Physiol Mol Biol Plants (August 2022) 28(8):1515–1534
https://doi.org/10.1007/s12298-022-01224-1

REVIEW ARTICLE

Know when and how to die: gaining insights into the molecular regulation of leaf senescence

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Received: 5 May 2022 / Revised: 17 August 2022 / Accepted: 21 August 2022 / Published online: 30 August 2022

Abstract Senescence is the ultimate phase in the life cycle of leaves which is crucial for recycling of nutrients to maintain plant fitness and reproductive success. The earliest visible manifestation of leaf senescence is their yellowing, which usually commences with the breakdown of chlorophyll. The degradation process involves a gradual and highly coordinated disassembly of macromolecules resulting in the accumulation of nutrients, which are subsequently mobilized from the senescing leaves to the developing organs. Leaf senescence progresses under overly tight genetic and molecular control involving a well-orchestrated and intricate network of regulators that coordinate spatio-temporally with the influence of both internal and external cues. Owing to the advancements in omics technologies, the availability of mutant resources, scalability of molecular analyses methodologies and the advanced capacity to integrate multidimensional data, our understanding of the genetic and molecular basis of leaf ageing has greatly expanded. The review provides a compilation of the multitier regulation of senescence process and the interrelation between the environment and the terminal phase of leaf development. The knowledge gained would benefit in devising the strategies for manipulation of leaf senescence process to improve crop quality and productivity.

Keywords Senescence · Regulation · Omics technologies · Crop productivity

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Introduction

An organism completes its life cycle by following a specific pattern of development; it grows, matures, and ultimately dies. The plants and their organs die likewise, and the decline of plant physiological events is called senescence which represents the ultimate stage of plant growth and development (Lim et al. 2007b). This physiological decline may be initiated and impactful at various specific cells, tissues, organs, or whole organism level. Individual organs, like leaves and flowers, possess a noticeably short lifespan. Briefly, plants grow for a limited period and start to senesce after the completion of reproduction. The degradation of chlorophyll and macromolecules such as proteins and nucleic acids often accompanies the senescence process in plants. Nevertheless, this deterioration during senescence is not necessarily terminal; instead, it is a reversible process that allows senescing leaves to re-green and restore their photosynthetic capacity under certain circumstances (Rapp et al. 2015). It is believed that the alterations that occur during senescence could constitute a trans-differentiation process which distinguishes it from programmed cell death (PCD). Cell death during senescence happens slowly, at the organelle or organism level, for efficient recycling of nutrients to the growing organs and offsprings which ensures fitness and reproductive success (Kim et al. 2016). In contrast, PCD involve localized, acute and rapid cell death. A frequently

used alternative term for 'senescence' in plant biology that often creates confusion is 'ageing'. However, ageing refers to the changes that occur with time and throughout the life cycle of the plant (Thomas 2013). Plant senescence is controlled by intrinsic developmental factors, such as age and phytohormones, and also by environmental (extrinsic) signals, including biotic or abiotic agents, namely pathogens attack, extreme temperatures, drought, nutrient deprivation, and exposure to ozone (Lim et al. 2003; Fig. 1A). The leaf senescence process can be divided into three phases: initiation, degenerative and terminal (Yoshida 2003; Fig. 1B). During the early leaf developmental stages, the younger leaves serve as sink, and the older ones act as the source. On maturation, the demand of younger leaves declines, which results in sugar accumulation in the older leaves and it initiates senescence. This transition from sink to source and the drop in anabolic activity is designated as the initiation phase of senescence. The subsequent catabolism of macromolecules, actuation of salvage and/or remobilization pathways and the dismantling of cellular components (organellar redifferentiation) are associated with the degenerative phase of senescence. The final stage of leaf senescence, the terminal phase, is characterized by the dissipation of ROS and loss of cell integrity and cell death (Yoshida 2003; Lim et al. 2007b).

Broadly, the plant senescence process can be categorized as natural or induced senescence (Miryeganeh 2022). Although both types of senescence are identical at the morphological level, they exhibit notable differences in the signalling and regulatory mechanisms (Wollaston et al. 2005). Natural senescence in plants has been widely studied with respect to the ageing of leaves, flowers, and coleoptiles. Leaf senescence is usually an organ level senescence but is also strongly associated with cellular or organismal death. Leaves serve as nutrient sources during the early stages of development as this source status is taken over by flowers during the very late stage of plant development (Guo et al. 2021). This review, emphasizes on how the external and internal factors influence the ageing of leaves, and the molecular basis of the evolutionarily acquired leaf developmental process. Efforts have been made to provide critical and up-to-date information on the recent advances in the study of leaf senescence, which would be beneficial in devising strategies for its manipulation to improve crop quality and productivity.

