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Tansley review

Senescence, ageing and death of the whole plant

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Summary

This review considers the relationship between the lifespan of an individual plant and the longevity of its component cells, tissues and organs. It begins by defining the terms senescence, growth, development, turnover, ageing, death and program. Genetic and epigenetic mechanisms regulating phase change from juvenility to maturity influence directly the capacity for responding to senescence signals and factors determining reproduction-related patterns of deteriorative ageing and death. Senescence is responsive to communication between sources and sinks in which sugar signalling and hormonal regulation play central roles. Monocarpy and polycarpy represent contrasting outcomes of the balance between the determinacy of apical meristems and source–sink cross-talk. Even extremely long-lived perennials sustain a high degree of meristem integrity. Factors associated with deteriorative ageing in animals, such as somatic mutation, telomere attrition and the costs of repair and maintenance, do not seem to be particularly significant for plant lifespan, but autophagy-related regulatory networks integrated with nutrient signalling may have a part to play. Size is an important influence on physiological function and fitness of old trees. Self-control of modular structure allows trees to sustain viability over prolonged lifespans. Different turnover patterns of structural modules can account for the range of plant life histories and longevities.

This review is dedicated to the memory of Philippe Matile, Professor of Plant Biology at the University of Zürich, who died in his 80th year on 29 October 2011. He was a brilliant, cultured, amusing, talented, unique man who made important contributions to our understanding of many aspects of botany, including senescence.

I. Endgame theory

I am not afraid of death, I just don't want to be there when it happens.

Woody Allen

To die, to be *really* dead, that must be glorious!

Bela Lugosi

The endgame is an arena in which miraculous escapes are not uncommon.

Leonid Shamkovich

How, if at all, is the lifespan of an individual organism related to the longevity of its constituent cells, tissues and organs? Plants present a challenge in the search for a definitive answer to this most fundamental of biological questions (Skulachev, 2011). Many species are semelparous: the endgame culminates in mating and the plant dies in an explosion of monocarpic senescence. But what of deciduous trees, for example? Here much of the plant body is made up of dead tissues, the canopy is renewed and discarded every year, root systems turn over, and reproduction takes place repeatedly over decades, centuries or even millennia. In iteroparous species, such as trees and clonal plants, there is a disjunction between the lifespans of the whole and parts. Can we discern unifying principles that account for such extreme variation in lifestyle across seed plants? The present review, which builds on a number of surveys of plant lifespan and its determinants (Thomas *et al.*, 2000; Thomas, 2002, 2003; Munné-Bosch, 2008; Borges, 2009; Peñuelas & Munné-Bosch, 2010; Issartel & Coiffard, 2011; Davies & Gan, 2012), seeks to frame the question and to look for answers. The topic is extensive and, consequently, discussion here has to be selective.

Senescence, ageing and death are subjects that notoriously attract semantic argument (for example, Thomas *et al.*, 2003; van Doorn & Woltering, 2004; van Doorn *et al.*, 2011). Moreover, the plant scientist's vocabulary of ageing, life history and senescence is (or should be) used in a different way from the gerontologist's, despite employing a number of common expressions. It is necessary, therefore, to begin with a brief discussion of the terminology used in this article. The intention is not to be prescriptive, but simply to avoid misunderstandings and to establish as clearly as possible where it is profitable to seek common mechanisms and controls.

II. Terms and conditions

1. Senescence

Senescence, which is part of a cloud of terms referring generally to the process or condition of growing old, has a specialized meaning in plant biology. A *Thesaurus* search for 'senescence' reveals words for maturity, ripeness, seniority and longevity, but the dominant associations are with notions of decay, decline, gerontology, morbidity and mortality. This reflects the etymological origin of the word (from the Latin *senescere*, to grow old) and its association with senility and the medical problems of human ageing.

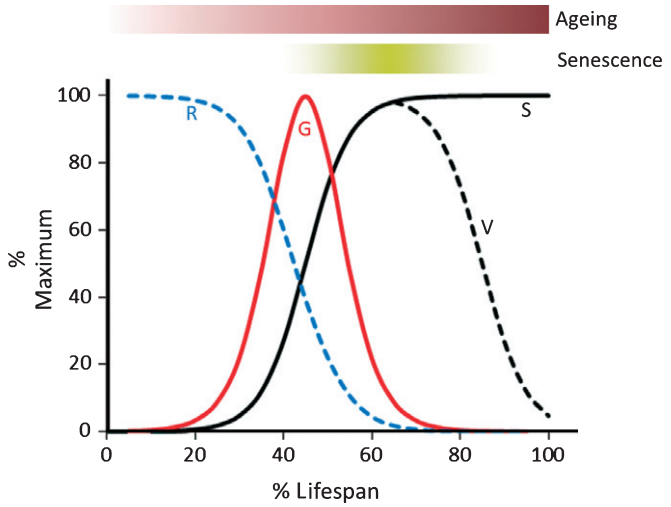

Fig. 1 Idealized growth and viability curves. The increase in size (S) of a cell mass, tissue, organ, whole plant or population follows a typical sigmoidal pattern. The instantaneous growth rate (G) is maximal at the inflexion point of the S curve. The relative growth rate (R = G/S) declines progressively over time. V represents viability, which begins to subside at some point following completion of growth. Declining viability is antagonistic to senescence. Note that ageing is considered here to refer to change (not deterioration) with time, and includes, but is not limited or defined by, the period of senescence and the terminal phase of decreasing V.

Current physiological understanding of the senescence condition, and its positive roles in plant growth, differentiation, adaptation, survival and reproduction, supports a definition that acknowledges senescence to be a phase of development that: (1) is a transdifferentiation episode following the completion of growth; (2) may or may not be succeeded by death; and (3) is absolutely dependent on cell viability and the expression of specific genes (Gan, 2007; Lim *et al.*, 2007; Guiboileau *et al.*, 2010; Breeze *et al.*, 2011; Liu *et al.*, 2011; Thomas, 2012; Fig. 1).

2. Growth and development

Development is the general term for the changes in form brought about through growth and differentiation. Because post-mitotic expansion processes in plants are largely driven by water, growth is not necessarily associated with an increase in dry mass. Growth is a scalable property of populations – from populations of cells to populations of phytomers to populations of individuals and on up to whole biomes and floras. The basic characteristics of population increase are shared across taxa and levels of organization. Typically, the pattern of accelerating and subsequent declining proliferation in biological systems is dependent on density. Growth begins slowly when population size is small, reaches a maximal rate when density is optimal with respect to metabolic and environmental constraints, wanes as limiting external and internal factors become increasingly influential, and finally approaches maximal size asymptotically (Fig. 1). Density dependence, interacting with demographic structure, is also an important factor in the evolution of optimal life histories, lifespan and senescence (Bonsall & Mangel, 2009).

Senescence is a normal and even essential feature of the post-mitotic phase of the plant cell life cycle and is immediately preceded

by (and sometimes partially overlaps with) the growth period. It is reasonable to suppose that intrinsic sigmoidicity, expressing the interplay between the potential for growth and the progressive imposition of limitations, is significant for triggering the senescence syndrome. Moreover, senescence, as already defined in section II.1, is negated directly by declining viability; thus the V curve in Fig. 1 represents the trailing edge of senescence capability in population lifespan. It follows that the template for the development of any and every plant cell, from any and every phytomer and the meristems that initiated them, has a built-in senescence module attuned to the growth–viability curve. Recent research in a range of organisms (Blagosklonny & Hall, 2009) is beginning to suggest that the link between growth and ageing processes is nutritional in nature (discussed further in sections IV.3 and IV.4).

Growth curves and their derivatives vary from tissue to tissue in their proportions and their positions on the time axis, and senescence responses will be correspondingly diverse. For example, cells of vascular tissue achieve full size and morphological maturity comparatively quickly, and programmed senescence followed by death of cell contents is completed soon after the growth asymptote is reached (Courtois-Moreau *et al.*, 2009; Ohashi-Ito & Fukuda, 2010). However, there is evidence that the stomatal guard cells of some species do not initiate a recognizable senescence program until long after structural and functional maturity, and may remain in the pre-senescent state when the leaf as a whole is senescent and shed (Zeiger & Schwartz, 1982; Keech *et al.*, 2010).

