PLANT SENESCENCE

Howard Thomas
IBERS, Edward Llwyd Building, Aberystwyth University, Ceredigion, SY23 3FG, UK

Keywords: Plant, senescence, ageing, longevity, death, gene, cell, tissue, leaf, flower, fruit, embryo, seed, root, germination, ripening, maturation, source, sink, season, stress, disease, crop, yield, food, waste

Contents

Summary

Senescence is a terminal stage of plant development. It often, but not invariably, ends in the death of cells, tissues, organs or the whole plant. At the cell level there are a number of different senescence pathways, most of which are autolytic, that is, the genetic and biochemical events originate within the senescing cell itself. Nucleus, vacuole, plastids and mitochondria interact during cell senescence. Up to the point where organelle integrity is lost, some kinds of senescence may be halted, extended or even reversed by various treatments, but beyond this threshold there is a rapid decline in viability leading to death. Developmental cell senescence and death occur during differentiation of xylem, floral tissues, embryos and seeds. Leaves, fruits and some flowers lose chlorophyll during senescence as chloroplasts differentiate into pigmented plastids. The products of chlorophyll breakdown are deposited in the cell vacuole. Proteins and nucleic acids are hydrolysed and the nitrogen and phosphorus liberated are exported from the leaf to sink tissues. Fruit ripening shares a number of regulatory and biochemical features with leaf and flower senescence. Senescence contributes to root turnover, an important factor in global carbon balance. Plants and their parts often must attain maturity before they are able to respond to signals that induce senescence. Floral induction and seed formation stimulate senescence. In monocarpic species the entire plant undergoes reproductive death. Polycarpic plants flower repeatedly during their lifetimes, and show no clear relationship between senescence and longevity. Senescence is a strategic and tactical response to seasonal and unpredictable stresses, including changing daylength, flooding, drought, excessive light, darkness, nutrient limitation and disease. The timing of senescence in relation to carbon capture and nutrient remobilization is a major determinant of crop yield. Senescence and related processes account for significant postharvest losses and food wastage.

1. What is Plant Senescence?

1.1. Terminology

1.1.1. Senescence

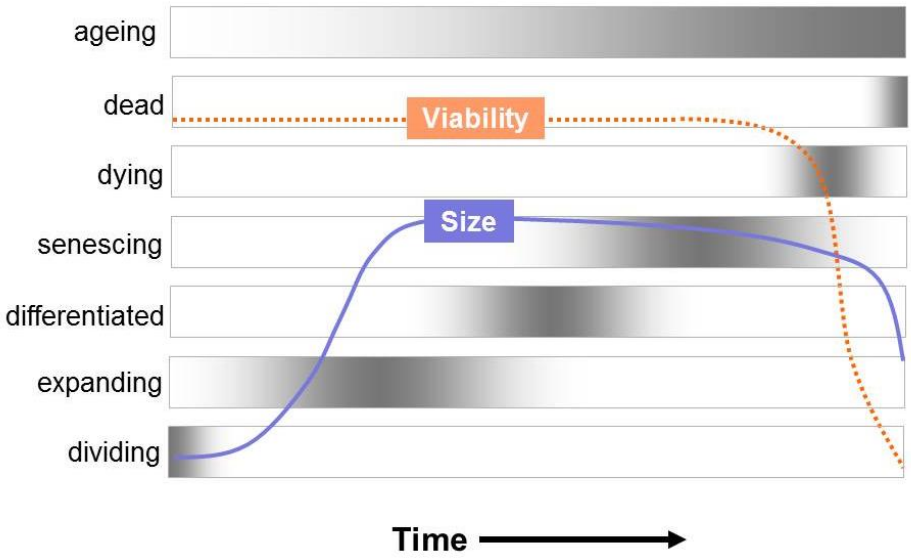

Figure 1. Senescence and related events in the life-history of a plant and its parts. The sequence of events is reiterated at all levels in the hierarchy of plant organization, from cells, tissues, and organs through to individual plants, communities, and even whole floras. Note that ageing includes, but is not limited or defined by, terminal senescence and death phases.

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 (Figure 1). 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 Latin *senescere* to grow old) and its association with senility and the medical problems of human ageing.

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 follows the completion of growth, is absolutely dependent on cell viability and which may or may not be succeeded by death.

1.1.2. Ageing

Ageing (sometimes also spelled 'aging') is another term, like senescence, that has become associated with deterioration. In the general biological context, however, it should be considered to refer to changes that occur with time, and therefore to embrace

the time-based processes of growth and differentiation as well as maturity and senescence.

1.1.3. 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". Application of the term 'cell death' to the physiology of senescence, though widespread, seems inappropriate. By definition, changes that occur in dead cells are post-mortem and non-biological. Biologists studying terminal events in development need to distinguish between the regulated activity of viable biological structures and the pathological outcomes of organic collapse.