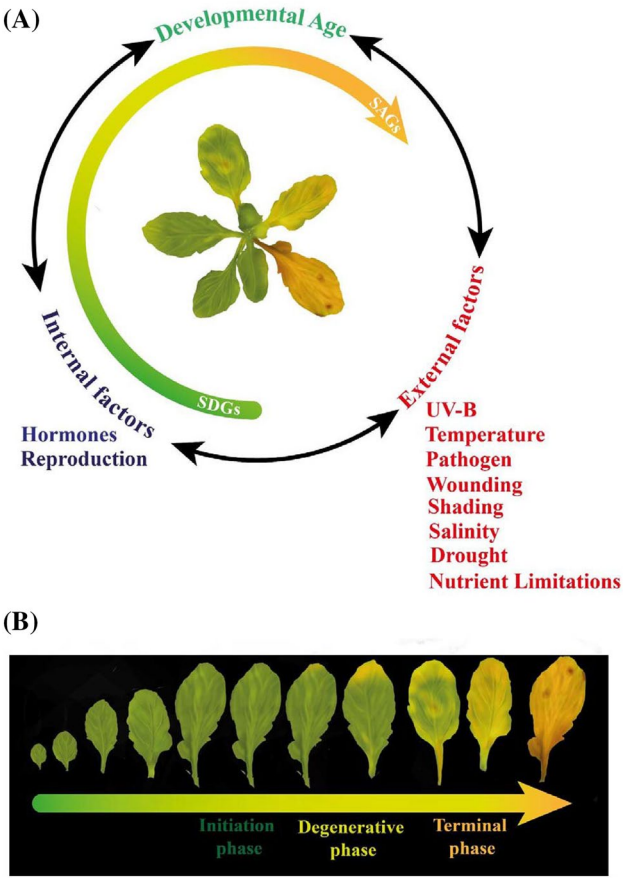


Fig. 1 Leaf senescence paradigm. Leaf senescence is primarily governed by developmental age; however, leaf senescence onset and progression are also regulated by various internal and external stimuli (**A**). These stimuli engage different signaling pathways, activating transcription factors that control leaf senescence. The senescence process is associated with the down-regulation of SDGs, including photosynthesis-related and metabolic process-related genes. In addition, enhanced expression levels of SAGs (hydrolytic enzymes, TFs, transporters, etc.) are also reported during leaf senescence. The leaf senescence can be divided into three phases: initiation, degenerative and terminal (**B**). The initiation phase includes the sink/source transition and the decline in photosynthesis. The second stage, the degenerative phase is the degradation phase, where macromolecules and cell organelles undergo degradation. And the ultimate terminal phase involves cell death

Leaf Senescence: A terminal stage of leaf development

Like plants, leaves follow a specific pattern of development wherein they grow, expand rapidly, import carbon and nitrogen and undergo rapid protein synthesis until they achieve full photosynthetic competence. After this photosynthetically active period, their contribution to photosynthate production declines, and leaves enter the terminal developmental stage of programmed cell death (Lim et al. 2003). In monocarpic plants, leaf senescence is developmentally associated with other organs or whole plant senescence and is triggered by reproductive development. However, in many tree species, leaf senescence can also occur regardless of other plant organs. Overall, leaf senescence is a degradation process that eventuates in a well-orchestrated manner and involves recycling of both macro- and micronutrients.

Induced Senescence

Plants encounter harsh growing conditions owing to exposure to several biotic and abiotic stress factors, which adversely affect their growth, development and productivity. Unlike animals, plants cannot move and escape such unfavourable environmental conditions and thus have evolved mechanisms to rapidly respond to and complete their life cycle even under deteriorating stressful situations (Wollaston et al. 2003). One such response is the onset of senescence that involves the mobilization of nutrients from the dying, no longer essential parts of the plant (e.g., a diseased leaf or an older leaf) to other growing parts (young leaves) or reproductive organs. The onset of senescence also ceases water consumption by older or diseased leaves and allows the plant to complete its life cycle. Several external factors that influence leaf senescence and their mode of action are discussed below. Table 1 presents information on important SAGs associated with natural and induced senescence in two model plants, Arabidopsis and rice.