Differentiation is the change in structure and function that results in cell, tissue and organ specialization. The capacity to reverse the process of differentiation is a characteristic of the plastic nature of plant development. Senescence and development interact at different levels. Senescence is part of the program that specifies cell fate. It is triggered differentially in tissues and organs, resulting in complex anatomies and morphologies that change and adapt over time (Gunawardena, 2008). It is the means by which resources are recycled from obsolete body parts to new developing structures (Feller *et al.*, 2007; Guiboileau *et al.*, 2010). Variations on the senescence program theme have been shaped by evolution to give rise to a diversity of structures within the angiosperm life cycle (Thomas *et al.*, 2009).

3. Turnover

Molecules, cells, phytomers, individuals and even whole floras typically turn over (Leopold, 1975). Turnover is defined as flux through a pool. In the case of leaf turnover, the pool is the canopy (Hikosaka, 2005). Newly initiated leaves are recruited to the canopy, grow and mature, become senescent and ultimately die and leave the pool. In this sense, the combined S–V curve of Fig. 1 is illustrative of the turnover kinetics of phytomers or other structural entities within a biological system. If the rate of organogenesis and recruitment exceeds the rate of exit through death, the plant is growing. At the steady state, recruitment and loss are balanced. An excess of departures over arrivals will result in death of the plant. Looked at this way, the difference between annuality and perenniality is essentially quantitative, the consequence of genetically determined variations in the balance between rates inward and rates outward. There is generally little correlation between turnover schedules of leaves and roots either in trees (e.g. Withington *et al.*, 2006) or in biennials (e.g. Heilmeier *et al.*, 1986).

4. Ageing

Ageing is another term, like senescence, that has become associated with deterioration. It is true that, in the long run, errors will accumulate and living tissues will show signs of wear and tear. In the general biological context, ageing should refer to changes that occur with time, and therefore will embrace the time-based processes of growth and differentiation as well as maturity, senescence and mortality (Fig. 1). Ageing in this sense is not simply another name for declining viability. The notion that we start to die as soon as we are born is a nice poetic conceit, but is not helpful in understanding the biology of ageing. According to some proposed models, senescence is an accelerated form of ageing. Senescing organs, tissues and cells are built either to fail quickly or to be deficient in the mechanisms that otherwise defend against physiological decline. Reactive oxygen-based cell death theories of plant senescence come into this category (e.g. Van Breusegem & Dat, 2006; De Pinto *et al.*, 2011). A related view is that, as ageing and eventual death are thermodynamically unavoidable, senescence has evolved as a developmental strategy, a syndrome of programmed self-immolation that pre-empts the inevitable, enabling the individual plant to control its own viability and integrity over the course of the life cycle (Thomas, 1994). This is sometimes called the Samurai Law of Biology ('it is better to die than to be wrong'). Skulachev (2011), in a paper with a, shall we say, idiosyncratic approach to the lexicon of botany, applies the term 'phenoptosis' to programmed ageing leading to death of the whole organism.

5. Death

Death is a condition or state and is the culmination of, and separate from, the process of dying. The philosopher Ludwig Wittgenstein wrote 'Death is not an event in life'. By definition, changes that occur in dead cells are post-mortem and nonbiological. Biologists studying terminal events in development need to distinguish between the regulated activity of viable biological structures and the pathological outcomes of organic collapse (Thomas *et al.*, 2003).

Senescing tissue is viable, dead tissue is not, and there is a transitional condition between the two states (the descriptive term 'acherontic' has been suggested – Thomas, 2003) during which metabolism modulates into abiotic chemistry. This terminal period is often rapid and always irreversible. During the preceding senescence phase, cell membranes and organelles remain intact, and organs stay turgid. In some cases, notably the senescence of mesophyll in leaves, this phase is reversible until almost all of the cells' macromolecules have been recycled and exported to the rest of the plant.

Cells within the same organ can be at different stages in the progression from growth to senescence to death. For example, there is a gradient of cell age from leaf base to tip in grass species such as maize (*Zea mays*; Fig. 2). Senescence proceeds from the tip

Fig. 2 Leaf senescence in maize (*Zea mays*). The plant on the left (a) is growing on low levels of nitrogen fertilizer and shows the typical gradient of yellowing within and between leaves. The plant on the right (b) has been genetically modified to reduce the expression of a gene encoding an enzyme of nitrogen mobilization in senescence. Green tissues in (b) pass directly from maturity to death without a clear intervening senescence phase. Such genetic interventions show that death neither requires senescence nor is the inevitable consequence of it. Photograph from a study by Iain Donnison and Howard Thomas.

downwards, and towards the veins from interveinal regions of the lamina. The shoot axis also senesces, initially from the bottom up, later from the top down. As described in section II.3, survival of the whole organism is a matter of turnover kinetics, reflecting the balance between continued proliferation in meristems and the wave of post-senescence mortality that consumes existing phytomers (Thomas *et al.*, 2000). Heterogeneity within a single structure can make it difficult to disentangle senescence phase, terminal and post-mortem events, and to determine when the plant as a whole has ceased to be viable.

6. Program

The expressions 'senescence program', 'programmed ageing' and 'programmed death' are used extensively (e.g. Thomas *et al.*, 2009; Parish & Li, 2010; Kirkwood & Melov, 2011; Skulachev, 2011). The idea of a program as applied to living systems has been taken from computer science. The purposeful nature of a particular biological process, such as senescence, is conceived to be the consequence of control by the equivalent of an executable machine routine: hormones and other signal molecules, kinases and transcription factors are activated in sequence, leading to physiological change. Senescence, like many events in the plant life cycle, proceeds according to a timetable determined by developmental and environmental factors and mediated by a genetic program (Jansson & Thomas, 2008). That a process proceeds towards a final state in a predictable fashion is not, by itself, evidence that it is genetically programmed; it may simply be that the system is built in a particular way and so always fails in more or less the same manner (Thomas *et al.*, 2003).

7. Mortality, life expectancy, lifespan

The present article focuses largely on the physiological nature and mechanisms of ageing and senescence. Scaling up these processes to the population level and beyond takes us into the fields of demographic and evolutionary studies, which have their own conventions and definitions (e.g. Bonsall, 2006; Lauenroth & Adler, 2008), in which terms such as mortality (chance of death at a given age), life expectancy (time to death at a given age) and lifespan (maximal life expectancy at birth) are quantifiable actuarial parameters. Where these terms occur in the present article, they are used empirically.

III. Juvenility and maturity

1. Phase change

The state of maturity has implications for the lifespan of a plant and the turnover of its organs. The plant must be competent to respond to the stimulus that triggers senescence and death. The progression from incompetence to competence is a developmental event marking the transition from juvenility to maturity, a phenomenon sometimes referred to as phase change or heteroblasty (Jones, 1999; Day *et al.*, 2002). A review of the demography and phenology of perennial herbs of temperate forests by Bierzychudek (1982) gave the age at first reproduction as being in the range 1–10 yr. The juvenile period is extremely prolonged in some trees – from 3 to 7 yr in *Pinus sylvestris* up to 60 yr in *Quercus* sp. (Wareing, 1956). Species of *Quercus* and a number of other tree genera, for example *Fagus* and *Carpinus*, are marcescent: abscission of leaves borne on lower, juvenile-stage branches is suppressed and dead foliage remains attached until displaced by new leaves in the spring, whereas leaves of adult branches are shed when senescent in autumn (Berkley, 1931). This change with maturity is not related to plant size, proximity to the root or number of dormancy–growth cycles, but is rather an intrinsic property of dividing cells at the shoot apex (Robinson & Wareing, 1969).

