Senescence

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Plants not only may delay senescence until after they reproduce successfully, but they may also bring senescence time forward, in order to reproduce in favored conditions. It demonstrates that even though senescence is part of aging, it does not necessarily mean plants have to reach a certain age to senesce. Experiments using different aged plants have suggested that in interest of their final outcome and fitness, plants carefully weigh out environmental cues and transit to next developmental phase at proper time, even if that means transiting to terminal senescence phase earlier and shortening their lifespan.

senescence timing aging climate change reproductive synchrony

1. Senescence and Aging in Plants

Senescence (from the Latin word “senēscere”: to grow weak, become exhausted, and to be in a decline) generally refers to the process of growing old and is associated with decay and mortality or decreased fertility with age [1], but it is actually a very widespread concept for plants. Plants have specific characteristics that violate the general, classical definition of senescence. For example, they are modular; meaning their architecture is made of a repetition of units which allows them to rejuvenate [2]. In addition, their cellular division does not always cause shorter telomeres [1]. There are even some plants for which the concept of senescence simply does not apply. Researchers addressed the hypothesis of senescence that assumes aging results from an accumulation of deleterious mutations, by studying extraordinary long living trees: bristlecone pine (*Pinus longaeva*), ranging in age from 23 to 4713 years. They studied viability traits, such as seed weight and germination rate, biomass, and frequency of mutations and found no significant relationship between these factors and age of the trees. They concluded that these trees do not senesce [3]. Another group of researchers [4] examined whether an extraordinarily long-living herb *Borderea pyrenaica* (Dioscoreaceae), which is known to live more than 300 years, experiences senescence. They investigated the relationship between age, reproductive value, and vital rates. No evidence for senescence was found as growth and fecundity did not decrease at older ages, and survival and reproductive value increased with age. Another study on these perennial herbs [5], tested age-related changes in several photo-oxidative stress markers and found no age-dependent signs of oxidative stress. Therefore, they suggested that age-induced senescence is not a universal feature of aging in perennial plants.

It is true that as organisms grow old, their performance declines. This might raise the question of why natural selection is not replacing individuals that perform poorly at older age by the ones performing stronger as aged group? The fitness decline caused by senescence has been suggested to be either because of accumulation of mutations [6] or results from genes that have been chosen by natural selection because of their positive effect in

earlier life, despite of their adverse effects in later life (pleiotropy) [7]. The classic theory of senescence evolution says that the power of natural selection decreases with age [6]. Hamilton W.D. (1966) [8] discussed that increased mortality and/or decreased fertility in older ages does not affect the fitness as much as it would have, had those happened at younger ages. He suggested that, as the individual grows old, mutations that have caused better performance and increased fertility at younger age, but have caused less performance later on in life, will be established in population, because high performance at early life is of a great advantage for plant and it is selectively favored [7][8][9]. In other words, mutations coding for “live fast, die young” performance, are favored and naturally selected over genes coding for average performance and longer life [10]. Senescence, in the other hand, is considered to have been evolved as an essential strategy associated with plant reproduction, adaptation, fitness, and survival [11]. Various gene expression profiling and transcriptome studies have shown a conserved pattern among plant species for senescence regulation, as similar catabolic pathways were found to be upregulated at senescence. This shows that senescence could be an evolutionary selected trait [12][13]. We know that plants not only delay death until after they reproduce successfully [14][15], but they sometimes even bring senescence time forward, in order to escape upcoming unfavored environmental conditions and maximize their productivity [16]. In fact, plants have been called “unusual organisms” that can have some control over their own life span based on environmental cues, above and beyond the aging process [14][15][17].

