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Evolutionary and Demographic Approaches to the Study of Whole Plant Senescence

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I. Introduction

There have been two distinct definitions of "plant senescence" which have developed within the literature. First, physiologists and cell biologists use the term senescence to describe the continual turnover of cells and plant parts that occurs within an individual as part of an internally controlled program of development. In cases of monocarpy (semelparity), this program can be responsible for the death of the whole organism. The details of this program of "physiological senescence" within individuals are addressed in the other chapters of this book. The second, alternative approach to senescence is termed "evolutionary senescence" and it addresses theories and experimental evidence explaining variation in mortality patterns among individuals within populations and between species. Senescence, as viewed by most animal and evolutionary biologists (see Chapter 1), is a decline in age-specific survival and reproduction with advancing age. The evolutionary theories of senescence are designed to explain *why* this senescence occurs in most species, and to explain the variation in the rates of evolutionary senescence between different species. It is this, evolutionary, population-level, approach to senescence that will be considered in this chapter. This chapter will apply a demographic approach to senescence that has traditionally been used exclusively by animal biologists and theoreticians, to the study of plants and the determination of mortality patterns and longevity.

From an evolutionary perspective, the phenomenon of senescence presents a paradox: Why should a trait that causes an individual to have an increased probability of dying with age persist in a population? The theory of evolution by natural selection suggests that heritable traits that improve the survival and reproduction of individuals should spread through a species because of their higher rates of transmission. We know that there is genetic variation in mortality patterns and longevity in populations. In classic studies with the fruit fly, *Drosophila melanogaster*, for example, researchers have successfully used artificial selection experiments to extend the life span and reduce rates of senescence (cf. Rose, 1984). This large degree of genetic variation in life span is expected in most species, and these results suggest that natural selection could potentially act on this genetic variation to change the time of onset and rate of senescence. Moreover, we also know that there are large differences between species in the rates of senescence. Birds, for example, generally live longer and have a lower rate of increase in mortality with age, in other words a lower rate of senescence, than mammals of comparative size. Some species are thus more effective at preventing or repairing damage than others are. The presence of genetic variation for senescence both within and between species needs to be explained from an evolutionary perspective. The objective of this chapter is to present the study of whole plant senescence within an evolutionary and demographic context. In the first part of this chapter the theories which have been proposed to explain the evolution and persistence of senescence will be discussed, and experimental tests of the theories will be evaluated. To study senescence at the level of the whole plant, demographic evidence for a decline in mortality and reproduction with age is essential. In the second part of this chapter, demographic evidence for senescence in plants will be evaluated and the techniques and problems that are unique to demographic studies of whole plant senescence will be discussed.

II. Evolutionary Approaches

A. Theories of Senescence

Both physiological and evolutionary senescence show genetic control, yet the details of our understanding of senescence at these two levels is very different (Bleecker, 1998). Physiological studies have demonstrated that within an individual plant, there is a genetic program which results in an orderly degenerative process leading either to the reabsorption of nutrients and the abscission of plant parts, or in the case of semelparous species, to the death of the whole organism (Noodén, 1988). Physiological senescence of plant parts is an active process that requires energy and protein synthesis and often involves the redistribution of nutrients and photosynthates. It is a beneficial process which increases an individual's chance for survival, for example by preparing a plant for harsh environmental conditions (e.g., winter), or by allowing a plant to shed unnecessary and inefficient structures.

Genetic control of evolutionary senescence is clear from the fact that there are species-specific differences in maximum life span and from the inheritance of longevity traits within populations. An evolutionary approach to the study of senescence seeks to understand the ultimate reasons why senescence occurs, and how we can explain variation in the rates of senescence between species. Given that there is genetic control of evolutionary senescence, it is interesting to ask why there has not been selection for increased longevity and perhaps even infinite life span. Instead, this phenomenon of senescence, which increases the probability of dying with increasing age, persists in populations and because of this, it is often considered to reflect a failure of natural selection to act against a deleterious trait.

The paradox of senescence from an evolutionary point of view is that, at the level of the individual, there is no scenario in which an increased rate of dying with age can be considered an adaptive trait, in other words a trait directly favored by natural selection. Historically, the one direct "adaptive" theory of senescence in the animal literature suggested that senescence was a mechanism to eliminate older, weaker, individuals from a population in order to conserve resources for younger more vigorous individuals (Weismann, 1889). Within the plant literature, it has been suggested that mortality will be selectively advantageous for an individual if it would result in a better chance for the immediate colonization of its offspring (Wilson, 1997). These hypotheses rely on the principles of group selection, and they can be shown to be theoretically improbable given that selection acts primarily at the level of the individual (Charlesworth, 1980).

Evolutionary theorists have alternatively suggested that senescence may be a non-adaptive trait which has evolved indirectly as a consequence of a selective premium on genes with favorable effects on survival or fecundity early in the life history. The key element to allow the evolution of senescence is the fact that the force of natural selection on survival and fertility necessarily decreases with age because organisms have a nonzero chance of dying from external causes such as predation and accidents. Medawar (1952) provided the foundation for the major theoretical progress on the evolution of senescence. He suggested that, even in the hypothetical complete absence of senescence, if constant fertility is assumed, the reproductive output of each age class declines with age, because survivorship from birth is a decreasing function of age. As a result, the relative importance of traits expressed at late ages, to the lifetime fitness of an individual, is less than the importance of traits expressed at earlier ages. This implies that the intensity of selection will be stronger on age-specific genes acting early in life, than it will on genes that effect traits later in life. Some genes, which have their effect at very late ages, after an organism is usually dead, can completely escape the influences of selection. The first direct experimental test of Medawar's hypothesis is presented later in this chapter (Section IIB).