2. Development of competence to senesce

Jing *et al.* (2005) demonstrated the age dependence of senescence inducibility by analysing the response of *Arabidopsis* plants to ethylene over the course of development (Fig. 3). In this experiment, plants grown in air begin to show signs of foliar senescence when they are *c.* 35 d old. If plants are exposed to ethylene, however, senescence can be induced at 25 d and increases strongly thereafter. These results demonstrate that, in terms of competence to express the senescence program in response to ethylene, the juvenile to mature phase transition occurs at *c.* 25 d. In the period between 25 and 35 d, the plant is competent, but does not initiate and execute

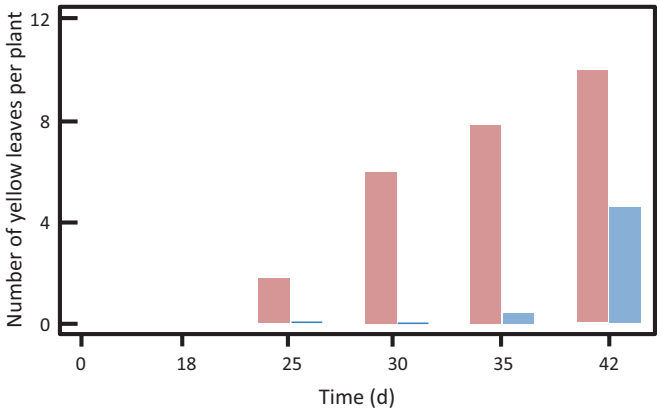

Fig. 3 The effect of ethylene treatment on visible leaf yellowing in *Arabidopsis*. Plants were grown either continuously in air for the indicated number of days (blue bars) or first in air for up to 4 d before the indicated days, followed by 3 d of growth in air supplemented with ethylene and one additional day of growth in air (red bars). Based on data from the study by Jing *et al.* (2005).

senescence because ethylene and/or some other endogenous regulator are limiting. Graham *et al.* (2012) refer to maturity-related ethylene sensitivity as the 'senescence window' concept.

3. Hormonal control of phase change

Transition from juvenile to mature morphology and physiology along the shoot is particularly clear in ivy (*Hedera helix*). If tissue taken from a juvenile ivy plant is cultured, plants regenerated from it have a stable juvenile phenotype; adult tissue yields adult-type regenerants (Polito & Alliata, 1981). Gibberellins (GAs) are important hormonal factors in the regulation of phase change, although their effect differs from species to species. Senescence of juvenile ivy leaves is accelerated by ethylene and prevented by GA (Horton & Bourguoin, 1992). Adult-phase ivy reverts to juvenility when sprayed with GA (Frydman & Wareing, 1974). By contrast, the transition from the vegetative phase to competence to flower is delayed in maize and *Arabidopsis* mutants with deficiencies in GA synthesis or perception (Evans & Poethig, 1995; Mutasa-Göttgens & Hedden, 2009).

4. Genetic and epigenetic regulation

The phase transition point in development is itself under genetic control. A series of *Arabidopsis* mutants, designated *old* (*onset of leaf death*), has been isolated (Shirzadian-Khorramabad *et al.*, 2008). These include at least 16 mutant lines in which the timing of the response to ethylene (Fig. 3) is delayed. It may be significant that 10 of the 16 are also late flowering. Many plant species must go through a juvenility–maturity transition before they become competent to flower.

Competence to flower and to respond to senescence signals are just two characters in a complex of traits that differ markedly either side of the phase change point. For example, leaves of juvenile maize plants are short, hairless and covered in epicuticular wax, whereas adult-phase leaves are long and narrow with hairs but no wax (Poethig, 2009). Analysis of phase change mutants in maize suggests that the signal triggering the transition is perceived directly in individual leaf primordia rather than by the shoot apical meristem, and is under the regulatory influence of microRNAs, among which miR156 has a decisive role. In maize, *Arabidopsis*, rice (*Oryza sativa*) and a number of woody species, miR156 has been shown to target a wide range of developmentally sensitive genes, including those encoding transcription factors of the SQUAMOSA PROMOTER BINDING-LIKE (SPBL) family (Poethig, 2010; Bergonzi & Albani, 2011; Wang *et al.*, 2011; Xie *et al.*, 2012). Traits displayed by plants overexpressing miR156 include prolonged expression of juvenile leaf characters, a higher rate of leaf initiation, increased branching and late flowering, all of which will have implications for terminal events in the life of the plant and its parts. Overexpression of miR156 in rice significantly inhibited expression, in young leaves, of miR164, a microRNA shown by Kim *et al.* (2009) to be part of a network regulating age-related cell death in *Arabidopsis* (Xie *et al.*, 2012). Bergonzi & Albani (2011) pointed out a limitation in relating these mechanisms to the question of life form and longevity, namely that most of the experimental evidence has been obtained from annual species, and a role for microRNAs in the phase change of perennials has yet to be confirmed. They suggested that the gene *TERMINAL FLOWER 1* (*TFL1*) functions in regulating the length of the juvenile period in perennials.

A possible mechanism for epigenetic regulation of maturity is DNA methylation, which stably silences particular genes. A study of giant redwood (*Sequoiadendron giganteum*) showed the DNA of juvenile-phase tissues to be 23% methylated, compared with < 14% for clonally identical adult tissues (Monteuuis *et al.*, 2008). Li *et al.* (2010) showed that phase change in maize is associated with heritable silencing of the *MuDR* transposable element by the naturally occurring derivative of *MuDR*, *Mu killer* (*Muk*), a process involving methylation and small interfering RNA (siRNA). A similar silencing system operates during phase change in *Arabidopsis* (Hunter *et al.*, 2006). Studies of such epigenetic mechanisms specifically in relation to maturity, senescence and ageing are likely to be rewarding.

IV. Sources and sinks

1. Supply and demand during development

A semelparous plant dies because its sink tissues kill its source organs by a kind of starvation (*Erschöpfungstod* – Molisch, 1929), by induction of senescence in response to nutrient diversion (Davies & Gan, 2012) or by export of a 'death hormone' (Wilson, 1997). A sink is defined as a net importer of nutrients (nitrogen (N), phosphorus (P), potassium (K), sulfur (S) and other minerals) and assimilates (carbon (C) derived directly or indirectly from photosynthesis). Developing seeds, bulbs, tubers and other structures that accumulate storage compounds are strong sinks, as are expanding leaves and branches during vigorous vegetative growth. Organs that supply the precursors for sink metabolism are sources. Sources and sinks communicate through the vascular system. During development of the endosperm of cereal grains and the parenchyma of

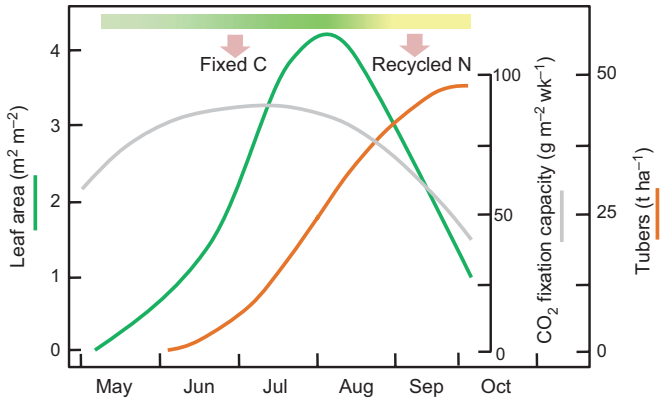

Fig. 4 Relationship between photosynthesis and senescence of source leaves and yield of potato (*Solanum tuberosum*) tubers (sinks). Photosynthetic capacity, which supplies the carbon for the accumulation of tuber starch, begins to decrease at the end of August. At this time, senescence is initiated, the leaf area begins to decline and remobilized amino acids support the synthesis of tuber storage protein. Based on data from Milthorpe & Moorby (1979).