Dark-induced senescence

The effect of light on triggering senescence is somewhat complicated and essentially relies on the intensity and wavelength of the incident light. High or sub-optimal intensities or dark conditions can induce premature leaf senescence in plants (Wollaston et al. 2003). Recently, the physiological, cytological and transcriptomic changes during dark-induced leaf senescence (DILS) in barley have been extensively reviewed by Nowicka et al. (2018). As reported, the DILS is characterized by prominent upregulation of macromolecular and metabolite degradation with a concomitant decline in photosynthesis. Weaver and Amasino (2001) have shown that senescence was delayed when whole Arabidopsis plants were placed in darkness. Nevertheless, when individual leaves were covered, senescence was induced. These results indicate that senescence is a highly localized phenomenon depending on the light status of the entire plant.

To pinpoint the conserved molecular forces driving the senescence process, microarray expression analysis of natural, dark-induced and sucrose starvation-induced suspension cultures was performed in Arabidopsis (Wollaston et al. 2005). The salicylic acid (SA), jasmonic acid (JA), and ethylene response pathways were involved in regulating natural, dark-induced and cell-suspension senescence, respectively. Comparative transcriptome studies of developmental, dark-induced (detached leaves) and dark-induced senescence of leaves attached to the plant revealed noticeable differences in the expression pattern of few senescence associated genes (SAGs) such as transcription factors, transporters, receptor like kinases, and hormone pathway genes among the different senescence processes. Developmental leaf senescence showed a higher accumulation of amino acid and oligopeptide transporters (Graaff et al. 2006). Several differentially expressed genes linked to plant hormone signal transduction pathways, TFs (WRKYs, NACs, HSFs, PIFs and bHLHs), and protein processing machinery were discovered by transcriptome analysis of dark-induced senescence in bermudagrass (Fan et al. 2019).

A delay in dark-induced senescence was reported in the transgenic rice plants overexpressing *SUBMERGENCE 1A (SUB1A),* an ethylene response factor (Schippers et al. 2015). The submergence tolerance gene SUB1A suppresses the effect of dark-induced senescence (DIS) by repressing the phytohormone signalling pathways, especially ethylene, JA and SA pathways in rice. Additionally, Sakuraba et al. (2014a) found that the transcript and protein levels of PHYTOCHROME INTERACTING FACTOR3, 4 and 5 (PIF3, 4 and 5) were substantially increased during age-triggered and dark-induced senescence of *Arabidopsis* leaves. A recent study by Hao et al. (2022) showed the implication of plastocyanin or PCY-SAG14 module in copper homeostasis under DIS in Arabidopsis. PCY-SAG14 is an endomembrane localized module that promotes DIS and is post-transcriptionally regulated by miR408. Under prolonged dark conditions, PIF3/4/5 are activated, release PCY-SAG14 module from miR408 regulation and subsequently promote DIS (Hao et al. 2022). Zareen et al. (2022) demonstrated the involvement of a multifunctional WD-40 repeat protein, HOS15 (high expression of osmotically responsive genes 15) as a positive regulator of dark-induced and natural leaf senescence in *Arabidopsis*. It was proposed that since HOS15 is the component of PWR-HDA9 repressor complex, it functions as a transcriptional corepressor and inhibits the acetylation of negative regulators of senescence, NPX1, PDG9 and WRKY57.

Salt stress-induced senescence

Salinity stress adversely impacts plant growth and productivity as it causes reduced growth, sink-source imbalance, leaf senescence and eventually the death of plants (Albacete et al. 2014). Salt-induced premature leaf senescence in sweet potato was shown to be associated with chlorophyll degradation, decreased photosynthesis and accumulation of ROS (Chen et al. 2012). Salt stress mainly causes osmotic stress by accumulating toxic sodium ions (Na^+) in leaves (Schippers et al. 2015). Elaborately, this imbalance in the sink-source relationship leads to plant growth decline during salt stress conditions. Studies on salt-tolerant versus sensitive wheat varieties revealed that salt-tolerant variety exhibits a comparatively delayed senescence phenotype due to an increased sink strength (Schippers et al. 2015).