With this major assumption, that natural selection is weaker at later ages, two major evolutionary theories, mutation accumulation and pleiotropy, have been proposed to explain the evolution and persistence of senescence in populations. The evolutionary theory of mutation accumulation suggests that due to the decline in the strength of selection on age-specific characters, mutations with a deleterious effect at the end of the life cycle will accumulate, resulting in a decrease in age-specific mortality and fecundity with increasing age (Medawar, 1952). In other words, because they will be effectively neutral, genes with deleterious effects will spread in the population because they effect only late-life traits. This type of mutation accumulation is distinct from the somatic mutations that may accumulate during the life of an individual. It refers instead to the large number of deleterious genes that are expressed at late ages and which persist in the population as a result of a change in the mutation–selection balance. The accumulation of mutations is thus due either to increasingly ineffective selection against recurrent deleterious mutations, or from genetic drift.

The second major evolutionary theory, antagonistic pleiotropy, suggests that genes effecting traits negatively late in life may get established in a population if those genes also have positive effects at an earlier age (Williams, 1957). This theory is based on an optimality approach in which late-life performance is sacrificed for early survival or reproduction (Partridge and Barton, 1993). A closely related theory, the disposable soma theory, is a variation of the pleiotropy theory and incorporates a trade-off between reproduction, on the one hand, and maintenance and repair, on the other (Kirkwood and Holliday, 1979). This

theory suggests that if there are a limited amount of resources available to the organism, then the allocation of energy to early reproduction will be favored at the expense of somatic maintenance and repair. Repair will consequently occur at a sub-optimal level leading to senescence. The lack of repair leads to an accumulation of damage to somatic cells and an increased probability of death at later ages. One of the primary assumptions of the disposable soma theory is that there is an early separation of germ line from the soma. This separation does not occur until just prior to reproduction in plants, yet as will be discussed later in this chapter, a violation of this assumption does not appear to be an important factor in the evolution of senescence.

The evolutionary theories of senescence suggest that the decline with age in reproduction and survival is the result of the failure of natural selection to act against genes with late age-specific effects (Charlesworth, 1980). The common thread of the evolutionary theories is that senescence is a consequence of a selective premium on genes with favorable effects on survival and fecundity early in the life history. The theories suggest that senescence is a trait that has evolved because of the absence of selection against degenerative changes in old age, or because of positive selection for mutations that increase early success even when they have later deleterious effects. There are several lines of evidence that can be used to evaluate the theories. First, the assumptions and predictions of the theories can be directly tested through experimental manipulation. Secondly, evidence needs to be obtained from demographic studies to determine whether there is, in fact, evidence for a change in mortality with increasing age, and to determine how species differ with respect to their mortality patterns. Both of these approaches will be used in the remainder of this chapter.

B. Tests of the Theories

1. Medawar's hypothesis

At the foundation of the evolutionary theories of senescence are the theoretical predictions of Medawar (1952). He suggested that in a natural environment a population would experience a high level of random, age-independent, mortality due to harsh environmental conditions. As a result, there is an ever-decreasing proportion of individuals that survive to reproduce at older ages. Harmful genes whose time of expression is beyond the natural range of life span for a particular species, can accumulate in a population with little or no check because the effect of mortality from external causes will be to reduce the force of selection in each successive portion of the life span. As a result, natural selection is ineffective at preventing an accumulation of deleterious genes that affect late-ages because they have a minimal impact on the population. Medawar concluded that aging is the result of the cumulative expression of deleterious genes in individuals that live longer than the average life span of the species in its natural environment. He expanded this further to suggest that in populations removed from their natural environment, a larger percentage of individuals will live to relatively late ages, and it is only under these circumstances that the expression of late deleterious genes would be evident and senescence could be observed. A direct test of Medawar's hypothesis would be to compare the demography of a single species under natural conditions and under artificial conditions in which life span was artificially extended. The previously unpublished study described here is the first direct test of this hypothesis.

The objective of this experiment was to evaluate the mortality of a single species in its natural environment and under idealized conditions. The experiment was done with *Rumex*

hastatulus, a dioecious, wind-pollinated, weedy colonizer of disturbed sites, which is known to show substantial variation in individual life span from annual, to biennial, to short-lived perennial (Radford *et al*., 1968). For the field experiment, the objective was to grow the plants under the same local conditions under which selection had been acting and had shaped the life history. The observed mortality pattern would then be an accurate assessment of natural field mortality. For this experiment, 646 three-week old seedlings were planted into the field, 20 cm apart, and except for an initial mowing, immediately prior to planting, the natural vegetation was undisturbed. For the experiment in the greenhouse, the objective was to grow plants under idealized conditions and to increase the proportion of individuals in the population living to late ages. To do this, there were two treatments. In the first, undisturbed census treatment, 637 plants were grown in individual pots, and watered and fertilized on a regular basis. In the second greenhouse treatment, 50 plants were inhibited from flowering by removing any flower buds from the base of the plant as soon as they appeared. This latter treatment was used to assess the effects of reproduction on mortality under these idealized conditions. The field and greenhouse populations were all censused monthly until every individual had died, and the mortality patterns of the different populations were contrasted.