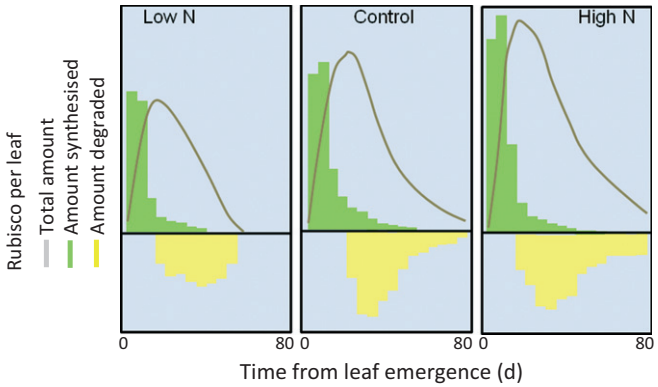

Fig. 5 Turnover of Rubisco from initiation to senescence of the 12th leaf of rice (*Oryza sativa*) plants fed with different levels of nitrogenous fertilizer. Rubisco is synthesized at a high rate in young growing leaves. Only when synthesis has stopped, at around full leaf expansion, does protein breakdown begin. Synthesis (green bars) and breakdown (yellow bars) overlap to only a very limited degree. The total amount (but not turnover pattern) of Rubisco changes with increasing amount of nitrogen fertilizer fed to the plant, indicating that Rubisco is bifunctional, acting as a storage protein as well as an enzyme. Based on data from Makino *et al.* (1984).

potato tubers, large amounts of starch are accumulated. These organs are supplied via the phloem with assimilated C mostly fixed by current photosynthesis. Some of the amino acid precursors for the synthesis of proteins with structural, reserve or enzymatic functions in sinks may be the products of newly assimilated inorganic N; however, in general, most amino acids imported by the sink are the recycled products of protein degradation occurring during senescence of source tissues (Xu *et al.*, 2012). Figure 4 relates leaf area, photosynthesis and senescence to tuber development in the potato crop and shows the transition in canopy function from C source to N source.

2. Leaves as storage organs

The CO_2-fixing enzyme Rubisco is the largest repository of recoverable N in vegetative tissues. Careful turnover measurements have led to the conclusion that there is little or no simultaneous synthesis and breakdown of Rubisco at any time in the life of green tissue. For example, Fig. 5 presents data from a study of Rubisco from birth to senescence of the 12th leaf of rice (Makino *et al.*, 1984). Rubisco is synthesized at a high rate in young growing leaves. Only when synthesis has stopped, at around full expansion, does breakdown of the protein start. Synthesis (green bars) and breakdown (yellow bars) overlap to only a very limited degree. Feeding the plant with N fertilizer increases the amount of Rubisco, but does not change the turnover pattern. This kind of N-responsive behaviour is characteristic of a storage protein.

The bifunctionality of Rubisco as both a photosynthetic enzyme and a reserve of mobilizable N is reflected in the dual role of foliage: leaves are organs of storage as well as assimilation, and the initiation of senescence may be considered to be the point of transition from C to N source (Fig. 4). Young, actively growing vegetative sinks, and storage organs accumulating reserve proteins, have a rapacious appetite for N. When the demand cannot be met by import from the rhizosphere alone, N is withdrawn from older tissues. In extreme cases – monocarpic reproduction, for example – N remobilization from older tissues can occur on such a scale that the plant 'self-destructs' (Sinclair & de Wit, 1975; Gregersen, 2011; Davies & Gan, 2012). Internal redistribution of N between sources and sinks can also account, at least in part, for other patterns of senescence. Before leaf fall in deciduous species, salvaged N is transferred to bark tissues, where it accumulates as defined storage proteins that will be mobilized to provide amino acids to support the resumption of growth in the spring (Cooke & Weih, 2005). Progressive or sequential senescence occurs during vegetative growth in herbaceous species when continued leaf production at the stem apex is frequently at the expense of the senescence of preceding leaves on the shoot. For example, in certain temperate pasture grasses, each vegetative branch (tiller) generally carries about three mature leaves at any given time, and every new leaf that appears must be balanced by senescence of the lowermost leaf (Yang *et al.*, 1998). Sequential senescence is a useful adaptation because it buffers growth against fluctuations in the supply of N and other mobile nutrients, and allows rapid response to defoliation. L-systems models of grass tiller development are consistent with self-regulatory feedback regulation between shoot architecture and phytomer turnover (Verdenal *et al.*, 2008).

3. Source–sink communication

How is sink demand communicated to the source, or source capacity signalled to a potential sink? Answers to these questions, which are critical for understanding how senescence is regulated in the whole plant, are various and depend on the species and circumstances. Elimination of strong sinks often delays senescence in source leaves, as seen in the dramatic example of the regreening response of basal *Nicotiana* leaves to removal of the flowering shoot above them (Zavaleta-Mancera *et al.*, 1999). However, there are also many instances (*Capsicum*, *Triticum*, *Zea*) where senescence is precipitated by reduced sink demand. These observations may be

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explained by a mechanism whereby source leaf senescence is triggered by some critical level of uncommitted assimilate. There is growing evidence that sugars exert a regulatory influence over leaf senescence (Rolland *et al.*, 2006; Wingler & Roitsch, 2008).

Plant cells have separate sensors for sucrose and hexoses (glucose, fructose). Changes in the sucrose to hexose ratio are detected by these systems and lead to different transduction pathways and inductive or repressive effects on gene transcription (Smeekens *et al.*, 2010). Extracellular invertase (CWinv, Fig. 6), which catalyses the hydrolysis of sucrose to form glucose and fructose, is a component of the pathway by which sugar from the source is unloaded from the photosynthate transport system on arrival at the sink. These functions mean that the enzyme has a central role in the transition from source to sink and in the pattern of sugar-mediated gene regulation (Lara *et al.*, 2004; Rolland *et al.*, 2006). One important sugar-sensing pathway is known to interact with the senescence program centres on the protein kinase SnRK1 (Snf1-Related Kinase1; Fig. 6). SnRK1 is a global regulator, acting as both a post-translational inhibitor and an inducer of transcription, with wide-ranging influence on development and environmental responses (Baena-Gonzalez *et al.*, 2007). It is activated by darkness, nutrient starvation and high cellular concentrations of sucrose or low glucose, or both, conditions associated with the induction of senescence (Jongebloed *et al.*, 2004; Parrott *et al.*, 2007). Extracellular invertase, by hydrolysing sucrose to produce hexoses, counteracts the influence of SnRK1. SnRK1 inhibits several key reactions of C and N metabolism by phosphorylating the corresponding enzymes. It also stimulates the transcription of genes that encode enzymes of C mobilization, such as α-amylase. Plants in which SnRK1 expression has been experimentally down-regulated display a number of developmental irregularities, including premature senescence (e.g. Thelander *et al.*, 2004).

The SnRK1 network interacts with a second senescence-regulating pathway through the enzyme hexokinase (HXK; Fig. 6). The HXKs encoded by the *Arabidopsis HXK1* and rice *HXK5* and *HXK6* genes function as glucose sensors. The glucose-sensing property of HXK resides mainly in the mitochondrion and is independent of its glycolytic role in converting glucose to glucose-6-phosphate. A fraction of HXK exists in the nucleus in high-molecular-weight complexes which repress the expression of photosynthetic genes and promote proteasome-mediated degradation of transcription factors that function in plant hormone signalling pathways (Smeekens *et al.*, 2010). *Arabidopsis hxk1* mutants are glucose insensitive, delayed in flowering and senescence, and provide evidence that sugars and cytokinins regulate in an antagonistic manner (Moore *et al.*, 2003).

