to the apparently nonspecific deterioration of the senescing higher animals.

## Plant Senescence

What can we deduce about the physiological forces that underlie the development of plant senescence? The most conspicuous factor associated with plant senescence is reproduction. Of course, many species of plants die as they complete reproduction, and long ago Molisch (*7*) pointed out that the century plant (*Agave americana* L.) is a centenarian only in climates where it cannot become reproductive for 100 years. In some other climates it flowers and fruits in 10 years, and there the century plant is only a decennarian (if I may coin a word). Molisch observed that if he prevented annual plants from flowering and fruiting by cutting them back he could prevent senescence. The deferment of senescence can be illustrated with soybeans by the systematic removal of flowers, which maintains the plants in a green and vigorous condition for many weeks after the normal time of senescence (Fig. 5). Molisch interpreted the association of reproduction and senescence as the mobilization of nutrients into the fruit from the rest of the plant, such a depletion leading to plant senescence (*Erschöpfungstod*). However, if one removes the reproductive organs at intervals from the time of first floral opening until just before fruit ripening, one finds that the signal causing plant senescence is accruing continuously during flowering and fruiting and is not simply associated with the filling of the fruits (*8*). It appears, then, that reproduction forces the plant into senescence, but the mobilization of nutrients into the fruit does not itself seem to account for senescence.

This point of view is further supported by the fact that in the case of dioecious species, the male plant and also the unpollinated female exhibit senescence, and in fact in the cases of both hemp and spinach, the male plants even die before the fruiting female plants.

The removal of flowers from the male spinach plant defers the senescence of the plant to about the same extent as does the removal of flowers from the female plants (*8*). The greatest deterrent to senescence is the removal of flowers before anthesis. Very similar effects are obtained with female plants, where the greatest deferment of senescence is achieved by removing flowers before pollination. The senescence signal coming from the tiny male parts is every bit as strong as the signal from the relatively large developing fruits. In both cases, however, the plants are plunging into a state of senescence, and the removal of flower or fruit only briefly defers their demise.

The flowering of spinach plants involves the development of a large bolt or enlarging flower stem, and perhaps the removal of flowers is only briefly effective in deferring senescence because already the mobilization of nutrients is being accomplished through the enormously enlarging stem. If the mobilization effects of stem growth are responsible for the senescence, then the application of gibberellin to spinach plants kept in a noninductive photoperiod offers a nice chance to test the effect, for application of gibberellin results in a bolting response without any flowering. We have found that spinach plants forced into bolting in this way do not undergo senescence, and that only the bolting plants which do develop flowers experience senescence (*9*).

The development of plant senescence is reflected in the growth curve of each plant, and mention has already been made of the termination phase of the sigmoid growth curve as an event related to the onset of flowering and fruiting. In some species with relatively weak tendencies toward senescence the removal of flowers and fruits defers the termination of growth. This has been nicely demonstrated for the tomato (*10*), a weakly senescing species of the progressively senescing type.

In considering plant senescence and its possible physiological causes, we have recognized reproductive growth as a powerful signal. Molisch interpreted the lethal effects of reproduction as being a consequence of mobilization effects, and yet at present we are unable to quantitatively measure these effects and can only say that flowering and fruiting bring about an intensification of a signal for plant senescence; that this is identical with mobilization effects is not certain. Study of the nature of the signal is greatly needed.

## Organ Senescence

The physiology of organ senescence and the forces which may control it are better understood. Studies by a wide variety of plant physiologists over the past 10 years have provided a rather interesting description of the senescing leaf or fruit. As a leaf grows old, its photosynthetic apparatus appears to become markedly less effective, as indicated by lowered photosynthetic rates and depressed net assimilation rates. Its auxin supply dwindles, and it appears that the cause of this effect is an increase in the enzyme systems which destroy auxins. Pilet (*11*) has even suggested that the deterioration of the auxin regime with age is responsible for organ senescence. As the leaf grows older there are changes in its metabolism of carbohydrates and proteins, and, as already mentioned, there is generally a respiratory climacteric and an associated deterioration of the chlorophyll pigments in favor of the carotenoids and anthocyanins. And as these metabolic shifts take place, there is a gross export of many of the organic and inorganic nutrients from the leaf, until abscission interrupts such traffic.

Are these senescence changes brought about by activities within the aging organ? In the case of fruits, it seems that in most instances they are, for under normal physiological circumstances maturation and ripening of the fruit appear to be triggered by the termination of development of the seed structures or by metabolic activities in the fruit itself. In the case of leaves, however, there appears to be considerable control by the rest of the plant.

The impact of the whole plant on leaf senescence was implied years ago in some observations of Stahl (*12*). He noted that when leaves of some trees had started to yellow, a crack or incision across the leaf would locally defer the yellowing. In a very clever experiment he punched a leaf disk out of a leaf of *Philadelphus* and showed that while the remaining parts of the leaf yellowed and died, the disk which had been removed remained green. Such evidence suggests that senescence in leaves may be a correlation effect—that is, it may be a function which is controlled by physiological events occurring in remote parts of the organism.