The results of these experiments show that survivorship in the field and in the greenhouse was very different (Fig. 23-1). The mean life span in the field was 146 ± 80 days. Only 4 of 646 individuals survived until the second growing season and only one of these individuals flowered. Mortality was high, constant, and age-independent, in this experimental field population. This pattern is consistent with Medawar's conjecture about species in their natural populations. A similar high rate of mortality was observed for a population of natural seedlings of this species, which were marked in the field following emergence (Roach, unpublished).

Survivorship in the greenhouse was very high: 84% of the population survived to the day of first flowering during the second growing season (day 350). Yet, post-flowering survival

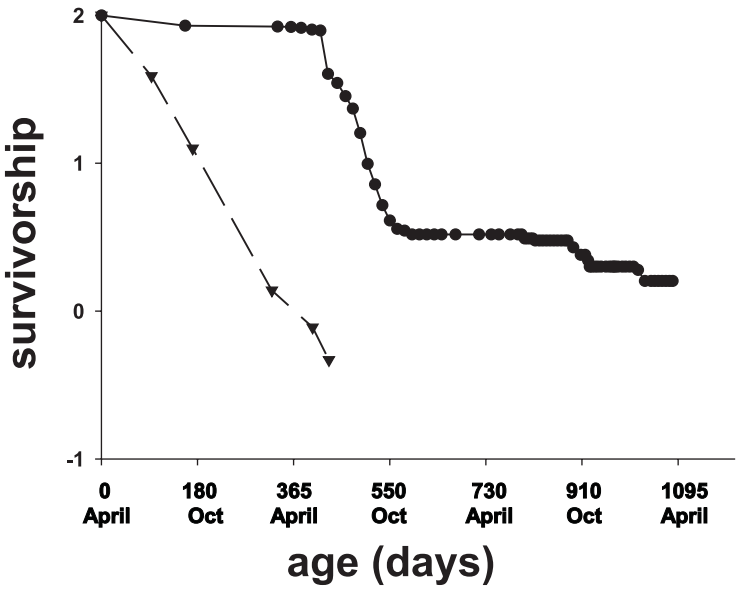

Figure 23-1. Survivorship curves (log percent) for *R. hastatulus* field (triangles) and undisturbed greenhouse (circles) populations.

was very low; only 9% of the undisturbed treatment plants were still alive post-flowering (day 525). Clearly, the developmental shift from vegetative to reproductive growth results in an increase in mortality. This may be an example of a pleiotropic trade-off between reproduction and survival, but the genetic correlations between these traits could not be measured with this experimental design. The mean life span in the undisturbed treatment in the greenhouse was 444 ± 259 days. There were no differences in mean life span for male and female plants that survived to flower.

The survival curves (Fig. 23-1) clearly show that, as predicted by Medawar, a larger proportion of the population in the greenhouse survived to later ages than in the field. Despite the fact that survivorship curves have traditionally been used in plant demography studies, it is in fact difficult to discern the pattern of mortality by looking at changes in the slope of a survival curve (Carey *et al*., 1992). Thus, in order to evaluate mortality patterns for the two greenhouse populations a plot of the age-specific mortality was calculated (Fig. 23-2). The mortality curve for the undisturbed treatment shows a mortality increase following reproduction and then a decline. There is a second, lower, increase in mortality following reproduction during the third growing season. For the 50 deflowered plants, 90% of the individuals were still alive at the post-flowering period during the second growing season. The mean life span for the non-flowering plants was 1064 ± 589 days. At the end of the third growing season, both populations showed a steady increase in mortality with increasing age of the plants, and there were no differences in mortality patterns for the undisturbed and non-flowering treatments.

The evidence from this study suggests that there may be multiple mechanisms that cause senescence in *R. hastatulus*. In a natural population, if random environmental factors do not cause a constant rate of mortality, then senescence may be manifest following the developmental shift to reproduction. If, on the other hand, flowering is inhibited, or plants

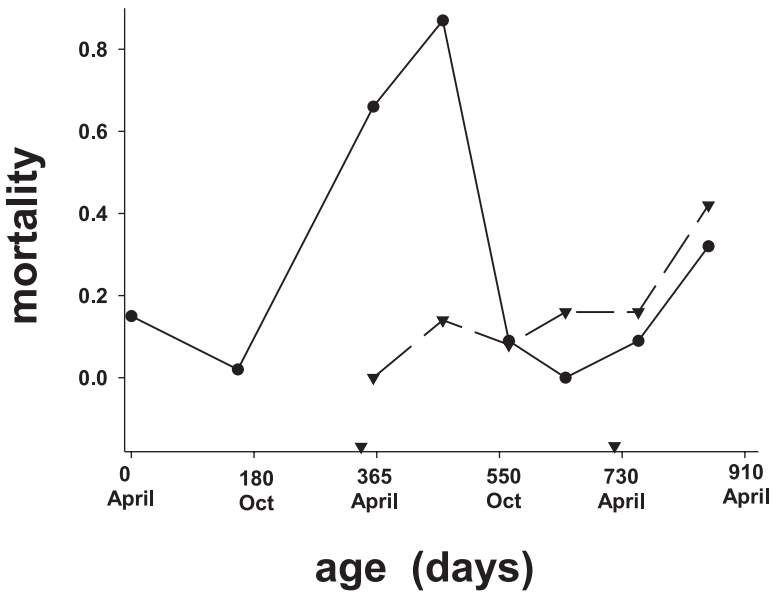

Figure 23-2. Mortality curves for *R. hastatulus* undisturbed (circles) and deflowered (triangles) greenhouse populations. Triangles on the age-axis designate time of reproduction.