2. Age-Dependent/-Independent Senescence in Plants

Even though senescence is part of the aging process [18], it does not necessarily mean plants always have to reach certain age to senesce. Recent studies have proposed that the timing of whole plant senescence is influenced by developmental age more than calendar age [16][19]. Besides age-dependent/developmental senescence, environmental conditions can also trigger senescence, and it has been shown that the timing and rate of senescence is highly affected by environmental cues such as photoperiod, temperature, and moisture in soil [18][20][21]. Therefore, studying senescence in natural populations is complicated because it is influenced by environmental factors that fluctuate seasonally or even daily and may also influence age-dependent mortality pattern [22]. To study the age-dependent/-independent dynamics of senescence, a group of researchers [23] investigated demographic aging in natural populations of *Plantago lanceolata* (Plantaginaceae), and reported synchronous changes in senescence across four cohorts (a cohort is a group of individual plants of same age) over time (i.e., environmental dependent senescence). Another study [24] of size-based/age-based senescence on *P. lanceolata* confirmed the Hamilton prediction that says the impact of selection decreases with age. Their analyses showed decline in size, lower inflorescence production, and reduced physiological strength prior to death, which were all best explained by size rather than age suggesting an important role for the environment in determining senescence.

Other researchers have shown that both initiation and termination of flowering (final senescence) are sensitive to environmental conditions [16][17][25][26], which give plants the advantage of flexibility in response to changing environment and allow setting seeds and senescing at a suitable time. In two recent publications, using groups of *Arabidopsis thaliana* that differed in age, it was also shown that whole senescence in plants is strongly

synchronized with their environmental condition All groups set seeds and senesced at the same time regardless of their age [16][17].

Studies using natural population of *A. thaliana* have identified genes that are involved in both local adaptation and senescence, and suggested that senescence may be helping with adaptation [27]. Under stressful environmental conditions (if the developmental time is appropriate), nutrients from vegetative organs such as leaves, reallocate towards reproductive organs. This is an important adaptation trait that plants have evolved, in order to accomplish their life cycle even under undesirable conditions [28]. Whether plants enter the senescence phase to avoid the stressful situation or they compromise for shorter life in exchange of better final outcome (as yield and seed set) is yet to be discovered. Compare to phenological studies focusing on bolting and flowering, whole-plant senescence and the effects of environmental changes on its patterns are still not well understood, because the timing of this last developmental stage is influenced by multiple factors affecting all previous developmental stages which makes it complicated [13][29].

3. Whole Plant Senescence

There are two main types of senescence in plants. The sequential or organ senescence, which happens in a continuum pattern at a certain developmental stage after accomplishing certain tasks, when senescing organs recycle their extra nutrients towards developing and growing ones [30][31][32][33][34][30]. For example, spring flowering is the result of consuming relocated nutrients from senescing autumn leaves. Organ senescence is mostly associated with age, but also with environmental condition [31]. The second type of senescence is reproductive senescence, which leads to the whole plant senescence in monocarpic plants and is usually called “monocarpic senescence”. It is the final stage of development and helps with final production and seed quality [32], and the way it is precisely programmed to occur after distinct sequential developmental phases, is very unique to monocarpic plants. Reproductive senescence initiates a gradual death and has been called the “natural cause of death” in plants [33]. Although most senescence studies have focused on leaf senescence rather than whole plant senescence, but in monocarpic plants leaf senescence is actually coordinated with whole plant developmental phases including the whole plant senescence [21][34].

Timing of whole plant senescence in monocarpic plants is important for fitness and natural selection, as close to the end of reproductive phase, plants try to invest all their resources and nutrients in final production [35]. One of the earliest observations of monocarpic senescence in plants was by Hildebrand (1881) [36], when he suggested that whole plant senescence happens after plants accomplish the reproductive phase, which is itself the result of remobilization of nutrients from vegetative to reproductive organs in order to provide resources for developing seeds. Therefore, in case of monocarpic plants, flowering senescence (“floral arrest”) is followed by whole senescence and can be studied as a senescence factor. An obvious definition for whole flowering senescence would be the time point when all the flowers are senesced and no more “flowering initiation” will occur [16][17][26]. Flowering senescence has an important role in determining the length of reproductive period, and also it affects the reproductive potential such as optimization of fruit and seed production. One of the first studies focusing on “flowering termination” and whole senescence was done over 26 years ago, when Hensel (1994) [37] studied the

