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# Ageing in plants

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**Abstract**

Ageing in green plants differs in some fundamental ways from the process in animals. The seasonal cycle and persistence of a plant is governed by a combination of the determinate or indeterminate status of meristems (growth centres) and the cell death and disposal strategies employed by plants to generate well-adapted anatomies and morphologies. The degree of perenniality depends on the balance between exploratory growth and the wave of tissue death that succeeds it, and extremes of longevity can arise by relatively minor changes in the quantitative relationship between growth and death. The senescence and elimination of organs and tissues are related to the internal reallocation of resources but are programmed phases in the integrated development of the whole plant and do not represent a kind of ageing by stress or starvation. Meristems of long-lived plants accumulate genetic damage but selection mechanisms exist within the organism to control genetic load, and even to exploit somatic mutations that confer adaptive benefits. It is concluded that most plants do not age in the strict gerontological sense and that extremely long-lived forms like trees and clonal creeping perennials are sustained by selection and correction at the level of semi-autonomous cell lineages. 

*Keywords:* Perenniality; Senescence; Resource capture; Somatic mutation

## 1. Introduction

Plants present challenges to general theories of biological ageing. The subject of ageing seen from the viewpoint of a plant scientist raises a series of questions, some of which may not feature prominently amongst the concerns that preoccupy gerontologists. For example, there is the issue of definitions. An organism gets from a state of viability to death by any of a number of routes, many of which either do not involve ageing at all, or in which ageing is a secondary or peripheral influence. By what criteria do we know that something is or is not (a) alive; (b) aged? Do we have definitions that allow us to say that an organism is really dead and not in diapause or some such suspended state? For instance, the only way to tell if a mature desiccated seed is still viable is to germinate it. The comparative biology of ageing reveals a whole range of lifestyles. There are individuals such as lobsters that appear not to age at all, organisms that certainly do age but usually die by accident (for example wild mice, which are almost invariably lost to predation or disease before they have a chance to grow old), species such as humans that age and in which death is frequently a consequence of ageing, and organ-

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isms that can be rejuvenated (some invertebrates, for example).

There are special difficulties in trying to translate ageing behaviour from one level of biological organisation to another. What do we mean by an individual? Is a tree, or a coral, or a sponge, or a slime-mould a single organism or a population? There is a 'scaling down' problem—what is the relationship (if any) between actuarial or demographic definitions of ageing and the behaviour of the individuals in the population? For example, life tables say that women in developed countries live on average 3 years longer than men do. Does this mean that women begin to age 3 years later, or at a slower rate than men, or what?

Turning to the nature of ageing itself, we see that all sorts of biological processes fail or decline with age, but which are symptoms and which causes, dictating the progress and nature of ageing? There is a problem of 'scaling up'—is there or is there not a relationship between ageing of component parts or systems, on the one hand, and of the whole organism on the other? What is the relationship between cell/tissue/organ death and survival of the whole plant? We know that eyesight, blood pressure, joints, mental processes all degenerate with age in humans, but what has this to do with dying? Is it meaningful to think in terms of a 'master' reaction, a specific component that deteriorates with age and passes one or more critical threshold leading directly to whole-organism decline?

Of particular significance for plants is the question of resource capture and allocation in relation to ageing. Is ageing a kind of starvation or neglect process, and what meaning does this have for autotrophic organisms, in which raw materials and energy are generally not limiting? What is the contribution of non-optimal (stressful) environments to ageing (is ageing the same thing as being weatherbeaten)? And how is the integration of an ageing period into the full lifecycle related to organism lifespan? Finally there are questions about the mechanisms of ageing. Is ageing a failure of processes that normally defend against it? What are the cost-benefit trade-offs of repair, maintenance and durable construction? How is this related to the distinction between germline and soma, and what does this mean for plants in which there is no such distinction? What about ageing avoidance, and is ageing a failure to escape from influences that invoke the ageing response? Have organisms been able to channel the inevitability of ageing into processes that benefit their ecological and evolutionary fitness and what influence has this had on the programmes for cell death and senescence? How can natural selection act to evolve genes with specific functions in ageing? What are 'ageing genes' like and can they be mutated, mapped and isolated? What are their environmental sensitivities? And can we do anything about ageing by tinkering with these genes?