Sugar metabolism may exert a signalling influence through the ratio of NAD to NADH in the cell. One of the *old* mutants of *Arabidopsis* (see section III.4), the early-senescing *old5*, has been shown to be deficient in quinolinate synthase, an enzyme required for the *de novo* synthesis of NAD (Schippers *et al.*, 2008). Another point of contact between sugar signalling and pathways regulating ageing is through Target of Rapamycin (TOR; Fig. 6), which is discussed in further detail in section IV.4

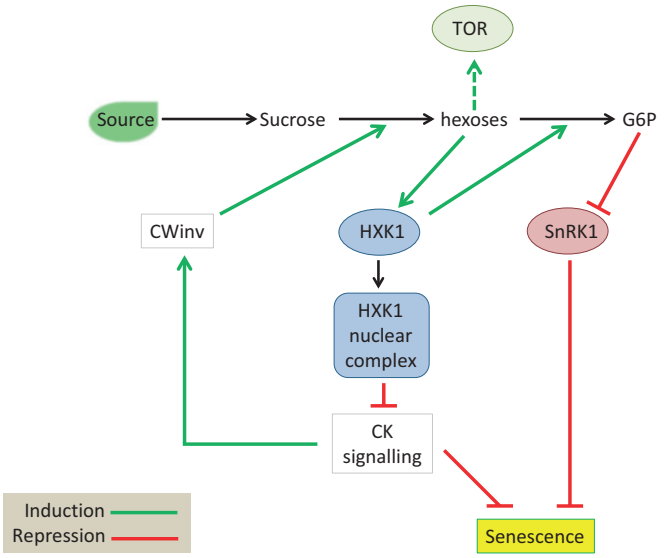

Fig. 6 Senescence is responsive to sugars, which act through the global regulators Snf1-Related Kinase 1 (SnRK1), Hexokinase 1 (HXK1) and Target of Rapamycin (TOR). SnRK1 delays senescence. HXK1 is part of a nuclear complex that promotes senescence by repressing cytokinin (CK) signalling. The sugar products of hydrolysis of sucrose by cell wall invertase (CWinv) are positive regulators of HXK1 and TOR. Glucose-6-phosphate (G6P), the product of the HXK1-catalysed phosphorylation of glucose, is a negative regulator of SnRK1.

4. Regulation of source senescence by hormones and nutrients

There is evidence that cytokinins exert their senescence-delaying influence by regulating source–sink relations (Davies & Gan, 2012). Tissues with the highest cytokinin levels are the strongest metabolic sinks and attract the majority of nutrients by outcompeting less active structures. Cytokinins are normally produced in roots and transported to leaves. One hypothesis to explain post-flowering leaf senescence suggests that, in many plants, cytokinins from the root are redirected into the developing seed instead of into the leaves. The seed, therefore, becomes a stronger sink and nutrients are diverted from leaves into seeds, triggering leaf senescence. The extracellular invertase associated with the transfer of translocated C from the vascular system to the sink is the common factor in cytokinin and sugar regulation (Moore *et al.*, 2003; Lara *et al.*, 2004; Fig. 6). The inhibition of extracellular invertase results in the inhibition of the cytokinin-mediated delay of leaf senescence.

An alternative, or additional, explanation for the regulation of source–sink interactions in senescence has been called the 'death hormone' hypothesis (Wilson, 1997). It proposes that developing sinks export a factor that triggers senescence in target leaves. Much of the evidence in favour of this idea comes from surgical and other manipulative experiments performed on soybeans. The removal of pods (Nooden & Murray, 1982) or the restriction of their development (Miceli *et al.*, 1995) prevents reproduction-associated foliar senescence in this species. No hormone has been convincingly identified as the explanation for sink-regulated senescence in soybean, but it is known that there is significant

transfer of sugar from developing seeds back to source leaves through the xylem (Bennett *et al.*, 1984). Moreover, the physiology of foliage in depodded plants undergoes a major change with implications for source status. Depodding releases uncommitted N already accumulated by the plant. N fixation by root nodules, which normally declines during fruiting in soybean, is maintained in depodded plants. Elevated levels of N represent a 'metabolic sink' for photosynthate, thus altering carbohydrate metabolism and sugar signalling in source leaves. There is a buildup of novel glycoproteins (vegetative storage proteins, VSPs) as a consequence of the specific induction of new genes (Feller *et al.*, 2007). Leaves of depodded plants also accumulate starch (Nakano *et al.*, 2000), another metabolic sink for sugars. It seems that the leaves of depodded soybean plants become modified into alternative sinks. All in all, the sugar-sensing model is the best understanding we currently have of source–sink regulation of senescence.

V. Monocarpy and polycarpy

1. Reproduction and whole-organism senescence

Flowering plants are divided into annuals, biennials and perennials on the basis of whether they complete their life cycles in one, two or many years, respectively. The different types are distinguished by the relationship of senescence of the whole plant to that of its parts. Annual plants, which grow, reproduce and die in a single season, obey the Romano Rule ('live fast, die young'; Pearl, 1928; Kaufmann, 1996; Issartel & Coiffard, 2011). Biennials generally devote the first year to vegetative growth and the second year to reproduction, senescence and death: their life cycles are qualitatively no different from those of annuals. There are also monocarpic perennial species, such as century plant (*Agave*) and Japanese timber bamboo (*Phyllostachys*), which may survive for many years in the vegetative condition, but then produce flowers and fruits and die. Death following reproduction is also common in animals – for example, mayfly, octopus and Pacific salmon. The general biological term for this reproductive strategy (sometimes referred to as 'big bang' senescence – Humphries & Stevens, 2001) is semelparity. Perennial plants that reproduce repeatedly throughout their lifespan are described as polycarpic; the term covering animals and plants is iteroparous.

2. The determinate or indeterminate apex

The shoot apex may be continuously meristematic (indeterminate), or may cease to make new structures (determinate) either by differentiating into a terminal organ or as a consequence of senescence of the apical meristem (Bull-Hereñu & Classen-Bockhoff, 2011; Davies & Gan, 2012). Plant habit, life cycle and senescence profile are intimately associated with the pattern of apical and primordium determinacy. In monocarpic species, all indeterminate vegetative shoot apices become determinate floral apices and the entire plant senesces once the seeds have been dispersed. Polycarpic perennials retain a population of indeterminate apices as well as those apices that become reproductive and determinate (Thomas *et al.*, 2000).

Determinacy of a terminal floral apex is usually irreversible. There are, however, numerous examples of reversion, in which the meristem is kick-started into further activity and produces abnormal vegetative or floral structures (Tooke *et al.*, 2005). Mutations in genes that determine and maintain the reproductive state often result in a form of floral reversion in *Arabidopsis*. Reversion suppresses flowering-associated leaf senescence. In species with so-called 'open' inflorescences (Bull-Hereñu & Classen-Bockhoff, 2011), where the apical meristem dies and there is no terminal flower, meristem senescence may be reversible during the early stages of lateral floral bud differentiation (Proebsting *et al.*, 1976). The genetic and hormonal regulation of apical senescence is best understood in pea (*Pisum sativum*), in which determinate and indeterminate types differ in interactions between loci controlling the photoperiodic flowering response, the influence of developing pods and the supply of GAs (Davies & Gan, 2012). The loss of apical dominance that occurs when the terminal shoot meristem dies would be expected to lead to cytokinin-mediated axillary bud regrowth. Expression of the regulatory gene *AtMYB2* is associated with the suppression of axillary bud outgrowth during monocarpic senescence in *Arabidopsis*. T-DNA *atmyb2* insertion lines are bushy, and senescence is delayed, as a consequence of suppressed cytokinin production (Guo & Gan, 2011).

Vegetative apices of biennial and perennial species may also become temporarily determinate, and differentiate into resting (dormant) buds. In deciduous woody species, dormancy of apical buds and the senescence and shedding of leaves in autumn are linked events in the annual cycle (Olsen, 2010).

3. Reproductive senescence in dioecious species

Senescence in semelparous organisms is often considered to be the cost of sex, reflecting the trade-off between survival and reproductive investment (e.g. Stearns, 1989). Source–sink regulation, as described in section IV, suggests a resource-based mechanism for monocarpic senescence. If developing seeds and fruits are indeed such strong sinks that they induce senescence in source leaves through perturbations in sugar sensing, hormonal signalling and nutrient diversion, senescence in sexually dimorphic (dioecious) species should be expressed differently in males and females. This proposition was not supported by classical experiments on spinach (*Spinacea oleracea*) carried out by Leopold *et al.* (1959), in which senescence was shown to occur simultaneously in individuals of the two sexes. Senescence in males was delayed by removing the tiny pollen-producing flowers, the sink size of which is negligible compared with those of females destined to carry the developing fruits. This subject was recently revisited by Sklensky & Davies (2011), who measured the partitioning of photosynthate during flower development in spinach plants of the two sexes, and showed that reprogramming apices for floral differentiation diverts sufficient resource away from vegetative tissues to trigger senescence. The results are broadly consistent with source–sink regulation through the hormone–sugar mechanism described in section IV.