1.1.4. Program

The expressions 'senescence program' and 'programmed cell death' are extensively used. 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 lifecycle, proceeds according to a timetable determined by developmental and environmental factors and mediated by a genetic program.

1.2. Relationship between Senescence 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 increase in dry mass. 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. It is the means by which resources are recycled from obsolete body parts to new developing structures. Finally variations on the senescence program theme have been shaped by evolution to give rise to a diversity of structures within the angiosperm lifecycle.

1.3. Relationship between Senescence and Ageing

As discussed in Section 1.1.2, the changes with time that fall under the general term ageing are not necessarily deteriorative, although in the long run errors will accumulate and living tissues will show signs of wear and tear. 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. A related view is that, since ageing and eventual death are thermodynamically unavoidable, senescence has evolved as a developmental strategy

that pre-empts the inevitable, enabling the individual plant to control its own viability and integrity over the course of the lifecycle. This is sometimes called the Samurai Law of Biology ('it is better to die than to be wrong'). Such is the current state of knowledge that it is difficult to resolve the interconnections between ageing and senescence mechanisms, and they will continue to be fascinating areas of research and speculation.

1.4. Relationship between Senescence and Death

Senescence and death differ fundamentally: by definition, senescing tissue is viable, dead tissue is not. There is a transitional condition between the two states during which metabolism modulates into abiotic chemistry. This terminal period is often rapid and always irreversible. The preceding senescence phase is usually comparatively extended. Cell membranes and organelles remain intact, and organs stay turgid. In some cases, notably the senescence of green (mesophyll) cells 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 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*; Figure 2). Senescence proceeds from the tip downward, and towards the veins from interveinal regions of the lamina. This heterogeneity within a single structure can make it difficult to disentangle senescence-phase, terminal and post-mortem events.

Figure 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 expression of a gene encoding an enzyme of nitrogen mobilization in senescence.

2. Senescence of Cells and Tissues

2.1. Mechanisms of Cell Senescence

2.1.1. The Growth Curve

Senescence of tissues, organs and individuals is an expression of processes going on at the cell level. In terms of comparative biology, a cell is a cell is a cell and senescence

may be expected to figure amongst the common features of structure and physiology. The basic characteristics of cell growth and proliferation are shared across the taxa. Senescence is properly recognized as a normal and even essential feature of the post-mitotic phase of the cell life-cycle and is immediately preceded by (and sometimes partially overlaps with) the growth period (Figure 1). Typically the pattern of growth in biological systems is density-dependent, beginning slowly when cell mass is small, reaching a maximal rate when density is optimal with respect to metabolic and environmental constraints, declining as limiting external and internal factors become increasingly influential, and finally approaching maximal size asymptotically. Such sigmoidal behavior is usually described mathematically by some variation of the logistic equation. The generalized, archetypal logistic-type function is:

$$G = \alpha + \beta y^t$$

where mass or size, G, is related to time t by three coefficients or groups of coefficients. y is always e^{-c} and represents exponential growth with a rate constant c. α is the value of G at the asymptote, or a transformation of it, and β refers to the initial value or state of the system. To fit this kind of function is virtually impossible without computers, and even then it took until the late sixties to solve the problem of fitting the generalized logistic with statistical rigor. Nowadays anyone can do it with absolute confidence that the error estimates all conform to the laws of non-linear estimation.

2.1.2. Senescence as a Normal Phase of Cell Lifespan

Growth curves are of interest in the study of senescence because a cell population in the period of decline between the point of maximal growth rate and the stationary phase is sometimes regarded as ageing. The relationship between ageing and senescence is discussed in Section 1.3. It is characteristic of plants that the logistic-type pattern seen in cell cultures is observed at progressively higher levels of organization up to organ, individual and even beyond (Figure 1). It is reasonable to conjecture that intrinsic sigmoidicity, expressing the interplay between the potential for growth and the progressive imposition of limitations, is an important factor in triggering the senescence syndrome. It follows that the template for the development of any and every plant cell, from any and every tissue and organ, has a built-in senescence module attuned to the growth curve. The curves themselves will vary in their proportions, the onset and rate characteristics of their ageing phases will differ accordingly and so will the senescence response. 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. On the other hand, there is evidence that the stomatal guard cells of some species do not initiate a recognisable 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.

2.1.3 Autolysis during Cell Senescence

Genetically programmed cell senescence is a form of suicide. Although there are instances of plant cells being killed by their neighbors, and a severe external stress can cause fatal trauma, in most cases the senescence process is autolytic; that is, cytoplasm

is both the source and the location of the degradative activities that ultimately bring about its own death (Figure 3). During autolysis, macromolecules are cleaved by lytic enzymes into oligomeric fragments and ultimately into monomers. As the location of peptidases, nucleases, peroxidases and other hydrolytic and oxidative enzymes, the vacuole has an essential function in most kinds of autolytic cell senescence. Macromolecules are either engulfed by, or transported across, the tonoplast (the bounding membrane of the vacuole) and degraded in the vacuolar space, or else the tonoplast ruptures, flooding the cytosol with lytic enzymes and rapidly killing the cell. Autolysis in plants takes a number of different forms, some of which are listed in Table 1.