Table 1 A list of SAGs known to regulate leaf senescence in Arabidopsis and rice

Species	SAGs	Functional Category	Regulation of Senescence	Induced by	Reference
Arabidopsis thaliana	13-LOX	Protease	Positive	Age	Springer et al. 2016
	AAF	Protease	Positive	Age	Chen et al. 2012
	ABF2	Transcription factor	Positive	Age	Gao et al. 2016
	ACS2	Ethylene biosynthesis	Positive	Age	Breeze et al. 2011
	AHK3	Kinase	Negative	Age	Kim et al. 2006
	ANAC016	Transcription factor	Positive	Dark	Sakuraba et al. 2015a, b
	ANAC019/055/072	Transcription factor	Positive	Age, dark and JA	Zhu et al. 2015
	ANAC046	Transcription factor	Positive	Age and dark	Oda-Yamamizo et al. 2016
	APX4	Redox regulation	Negative	Age	Panchuk et al. 2005
	ARF2	Transcription factor	Positive	Age, dark	Lim et al. 2010
	ARR4	Response regulator	Negative	Age, dark/starvation	Buchanan-Wollaston et al. 2005
	ATAF1	Transcription factor	Positive	Age, ABA, H_2O_2	Garapati et al. 2015
	ATAF2	Transcription factor	Positive	Age and dark	Nagahage et al. 2020
	ATG12A	Autophagy	Negative	Age	Thompson et al. 2005
	ATG6	Protease	Positive	Age	Patel and Kumar 2008
	ATMYB2	Transcription factor	Positive	Age	Guo and Gan 2011
	AtNAP	Transcription factor	Positive	Age, dark and ABA	Yang et al. 2014
	AtNOS1	Nitric oxide biosynthetic process	Negative	Dark	Guo and Crawford 2005
	AtOM47	Transporter	Positive	Age, Dark	Li et al. 2016
	AUX1	Transporter	Negative	Age	Xu et al. 2011
	bHLH03/13/14/17	Transcription factor	Negative	JA	Qi et al. 2015
	BRI	Receptor kinase	Positive	Age	Li and Chory 1997
	CBF2/3	Transcription factor	Negative	Age, dark, ABA, SA and JA	Sharabi-Schwager et al. 2010
	CDF4	Transcription factor	Positive	Age and ABA	Xu et al. 2020
	CLE14	Transcription factor	Negative	Age, Salt, ABA, SA, and JA	Zhang et al. 2022a, b
	ClpC	Protease	Positive	Age and dark	Nakabyashi et al. 1999
	CRF5	Transcription factor	Positive	Age	Raines et al. 2016
	EDF1/2	Transcription factor	Positive	Ethylene	Chen et al. 2011
	ELS1	Transporter	Positive	Age, Dark	Wang et al. 2016
	ERF4/ERF8	Transcription factor	Positive	Age	Koyama et al. 2013
	FtSH4	Protease	Negative	Age	Zhang et al. 2017
	GBF1	Transcription factor	Negative	Age and Oxidative stress	Smykowski et al. 2010
	JAZ7	Transcription factor	Negative	Dark	Yu et al. 2016
	JUB1	Transcription factor	Negative	Age and Oxidative Stress	Wu et al. 2012
	KHZ2	Transcription factor	Positive	Age	Yan et al. 2017
	LOX1	Lipoxygenase	Positive	JA	He et al. 2002
	LOX3	Lipoxygenase	Positive	JA	He et al. 2002
	MAPKKK18	kinase	Positive	ABA	Matsuoka et al. 2015
	MYB44	Transcription factor	Negative	ABA	Jaradat et al. 2013
	NAC016	Transcription factor	Positive	Age, Salt, Dark, Oxidative	Kim et al. 2013
	NTL4	Transcription factor	Positive	Drought	Lee et al. 2012
	ORE1	Transcription factor	Positive	Age, Ethylene	Rauf et al. 2013
	PIF3/4/5	Transcription factor	Positive	Dark	Hao et al. 2022
	PIF4/5	Transcription factor	Positive	Age, dark and ethylene	Sakuraba et al. 2014a
	PYL9	Receptor	Positive	Age and drought	Zhao et al. 2016

Table 1 (continued)