Here I select some aspects of plant ageing that address questions posed above. One concerns the relationship between longevity of the whole organism and that of its parts. Then the significance of resource capture for senescence is considered. The discussion ends with some comments from the botanical perspective on the significance of accumulated mutations in the ageing of long-lived plant species.

## 2. Annuality and perenniality

### *2.1. Meristems*

Although totipotency (the potential to develop into a complete organism) is an intrinsic property of most viable plant cells, the anatomy and morphology of the individual plant is usually generated by differential cell division and expansion initiated in defined growth centres (meristems—Meyerowitz, 1997). The meristems at the terminus of the shoot axis and at the insertion points of leaves are critical for longevity of the whole plant (which is not to devalue the importance of roots and their meristems, but for the purposes of discussion I will concentrate on events above ground). As long as the apical meristem initiates new vegetative organs while retaining a core of what in animals would be termed (confusingly for plant scientists) 'stem cells', the shoot is indeterminate and its potential lifespan open-ended. But if a terminal set of organs is established simultaneously with, or even at the expense of, a decline

in proliferative capacity, the shoot becomes determinate. Death of a determinate meristem is often preceded by a more or less prolonged and reversible period of proliferative arrest (Wang and Woolhouse, 1982; Bleecker and Patterson, 1997). A perennial plant is one where the apical meristem of at least one of its shoot axes remains indeterminate beyond the first growth season. Determinacy is usually associated with a change in the nature of the organs initiated at the apical meristem from vegetative structures, such as leaves, to reproductive structures (flower parts). Conversely, the shoot apical meristem is normally indeterminate for as long as it remains vegetative. But there are many exceptions to these generalisations. In several species, genetic variants have been described in which axes bearing inflorescence that are usually terminal can resume vegetative growth. Conversely, there are examples of reproductive apices that produce leaves instead of floral organs, but where the meristems remain determinate (Jones and Stoddart, 1971; Battey and Lyndon, 1990; Thomas et al., 2000).

### 2.2. Death of cells and organs

Meristem determinacy is important for annual/perennial status, but it is not the whole story. Progressive programmed senescence and death make critical contributions too. Plant form is modular and each and every module is ultimately disposable. Disposability is also a feature of plant anatomy. The surface area of a tissue or organ needs to keep pace with volume to sustain vital transport and exchange processes. This is generally achieved by pervading the structure with holes. In animals, tubes are often formed by cell migration, such as occurs in gastrulation. But the rigid wall limits cell movement in plants, though there are examples of intrusive cell migrations in plant differentiation (Lev-Yadun, 2001). The commonest biological mechanism for creating perforations in plant tissues is selective cell death. Plant cells developed the capacity for controlled autolysis very early in evolution (Park et al., 1999; Moriyasu, 1995) The first plants that colonised the land were already actively exploiting lysigeny (intracellular dissolution of protoplasm) and schizogeny (cell separation) to differentiate conducting tissues and shed reproductive structures and other parts (Raven, 1986; Edwards, 1993). The controlled death and disposal of cells is a way of life for plants, generating their anatomies (inter- and intra-cellular apertures) and building well-adapted morphologies.

### 2.3. Life-forms

A creeping perennial such as clover pushes out into new areas of the environment by proliferation at its apices, occasional branching, and subsequent elongation growth. Behind the zone of environmental invasion is a wave of cell senescence, death and necrotrophic disappearance. As long as the rate of exploration and proliferation does not fall below the pace of pursuing tissue death and disappearance, the plant will persist. In this way creeping perennials appear to move around their environment, foraging for resources (Campbell et al., 1991). Clonal perennials can be very long-lived. For example bracken communities have been estimated to date back more than 1500 years, and prairie grass clones maybe twice this (Molisch, 1938; Stebbins, 1958; Oinonen, 1967). Shrubs and trees penetrate the environment in the vertical plane. The tissues that have been through programmed senescence and death, instead of disappearing through post-mortem decay as they do in creeping perennials, persist as mummified corpses—namely as wood. The extent to which apical meristems can stay at least one step ahead of the succeeding wave of cell death determines the life forms of ephemerals, annuals or perennials.