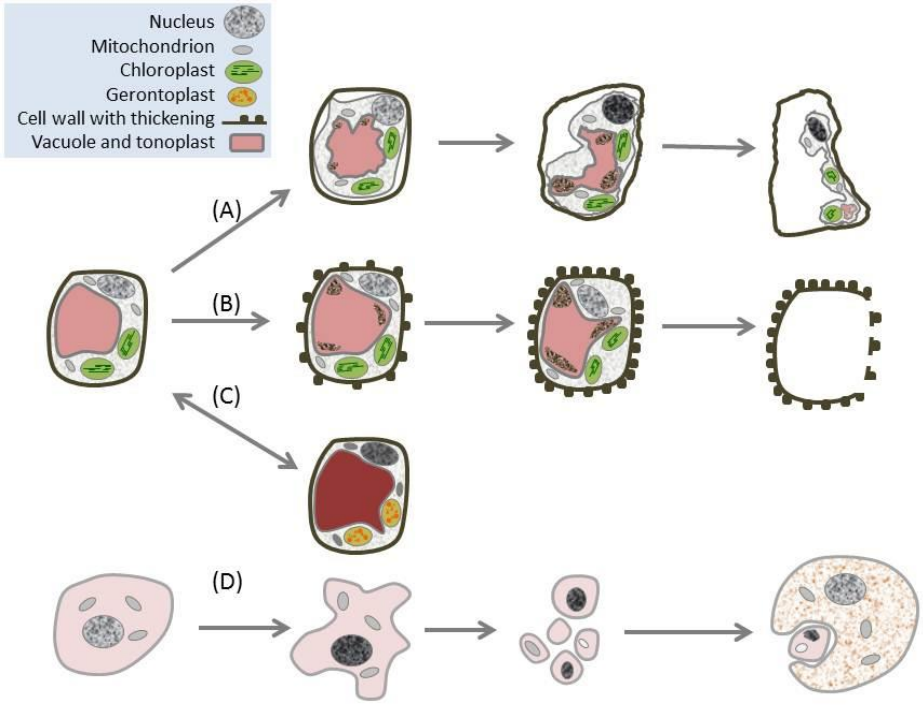

Figure 3. Cytological features of three modes of programmed senescence of plant cells compared with the apoptotic pathway of animal cell death. (A) Hypersensitive cell death, a resistance response to pathogen attack. Condensation and cleavage of DNA in the nucleus precedes vacuole disruption and blebbing of tonoplast and plasma membranes; the process ends with destruction of organelles, plasma membrane collapse and leakage of the dead cell's contents. (B) Tracheary element differentiation, an example of developmental cell senescence and death. Swelling and rupture of the vacuole happens as the cell walls undergo secondary thickening and restructuring. Nuclear DNA fragmentation occurs in the later stages, after vacuolar collapse. Finally autolysis eliminates the remaining cytoplasm, leaving an empty cell enclosed by a thickened wall. (C) Mesophyll cell senescence, an example of transdifferentiation in which the change in plastid structure reflects the functional transition from provision of assimilated carbon to source of salvaged nitrogen and phosphorus. The senescence process is distinct from cell death and in some cases is reversible. (D) Apoptosis in animal cells. Chromatin condensation and fragmentation are early morphological events. The plasma membrane is disrupted and cell contents are repackaged into apoptotic bodies, which are finally engulfed by neighboring macrophage cells.

Type Of Cell Senescence	Characteristics	Discussed Further
Autophagy	A form of cell senescence characterized by the regulated assembly of specific lytic structures that break down cytoplasm under the control of signal cascades and differential gene expression.	Autophagy also occurs in animals and is distinct from apoptosis. The mechanism is discussed in Section 2.1.5.
Transdifferentiation	Remodelling of structure and function of cytoplasmic organelles in post-mitotic cells. Only in the final stages is there loss of integrity and viability as lytic processes take control.	Senescence of green cells of leaves or pigmented tissues of fruits. See Sections 3.1, 3.3.
Hypersensitivity	A kind of cauterization or containment reaction to attempted infection by a pathogen.	Related to some kinds of spontaneous lesion formation in mutants. Hypersensitivity is discussed in further detail in Section 5.3.
Lysigeny	The formation of glands, channels and secretory ducts by the disintegration of cytoplasm.	Section 2.2. Air spaces formed in roots in response to low oxygen stress are lysigenous in origin.
Schizogeny	Senescence of schizogenous cells is a process of cell separation during which the middle lamella of the cell wall breaks down.	Section 2.2.
Altruism	Self-sacrificial elimination of cells for the benefit of the whole plant.	An example is corolla cell autolysis in fertilized flowers, which reduces competition with unfertilized blooms for the attention of pollinating insects. Section 3.2.
Types of Animal Cell Death		
Apoptosis	Type of programmed cell death characterized by blebbing, cell shrinkage, nuclear fragmentation, chromatin condensation DNA fragmentation.	Section 2.1.4.
Necrosis	Traumatic cell death resulting from acute cellular injury.	Non-physiological mortality of plant cells in response to trauma resembles necrosis in animals

Table 1. Modes of autolytic cell senescence in plants. Apoptosis and necrosis, the two principal forms of cell death in animals, are listed for comparison.