Species	SAGs	Functional Category	Regulation of Senescence	Induced by	Reference
	PRR9	Transcription factor	Positive	Age and dark	Kim et al. 2018
	PVA31	Transporter	Positive	Age	Ichikawa et al. 2015
	RAV1	Transcription factor	Positive	Age	Woo et al. 2010
	RD26	Transcription factor	Positive	Dark	Kamranfar et al. 2018
	RGL1	Transcription factor	Negative	Age	Chen et al. 2014
	SAG113	Protease	Positive	Age	Zhang et al. 2012
	SAG12	Protease	Positive	Age	Lohman et al. 1994
	SAG2	Protease	Positive	Age	Hensel et al. 1993
	SALICYLIC ACID INSENSITIVE 1	Defense response	Positive	Age	Morris et al. 2000
	SGRL	Chlorophyll degradation	Positive	Age	Sakuraba et al. 2014b
	TCP20	Transcription factor	Negative	JA	Danisman et al. 2012
	TCP4	Transcription factor	Positive	Age	Schommer et al. 2008
	WRKY45	Transcription factor	Positive	Age and dark	Chen et al. 2017; Barros et al. 2022
	WRKY57	Transcription factor	Negative	JA	Jiang et al. 2014
	WRKY6	Transcription factor	Positive	Age and *Pseudomonas syringae* infection	Robatzek and Somssich 2001
	WRKY75	Transcription factor	Positive	SA and oxidative stress	Guo et al. 2017
	XDH1	Protease	Positive	Age	Brychkova et al. 2008
	MET1	Methyltransferase	Positive	Age	Kim et al. 2008
	CMT3	Chromomethylase	Negative	Age	Feng et al. 2010
	DLM2/3	Demethylase	Positive	age	Yuan et al. 2020
	DME	Demethylase	Positive	age	Yuan et al. 2020
	ROS1	DNA Glycosylase	Positive	age	Yuan et al. 2020
	SUVH2	Histone methyltransferase	Negative	age	Ay et al. 2009
	HDA6	Histone deacetylase	Negative	Ethylene and JA	Wu et al. 2008
	HDA9	Histone deacetylase	Positive	Age, Dark	Chen et. al. 2016
	HDA15	Histone deacetylase	Negative	Age	Huang et al. 2018; Huang et al. 2022
	HAC1	Histone acetyltransferase	Positive	Dark	Hinckley et al. 2019
	JMJ16	Histone demethylase	Negative	Age	Liu et al. 2019
	NMR19	Retrotransposon	Positive	Age	He et al. 2018
	ERF34	Transcription factor	Negative	Age,Dark and Salt	Park et al. 2022
	PCAP2	ABA signaling pathway member	Positive	Cold and Dark	Panigrahy et al. 2021
	CYP708A3	Brassinosteroid signaling pathway member	Positive	Cold and Dark	Panigrahy et al. 2021
	WRKY22	Transcription factor	Positive	Dark	Zhou et al. 2011
	CLE42	Peptide hormone	Negative	Age	Zhang et al. 2022a, b
	ULS1/2	RING-box proteins	Negative	Age	Wei et al. 2022
	HOS15	Chromatin modulator	Positive	Dark	Zareen et al. 2022
	Ran1-GTP	Nuclear transport protein	Positive	Age and Dark	Pham et al. 2022
	WHIRLY1	Regulation of transcription	Negative	Age	Huang et al. 2022
	AHL9	Transcription Factor	Positive	Age and Dark	Zhou et al. 2022
	FBN2	Fibrillin	Negative	JA-induced	Kim et al. 2022
	PLDα1	Phospholipase	Negative	Mg + +	Kocourkova et al. 2021
	Tic55	Chloroplast translocon	Positive	Dark	Hsu et al. 2022
	REV	Transcription factor	positive	Age and redox changes	Xie et al. 2014

Table 1 (continued)

Species	SAGs	Functional Category	Regulation of Senescence	Induced by	Reference
Oryza sativa	Ghd2	Transcription factor	Positive	Drought	Liu et al. 2016
	GNT1	protein modification	Negative	Age, Dark	Fanata et al. 2013
	Lhca4	Light signalling	Positive	Age	Yamatani et al. 2018
	NYC1	Reductase	Positive	Dark	Sato et al. 2009
	NYC4	Chlorophyll degradation	Positive	Age, Dark	Yamatani et al. 2013
	OsHox33	Transcription factor	Positive	Age	Luan et al. 2013
	OsLOX2	Lipoxygenase	Positive	Age	Huang et al. 2014
	OsMYB102	Transcription factor	Negative	Dark and ABA	Piao et al. 2019
	OsNAC054	Transcription factor	Positive	Dark and ABA	Sakuraba et al. 2020
	OsNAC106	Transcription factor	Negative	Age, Dark	Sakuraba et al. 2015a
	OsNAC2	Transcription factor	Positive	Age, ABA	Mao et al. 2017
	OsNAP	Transcription factor	Positive	Age, ABA	Liang et al. 2014
	OsSIK2	Transcription factor	Negative	Age, Salt, drought, cold, dark, and ABA	Chen et al. 2013
	OsWRKY5	Transcription factor	Positive	Age, Dark	Kim et al. 2019
	SPOTTED LEAF3	Transcription regulation	Positive	Age, Dark	Wang et al. 2015
	OsPSL50	Transporter	Negative	Heat	He et al. 2021
	OsWRKY23	Transcription factor	Positive	Dark	Jing et al. 2009