survive flowering and live to extremely late ages, another type of senescence may still occur but for different reasons. The experimental manipulations show that the late-age increase in mortality after three years of growing in the greenhouse was not linked to reproduction, because there were no differences in the mortality rates between the reproducing and deflowered plants. There are two possible explanations for the late-age mortality increase for these greenhouse populations. First, it is possible that there may have been an environmental change caused by a cumulative deterioration of the soil environment. The plants were grown in the same small pots for the entire experiment. There was no visual evidence that the plants had become pot-bound (pers. obs.) but there may have been a deterioration of the soil environment, caused possibly by an accumulation of root pathogens or other soil microbes, which may have caused an increase in mortality over time and age. Long-lived plants grown in the field may also experience a deterioration of their immediate soil environment. Experimental work with *Krigia dandelion*, for example, showed that there can be a negative feedback between species and their soil communities, which can result in reduced survival (Bever, 1994). Changes in mortality caused strictly by a progressive deterioration of the environment are not considered evidence for senescence. However, alternatively, the increase in mortality at the later ages may be due to deleterious genetic traits expressed at ages not normally attained by this species. The increases in mortality in the greenhouse plants occurred when the individuals were between 500–1100 days old. The mortality rates from the field study clearly show that few, if any, individuals could be expected to live under natural conditions to these extreme ages. In this case the increase in late-age mortality would be due to the expression of genes with deleterious effects at late ages, which have persisted in the population because they have not been exposed to strong selection. In order to test this hypothesis more vigorously, one would need to measure the changes in additive genetic variance in life history traits with age (Charlesworth, 1990).

2. Evidence for selection on patterns of senescence

The best test of the predictions of senescence would be an experimental change in late-age mortality and reproduction in response to a change in age-specific selection. Whereas these types of selection experiments have never been directly done with plants, there is indirect evidence from comparative intra- and inter-specific studies of plants growing under contrasting environmental conditions. These studies support the hypothesis that patterns of senescence have been, and can be, molded by natural selection. The theoretical prediction is that senescence rates change in a predictable fashion when there are long-term external environmental conditions that increase or decrease survivorship. Furthermore, it suggests that the evolution of species life span differences is explained by the evolution of senescence. The intensity of selection on early performance varies with ecological circumstances, and this leads to variation in the rate of senescent decline in different species. An externally imposed increase in survivorship will favor the evolution of postponed senescence. Conversely, when the level of externally imposed mortality is high, species that invest in rapid growth and reproduction have a selective advantage over species or individuals that invest in prolonged growth and perhaps never have an opportunity to reproduce. A high level of environmental mortality should be associated with short life span and a rapid rate of senescence.

Testing the effect of exogenous mortality on rates of senescence is difficult in a natural environment, because the variation among populations in exogenous mortality must be

separated from any other possible evolutionary differences in the life histories among the study populations (Partridge and Barton, 1996). To date, the best examples of the influence of external environmentally imposed mortality patterns on life span and mortality rates in natural plant populations are two studies, one with *Poa annua*, and one comparative study with two species of *Lobelia*. In what has now become a classic example of the comparative effect of exogenous mortality under natural conditions, populations of annual meadow grass (*P. annua*), from frequently disturbed meadows and less disturbed meadows, were compared in a common garden experiment (Law *et al*., 1977; Law, 1979). The results of this experiment showed that seeds derived from plants from the frequently disturbed sites showed higher early reproduction but shorter life spans and higher rates of mortality. Whereas the design of this experiment did not eliminate the possibility that there may have been correlated environmental variables which selected for different life histories in the disturbed and undisturbed meadows, the experiment was sufficiently large to suggest that the results describe underlying genetic differences between the populations found in these two environments.

In a second study with two species of *Lobelia*, one semelparous and one iteroparous, Young (1990) found that the different life histories of these species could be attributed to demographic variation between sites caused by different environmental conditions. In the drier sites, where there was a higher risk of mortality, selection favored an annual, semelparous life history. Under these extreme environmental conditions, individuals flowered so infrequently, and suffered such high mortality between reproductive episodes, that the probability of longer life span and future reproduction was outweighed by the greater fecundity associated with semelparity. Both of these comparative studies show that, as predicted by the evolutionary theories, there can be differential evolution of mortality patterns, either within species or among closely related species, in response to different levels of exogenous mortality. In other words, genetic differences between populations in patterns of senescence can evolve in response to different levels of age-specific selection caused by external environmental factors.