Nucleases catalyse the hydrolysis of nucleic acids. They may be specific for RNA (ribonuclease, RNase) or for DNA (DNase) or they may be bifunctional, able to use either nucleic acid as a substrate. Exonucleases remove nucleotide monomers sequentially from the ends of the substrate molecule; endonucleases hydrolyse linkages between monomers within the polynucleotide chain. Proteins are hydrolyzed by peptidases (also called proteases or proteinases). Endopeptidases, which cleave internal peptide bonds in their protein substrates, are particularly important in cell senescence. Cysteine endopeptidases, so-called because each has a reactive cysteine residue in its catalytic centre, are active during plant senescence and animal cell death (apoptosis). Caspases, the cysteine proteases responsible for apoptosis, have little or no structural similarity to the functionally equivalent proteases of senescing plant cells. The latter are sometimes referred to as metacaspases and include a class of cysteine endopeptidase referred to as vacuolar processing enzymes (VPEs). Among other types of protease with roles in plant cell senescence are serine proteases, metalloproteases, ATP-dependent proteases and components of the ubiquitin/proteasome system (UbPS).

-

-

-

Bibliography

Archetti M., Döring T.F., Hagen S.B., Hughes N.M., Leather S.R., Lee D.W., Lev-Yadun S., Manetas Y., Ougham H.J., Schaberg P.G., H Thomas H. (2009). Unravelling the evolution of autumn colors - an interdisciplinary approach. *Trends in Ecology and Evolution* 24, 166-173 [Critical review of the major hypotheses concerning the biological significance of changes in leaf color during autumnal senescence]

Armstead I., Donnison I., Aubry S., Harper J., Hörtensteiner S., James C., Mani J., Moffet M., Ougham H., Roberts L., Thomas A., Weeden N., Thomas H., King I. (2007). Cross-species identification of Mendel's I locus. *Science* 315, 73. [Identification of SGR by positional cloning, comparative genomics and functional testing]

Bassham D.C. (2007). Plant autophagy—more than a starvation response. *Current Opinion in Plant Biology* 10, 587–593 [A detailed account of the regulation and mechanism of autophagy]

Bhalerao R., Keskitalo J., Sterky JF., Erlandsson R., Björkbacka H., Birve S.J., Karlsson J., Gardeström P., Gustafsson P., Lundeberg J., Jansson S. (2003). Gene expression in autumn leaves. *Plant Physiology* 131, 430-442 [Survey of genes associated with senescence of a field-grown aspen tree in relation to environmental signals at the onset of autumn]

Bond B.J. (2000). Age-related changes in photosynthesis of woody plants. *Trends in Plant Science* 5, 349-353 [Discussion of the roles of genetics, size, changing conditions and cumulative environmental stress in aging trees]

Breeze E., Harrison E., Page T., Warner N., Shen C., Zhang C., Buchanan-Wollaston V. (2008) Transcriptional regulation of plant senescence: from functional genomics to systems biology. *Plant Biology* 10 (Supplement 1), 99-109 [Application of network analysis to identify interactions between regulatory components and pathways in leaf senescence]

Buchanan-Wollaston V., Earl S., Harrison E., Mathas E., Navabpour S., Page T., Pink D. (2003). The molecular analysis of leaf senescence - a genomics approach. *Plant Biotechnology Journal* 1, 3–22 [Application of global transcription screening to the study of leaf senescence]

Buchanan-Wollaston V., Page T., Harrison E., Breeze E., Lim P.O., Nam H.G., Lin J-F., Wu S-H., Swidzinski J., Ishizaki K., Leaver C. (2005). Comparative transcriptome analysis reveals significant differences in gene expression and signalling pathways between developmental and dark/starvation-induced senescence in *Arabidopsis*. *Plant Journal* 42, 567-585 [Genetic dissection of the responses of leaf senescence to different triggers]

Courtois-Moreau C.L., Pesquet E., Sjodin A., Muniz L., Bollhoner B., Kaneda M., Samuels L., Jansson S., Tuominen H. (2009). A unique program for cell death in xylem fibers of *Populus* stem. *Plant Journal* 58, 260–274 [A detailed study showing that development of xylem fibers, unlike tracheary elements, culminates in autophagic elimination of cell contents]