In the progressive senescence of organs, the first organ to go is the cotyledon. Its senescence is not simply a running-out or deterioration but is controlled by the apex, as is readily demonstrated by the removal of the apex of bean seedlings at different intervals after germination. In the snap bean, removal of the apex defers cotyledon senescence from the usual 7 days to almost a month. In soybeans, removal of the apex entirely prevents cotyledon senescence. The same apical influence applies to leaves. For example, the primary leaves of beans normally experience senescence at about 40 days of age, but removal of the stem apex at any time before the development of four or five trifoliate leaves appears to prevent quite completely the development of leaf senescence (Fig. 6). Removal of the apex not only defers yellowing and abscission of leaves and cotyledons but permits their continued growth, in a manner roughly analogous to the deferment of plant senescence by removal of the reproductive parts.

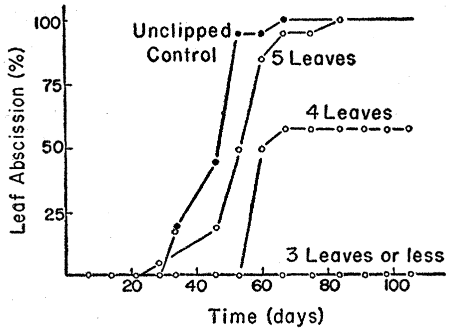

Fig. 6. The abscission of primary leaves of red kidney beans as it is modified by clipping off the plant apex after the development of 5, 4, or 3 trifoliate leaves. Removal of the apex after three leaves or less had developed prevented senescence and abscission of the primary leaves.

In the case of plant senescence I spoke of an increasing signal for senescence. In the case of the leaf, a similar intensification of a senescence signal is provided by the apex during the functional life of the leaf. This has been illustrated nicely by Rubinstein (*13*) in some current studies of abscission. He excised the abscission zones of bean leaves of different ages and measured the effects of stem decapitation on the abscission processes in the excised petioles. He found (Fig. 7) that the tendency to abscise becomes increasingly greater as the leaf ages, as shown by the lessening time interval before abscission in the excised petioles. However, removal of the stem apex when the seedlings were 11 days of age prevented this physiological aging effect. Decapitation at 15 or at 17 days also deferred further increases in the tendency to abscise.

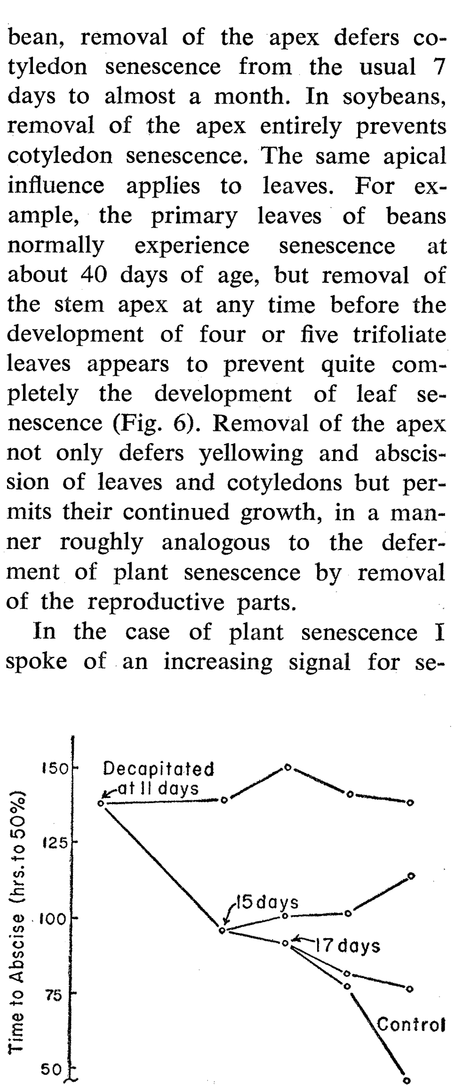

Fig. 7. Changes in the tendency of primary leaves of red kidney beans in the excised condition to abscise, as affected by age and by removal of the plant apex. Explants taken at intervals from day 11 to day 21 show an increase in speed of abscission; removal of the plant apex prevents this increase (*13*).

The influence of the growing point is further evidenced in the observed tendency of leaves of decapitated plants to continue to grow. For example, the cotyledons of tomato seedlings respond to decapitation of the stem by growing to as much as three times their ordinary size.

## Mobilization Effects

The ability of some parts of plants to mobilize substances from other parts has been known for years, though the phenomenon has not received general attention. The abilities of flowers and fruits to mobilize carbohydrates, phosphorus, potassium, and organic nitrogen was described beautifully by Mason and Maskell (*14*). Curtis and Clark (*15*) have observed that mobilizing forces seem to be strongest in flowers and fruits, less strong in growing points, still less strong in lateral buds, weakest in roots. While the means by which such mobilizations occur remains completely obscure, there is good reason to accept the concept that such forces pull materials from one part of a plant to another, and to believe that the reproductive organs of the plants are among the most effectual mobilizing centers.