III. Demographic Approaches

A. Distinguishing Age and Environment

In order to demonstrate a change in mortality patterns with age, studies on the evolution of senescence require, first and foremost, an accurate accounting of the age-specific mortality of individuals. This requires that marked individuals be followed for their entire life span. In one of the few studies which has documented the age-specific mortality from emergence to death for a perennial plant in its natural environment, Canfield (1957) found evidence for increased mortality with increasing age in several tussock range grasses (Fig. 23-3). This study provides excellent data on a long-lived iteroparous species, and it was the first study to document a senescence-like increase in mortality with age (Harper, 1977; Watkinson, 1992; Roach, 1993). It should be noted however, that one of the major difficulties associated with studying mortality patterns in natural populations is that there may be large season- or environment-dependent increases in mortality, and it may be difficult to distinguish endogenous causes of mortality due to senescence from these exogenous changes over time. In Canfield's study, the increase in mortality over time may have been due to the fact that the external environmental conditions became less favorable for survival. It is not

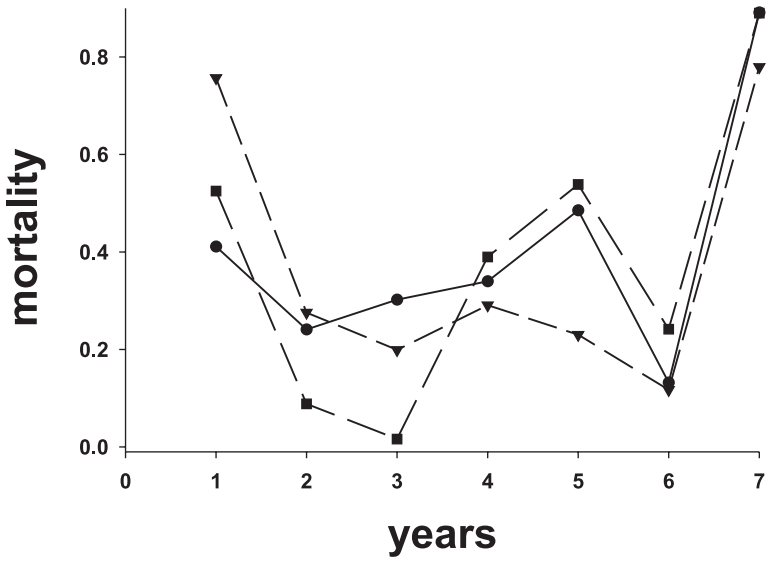

Figure 23-3. Mortality curves for three perennial range grasses: *Bouteloua hirsuta* (circles), *B. chondrosiodes* (squares), and *B. eriopoda* (triangles). Data derived from Canfield (1957), ungrazed treatments.

clear whether the increase in mortality was due to the increasing age of individuals in the population, or to a deterioration of the extrinsic environment. In order to definitively show that senescence is occurring in natural plant populations, experiments need to be designed with the proper controls to distinguish these different causes of mortality.

In order to differentiate between age- and environment-dependent changes in mortality, an additional cohort, of a different age, but experiencing the same environment, is needed. In the first study of this kind, an experiment was done with *Ipomea purpurea* (common morning glory) an indeterminate annual, which continues to grow, flower, and set seed, until an exogenous climatic event, i.e., frost, causes the death of individuals at the end of the growing season. This study was designed to determine: (1) whether there was a change in mortality patterns within the growing season prior to the abrupt change in mortality due to environmental deterioration; (2) whether there was a change in reproduction within the growing season; and (3) whether changes in mortality and reproduction were due to increasing age of the individuals in the population or to changes in the environment. In order to distinguish age- and environment-dependent changes, two plantings were made six weeks apart. In this species, nearly all seeds that are planted at the same time germinate synchronously; thus the two plantings created two cohorts that differed in age by six weeks. Given that the plants from the two cohorts were grown together in the same field, any differences in cohort performance could be attributed to age-specific variation, rather than to changes in the environment over time. Plants were censused regularly for mortality, and mature seeds were collected every three days. All plants that were still alive in mid-November were killed by a hard frost.

The comparative analysis of the mortality patterns of the two cohorts demonstrates a senescent decline in the oldest cohort during the growing season (Fig. 23-4). As seedlings, each cohort showed an increase in mortality after which mortality decreased. As the growing season progressed, the older cohort showed an increase in mortality, much higher than the

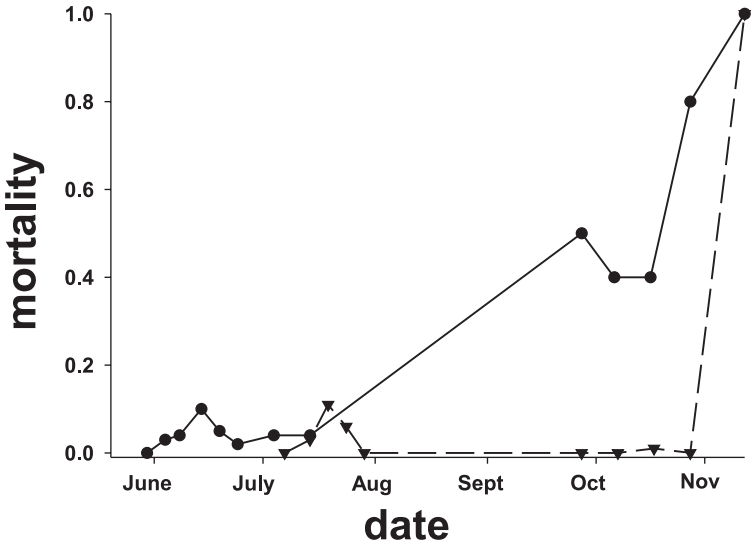

Figure 23-4. Mortality curves for two different-aged cohorts of *Ipomea purpurea*. Circles represent cohort 1; triangles (with dashed line) represent the later planted, younger, cohort 2. All remaining plants died in mid-November following a hard frost.

younger cohort, suggesting that it was age rather than changes in environmental conditions which were causing these mortality patterns. The younger cohort may have shown a similar increase at a later time if the populations had not been killed by the frost at the end of the growing season.