### 2.4. The genetic basis of perenniality

Plants are excellent subjects for the genetic analysis of lifespan. Within a single inter-fertile taxonomic grouping there may be a very wide range of longevity and extreme hybridisation can generate segregating populations within which the annuality/perenniality trait has been genetically dissected. Grasses of the genera *Lolium* and *Festuca* constitute such a complex and include virtual ephemerals such as *Lolium temulentum* at one

extreme, highly persistent perennials such as *Festuca arundinacea* at the other and many interspecific and intergeneric hybrids exhibiting every kind of life history character (Thomas et al., 2000). Moreover, selective transfer of agronomically useful traits between annuals and perennials has long been a mainstay of crop improvement. An example of transferring a perennial trait into annual backgrounds is the introduction of the stay-green or non-senescence character into sorghum (Thomas and Howarth, 2000). The conclusion from genetic studies of plant longevity is that perenniality/annuality is not a simple qualitative trait. Many genes have been described that individually participate in the progression from cell division, growth, maturity, senescence and death, but life history is clearly a matter of the quantitative relationships between expression of these genes than of simple on-off switching. It follows that perenniality and associated characters are analysable by genetic mapping as QTL (quantitative trait loci). Senescence in sorghum has been approached in this way (Crasta et al., 1999). One of the senescence loci mapped on top of a maturity (time of reproduction) locus. This co-localisation is consistent with the notion of life history as an expression of programmed senescence and death in combination with apical determinacy, since meristem activity and the timing of floral induction are likely to be major components of maturity.

## 3. Senescence in autotrophs

### *3.1. Resource capture in plants*

We have seen that special characteristics of plant life-forms and morphogenesis create a context for the ageing process that distinguishes multicellular plants from animals in some quite fundamental ways. Another defining feature of plants is their relationship to the capture and use of resource and energy. For an autotroph like a green plant, the conflicting demands of repair and maintenance activities on the one hand and investment in growth and reproduction on the other mean something quite different from the corresponding trade-off in animals. Of course, every plant will die if light, water or nutrients are withheld for long enough, and the productivity of many natural communities will usually be limited by one or more of these resources. But in general the kinds of resource capture imperative that drive green plants, if they have any significance for ageing at all, bear only the most distant resemblance to those to which animals and other heterotrophs are subject. Indeed, it has been argued that the profligacy of some characteristic developmental and metabolic processes in plants represents a kind of wilful inefficiency of resource use, which may be essential for success as a terrestrial autotroph. In a resonant phrase, Harper (1977) referred to the green plant as 'a pathological overproducer of carbohydrates'. Thomas and Sadras (2001) have discussed the evolutionary origins and physiological consequences of promiscuous resource capture.

### *3.2. Death by exhaustion*

Even so, resource allocation within an individual plant has often been invoked as an explanation of patterns of whole-plant senescence and longevity. Normally an annual such as soybean or sunflower will exhibit self-destructive (monocarpic) senescence in the reproductive phase so that every vegetative part of the plant, including its meristems, is consumed until the only viable structures remaining are the seeds (Sinclair and de Wit, 1975; Sadras et al., 2000). As gardeners have long known, preventing seed set by, for example, removing flower buds as they appear, greatly promotes vegetative growth and extends the longevity of monocarpic species. These observations led Molisch (1938) to suggest that senescence in such plants is a consequence of exhaustion or starvation promoted by the nutritional demands of the developing seeds. It can certainly be demonstrated that there is a functional conflict in some monocarpic species arising from the dependence of seed on leaves for the supply of both reduced N, which requires degradation of leaf protein, and reduced carbon, which requires maintenance of the N-rich photosynthetic apparatus (e.g. Sadras et al., 1993).