Some very telling experiments on mobilization in connection with senescence have been carried out recently by Mothes and his group in Germany. In the course of these studies he found that, while excised leaves of tobacco lose their color and senesce rapidly, leaves which are rooted escape senescence entirely (*16*). Extrapolating

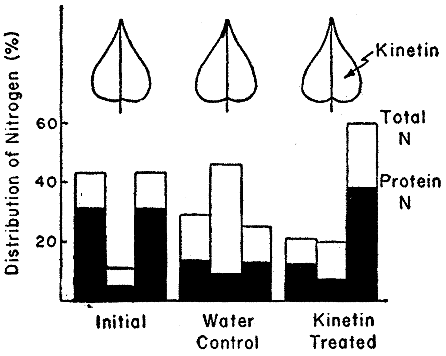

Fig. 8. Changes in distribution of total nitrogen and protein nitrogen in tobacco leaves and the modification of these changes with kinetin treatment. (Left) Initial distribution of nitrogen fractions in the left and right halves of the leaf blade and the central vein; (middle) distribution after 9 days in untreated leaves; (right) distribution after 9 days in leaves treated on one side with kinetin (30 parts per million) (*25*).

to the situation in the plant, he suggested that the chief mobilizing centers in the plant compete for organic nutrients which are synthesized principally in the roots, and hence an exclusive root supply to an excised leaf will avert its senescence. At the time of flowering and fruiting many plants experience a decline in root activity and then the competition by mobilizing centers may become lethal to the entire plant.

Mothes and Engelbrecht (*17*) have made a model to simulate the action of mobilizing centers in the plant, using kinetin as a synthetic mobilizing stimulant. Richmond and Lang (*18*) earlier observed that applications of kinetin to excised *Xanthium* leaves effectively retarded their senescence, measured as color deterioration or as protein degradation. Mothes' group has established that kinetin can actually produce a mobilizing effect by pulling organic nitrogenous materials into the treated area of the leaf. Thus, for example, as excised *Nicotiana* leaves aged, the right and left halves of the leaf blade lost the bulk of their protein nitrogen contents, and there appeared instead a large component of soluble nitrogen components accumulating in the region of the midrib, as shown in Fig. 8. When such a leaf was treated with kinetin on one side of the midrib, there was a mobilization of nitrogen components in the treated side, reflected in an increase in both the protein nitrogen and the total nitrogen fractions.

To determine whether the kinetin effect might be a consequence of a stimulation of protein synthesis, Mothes *et al.* (*19*) applied an amino acid which would not be incorporated into proteins: alpha aminoisobutyric acid. This amino acid, too, was mobilized into the kinetin-treated areas—a finding which led these workers to the conclusion that kinetin was bringing about a mobilization effect first, and that a synthesis of protein might follow the mobilization function. They suggest, then, that kinetin may be a model of the forces that cause mobilization of food materials in plants.

The suggestion that kinetin treatment may alter the effectiveness of ribonucleic acid (RNA) in causing protein synthesis is attractive. In this connection Martos (*20*) observed that the nucleic acids of leguminous cotyledons declined with aging, an observation which has been extended by Oota and his group in Japan. Oota and Takata (*21*) observed that the decline in RNA in aging cotyledons was matched by a rise in RNA in the growing points of the plants, and they proposed that the transport of RNA out of aging organs was an integral part of the development of senescence. Wohlgiehn (*22*) has correlated a high RNA content with the conditions which defer senescence in tobacco leaves, using Mothes' type of experimental approach.

The opportunity to examine at the level of modern biochemistry the phenomenon of mobilization actions in plants is now at hand. While there is not yet an explanation of these effects, there is a suggestive implication of an association of protein synthesis and the RNA components of organs with mobilization effects.

It is interesting to speculate as to whether such biosynthetic units as these may hold at least a partial explanation of the mechanism of plant senescence.

## Conclusions

The phenomenon of senescence achieves its most dramatic expression in plants, both as over-all plant senescence and as organ senescence. I believe that this important step in the life cycle of plants is, or can be, a positive force in the ability of the plant species to adapt to limitations of the environment, either climatic or competitive, and that senescence **can** be a strong factor in the evolutionary adaptability of plants.

While the mechanism controlling the more or less sudden onset of plant senescence is obscure, descriptive experiments have established that there are signals from the flowers and fruits which trigger the onset of death. A somewhat parallel situation has been described for the signals controlling the organ senescence of leaves, signals, which in this case come mainly from the stem apex. There is a growing body of evidence suggesting that mobilizing forces are involved in the senescence signals, and in the case of leaf senescence these can be mimicked, at least, through the application of kinetin (*23*).

It is high time that we should understand more exactly what the physiological cause of plant senescence may be. We are still unable to adequately answer the classic question posed by St. Paul to the Corinthians almost 2000 years ago (*24*): "Oh death, where is thy sting?"

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