Senescence may also be demonstrated through a decrease in the number or quality of offspring produced with age. In plants, seed weight is a major determinant of offspring quality (cf. Wulff, 1986). Changes in seed mass over age and time were compared for the two morning glory cohorts. At the end of the growing season, when both cohorts were producing seeds, there was a significant decline in mean seed mass for both cohorts (Fig. 23-5). This decline in offspring quality was due to a decline in the quality of the environment as the growing season progressed and concurrently as the plants aged. However, when a comparison is made across the two cohorts, there was a greater decline in seed mass in the older cohort. This difference is manifest as a significant difference between the two cohorts in the slope of the regression of individual seed mass on date (cohort 1: slope $= -0.11$; cohort 2: slope $= -0.03$). During this same period, there was no evidence for a trade-off between seed mass and seed number. The younger cohort showed an increase in the total mass of all seeds produced per day (slope $= +0.83$, $p < 0.0001$), and a positive correlation between seed mass and seed number ($r = 0.11$, $p < 0.02$). The older cohort showed no significant change in total seed mass per day during this same period (slope $= -0.10$, $p > 0.10$) and no significant correlation between these two traits ($r = 0.02$, $p > 0.5$). This analysis shows clear evidence for a senescent decline in reproduction. The results of this study with an annual morning glory show that even within short-lived semelparous species, there may be age-dependent variation in mortality and reproduction. Yet, it is only with the proper experimental controls, in this case a set of different-aged cohorts, that we can distinguish age-dependent and environment-dependent patterns.

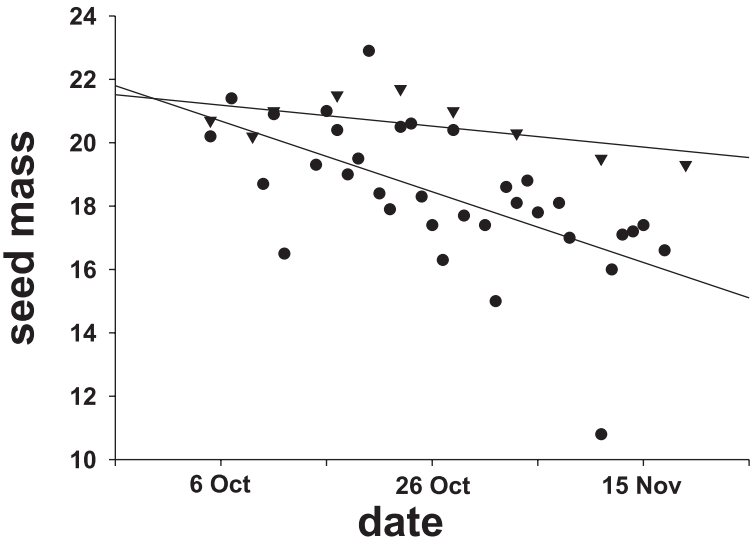

Figure 23-5. Changes in mean seed mass over time for two different-aged cohorts of *Ipomea purpurea*. Cohort 1 (circles) regression: $y = -0.11x + 51.9$ ($r^2 = 0.37$). Cohort 2 (triangles) regression: $y = -0.03x + 30.4$ ($r^2 = 0.38$).

B. Unique Features of Plants

1. Growth forms and theoretical expectations

A unique feature, distinguishing plants and animals is the indeterminate growth that takes place in the meristems of plants. Meristem organization may be an important factor determining variation in life span and mortality patterns of different species. Variation within the plant kingdom ranges from unitary organisms to clonal species. A plant species with a unitary organization and determinate growth is expected to follow demographic patterns similar to model animal systems such as *Drosophila*. On the other hand, plants with unitary organization and indeterminate growth, or clonal species with more modular construction, will have multiple meristems, and this can have important implications for population dynamics. Production of new meristems may allow a clonal species to escape determinacy and may facilitate an escape from whole organism senescence.

Historically, the demography of plants was considered to be relatively intractable because of this variation in growth forms, specifically because an "individual" is sometimes difficult to delineate. For unitary species, identification of an individual is not difficult and age- or size-specific mortality and reproduction are relatively easy to follow. For species with clonal growth, Harper (1977) introduced the term "genet" to refer to all individuals that were derived from the same zygote, and "ramet" as the modules of a genet, which may in some cases become severed from the parent plant and grow as independent individuals. As long as the birth rate of ramets exceeds the death rate, a genet will survive in the population. Ramet demography may, in some cases, act as a buffer for the dynamics of a genet. In a computer simulation based on several years of data from populations of *Ranunculus repens*, it was found that the decay rate of the genet can be buffered by the decay rate of the ramets, which has the effect of conserving genetic variability within the population (Soane and Watkinson, 1979).

