LONGEVITY IN PLANTS

INTRODUCTION

There is a large range of life spans in the plant kingdom. Certain tree species can live for well over a century whereas other plants complete their life cycle in a few weeks. When considering the lifespan of a particular plant species, it is important to keep in mind that there is a high rate of cell death occurring throughout the life of the plant. For example, much of the mass of a tree consists of wood, which is dead vascular tissue. Individual organs of a plant such as leaves have lifespan that can substantially differ from that of the whole organism. Moreover the lifespan of leaves from different species varies.

Longevity in plants is somewhat different from animal kingdom. Compared to humans' century-long life span, some plants—evergreens in particular—have the capacity to live for an exceptionally long time, even millennia. In a study published in *Current Biology*, scientists from the University of Bern in Switzerland presented evidence for a potential mechanism that could help explain some plants' everlasting longevity, i.e. minimal stem cell divisions to avoid mutational changes

Plants are referred to as annuals which live only one year, biennials which live two years, and perennials which live longer than that. The longest-lived perennials, woody-stemmed plants such as trees and bushes, often live for hundreds and even thousands of years (one may question whether or not they may die of old age). A giant sequoia, General Sherman is alive and well in its third millennium. A Great Basin Bristlecone Pine called Methuselah is 4,848 years old (as of 2017) and the Bristlecone Pine called Prometheus was a little older still, at least 4,844 years (and possibly as old as 5,000 years), when it was cut down in 1964. The oldest known plant (possibly oldest living thing) is a clonal Quaking Aspen (*Populus tremuloides*) tree colony in the Fishlake National Forest in Utah called Pando at about 80,000 years. Lichen, a symbiotic algae and fungal proto-plant, such as *Rhizocarpon geographicum* can live upwards of 10,000 years.

AREAS OF STUDY

Plants longevity is supposed to be apparent with the senescence, although it is not the fact. There are three major areas under which the longevity may be discussed and understood. These are as follows:

1. Dormancy
2. Meristem activity
3. Senescence

Dormancy

Dormancy is a state of metabolic arrest that facilitates the survival of organisms during environmental conditions incompatible with their regular course of life. Many organisms have deep dormant stages to promote an extended life span (increased longevity). In contrast, plants have seed dormancy and seed longevity described as two traits. Seed dormancy is defined as a temporary failure of a viable seed to germinate in conditions that favor germination, whereas seed longevity is defined as seed viability after dry storage (storability).

Meristem activity

The team zeroed in the formation of axillary meristems—stem cells that give rise to branches—in *Arabidopsis thaliana* and tomato, finding few cell divisions between the apical meristem located at the very top of a plant and the axillary meristems. With such little proliferation comes less opportunity to accumulate potentially deleterious genetic mutations in somatic cells that could kill the organism, the authors reasoned.

“Meristem aging is not a problem for perennial plants, in other words,” said Sergi Munné Bosch, a plant physiologist at the University of Barcelona who was not part of the study. “The meristems are the growing units. If they don’t senesce, then the plant will keep the capacity to grow and reproduce forever, at least potentially.” Instead, he added, structural defects or pathogens most often kill plants. it’s possible the researchers’ observations of the meristem constitute a mechanism for life span maintenance, but it is likely not the only mechanism. “The relationship of mutation accumulation to aging and death is not clear,” he said.

Senescence

It is advantageous to maintain leaves only for as long as they contribute to the survival of the plant. In temperate climates, for example, the shortening days and colder temperatures of the approaching winter limit productivity and in deciduous plants triggers a massive programmed cell death that often leads to beautiful autumn colors and ultimately death and loss of the leaves.

This “yellowing” of leaves is often referred to in the plant biology literature as leaf senescence or the senescence syndrome. Specifically the senescence syndrome refers to the process by which nutrients are mobilized from the dying leaf to other parts of the plant to support their growth. Nutrient availability, particularly nitrogen, has been a major limit to growth and reproductive success throughout plant evolution. Moreover, plants are fixed in a particular location in the soil and deplete their local environment. Thus, there has been strong selective pressure to evolve systems to retain and recycle nutrients.

There are many factors that can initiate the nutrient-recycling program. In some species, a need for nutrients elsewhere, such as for developing seeds, can trigger the senescence syndrome. Another likely factor is the loss of photosynthetic productivity due, e.g., to shading by upper parts of the plant or to the accumulation of damage to leaf cells. Certain reactions of photosynthesis can generate oxidative damage, and, as in animals, different species have invested to differing extents in systems to prevent and repair such damage. To be consistent with the literature on animal aging, the age-related declines in leaf productivity caused by metabolism and other types of cumulative damage would constitute leaf senescence and the recycling program to salvage nutrients from the senescing leaf is the senescence syndrome.

The senescence syndrome is characterized by distinct cellular and molecular changes. The chloroplast is the first part of the cell to undergo ultrastructural changes resulting from its disassembly (the concomitant loss of chlorophyll results in the characteristic color changes of leaves). Because the chloroplast contains the bulk of the nutrients in a leaf cell, it is not surprising that it is the target of the recycling program. During senescence, the mRNA level of most genes declines rapidly, but the mRNA level of specific genes (senescence-associated genes) increases during this process. Many senescence-associated genes encode proteins that accomplish parts of the recycling program

such as proteases, nucleases, and proteins involved in metal binding and transport.

In many plant species, certain hormones can either enhance or delay senescence. For example, delayed leaf senescence is observed in *Arabidopsis* plants containing a mutation that confers ethylene insensitivity. Cytokinin levels decline in senescing leaves and treatments that reverse or prevent senescence such as the removal of seed pods restores the flux of cytokinins to leaves. Thus, changes in levels of or sensitivity to the hormones ethylene and cytokinin may be part of the normal system that regulates leaf senescence.

Further support that cytokinin regulates senescence is provided by transgenic plant studies. To deliver cytokinin specifically to leaves at the onset of senescence, the promoter of one of the senescence-associated genes that is uniquely active during senescence was used to drive expression of the structural gene for an enzyme of cytokinin synthesis. When leaves of plants that contain this "engineered" gene initiate senescence, the promoter is activated, cytokinin is produced, and the leaves do not senesce. The system is auto regulatory: just enough cytokinin is produced to block leaf senescence because the prevention of senescence attenuates promoter activity. This autoregulatory system avoids the toxic effects that result from high levels of cytokinin.

Although genes that are expressed during the senescence syndrome as well as ways to manipulate senescence have been identified, much remains to be done to understand the molecular basis of aging in plants. For example, nothing is known about the signal transduction pathways that lead to altered gene expression during senescence or how hormones such as cytokinin influence senescence. However, we now have many of the tools to explore this process.

Conclusion

It remains to be seen whether common mechanisms link the aging process in diverse organisms. We, however, can depend on the knowledge that aging mechanisms will be worked out in several systems in the relatively short term. Over the longer term we can hope to continue the tradition of biomedical

research by intervening in this process to maintain our vitality and quality of life for a greater fraction of our life time.

References

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