RNA-seq analysis of salt stress-induced leaf senescence in *Medicago truncatula* revealed regulation of more than 4000 SAGs. Of these, 1546 were also reported to express commonly in dark and salt-induced senescence (Dong et al. 2021). Studies on the rice early leaf senescence mutant, *bilateral blade senescence 1* (*bbs1*), demonstrated its hypersensitivity to salt stress indicating a close relation between leaf senescence and salt stress pathways (Zeng et al. 2018). Detailed analysis of the *bbs1* mutant identified an insertion in the coding region of a receptor-like cytoplasmic kinase, OsRLCK109. Furthermore, it was found that the gene coding for *OsRLCK109* was induced during salt stress. A recent study by Park et al. (2022) revealed the intersection of salt stress response with leaf senescence through the characterization of Arabidopsis ETHYLENE RESPONSIVE FACTOR34 (ERF34). ERF34 exhibited a differential expression pattern during developmental senescence, and its negative regulatory role in salt stress-induced and developmental senescence is also reported. The crosstalk between salt stress and senescence is supported by ER34-mediated transcriptional activation of salt-stress responsive genes, EARLY RESPONSIVE TO DEHYDRATION10 (ERD10) and RESPONSIVE TO DESICCATION29A (RD29A) (Park et al. 2022).

Drought stress and senescence

Drought stress triggers responses ranging from altered gene expression to changes in plant metabolism and growth, including leaf senescence which causes a substantial decrease in canopy size and reduced yield. Some commonality has been observed in the symptoms of senescence induced by different stressors such as drought, salinity as well as developmental age-related leaf senescence. For example, drought-induced senescence involves stomatal closure, the most notable phenomenon in older senescing leaves for allowing reduced water loss through transpiration at the whole plant level (Bosch and Alegre 2004). Carbon/nitrogen balance is another critical factor for regulating drought-induced leaf senescence that was studied in *Sorghum bicolor* leaves (Chen et al. 2015). A direct role of cytokinins and ABA in gene reprogramming during drought-induced senescence has also been proposed given that the high ABA enhanced carbon mobilization from senescing leaves to grains in the drought-stressed rice and wheat (Reguera et al. 2013). A study on wheat landraces subjected to drought stress revealed that the biosynthesis of stem-specific proteins halts and to compensate for the lower assimilate synthesis rate, stem senescence and remobilization processes are triggered (Bazargani et al. 2011). In tobacco, the expression of *Isopentenyltransferase* (*IPT*) driven by stress and maturation-inducible promoter (pSARK) enhanced drought tolerance by delaying leaf senescence (Rivero et al. 2007).

Nutrient limitation and senescence

Plants require both macro- and micro-nutrients for proper growth and development. The unavailability of these

nutrients often causes starvation, which eventually leads to premature leaf senescence in plants. The nitrogen-limiting condition triggers chloroplast degradation to initiate nitrogen recycling in plants. A field experiment with sixteen tropical maize varieties exhibiting wide variation for grain yield at a low nitrogen supply (N) showed a negative correlation between leaf senescence at the grain filling stage and N-use efficiency (Schulte et al. 2007). The authors also pinpointed the possibility of screening these genotypes for N deficiency-induced senescence survival as a parameter for identifying N-use efficient maize cultivars. Another indispensable macroelement necessary for optimal growth and development of all crops is sulfur (S), which is available as sulphate ($SO4^{2-}$) in soil and as sulphur dioxide (SO_2) in the environment. Interestingly, the effects of S deprivation are influenced by the availability of N as observed in oilseed rape wherein delay in leaf senescence was reported under the low S-high N condition (Dubousset et al. 2009). Delay in senescence under S-deficient conditions has been observed in barley, where the process is accompanied by down-regulation of two cysteine- and one serine-protease gene (Veliz et al. 2020). These results highlight the requirement of S for the proper onset of leaf senescence, which ultimately affects the grain quality.