Donnison I.S., Gay A.P., Thomas H., Edwards K.J., Edwards D., James C.L., Thomas A.M., Ougham H.J. (2007). Modification of nitrogen remobilisation, grain fill and leaf senescence in maize (*Zea mays* L.) by transposon insertional mutagenesis in a protease gene. *New Phytologist* b, 481–494 [Gene knockout shows a regulatory role for VPE in senescence and development]

Eissenstat D.M., Yanai R.D. (1997). The ecology of root lifespans. *Advances in Ecological Research* 27, 1-60 [Review of root turnover and the part played by senescence]

Encarnacion M., Lara B., Garcia M-C.G., Fatima T., Ehness R., Lee T.K., Proels R., Tanner W., Roitsch T. (2004). Extracellular invertase is an essential component of cytokinin-mediated delay of senescence. *Plant Cell* 16, 1276–1287 [The role of sugar sensing in the hormonal regulation and source-sink relations of leaf senescence]

Farage-Barhom S., Burd S., Sonego L., Perl-Treves R., Lers A. (2008). Expression analysis of the BFN1 nuclease gene promoter during senescence, abscission, and programmed cell death-related processes. *Journal of Experimental Botany* 59, 3247-3258 [Study of the transcriptional regulation of a nucleic acid degrading enzyme]

Feller U., Anders I., Mae T. (2007). Rubiscolytics: fate of Rubisco after its enzymatic function in a cell is terminated. *Journal of Experimental Botany* 59, 1615-1624 [Critical review of the options for catabolism of plastid proteins during senescence]

Gan S., editor (2007). *Annual Plant Reviews Volume 26: Senescence Processes in Plants*. Oxford: Blackwell [Collection of reviews of all aspects of plant senescence]

Giovannoni J.J. (2007). Fruit ripening mutants yield insights into ripening control. *Current Opinion in Plant Biology* 10, 283–289 [Genetic analysis of tomato fruit ripening]

Guerra J.C.P., Coussens G., De Keyser A., Rycke R., De Bodt S., Van De Velde W., Goormachtig S., Holsters M. (2010). Comparison of developmental and stress-induced nodule senescence in *Medicago truncatula*. *Plant Physiology* 152, 1574–1584 [Detailed analysis of gene expression in senescing cells of a legume species with indeterminate root nodules]

Gunawardena, A.H.L.A.N. (2008). Programmed cell death and tissue remodeling in plants. *Journal of Experimental Botany* 59, 445-451 [A review of the ways in which plants develop holes, airspaces, cavities and tubes]

Gunawardena A., Pearce D.M., Jackson M.B., Hawes C.R., Evans D.E. (2001) Characterisation of programmed cell death during aerenchyma formation induced by ethylene or hypoxia in roots of maize (*Zea mays* L.). *Planta* 212, 205–214 [Cell biology of a stress-triggered cell senescence response]

Gunawardena A.H.L.A.N., Greenwood J.S., Dengler N.G. (2007). Cell wall degradation and modification during programmed cell death in lace plant, *Aponogeton madagascariensis* (Aponogetonaceae). *American Journal of Botany* 94, 1116–1128 [A dramatic example of hole formation during normal leaf development]

Hatsugai N., Kuroyanagi M., Nishimura M., Hara-Nishimura I. (2006). A cellular suicide strategy of plants: vacuole-mediated cell death. *Apoptosis* 11, 905–911 [Compares and contrasts cell death mechanisms in plants and animals]

Hatsugai N., Kuroyanagi M., Yamada K., Meshi T., Tsuda S., Kondo M., Nishimura M., Hara-Nishimura I. (2004). A plant vacuolar protease, VPE, mediates virus-induced hypersensitive cell death. *Science* 305, 855-8 [Function of 'metacaspase' in responses to infection by a pathogen]

Jansson S., Thomas H. (2008). Senescence: developmental program or timetable? *New Phytologist* 179, 575–579 [Critically examines the concept of a program as applied to the regulation and execution of the senescence syndrome]

Jing H.C., Schippers J.H., Hille J., Dijkwel P.P. (2005). Ethylene-induced leaf senescence depends on age-related changes and *OLD* genes in *Arabidopsis*. *Journal of Experimental Botany* 56, 2915-23 [Genetic basis of a change from juvenile to mature responses to senescence induction by ethylene during *Arabidopsis* development]

Jones R., Ougham H., Thomas H., Waaland S. (in press) *The Molecular Life of Plants*. Chichester: Wiley [Textbook covering all aspects of plant development, including senescence and ripening, sexual and vegetative reproduction, stress responses and seed germination]

Jovanovic M., Lefebvre V., Laporte P., Gonzalez-Rizzo S., Lelandais-Brière C., Frugier F., Hartmann C., Crespi M. (2008). How the environment regulates root architecture in dicots. *Advances in Botanical Research* 46, 35-74 [Reviews the factors determining root development and survival]