### 3.3. Programmed senescence

The death-by-starvation hypothesis may be too superficial. For one thing, there are many monocarpic species—most cereals, for example—where suppressing reproduction and seed set has little effect on, or even accelerates, whole-plant senescence. In spinach and many other species, male and female flowers are borne on separate plants; yet, despite the absence of developing seeds, males undergo terminal senescence simultaneously with females (Leopold et al., 1959). Artificial attempts to meet the voracious demands of developing seeds by, for example, foliar feeding of nutrients are generally ineffective in preventing monocarpic senescence. It has been argued that the evolution of accelerated or delayed senescence in sterile plants may have been favoured depending on the relative importance of herbivory and competition (Thomas and Sadras, 2001). One way of rationalising the significance of internal competition for resources as a factor in plant ageing is to think of it as a symptom of interacting developmental programmes, one of which specifies a senescence syndrome that is intrinsic, with variations, to every plant tissue. It is important in this connection to emphasise that the term, senescence, is used in a specialised way by plant scientists. It describes the physiological stage preceding death in most cells, tissues and organs, but it is not itself an ageing or death process. There are many examples of death without senescence, and of senescence without death. For instance, it is possible for a tissue to pass directly from viability to death, omitting a recognisable senescence phase. The triggering of localised cell death (the so-called hypersensitive response) is what happens when plant cells react to attack by a pathogen. This, therefore, effectively cauterises the point of attempted invasion (Dangl et al. 2000). Conversely, the fact that senescence is readily reversible in many species (Zavaleta-Mancera et al., 1999a,b; Thomas and Donnison, 2000) establishes clearly that it is mechanistically independent of, and by no means inevitably leads to, degeneration and death.

### 3.4. Building on the ruins

We have seen that senescence, which generally follows organ maturity and occurs without growth or morphogenesis, is a way of remodelling the form of the plant by disposing of unwanted or inappropriate cells and tissues, while simultaneously reallocating resources. In a sense, programmed senescence in the plant life-cycle pre-empts ageing and if it culminates in declining viability and death, then it does so on the plant's own terms, so to speak (Thomas, 1994). The mode of development that leads to the construction of trees consists of successively building new, young structures on the accumulated corpses of previous generations of tissues and organs. Even the most ephemeral monocarpic annuals conform in principle to this plan—but in this case the fate of the youngest tissues at the end of the first season of the lifecycle is to disperse rather than to cling to the embalmed remains of their parent. Looked at this way, there is nothing special about the ageing characteristics of giant redwoods or centuries-old clonal populations of ferns. They simply represent the successful activity of the universal biological processes that maintain genetic integrity in cell lineages.

## 4. Accumulated genetic damage and plant ageing

### 4.1. Somatic mutations

Nevertheless, some of the meristems of a 4000-year-old bristlecone pine will have been proliferating for the entire lifetime of the plant and will certainly have experienced events that induce mutational changes. Intuitively, it might be expected that somatic mutations might ultimately accumulate in each indeterminate meristem to a point where viability is compromised. Ageing of a long-lived perennial plant would take the form of the progressive loss of indeterminate axes due to meristem mutational load. Such somatic mutations demonstrably occur and their propagation through cell lineages accounts for the increased frequency with age of chimeras and what garden-

ers call 'sports'. But it would be a mistake to picture ageing as directly related to the accumulation of somatic mutations in meristems. Many mutations of this sort are beneficial and may be important sources of adaptive fitness (Gill et al., 1995; Salomonson, 1996; Pineda-Krch and Fagerstrom, 1999). Meristems are sites where transposons can become active (Chaparro et al., 1995) and again this will often lead to new genotypes better adapted to variable environments. Klekowski (1988) made the interesting suggestion that a meristem is analogous to a microbial chemostat and showed that a model developed for the propagation of mutations in a microbial cell culture described the genetic load characteristics of two fern species differing in the longevity of clonal genotypes. Models of genetic mosaicism in plants and other organisms that reproduce clonally show that most deleterious somatic mutations are efficiently purged by intraorganismal selection (e.g. Pineda-Krch and Fagerstrom, 1999; Orive, 2001).

### 4.2. The hierarchy of plant ageing

This brings us back to the question of what we mean by an individual in relation to plant ageing. The whole plant, comprising root, shoot, leaves, flowers and so on, is clearly an integrated unit and has a definable lifespan. But each of the organs that represent the structural modules from which the whole plant is built also has a lifespan and interacts with other modules essentially as does an individual organism with other individuals in a population. The accumulation and propagation of somatic mutations in meristems according to models of within-organism selection takes the fractal-like scaling-down of identity to the level of individual cells. We conclude that, as a consequence of developmental and adaptive strategies, which resist, avoid and pre-empt ageing, plants can hardly be said to age at all in any sense recognisable from animals. Ageing is a fate that probably awaits all living organisms: it is just that plants are organised so that they are not there when it happens.

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