Lecture 18:

Senescence and Programmed Cell Death

Plants exhibit various types of senescence

Senescence occurs in a variety of organs and in response to many different cues.

Many annual plants, e.g. wheat, maize, soybean, abruptly yellow and die following fruit production.

Senescence of the entire plant after a single reproductive cycle => **monocarpic senescence**

Monocarpic senescence in soybean

Left:

Entire plant underwent senescence after flowering and production of fruits.

Right:

Plant remained green and vegetative because its flowers were continually removed.

Other types of senescence

Senescence of aerial shoots in herbaceous perennials

Seasonal leaf senescence (e.g. deciduous trees)

Sequential leaf senescence (e.g. leaves die when they reach a certain age)

Senescence (ripening) of fleshy fruit; senescence of dry fruit

Senescence of specialized cell types (e.g. trichomes, tracheids)

Senescence of storage cotyledons and floral organs

Stages of flower senescence in morning glory



Triggers of senescence

Internal → monocarpic senescence

External → day length and temperature in autumnal leaf senescence of deciduous plants
→ abiotic and biotic stress

Regardless of the initial stimulus, different senescence patterns share common internal programs in which regulatory senescence genes initiate a cascade of secondary gene expression that brings about senescence and death.

Senescence is an ordered series of cytological and biochemical events

Senescence is genetically encoded, allowing a predictable course of cellular events.

Some organelles are destroyed while others remain active.

- chloroplast – first organelle to deteriorate during onset of leaf senescence (destruction of thylakoid protein components and stromatal enzymes)
- nuclei remain structurally and functionally intact until the late stages of senescence

Senescent tissues carry out catabolic processes that require *de novo* synthesis of

- proteases
- nucleases
- lipases
- chlorophyll-degrading enzymes

Senescence is an ordered series of cytological and biochemical events

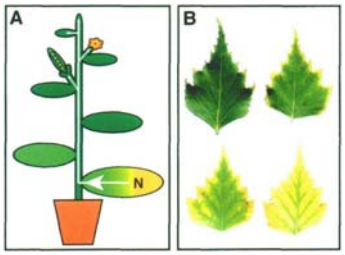
Senescence down-regulated genes (*SDGs*) – their expression decreases during senescence
e.g. photosynthetic genes

Senescence-associated genes (*SAGs*) – their expression is induced during senescence

Group A (Class I): proteases, ribonucleases, lipases, ACC synthase, ACC oxidase

Group B (Class II): glutamine syntethase (converts NH_4^+ to glutamine, nitrogen recycling from leaves)

Senescence is a recycling process

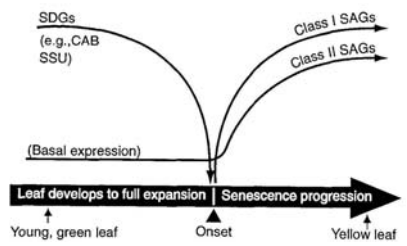


Some of the released nutrients (N) such as nitrogen are transported to developing seeds and young organs at the shoot apex.

Progression of leaf senescence in a species of birch. Senescence proceeds from leaf margins toward the center. Note that cells surrounding the vascular tissues senesce relatively late to facilitate nutrient mobilization from adjacent senescing cells.

Gan & Amasino (1997)
Plant Physiol. 113: 313

Differential gene expression during leaf senescence



Senescence down-regulated genes ***(SDCs)*** include chlorophyll a/b-binding protein gene *(CAB)*, Rubisco small subunit gene (*SSU*).

SAGs **-** expression up-regulated during leaf senescence.
Class I ***SAGs*** - expressed only during senescence (senescence-specific).
Class II ***SAGs*** **-** have basal level of expression during early leaf development, but expression increases during senescence.

Gan & Amasino (1997)

Salicylic acid has a role in regulating gene expression during leaf senescence

Arabidopsis mutants defective in SA-signaling:

Morris et al. 2000 Plant J 23: 677

npr1 = NONEXPRESSER OF PR GENES 1 (ankyrin repeat protein)
pad4 = PHYTOALEXIN-DEFICIENT 4 (lipase-like protein)
NahG = expresses salicylate hydroxylase, unable to accumulate SA

Plants grown at 12h L/8h D.

pad4 showed uniform chlorosis over the entire leaf lamina, but negligible signs of necrosis.

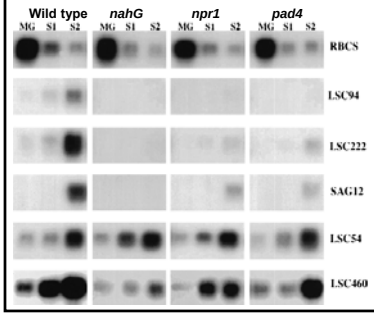
Effect of *pad4* mutation appears to be to delay or even inhibit necrosis in the senescing tissue.

As leaves started to senesce, chlorosis became visible at the edges, gradually spreading up the leaf. This chlorosis was rapidly followed by necrosis, the senescing tissue normally dying well before the entire leaf became chlorotic.

Salicylic acid has a role in regulating gene expression during leaf senescence

SAG12 = cysteine protease gene; expression partially depends on presence of SA
a marker of natural leaf senescence

Its expression is closely linked to senescence and chlorosis.
Its expression is highly affected by the presence of the SA-signaling pathway.

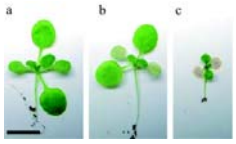


Northern blots on
MG, mature green
S1, early senescence
S2, mid-senescent showing signs of yellowing

RBCS, ribulose bisphosphate carboxylase small subunit;
LSC94, *PR1a;*
LSC222, chitinase;
SAG12, cysteine protease;
LSC54, metallothionein;
LSC460, glutamine synthetase.