Male–female difference in survival related to reproductive investment is a live issue in debates about evolutionary models of

sex and ageing in animals (Bonduriansky *et al.*, 2008). In these organisms, however, the primary and secondary differentiation of male and female reproductive strategies is complicated by sexual behaviour, environmental factors that influence life expectancy or ageing rate, and a range of sexually antagonistic interactions. It is doubtful, therefore, that animal models could be helpful for understanding senescence in dioecious monocarpic plants.

4. Selection for life history traits

There are many instances of interfertility in crosses between annual/monocarpic genotypes or species and related perennial/polycarpic types. In general, the hybrid offspring tend towards a perennial phenotype, an inheritance pattern consistent with the origin of annuality as a loss-of-function derivative of an ancestral perennial habit (e.g. Thomas *et al.*, 2000, 2011). Selection for monocarpy has been a significant factor in crop domestication (Allaby *et al.*, 2008). Arable weeds are subject to the same selection pressure as the cultivated species they infest, and show the same trend from perenniality to annuality. An example is *Lolium temulentum* (darnel), a weed of wheat and barley, more or less extinct in modern intensive agriculture, but historically, and in developing countries, a poisonous scourge with a reputation for harmful contamination of the food chain (Camporesi, 1989). *Lolium* speciation 2–3 million yr ago, and the subsequent appearance of darnel *c.* 10 000 yr ago, were contemporaneous with the evolution and domestication of the genus *Triticum* in the Fertile Crescent region (Charmet *et al.*, 1997; Catalan *et al.*, 2004; Senda *et al.*, 2005; Inda *et al.*, 2008). It is clear that co-selection has converted the perennial *Lolium* ancestor of darnel into a cereal analogue, thereby fitting it so closely to its agricultural niche that its geographical range precisely followed the spread of temperate arable agriculture in prehistory (Thomas *et al.*, 2011). Cultivated oat and rye are thought to have originated, like darnel, as weeds of wheat, but to have taken the extra step of becoming adopted as cereals in their own right (Ladizinsky, 1998). In each case, the life history of the weed mimics the monocarpic, high-harvest-index characteristics of the crop with which it coexists.

VI. Longevity and ageing

1. Lifespans

The life expectancies of polycarpic perennials range from < 10 yr in some herbaceous species to more than 2000 yr in woody conifers (Table 1). Many plants that form clones by asexual reproduction can proliferate to establish community-sized 'individuals' of extraordinary longevity possibly, as is the case for *Lomatia tasmanica* (Lynch *et al.*, 1998), in excess of 40 000 yr. Over the course of these extremely extended lifetimes, the cycle of initiation, maturation, senescence and death of individual structural units will have been recurrent, apparently continuing independently of whatever processes determine ageing and longevity of the plant as a whole. This contrasts with the monocarpic strategy, in which the progressive senescence and death of the entire vegetative body is invoked to ensure mass nutrient mobilization and transfer to the seeds that survive to restart the life cycle in the next generation.

Table 1 Maximal lifespans of individual and clonal plants (Lynch *et al.*, 1998; Thomas, 2003; Johnson & Abrams, 2009; de Witte & Stöcklin, 2010)

Species	Age (yr)
Single plants	
Bristlecone pine (*Pinus longaeva*)	4600
Giant sequoia (*Sequoiadendron giganteum*)	3200
Huon pine (*Dacrydium franklinii*)	2200+
Common juniper (*Juniperus communis*)	2000
Stone pine (*Pinus cembra*)	1200
Queensland kauri (*Agathis microstachya*)	1060
European beech (*Fagus sylvatica*)	930
Olive (*Olea europaea*)	700
Blackgum (*Nyssa sylvatica*)	679
Hemlock (*Tsuga canadensis*)	555
Scots pine (*Pinus silvestris*)	500
White oak (*Quercus alba*)	464
Chestnut oak (*Quercus montana*)	427
Pitch pine (*Pinus rigida*)	375
Red oak (*Quercus rubra*)	326
Pear (*Pyrus communis*)	300
Black oak (*Quercus velutina*)	257
Black walnut (*Juglans nigra*)	250
European ash (*Fraxinus excelsior*)	250
Apple (*Pyrus malus*)	200
English ivy (*Hedera helix*)	200
Arctic willow (*Salix arctica*)	130
Flowering dogwood (*Cornus florida*)	125
European white birch (*Betula verrucosa*)	120
Bigtooth aspen (*Populus grandidentata*)	113
European grape (*Vitis vinifera*)	100
Scots heather (*Calluna vulgaris*)	42
Myrtle whortleberry (*Vaccinium myrtillus*)	28
Spring heath (*Erica carnea*)	21
European elder (*Sambucus racemosus*)	20
Scandinavian thyme (*Thymus chamaedrys*)	14
Crossleaf heather (*Erica tetralix*)	10
Clonal plants	
King's lomatia (*Lomatia tasmanica*)	43 000+
Huckleberry (*Gaylussacia brachycerium*)	13 000+
Creosote (*Larrea tridentata*)	11 000+
Quaking aspen (*Populus tremuloides*)	10 000+
Bracken (*Pteridium aquilinum*)	1400
Velvet grass (*Holcus mollis*)	1000+
Sheep fescue (*Festuca ovina*)	1000+
Red fescue (*Festuca rubra*)	1000+
Ground pine (*Lycopodium complanatum*)	850
Lily of the valley (*Convallaria majalis*)	670+
Reed grass (*Calamagrostis epigeios*)	400+
Black spruce (*Picea mariana*)	330+
Wood sage (*Teucrium scorodonia*)	100

2. Stochastic events and deteriorative ageing

Some of the meristems of the oldest individuals of the species listed in Table 1 will have been proliferating cells, tissues and organs for up to 3000 yr and more. Even a cell replication mechanism of the highest fidelity would be expected to propagate a significant number of errors over such an extended timescale (Salomonson, 1996; de Witte & Stöcklin, 2010). Somatic mutations can also be a

consequence of genetic and metabolic damage caused by reactive oxygen species and free radicals, factors that tend to build up with age (Munné-Bosch, 2007). The rate of somatic mutation functions as a clock and has been used to estimate lifespan (Heinze & Fussi, 2008; Warren, 2009).

There have been relatively few experimental tests of the mutational load hypothesis of physiological deterioration during plant ageing, and results are contradictory. A study of bristlecone pine plants up to 4700 yr old found no statistically significant relationship between the age of the individual and the frequency of mutations in pollen, seed and seedlings (Lanner & Connor, 2001). However, Ally *et al.* (2010) observed that increasing clone age in *Populus tremuloides* was associated with a significant decline in the average number of viable pollen grains per catkin per ramet. Assuming the rate of deterioration to be constant over time, it would take *c.* 500–20 000 yr for a clone to lose male sexual function completely. The maximal lifespan for clonal *P. tremuloides* is estimated to be 10 000 yr (Table 1). An age-related increase in the frequency with which chimeras and sports arise is observed in many perennial species, but their review of the evidence led de Witte & Stöcklin (2010) to conclude that the case for somatic mutation as a mechanism of whole-plant deteriorative ageing is weak. Mutations of this sort may even be important sources of adaptive fitness for a long-lived organism, generating tissues with new genotypes better adapted to variable environments (Salomonson, 1996; Pineda-Krch & Fagerström, 1999; Folse & Roughgarden, 2012). Models of genetic mosaicism in angiosperms show that most deleterious somatic mutations are efficiently purged by selective pressures acting on the population of cells newly derived from the meristem – in the words of Klekowski (2003) 'stratified meristems would be expected to be the least liable to undergo mutational meltdown'.

3. Telomeres and plant ageing

Some age-related changes to the genome arise physiologically rather than by chance. An example is DNA methylation, as discussed above in relation to phase change (section III.4). Another process that has attracted much gerontological research interest is telomere shortening, a mechanism with its conceptual origins in the hypothesis that the limited lifespan of isolated animal cells *in vitro* is the cellular expression of the ageing process (Swim, 1959; Hayflick & Moorhead, 1961).