Keskitalo J., Bergquist G., Gardeström P., Jansson S. (2005). A cellular timetable of autumnal senescence in aspen. *Plant Physiology* 139, 1635-1648 [Inventory of physiological, cytological and molecular events before and after the induction of senescence in a field-grown aspen tree]

Kim J.C., Laparra H., Calderon-Urrea A., Mottinger J.P., Moreno M.A., Dellaporta S.L. (2007). Cell cycle arrest of stamen initials in maize sex determination. *Genetics* 177, 2547-2551 [Genetic regulation of unisexual flower development in maize]

Kozela C., Regan S. (2003). How plants make tubes. *Trends in Plant Science* 8, 159-164 [Review of lysigenous and schizogenous cell senescence]

Lam E. (2004). Controlled cell death, plant survival and development. *Nature Reviews of Molecular Cell Biology* 5, 305-315 [Compares different modes of cell senescence and death in plants and animals]

Lanner R.M., Connor K.F. (2001) Does bristlecone pine senesce? *Experimental Gerontology* 36, 675-685 [Surveys the data on the physiology of one of the longest-lived of plant species and concludes that it does not]

Lim P.O., Kim H.J., Nam H.G. (2007). Leaf senescence. *Annual Review of Plant Biology* 58, 115–36 [Review of the molecular regulation of senescence]

Liu F., VanToai T., Moy L.P., Bock G., Linford L.D., Quackenbush J. (2005). Global transcription profiling reveals comprehensive insights into hypoxic response in *Arabidopsis*. *Plant Physiology* 137, 1115-1129 [Genomics applied to molecular responses to oxygen depletion]

Liu Y., Schiff M., Czymmek K., Tallóczy Z., Levine B., Dinesh-Kumar S.P. (2005). Autophagy regulates programmed cell death during the plant innate immune response. *Cell* 121, 567–577 [A study showing that a key ATG is expressed during the HR]

Lorrain S., Lin B., Auriac M.C., Kroj T., Saindrenan P., Nicole M., Balagué C., Roby D. (2004). VASCULAR ASSOCIATED DEATH1, a novel GRAM domain–containing protein, is a regulator of cell death and defense responses in vascular tissues. *Plant Cell* 16, 2217–2232 [Describes a lesion mutant impaired in both vascular development and pathogen defense response]

Lorrain S., Vailleau F., Balagué C., Roby D. (2003). Lesion mimic mutants: keys for deciphering cell death and defense pathways in plants? *Trends in Plant Science* 8, 263-71 [Survey of spontaneous death mutants and the regulatory pathways they reveal]

Lozano R., Giménez E., Cara B., Capel J., Angosto T. (2009). Genetic analysis of reproductive development in tomato. *International Journal of Developmental Biology* 53, 1635-1648 [Molecular model of fruit ripening based on analyses of tomato]

Makino A., Mae T., Ohiro K. (1984). Relation between nitrogen and ribulose-1,5-bisphosphate carboxylase in rice leaves from emergence through senescence. *Plant and Cell Physiology* 25, 429-437 [Definitive study of Rubisco turnover]

McCullagh P., Nelder J.A. (1989). *Generalized Linear Models*, 2nd edition. London: Chapman and Hall [Comprehensive account of the theory and practice of modeling growth]

Munné-Bosch S. (2008). Do perennials really senesce? *Trends in Plant Science* 13, 216-220 [Discusses the evidence relating death of the whole plant and senescence of its parts, with special reference to perennial species]

Mur L.A.J., Aubry S., Mondhe M., Kingston-Smith A., Gallagher J., Timms-Taravella E., James C., Papp I., Hörtensteiner S., Thomas H., Ougham H. (2010). Accumulation of chlorophyll catabolites photosensitizes the hypersensitive response elicited by *Pseudomonas syringae* in *Arabidopsis*. *New Phytologist* 188, 161–174 [Shows the chlorophyll catabolism pathway to be a link between senescence and the HR]

Mur L.A.J., Kenton P., Lloyd A.J., Ougham H., Prats E. (2008). The hypersensitive response; the centenary is upon us but how much do we know? *Journal of Experimental Botany* 59, 501-520 [Review of the nature of the HR]

Navarre D.A., Wolpert T.J. (1999). Victorin induction of an apoptotic/senescence-like response in oats. *Plant Cell* 11, 237-49 [Example of senescence triggered by a host-specific toxin secreted by a pathogen]

Ohashi-Ito K., Fukuda H. (2010). Transcriptional regulation of vascular cell fates. *Current Opinion in Plant Biology* 13, 670–676 [Describes current insights into regulation of vascular development by hormones, signaling cascades and transcription factors]

Ougham H., Hörtensteiner S., Armstead I., Donnison I., King I., Thomas H., Mur L. (2008). The control of chlorophyll catabolism and the status of yellowing as a biomarker of leaf senescence. *Plant Biology* 10 (Supplement 1), 4-14 [Mechanism of chlorophyll breakdown and its function in senescence]