relationship between the proliferative capacities of inflorescence meristems and final fruit development in *A. thaliana*, and provided strong evidence that floral arrest is mediated by a communication between inflorescence meristems and developing final fruits and seeds. A recent study of floral termination in *A. thaliana*, expanded the classic model of Hensel, and examined the mechanism by which final fruits affect flowering termination [38]. They suggested that inflorescences only arrest at certain developmental age and in response to a highly localized auxin signal from recently produced fruits. Another group of researchers [39] investigated the correlative control of *A. thaliana*'s seeds over inflorescences and studied how reaching to a certain number of seeds inhibit further maternal growth. They identified expression of stress- and senescence-related genes right after fruiting and inflorescence meristem arrest. They also reported sudden arrest in mitotic activity upon fruit removal, meaning the term "mitotic senescence", a proposed name for growth arrest after fruit production when meristem cells lose their ability for mitotic cell division [31][40][41], may have not been used properly. In *A. thaliana*, it is shown that producing certain number of flowers and fruits will lead to reproductive meristem arrest and if fruit numbers are low, inflorescence meristem continues its activity and can also be reactivated in case of fruit removal [38][42].

A correlation control between developing fruits/seeds and senescence timing in monocarpic plants has been also observed when removal of reproductive structures or preventing their development, delayed terminal senescence [40][43][44][45][46]. This is suggested to be either through source–sink relationship [43] or in another point of view, via signals from offspring [44][47][48]. However, recent studies have shown that the connection is more complicated than that [49]49. Several quantitative trait loci (QTL) analyses using recombinant inbred lines (RILs) populations of *A. thaliana* have reported accession- and/or condition-specific QTLs for advancing or delaying senescence (see, e.g., in [50][51]), suggesting plants have evolved natural genetic variations according to their evolutionary and also ecological history. Woolhouse [52] suggested that because of polyphyletic origin of monocarpic senescence, there may have been independently evolved control strategies in different plant groups and encouraged scientists to avoid generalizing and simplifying the concept of monocarpic senescence and instead explore senescence separately in different species. Investigating senescence in pea plants (*Pisum sativum* L.) led scientists to reject the simplified source–sink view that says senescence is induced by developed flowers and fruits [30]. They suggested that senescence is the "consequence" of reproductive phase; meaning the commitment of plant to redirect the nutrients towards reproductive sinks is "required" but "not enough", and monocarpic senescence timing is influenced by many factors including environmental condition and also the previous developmental life stages. For example, low nitrogen level has been reported to induce early senescence [53], as opposed to high nitrogen level which delays senescence [54]. Studying the effect of day length on senescence, as one of the main environmental factors, using different ecotypes of *A. thaliana* showed that long day only causes earlier senescence in early flowering accessions, and not in late flowering accessions, which shows senescence being influenced by both environment and genetic [55]. They also found that senescence was linked to other developmental traits such as flowering and fruit number, which was evidenced in other studies as well where correlation between flowering time and seed set/senescence was reported (see, e.g., in [56][57][58][59][60]). They suggested that the effect of genetic and environment on senescence and related developmental traits might be through common regulatory pathway as the pattern of association between senescence and other traits was the same, regardless of senescence variation being caused by ecotype or day-length [55]. This was seen in similar cases before, where QTL studies using *A.*

thaliana RILs reported overlapping flowering and senescence genes with the loci affecting either of those traits and suggested senescence and flowering may be genetically linked and sharing regulatory loci [17][61]. Other lines of studies have shown that senescence of first few emerging leaves, will send nutrients such as nitrogen to later-emerging leaves and affect whole plant senescence [50][62][63]. Studying 45 accessions of *A. thaliana* and 155 RILs also showed that *A. thaliana* plants may have evolved to use various methods to accomplish developing fruits, seeds, and then senesce, which seemed to be dependent on flowering time [51]. Later-flowering groups used reallocated nutrients from senescing leaves, whereas earlier-flowering group of plants used photosynthates. However, even when senescence is flowering-dependent, it does not necessarily follow the pattern in which later flowering means later senescence, yet it might mean plants adjust their flowering time, in order to coordinate their senescence time with appropriate environmental condition that is in interest of plant fitness and productivity [16][17][26]. Even though experimental studies have shown that removal of reproductive organs will prolong vegetative phase and delay flowering, which then may increase plant life span and delay senescence, this may not simply be the case. In order to do the proper adjustment with the environment, flowering and senescence seem to interact with each other, and studies have shown that whole plant senescence is associated with both flowering-dependent and flowering-independent pathways[16][17][29][34][42][61].

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