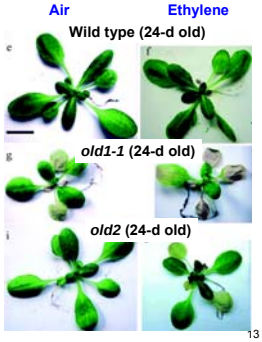
The onset of leaf senescence is controlled by leaf age and ethylene can promote leaf senescence

Investigation of the interaction between leaf age and ethylene on mutants with altered leaf senescence that are named as ***o**nset of **l**eaf **d**eath* (*old*) mutants.



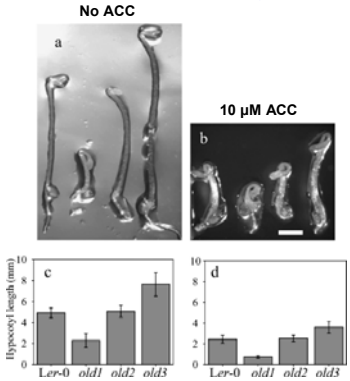
16-day air-grown

Ler-0 *old1-1* *old3*



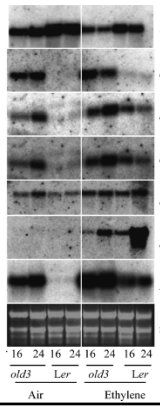
Jing et al. (2002)
Plant J 32: 51

old mutants exhibit different ethylene responses



Jing et al. (2002) Seedlings grown in darkness at 21°C for 5 days

Differential gene expression in *old* mutants



CAB Chlorophyll *a/b* binding protein

SAG13 Short chain alcohol dehydrogenase

SAG14 Blue copper-binding protein

SAG21 LEA protein

SAG2 Cysteine protease

SAG12 Cysteine protease

PR-1 pathogen-related protein

rRNA

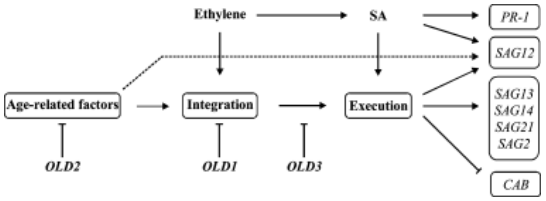
Expression of genes conferring resistance to pathogen attack (*PR-1*) enhanced *old* mutants.

Expression of *SAG12* is down-regulated in *old3* mutants despite their enhanced senescence phenotype.

SAG12 is not essential for progression of leaf senescence in *old3* (and *old1*) mutant. *OLD* genes may control a common pathway that is required but not sufficient for full expression of *SAG12*.

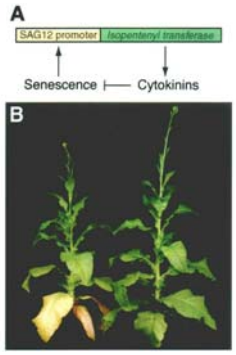
Jing et al. (2002)

The old mutants may work in a common regulatory pathway

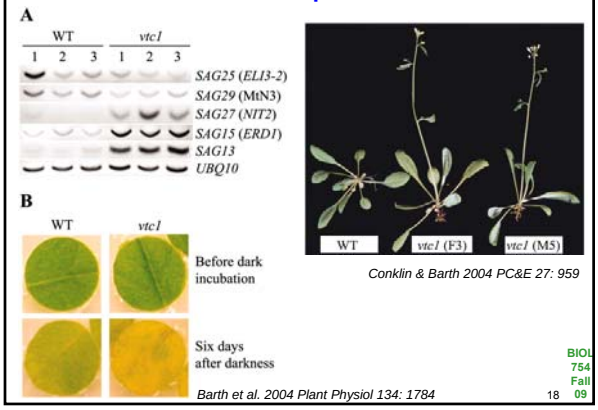


Jing et al. (2002)

Retardation of leaf senescence by autoregulated production of cytokinins



Low levels of vitamin C promote senescence



Conklin & Barth 2004 PC&E 27: 959

Barth et al. 2004 Plant Physiol 134: 1784

Programmed cell death is a specialized type of senescence

Senescence can occur at the level of:

whole plant (monocarpic senescence)
organ (leaf senescence)
cell (tracheary element differentiation)

Process whereby individual cells activate an intrinsic senescence program = **Programmed Cell Death (PCD)**

In animals, PCD may be initiated by specific signals (errors in DNA replication during division)

- involves expression of a characteristic set of genes, resulting in cell death
- accompanied by morophological and biochemical changes (**apoptosis**, Greek: "falling off")
- during apoptosis, cell nucleus condenses and DNA fragments in a specific pattern

Programmed cell death is a specialized type of senescence

PCD in plants, less well characterized

- but similar histological changes as in animals observed
- PCD occurs during differentiation of xylem tracheary elements, during which nuclei and chromatin degrade and cytoplasm disappears → activation of genes encoding nucleases and proteases
- protection against pathogenic organisms
  - infection by pathogen causes plant cells to quickly accumulate high concentrations of toxic phenolic compounds and die (it's not quite as simple) → dead cells form small circular island of cell death (**necrotic lesion**)
  - necrotic lesions isolate and prevent infection from spreading to surrounding healthy tissues by surrounding the pathogen with a toxic and nutritionally depleted environment (**hypersensitive response**)