Animals with dysfunctional telomeres develop features of premature deteriorative ageing caused by the activation of cell death pathways (Sharpless & DePinho, 2007). However, evidence for a correlation between lifespan and mean telomere length or telomerase activity across vertebrate groups is weak (Monaghan & Haussmann, 2006). The telomere attrition model of age-related decline in function and viability proposes that loss of telomerase activity over time results in progressive reduction in telomere length until, at a critical point, chromosomal fusions and rearrangements become so frequent that the cell is diverted from division into the pathway leading to senescence and death (Forsyth *et al.*, 2002). Telomerase is a ribonucleoprotein complex that extends the ends of telomeres after replication through the activity of telomerase reverse transcriptase (TERT; Watson & Riha, 2010). In *Arabidopsis*, the minimal amount of telomeric DNA required for protection from chromosome end-to-end fusion is within the range 260–450 nucleotides, with the first telomeric fusions becoming detectable when the shortest telomere dips below 1 kb (Heacock *et al.*, 2007).

The telomere–telomerase system is highly conserved, and there have been a few observations of apparent age-related telomere shortening in some plant species. An early study reported that the telomeres of barley became progressively shorter during whole-plant development, but longer in callus culture (Kilian *et al.*, 1995); however, observations on bristlecone pine, and also *Ginkgo biloba* individuals up to 1400 yr old, have failed to demonstrate significant shortening of telomeres with age (Flanary & Kletetschka, 2005; Song *et al.*, 2010). Moreover, telomerase mutants do not show consistent alterations in deteriorative ageing or related processes. *Arabidopsis* mutants carrying a T-DNA disruption of the *AtTERT* gene lack telomerase and survive for up to 10 generations. Telomere attrition occurs at the rate of *c.* 250–500 base pairs per generation. Beyond the fifth generation, severe cytogenetic defects begin to accumulate. Plants of later generations develop malformations of organs and meristems and ultimately become arrested in a vegetative and partially de-differentiated condition. Significantly, mutants at this terminal stage are longer lived than comparable wild-type individuals (Riha *et al.*, 2001). Studies on rice have identified RICE TELOMERE BINDING PROTEIN1 (RTBP1) as a negative regulator of telomere length, with a possible role in telomere architecture. Knocking out RTBP1 expression resulted in rice mutants with significantly longer telomeres than those of the wild-type. Over four generations, growth and development were severely disturbed in *rtb1* plants, and there was increased frequency of anaphase bridge formation and chromosomal fusions (Hong *et al.*, 2007). Plants and animals clearly have fundamentally different responses to telomere disruption, probably as a consequence of contrasting developmental and genomic architectures. McKnight & Shippen (2004) pointed out that plants have 'an amazing capacity to withstand raging genomic instability' and suggest that animals respond to dysfunctional telomeres in such a fundamentally different way because they, unlike plants, have recourse to cell elimination via p53-mediated cell death pathways.

4. Costs of repair and maintenance

The contrasting relationship of multicellular plants and animals to the capture and use of resource and energy has major implications for theories of biological ageing. Animals, being heterotrophic, have to reconcile the energy demands of repair and maintenance activities with those of growth and reproduction. Some theories consider age-related physiological deterioration and mortality to be a reflection of a progressive imbalance in this relationship (Kirkwood, 2002). It is questionable, however, whether such an explanation can apply to autotrophic organisms such as green plants. In general, plants are material and energy rich, capturing resources in a manner that has been described as promiscuous and even pathological (Harper, 1977; Thomas & Sadras, 2001). Resource allocation between repair and growth activities is therefore likely to mean something quite different for plants compared with animals.

Nevertheless, much current research activity has become centred on caloric restriction (Mair & Dillin, 2008), an area of resource availability bearing on age-related deterioration, where plants and animals may share functions and mechanisms. The headline-grabbing drive behind biomedical interest in this subject is, of course, the clinical and sociological problem of obesity; however, although it seems clear that excess calories are bad for life expectancy, the contrary idea that restricting calorie intake can extend life is more controversial (see Mattison *et al.*, 2012). Here, the discussion considers recent research that suggests a cell biological mechanism that operates in animals, fungi and plants to link nutritional state, growth, deteriorative ageing and autophagy. Autophagy, a major catabolic process of eukaryotic cells, participates in repair and maintenance activities by degrading and recycling damaged macromolecules and organelles, and has been implicated in the incidence of diverse age-related physiological and pathological changes (Vellai *et al.*, 2009).

Figure 7 summarizes the mechanism of autophagy. The expression of autophagy genes (*ATG*s) and the activities of ATG proteins are controlled by a network of regulatory kinases. Autophagy begins with a phase of vesicle induction, followed by vesicle expansion, docking and fusion with the tonoplast and, finally, digestion. The signalling pathways that direct the cell into autophagy converge on the expression and activity of ATG1 and ATG13. ATG1 is phosphorylated by protein kinase A, a negative regulator of autophagy, which is itself regulated by the kinase TOR. Phosphorylation of ATG1 releases it into the cytosol from the pre-autophagosomal structure (PAS), a complex with ATG13. Expression of *ATG1* and *ATG13* is activated by the transcriptional regulator GCN4 (General Control Nonderepressible4). GCN4 is regulated by phosphorylated elongation initiation factor 2α, which, in turn, is activated by the starvation-induced kinases GCN2 and AMK (AMP-activated kinase). In addition to its role in *ATG* expression, AMK stimulates autophagy by phosphorylating and inactivating TOR. A vesicle is formed by coalescence of PASs into a cage that captures a portion of the cytoplasm. Expansion of the autophagosome vesicle is aided by many ATG proteins. Ultimately, the autophagosome is linked to cytoskeletal microtubules and targeted to the vacuole (Bassham, 2007; Kundu & Thompson, 2008; Liu & Bassham, 2012).

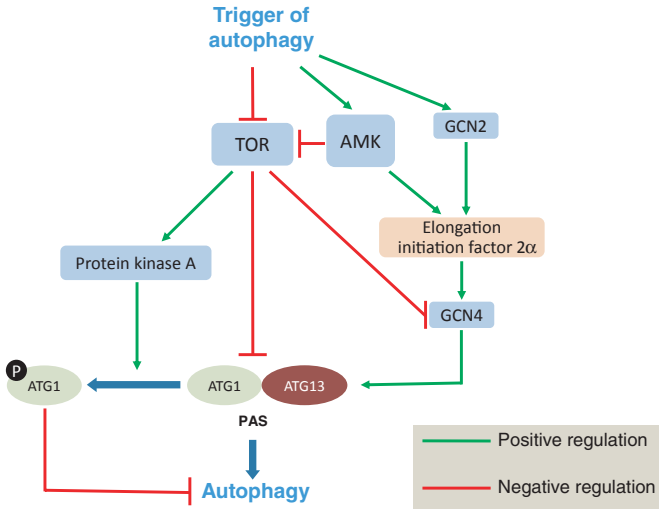

Fig. 7 Target of Rapamycin (TOR) network regulating autophagy. Multiple signalling pathways converge on the expression and activity of autophagy genes *ATG1* and *ATG13*. TOR, protein kinase A, AMK (AMP-activated kinase) and GCN2 (General Control Nonderepressible2) are kinases operating in autophagy signalling pathways. Elongation initiation factor 2α and the transcription factor GCN4 regulate the expression of *ATG1* and *ATG13*. PAS (pre-autophagosomal structure), the complex containing ATG1 and ATG13, is the precursor of the autophagosome.

TOR is emerging as a point of convergence for a regulatory network coordinating energy status, sugar content, N availability, cell fate and longevity, although the extent to which the architecture of the TOR signalling system of animals and fungi is conserved in plants is not yet completely settled (Baena-González & Sheen, 2008). In their discussion of the relationship of ageing to growth, Blagosklonny & Hall (2009) stated that 'life-promoting TOR signalling seems also to contain seeds of death' – that is, it is a true regulator of ageing as defined in section II.4. TOR is absolutely essential for development, controlling cell growth and proliferation by altering mRNA translation (Oldham & Hafen, 2003). However, on completion of differentiation, it causes age-related deterioration, and lifespan can be increased by reducing TOR signalling (Mair & Dillin, 2008). In plants, mutants in autophagy show accelerated senescence and cell death (Thompson *et al.*, 2005), and silencing TOR kinase induces early leaf yellowing and sugar accumulation (Deprost *et al.*, 2007), probably through the interaction of TOR with the hexokinase and SnRK1 signalling pathways (Fig. 6). The case for TOR as a nutrient-sensing regulator of plant growth and senescence is reasonably strong, but definitive evidence for a role in deteriorative ageing remains to be established (Liu & Bassham, 2012).