Parfitt J., Barthel M., Macnaughton S. (2010). Food waste within food supply chains: quantification and potential for change to 2050. *Philosophical Transactions of the Royal Society, London. B*. 365, 3065–3081. [A recent survey identifying the postharvest stage in developing countries and the retail and consumer level in developed economies as points where major spoilage and waste occurs]

Parish R.W., Li S.F. (2010). Death of a tapetum: a programme of developmental altruism. *Plant Science* 178, 73–89 [Discusses the relationship between pollen development and the senescence and death of tapetum cells]

Perez-Amador M.A., Abler M.L., De Rocher E.J., Thompson D.M., van Hoof A., LeBrasseur N.D, Lers A., Green P. (2000). Identification of BFN1, a bifunctional nuclease induced during leaf and stem senescence in *Arabidopsis*. *Plant Physiology*, 122, 169–179 [Study of a lytic enzyme with functions in developmental cell senescence of a number of different plant organs]

Pickard W.F. (2008). Laticifers and secretory ducts: two other tube systems in plants *New Phytologist* 177, 877-888 [Examples of developmental cell elimination]

Plaxton W.C., Podestá F.E. (2006) The functional organization and control of plant respiration. *Critical Reviews in Plant Sciences* 25. 159–198 [Includes a discussion of the climacteric]

Price A.M., Aros Orellana D.F., Mohd Salleh F., Stevens R., Acock R., Buchanan-Wollaston V., Stead A.D., Rogers H.J. (2008). A comparison of leaf and petal senescence in wallflowers (*Erysimum linifolium*) reveals common and distinct patterns of gene expression and physiology. *Plant Physiology* 147, 1898-1912 [Genomic study comparing senescence of leaves and petals in the same species]

Roberts J.A., Elliott K.A., Gonzalez-Carranza Z.H. (2002). Abscission, dehiscence, and other cell separation processes. *Annual Review of Plant Biology* 53, 131-158 [A comprehensive survey of the many different instances of cell separation and its regulation in plant development]

Robson P.R.H., Donnison I.S., Wang K., Frame B., Pegg S.E., Thomas A., Thomas H. (2004). Leaf senescence is delayed in maize expressing the *Agrobacterium* IPT gene under the control of a novel maize senescence-enhanced promoter. *Plant Biotechnology Journal* 2, 101-112 [Physiological characteristics of transgenic *stay-green*s in maize]

Rogers H.J. (2006). Programmed cell death in floral organs: how and why do flowers die? *Annals of Botany* 97, 309-315 [Review of the occurrence and mechanisms of senescence processes during reproductive development]

Sakamoto W. (2006). Protein degradation machineries in plastids. *Annual Review of Plant Biology* 57, 599–621 [The roles of Clp, FtsH and other proteases in the breakdown of plastid proteins]

Samuels A.L., Kaneda M., Rensing K.H. (2006). The cell biology of wood formation: from cambial divisions to mature secondary xylem. *Canadian Journal of Botany* 84, 631-639 [Includes discussion of cell senescence during xylem development]

Silvertown J., Franco M., Perez-Ishiwara R. (2001). Evolution of senescence in iteroparous perennial plants. *Evolutionary Ecology Research* 3, 393–412 [Application of a number of tests to determined the role of natural selection in the reproductive strategies and lifespans of plants]

Sklensky D.E., Davies P.J. (2011). Resource partitioning to male and female flowers of *Spinacia oleracea* L. in relation to whole-plant monocarpic senescence. *Journal of Experimental Botany* 62, 4323-4336 [A study of the pattern of photosynthate allocation in dioecious spinach]

Tanaka Y., Sasaki N., Ohmiya A. (2008). Biosynthesis of plant pigments: anthocyanins, betalains and carotenoids. *Plant Journal* 54, 733-749 [Review of the structures and biosynthetic origins of pigments, including those of senescing and ripening tissues]

Thomas H. (1992). Canopy survival. In: *Crop Photosynthesis: Spatial and Temporal Determinants*. (ed. Baker N.R., Thomas, H.). pp. 1-41. Amsterdam: Elsevier [Considers in detail the influence of senescence on crop productivity]

Thomas H. (2003). Do plants age and if so how? *Topics in Current Genetics* 3, 145-171 [Discusses the relationships between the senescence and lifespan of plants and their parts]

Thomas H. (2010). Leaf senescence and autumn leaf coloration. *McGraw Hill 2010 Yearbook of Science and Technology* pp 211-214. [Considers how and why leaves change color when they senesce]

Thomas H., Howarth C.J. (2000). Five ways to stay green. *Journal of Experimental Botany* 51, 329-337 [Definition of different classes of senescence mutant and discussion of the implications of the *stay-green* phenotype for crop production]