To date, there have been no comparative studies of the demography of ramets and genets, thus it is not known whether the demography of a population of genetically identical ramets reflects the vigor of the genet. With respect to studies on evolutionary processes such as senescence, it is critical that demographic studies be made at the level of the genet (Silander, 1985). There is limited evidence showing variation in age-specific demography of genets. In a study of the pasture grasses *Agrostis stolonifera* and *Lolium perenne*, Bullock *et al.* (1996) showed tiller production for different-aged genets changed in response to grazing. The existence of extremely long-lived clones is often cited as evidence that some clonal species lack whole organism senescence (cf. Watkinson and White, 1985; Noodén and Guiamét, 1996; Gardner and Mangel, 1997). Unfortunately, given their extreme longevity, it may be impossible to get a true understanding of late-age dynamics. There is clearly a need for more comparative studies of the demography of genets and ramets.

Variation in growth forms within the plant kingdom may necessitate a reevaluation of some of the evolutionary theories of senescence. In particular, the assumption of the theories that a species shows age-specific gene expression may be violated. A genet which grows to consist of a collection of ramets of different ages, cannot be expected to show age-specific gene action, particularly if the ramets are detached from the parent plant and grow independently. There is evidence, however, from bamboo that independent ramets show synchronized genetic control of flowering time, but the nature of this internal genetic clock and its interaction with the environment is not known (John and Nadguada, 1999). Moreover, even without independent growth, the turnover of tissues within an individual may allow an escape from senescence. For example, in the forest herb *Arisaema*, old tissue is sloughed off from the bottom of corms as new tissue is added to the top so that no part of any plant, even one 20 years old, is ever really over 4 years old. If there is complete turnover of tissue within an individual, in other words if no tissue ever achieves a chronologically old age, then there may be no reason to expect evolutionary senescence or any increasing mortality with age (Bierzychudek, 1982).

A second assumption of the evolutionary theories of senescence, which requires further evaluation with respect to clonal species, is the assumption that the force of selection declines with increasing age. This issue has been addressed in several recent theoretical papers. An analysis by Gardner and Mangel (1997) suggests that it is the rate of sexual reproduction relative to vegetative growth that is the important determinate of the strength of selection. Higher rates of sexual reproduction and lower rates of clonal reproduction result in a more rapid drop in the strength of selection with clonal age following an initial peak [see also, Orive (1995) and Pedersen (1995)]. Experimental results by Bell (1984) with six asexual freshwater invertebrates support this generalization, but there have been no comparative studies with plants of the demography of closely related species with different rates of sexual reproduction and clonal growth.

2. Somatic mutations and age

Another important distinction between plant and animal development is the timing of the differentiation between germ and soma plasma. In mammals, insects, and many other animals, the germ cells are segregated early in development, whereas in plants, the germ line does not segregate from the soma until just before reproduction. It was theoretically suggested that senescence should be found to be inevitable for any organism for which there is an early segregation between germ and soma (Williams, 1957). This has led several researchers to predict that patterns of evolutionary senescence may not be the same in animals and plants

(cf. Kirkwood and Holliday, 1979; Rose, 1991). Yet, the reason why this segregation is necessary for the evolution of senescence has never been explicitly stated (Roach, 1993). Two scenarios are presented below, in which the presence of multiple meristems and the late separation of germ and soma may influence patterns of plant senescence.

Individual longevity may be affected if somatic mutations result in intraorganismal selection. In long-lived plants with multiple meristems, a genetic mosaic may develop within individual plants, and this may have one of two consequences. On the one hand, intraorganismal selection has primarily been seen as a mechanism for eliminating deleterious somatic mutations (cf. Klekowski and Kazarinova-Fukshansky, 1984; Otto and Orive, 1995). Natural selection can act among cells within an individual to eliminate deleterious mutations that appear in a ramet or module, with little or no impact on the fitness of the genet. Conversely, selection may allow the spread of a favorable mutation within a clonal plant, which may result in an evolutionary change if an advantageous mutation goes to fixation (Pineda-Krch and Fagerström, 1999). If somatic mutations occur at a level high enough to allow selective changes within the life span of an individual, then this could allow for a closer tracking of an individual with its environment thus potentially resulting in increased longevity. A recent stochastic model demonstrates that there is a high probability of an advantageous somatic mutation going to fixation through a mitotic cell lineage in the presence of intraorganismal selection (Pineda-Krch and Fagerström, 1999). Genetically different individuals may thus originate through a succession of chimeric ramet generations without sexual reproduction (Fagerström *et al*., 1998). The genetic mosaic within an individual may be evolutionarily important, particularly as a defense against rapidly evolving pathogens, parasites, and herbivores (Whitham and Slobodchikoff, 1981). Recent work on genetic polymorphism within the chloroplast genome in *Senecio vulgaris* has shown that there can be within-plant selection between different DNA chloroplast types (Frey, 1999). Additionally, work with *Chenopodium album* (Darmency, 1994) suggests that within-individual selection may be an important mechanism for the development of within-generation resistance to herbicides. With respect to the evolution of senescence, the important factor is whether or not there are some species which can escape senescence because intraorganismal selection allows individual genotypes to increase their longevity by becoming more "fine-tuned" to their environment over time. The evolutionary importance of intraorganismal selection in plants cannot be assessed until further studies are conducted (Pineda-Krch and Fagerström, 1999; Sutherland and Watkinson, 1986; Otto and Orive, 1995). Moreover, the importance of this to senescence across the plant kingdom is not known, but it will only potentially be relevant to extremely long-lived species with multiple meristems.