Iron (Fe) is an essential micronutrient indispensable for the functioning of key biological processes, including photosynthesis and respiration (Morrissey and Guerinot 2009). Studies on wheat seedlings revealed that Fe and N deficiency induces leaf senescence. A significant reduction in Fe concentration was observed under − N / + Fe compared to + N/ + Fe condition indicating a possible interplay between varied levels of N supply and Fe accumulation. Furthermore, inhibition of Fe export from senescing leaves to younger leaves under high N supply and an enhanced Fe export under N-deprived conditions was also reported (Parveen et al. 2018). Zakari et al. (2020) investigated the relationship between N deficiency-induced leaf senescence and ABA concentration in *psf* (premature senescence of flag leaf) mutant versus wildtype (WT) rice plants. They further demonstrated a significant level of ABA accumulation and up-regulation of ABA biosynthesis genes (9-cis-epoxycarotenoid dioxygenases or NCEDs), ROS burst and enhanced expression of SAGs in *psf* mutants compared to the WT plants under N deficiency condition. In addition, ABA accumulation showed a reversible pattern on N supplementation with an enhanced expression of ABA catabolic genes. Based on these results, it was concluded that adequate N supply has an inhibitory effect on ABA levels resulting in low ROS levels and delayed leaf senescence. Optimal levels of another macronutrient, Magnesium (Mg^{2+}) are also crucial for plant growth and development given that it is required for several essential cellular processes, including photosynthesis, protein and nucleic acid synthesis and energy metabolism (Guo et al. 2016). As per the study by Kocourkova et al. (2021), a phospholipase Dα1 (PLDα1) acts as a negative regulator of leaf senescence induced by high Mg^{2+} levels in *Arabidopsis*. Higher accumulation of ABA and JA were detected in *pldα1* mutant under high Mg^{2+} conditions. These studies carried out on *pldα1* and *psf* mutants highlight the overlap between the pathways regulating nutrient stress-induced senescence and natural ageing in plants.

Biotic stress and senescence

Various biotic stress factors, including pests and pathogens, challenge the plants during their growth and development. The pathogenic infection mechanisms interact with the developmental pathways to mutually influence each other owing to the cross-talk between their signalling pathways and convergence at several regulatory nodes (Guo et al. 2021). The complexity increases with the highly variable lifestyle of different pathogens influencing the plant developmental program in an unusual manner leading to an adverse effect on plant productivity. Necrotrophs induce premature senescence, while biotrophs delay host plants' ageing progression. WRKY TFs (WRKY6, 53, 70 and 30) are associated with the senescence program and are also well-known regulators of defense responses (Guo et al. 2021). Expression analysis of genes encoding for WRKY TFs indicated that five senescence-inducible OsWRKY genes (*OsWRKY 2, 6, 14, 26,* and *93*) were also upregulated in rice infected with *Magnaporthe oryzae* (Wei et al. 2013). Dark-induced leaf senescence was delayed in *Magnaporthe*-resistant transgenic rice lines overexpressing *OsWRKY93*, whereas an opposite phenotype was observed in *oswrky*93 mutant. It is thus believed that OsWRKY93 is a potential candidate for the breeding of rice cultivars with enhanced yield and resistance to *Magnaporthe* infection (Li et al. 2021b). Remarkably, the mutants of positive regulators of senescence, *ein2, ore1* and *nac055* (stay-green mutants), showed an altered age-related resistance against *Pseudomonas syringae* pv *tomato* (Schippers et al. 2015). In *Arabidopsis,* infection with *Botrytis cinerea* induced several senescence-associated genes (SAGs) and suppressed the expression of photosynthesis and starch metabolism genes (Windram et al. 2012). Interestingly, several phytohormones play a crucial role in regulating senescence and host defense responses. Several factors and signalling pathways regulate pathogen-induced senescence as the host needs to maintain developmental homeostasis and simultaneously activate defense responses for achieving resistance to infections. For instance, ethylene, ABA and SA signalling were activated upon *Botrytis cinerea* infection in Arabidopsis, indicating a crosstalk between developmental and biotic stress-induced senescence processes (Windram et al. 2012).