5. Ageing in relation to size and modular structure

Senescence and ageing in plants diverge radically from the equivalent processes in animals because of differences in organization and development. In particular, the animal body comprises distinct germline (gamete-producing) and soma (nonreproductive) cells, whereas there is no differentiation into germline and soma in plants (Walbot & Evans, 2003). Also of special significance for ageing is the body plan. Unlike animals, plants develop by the open-ended repetitive proliferation of homologous structural units. The vast range of plant form and life cycle arises by variations in the spatial arrangement of phytomers, or in the timing of initiation, development and senescence of these modules (Sachs *et al.*, 1993; Borges, 2009). Individual plants behave as competing populations of genetically equivalent organs integrated through source–sink interactions, a mode of organization that ensures adaptation to heterogeneous environments.

Because a single plant is structured as a population of phytomers, it appears to be more like a colonial organism, such as a coral, rather than an individual (Thomas, 2003). Closer examination reveals this resemblance to be perhaps superficial at best. Ageing has been measured in some colonial animals. For example, cohorts of *Hydra* polyps in the asexual phase exhibit extremely low mortality rates

and no significant decline in reproductive capacity over periods of years (Martinez, 1998). Once sexually differentiated, however, *Hydra* populations display age-related declines in the capacities for food capture, contractile movements and reproduction, and an exponential increase in mortality rate (Yoshida *et al.*, 2006). A recent study found evidence of deteriorative ageing (measured as asexual propagation rate, telomerase activity and relative telomere length) in 7–12-yr-old asexual lineages of the colonial ascidian *Diplosoma listerianum*. Significantly, total rejuvenation permitting indefinite propagation and growth was achieved by passage between sexual generations (Sköld *et al.*, 2011). Yoshida *et al.* (2006) considered the behaviour of colonial metazoans to conform to the expectations of the pleiotropism theory of ageing (Williams, 1957), which can be discounted as an explanation of age-related decline in trees (Mencuccini *et al.*, 2005). Meiosis and sexual reproduction reset the ageing clock; something similar happens during the mitotic cycle in meristems.

Most perennial plants get bigger as they grow older, and of course organisms that are large and old will also be weatherbeaten, the visible signifier of ageing. Size has physiological consequences too. It is a significant factor in the development of symptoms of ageing-related deterioration (Day *et al.*, 2002); however, although there is a general inverse relationship between growth rate and increasing age class in trees, growth rates in the oldest and largest trees are frequently sustained for the remainder of their lives (Johnson & Abrams, 2009). Size affects nutrient allocation and the ratio of photosynthesizing to respiring tissues (Mencuccini *et al.*, 2005). As a tree grows, increasing distances between the roots and the extremities of the crown impose increasing stress on the hydraulic functions of the vascular system (Woodruff & Meinzer, 2011). Ryan & Yoder (1997) considered this to be more likely than nutrient allocation, respiratory patterns or increasing mutational load as a determinant of tree growth, form and age-related physiological deterioration. Declining photosynthesis in older ponderosa pine (*Pinus ponderosa*) and other species has been shown to be associated with decreases in the flow of water and sap in the whole tree (Hubbard *et al.*, 1999). However, Lanner & Connor (2001) found no evidence for age-related deterioration in vascular function of bristlecone pines over the age range 23–4713 yr. Ishii *et al.* (2007) suggested that modular structure allows trees to sustain crown productivity and longevity by a process of adaptive reiteration, which decreases the ratio of respiration to photosynthesis, enhances hydraulic conductance to newly developing foliage, reduces nutrient loss, rejuvenates apical meristems and increases lifetime reproductive output.

6. Patterns of annuality and perenniality

The life expectancy outcome for a plant is the product of the modular, fractal design of the plant body and the quantitative association between the making, senescence and death of structural modules. The diversity of plant forms can be related to different combinations of vegetative growth, apical determinacy, senescence and persistence of dead tissues (Thomas *et al.*, 2000). This may be illustrated with some simple animations (Supporting Information Videos S1–S4).

- In Video S1, shoot growth is represented as the repeated production of new structural modules (colour coded green for pre-senescent, yellow for senescing). The animation shows the vegetative phase of the plant's life cycle, during which branches develop from axillary buds and grow out as new shoots. The wave of growth and branching is pursued by a wave of senescence. As long as the senescing zone keeps its distance from the proliferating meristems at the shoot apices, the plant as a whole survives. Retention of at least one viable vegetative apex is also the key to the survival of extreme environmental challenges. For example, intercalary meristem organization is a morphological adaptation that gives perennial grasses the capability to withstand grazing and fire.
- The second example (Video S2) follows the pattern of the first, but, in this case, the senescent modules are lost by shedding, death and decay. This is what happens in populations of creeping clonal plants. As the wave of senescence and death moves through the plant body, individual branches become isolated as genetically identical ramets and continue to develop as separate individuals. As we have seen (Table 1), this mode of development allows some clonal herbaceous species to achieve immense ages. Horizontal perenniality enables members of the clone to move around the environment – an effective way of finding and systematically exploiting new resources.
- Video S3 is a variant of horizontal perenniality in which senescent modules, instead of disappearing, become lignified and persist as woody tissue (colour coded brown). The result is a tree – an example of vertical perenniality.
- Video S4 shows the consequence of a change in the type of structural module from vegetative to reproductive during growth. In this case, a terminal flower (blue) is produced at the apex of each branch. Again, a wave of senescence advances through the plant body, but, this time, it completely overtakes the production of new modules. The result is monocarpy – suicidal reproduction. Mortality in many relatively short-lived polycarpic perennials resembles that of monocarpic annuals or biennials in occurring without any sign of age-related decline in fitness.

Looked at in this way, the differences between annuals, biennials and perennials, or between monocarps and polycarps, are essentially a matter of the relative timings and rates of initiation and execution of programs for organogensis and senescence. It is easy to imagine selection for quantitative variations in these processes contributing significantly to the evolutionary origins of the great diversity of plant habit and life history.

VII. Last words

As this discussion reaches its terminal stage, it is perhaps appropriate to return to the question of biological ageing and what it means for green plants. The term stress, as used by an engineer, describes an environmental influence that invokes a corresponding strain. Time is stressful for living organisms. It is customary to think about responses to a nonoptimal environment in terms of specific stress genes, stress proteins and stress metabolites (Shulaev *et al.*, 2008; Hirayama & Shinozaki, 2010). Ageing is the *time-stress response*. A camera stops time: the baby in

the photograph remains a baby. Time is really *thermal time* and can be slowed with a refrigerator: peas at ambient temperature deteriorate faster than frozen peas. Time can be frustrated by playing dead: a leafless winter dormant tree looks like a dead tree.

As an individual organism and its substructures age, metabolic and physiological changes will express the activities of time-stress genes and their products. The strategies open to viable organisms and their components, if they are to avoid succumbing to mortality under the influence of the ever-ticking clock of entropy, are those that apply to environmental stresses in general: avoidance, resilience or adaptation. In the specific case of time-stress, the options are: (1) to outrun it – in other words, to grow, develop and differentiate; (2) to resist it – through building in structural and functional durability and by repairing wear and tear; or (3) to pre-empt it – using programmed senescence as a developmental and adaptive resource so that ageing and death take place on the organism's own terms, so to speak (Thomas, 1994). By adopting these measures, plants have been able to realize Woody Allen's aspiration and organize their lives so that they are not there when death happens. The capacity to control the ageing process is one of the essential attributes that enables plants to thrive in almost every habitat on Earth.

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Supporting Information

Additional supporting information may be found in the online version of this article.

Video S1 Growth and senescence in an idealized plant.

Video S2 Growth and senescence in a clonal horizontal perennial.

Video S3 Growth and senescence in a woody vertical perennial.

Video S4 Growth and senescence in a monocarpic plant.

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