The lack of separation between germ line and soma may also be important in the evaluation of the age-specific quality of offspring, particularly if somatic mutations become incorporated into the germ line. If there is a decline in the quality of offspring produced at later ages, then the intensity of selection on late-life traits will decline and will result in the evolution of more rapid senescence. The chance of incorporating a somatic mutation into a gamete depends on how many cell generations there are before gametes are formed and how many cell lines produce germ cells (Jerling, 1985). It is clear that the individuals in some species can live to extremely old ages, for example, clones of creosote bush are estimated to be 9,000–10,000 years old (Vasek, 1980). Furthermore, some species cover extensive areas, and thus can potentially have large numbers of meristems. Over time, and with a large number of meristems, there is thus the theoretical potential for mutations to become incorporated into the germ line. In a study of the effects of tree age on pollen, seed, and seedling characteristics in Great Basin bristlecone pine trees (*Pinus longaeva*),

Connnor and Lanner (1991) evaluated trees ranging in age from 23 to 4,257 years at both high and low elevation sites. They had hypothesized that there should be an increase in somatic mutations with age, particularly at high elevation sites since radiation can severely damage pollen. Their results showed no relationship between tree age and any reproductive variables and no differences between the two sites. To date, evidence suggests that vertical inheritance of somatic mutations is rare (reviewed in Schmid, 1990). Thus, with the possible exception of variation arising from intraorganismal selection, the late separation of germ line and soma does not appear to be an important factor in the evolution of senescence.

IV. Conclusions

Studies of evolutionary plant senescence use both demographic and evolutionary approaches to understand *why* the deleterious phenomenon of senescence persists in populations. Evaluating plant senescence within a demographic and evolutionary context is a relatively new approach (Watkinson, 1992; Roach, 1993; Pedersen, 1999), and there are many studies which will need to be done before we will have a clear understanding of the processes that have shaped life histories at the latest ages. Some guidelines and issues that should be considered in future studies are discussed below.

There is currently a paucity of high quality data on age-specific demography from plant populations. Despite the fact that the demography of plants is comparatively easy, few studies have followed a population of individuals for their entire life cycle, particularly for species which live more than a few years (reviewed by Watkinson, 1992; Roach, 1993). Geographically and taxonomically, studies that have been done, to date, have barely begun to sample the diversity of the plant kingdom (Franco and Silvertown, 1990). The studies which have been done have used small sample sizes, and there have been very few attempts to estimate the variance in age-specific life history traits.

One of the limitations of gerontological studies, in all species, has been sample size (Finch, 1990). Survivorship curves based on small sample sizes can provide estimates of life expectancy at birth, but it is not possible to estimate mortality rates late in life because so few individuals remain alive at older ages. Moreover, in field experiments there is a high rate of random mortality affecting survival at all ages. With a large sample size, it will be possible to attain a more accurate understanding of the inherent changes in mortality at different ages.

It is important to emphasize that data on age-specific mortality, not just life span data, are needed to understand the evolution of senescence. Plant species show a wide range of variation in their life spans, ranging from a few weeks, for some ephemeral annuals, to over 1,000 years for many conifer species. The presence of senescence cannot be inferred from life span measures for several reasons. First, evidence for senescence is derived from a change in the shape of the mortality curve for the population. Data on the maximum longevity of a species give the endpoints for the curve, but tell us nothing about a change in the shape of the curve with age (Bell, 1992). Secondly, two species can show different longevities due to differences in their annual mortality rates irrespective of any differences in senescence (Partridge and Barton, 1996). Thus, more complete demographic data on age-specific mortality are critical.

As it has been defined here, senescence refers to an increase in adult mortality with age. Theoretical studies have shown that factors other than senescence may cause mortality to increase with age (Abrams, 1993; Blarer *et al.*, 1995, McNamara and Houston, 1996).

Specifically, the theoretical expectation is that an optimized life history may show an increase in mortality due to an increased reproductive effort late in life. For plant species with determinate growth, in other words a fixed size at sexual maturity, reproductive output is expected to decline with age, but for some plant species, which increase in size with age, an increase in late reproduction may be observed. The change in age-specific reproductive effort, and its consequences for mortality rates has not been experimentally evaluated. Furthermore, selection acts jointly on survival and reproduction. Consequently, future studies with complete data on age-specific mortality and age-specific fecundity may demonstrate that some combination of mortality and fecundity will be a better measure, to detect the presence of aging in a population and to compare rates of aging, than mortality measures alone (Partridge and Barton, 1993, 1996). Plants are good experimental organisms to test these ideas, because age-specific reproduction is relatively easy to quantify.

The comparative biology of different species is one of the most useful approaches in evolutionary biology. There are a wide variety of growth forms, life spans, and mortality patterns within the plant kingdom and species have traditionally been classified either by their longevity, annual, biennial, perennial, or by their number of reproductive episodes, monocarpic, polycarpic. With more complete data on a wider range of species, perhaps we can look forward to the time when a thorough phylogenetic analysis of the evolution of whole plant senescence can be considered.

Finally, it is hoped that future research will begin to make a link between physiological and evolutionary approaches to plant senescence. Perhaps then, we will be able to make a bridge between our understanding of the physiological processes which occur within an individual, how the processes of physiological senescence change with age, and how they effect life history traits and the evolution of senescence.

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