Thomas H., Huang L., Young M., Ougham H.(2009). Evolution of plant senescence. *BMC Evolutionary Biology* 9, 163 [Phylogenetic study of the evolutionary origins of senescence]

Thomas H., Ougham H., Mur L.A.J., Jansson S. (in press). Senescence and programmed cell death. In: *Biochemistry and Molecular Biology of Plants*. 2nd edition (ed. Buchanan B., Gruissem W., Jones R.). NY: Wiley [Comprehensive review of the cell and molecular biology of plant senescence]

Thomas H., Ougham H.J., Wagstaff C., Stead A.J. (2003). Defining senescence and death. *Journal of Experimental Botany* 54, 1127-1132 [A discussion of the extent to which senescence and programmed cell death share similar mechanisms and controls]

Turner G.W. (1999). A brief history of the lysigenous gland hypothesis. *Botanical Review* 65, 76-88 [A useful review of the development of changing ideas about lysigeny and schizogeny]

Turner S., Gallois P., Brown D. (2007). Tracheary element differentiation. *Annual Review of Plant Biology* 58, 407-433 [A comparative account of the development of xylem cells in two model species, *Zinnia* and *Arabidopsis*]

Uauy C., Distelfeld A., Fahima T., Blechl A., Dubcovsky J. (2006). A NAC gene regulating senescence improves grain protein, zinc, and iron content in wheat. *Science* 314, 1298-1301 [Identification, evolution, function and agricultural implications of a regulator of the transition from the photosynthetic to remobilization phase of leaf development]

van Doorn W.G., Woltering E.J. (2004). Senescence and programmed cell death: substance or semantics? *Journal of Experimental Botany* 55, 2147-2153 [Review of evidence in favor of classifying senescence as a type of programmed cell death process]

Wingler A., Marès M., Pourtau N. (2004). Spatial patterns and metabolic regulation of photosynthetic parameters during leaf senescence. *New Phytologist* 161, 781-789 [Analysis of basis of changing photosynthetic capacity during senescence]

Wingler A., Roitsch T. (2008). Metabolic regulation of leaf senescence: interactions of sugar signaling with biotic and abiotic stress responses. *Plant Biology* 10 (Supplement 1), 50–62 [Reviews the role of sugars in regulating source-sink interactions during senescence]

Withington J.M., Reich P.B., Oleksyn J., Eissenstat D.M. (2006). Comparisons of structure and life span in roots and leaves among temperate trees. *Ecological Monographs* 76, 381–397 [Tests and disproves the theory that similar root and leaf traits, including tissue longevity, are physiologically linked and should therefore be positively correlated]

Zavaleta-Mancera H.A., Franklin K.A., Ougham H.J., Thomas H., Scott I.M. (1999). Regreening of senescent *Nicotiana* leaves. I. Reappearance of NADPH-protochlorophyllide oxidoreductase and light-harvesting chlorophyll a/b-binding protein. *Journal of Experimental Botany* 50, 1677-1682 [Study of the conditions under which leaf senescence may be reversed, and demonstration of changes in structure and function of regreening cells]

Zavaleta-Mancera H.A., Thomas B.J., Thomas H., Scott I.M. (1999). Regreening of senescent *Nicotiana* leaves. II. Redifferentiation of plastids. *Journal of Experimental Botany* 50, 1683-1689 [Quantitative measurements of the ultrastructure of regreening leaf cells]

Zeiger E., Schwartz A. (1982). Longevity of guard cell chloroplasts in falling leaves: implication for stomatal function and cellular aging. *Science* 218, 680-682 [Provides evidence that the green cells of different leaf tissues undergo senescence according to different timetables]

Zimmermann P., Heinlein C., Orendi G., Zentgraf U. (2006). Senescence-specific regulation of catalases in *Arabidopsis thaliana* (L.) Heynh. *Plant Cell and Environment* 29, 1049-1060 [Evidence supporting a central role for H_2O_2 metabolism in senescence]

Zimmermann P., Zentgraf U. (2005). The correlation between oxidative stress and leaf senescence during plant development. *Cellular and Molecular Biology Letters* 10, 515–534 [Identification of common regulatory pathways in stress responses and senescence]

Biographical sketch

Howard Thomas is Emeritus Professor of Biological, Environmental and Rural Sciences at Aberystwyth University, Wales, UK. He has held visiting professorships at Universities in Switzerland and the United States. His research interests include investigation and modification of cellular, biochemical and evolutionary mechanisms of plant senescence and death, with special emphasis on pigment and protein metabolism. Approaches include cloning, mapping and exploiting genes determining senescence, late-season and post-harvest deterioration, food, feed and bioprocessing quality and consumer perception in a range of crop and amenity species. He has been active in the application of imaging and machine-learning methods for non-destructive analysis of plant development and geneflow, and remote field to landscape-level monitoring of crops and ecosystems. He also has a special interest in the cultural significance of scientific research and promotion of links between science and the arts.