Oxidative stress and senescence

Most of the abiotic and biotic stresses trigger the accumulation of reactive oxygen species (ROS) and oxidative stress (Jajic et al. 2015). Resultantly, a high level of ROS mainly drives the oxidation of lipid membranes and damage to cellular biomolecules, which ultimately culminates into cellular, structural and functional damage. During oxidative stress plants activate the gene expression pathways of antioxidative enzymes, such as catalases, superoxide dismutases (SODs) and ascorbate–glutathione to maintain the redox homeostasis. Studies on plant chloroplasts by Munné-Bosch and Alegre (2002) revealed that a higher accumulation of ROS occurs in chloroplasts upon ageing which act as signalling molecules to activate the expression of several TFs and SAGs that are key for the progression of senescence (Garapati et al. 2015). Similarly, the singlet oxygen has been shown to induce the expression of WRKY6, which is vital for the senescing process (Jajic et al. 2015).

Moreover, a senescence-associated metallothionine protein, LSC54, has been shown to accumulate and correlate with the rising ROS levels during oxidative stress in *Arabidopsis*. Application of catalase inhibitors on leaves increased the expression of *LSC54,* while the treatment of leaves with quenchers of ROS downregulated the expression of *LSC54* (Navabpour et al. 2003). Additionally, the analysis of delayed leaf senescence in *Arabidopsis* mutants, *ore1, ore2* and *ore9*, revealed that they were highly tolerant to oxidative stresses (Woo et al. 2004). REV or REVOLUTA is a redox-sensitive HD-ZIPIII TF that has been shown to positively regulate age-triggered leaf senescence in *Arabidopsis* (Xie et al. 2014). A recent study on oxidative stress-sensitive rice T-DNA mutant, *RLS1* (reactive oxygen species-sensitive leaf senescence1) further highlighted the relationship between oxidative stress and senescence given that several SAGs and autophagy-related genes were upregulated during oxidative stress (Chen et al. 2018).

Heat stress and senescence

Generally, the elevation in temperature above the threshold level adversely impacts plant growth and crop yield (He et al. 2021). Severe heat stress induces cellular senescence by chloroplast disruption, photosynthesis impairment, initiation of DNA damage, ROS accumulation, and cell death (Fedyaeva et al. 2014). Heat stress-induced premature senescence has been attributed to accumulation of soluble sugars, decline in starch levels, degradation of soluble proteins, and accumulation of H_2O_2 with a concomitant decrease in antioxidant activity in sunflower primary leaves (Haba et al. 2014). Recently, *PSL50* (Premature senescence leaf 50) was shown to play an important role during heat-induced premature leaf senescence by modulating the H_2O_2 signaling pathway in rice (He et al. 2021). Nevertheless, the involvement of phytohormones in regulating stress responses in plants has also been well-documented (Guo et al. 2021). While there is an elevated production of both ethylene and ABA, a decline in cytokinin levels during senescence triggered by high temperature has been recorded in leaves of bentgrass (Xu and Huang 2007). Remarkably, a transcriptome-based study also revealed differential regulation of several heat shock transcription factors (HSFs) during leaf senescence in *Arabidopsis* (Raxwal et al. 2012). More details on HSFs and senescence are provided in the following section.

Cold stress and senescence

Cold stress affects plant growth and development by indirectly imposing osmotic and oxidative stresses as well as by changing the metabolic reactions and membrane properties, which eventually triggers plant senescence. Cold stress-induced leaf senescence indeed is an effective strategy for plants growing in a low temperature area to overcome extreme low temperatures (Caselles et al. 2021). Initiation of senescence in the aboveground tissues of *Iris pseudacorus* was documented during the winter seasons. In contrast, the underground rhizome remains dormant and re-establishes growth when favourable conditions return. Interestingly, the cold-acclimated *Arabidopsis* plants showed delayed senescence in rosette leaves with a marked recovery of Fv/Fm ratio after an initial decline (Daubresse et al. 2007). Cold stress induces the accumulation of *SAGs* in different plant species (Yang et al. 2017). Chilling stress treatment, alone or in combination with *Alternaria* sps. infection, resulted in pronounced leaf senescence in cotton (Zhao et al. 2012). This was evident by an increase in malondialdehyde activity, electrolyte leakage, and a decline in chlorophyll and soluble protein content. In *Iris pseudacorus*, induction of leaf senescence and an appreciable increase in ABA/cytokinin ratio has been observed during winters (Caselles et al. 2021). Transcriptome analysis of *Arabidopsis* plants exposed to cold and dark conditions showed that the ABA pathway positively regulates the leaf senescence process, while it is negatively regulated by the brassinosteroid pathway (Panigrahy et al. 2021).

Regulation of Leaf Senescence

Owing to the importance of leaf senescence in crop productivity, extensive physiological, biochemical, molecular and genetic studies have been conducted to unravel the complex, multi-tiered regulatory processes in action under

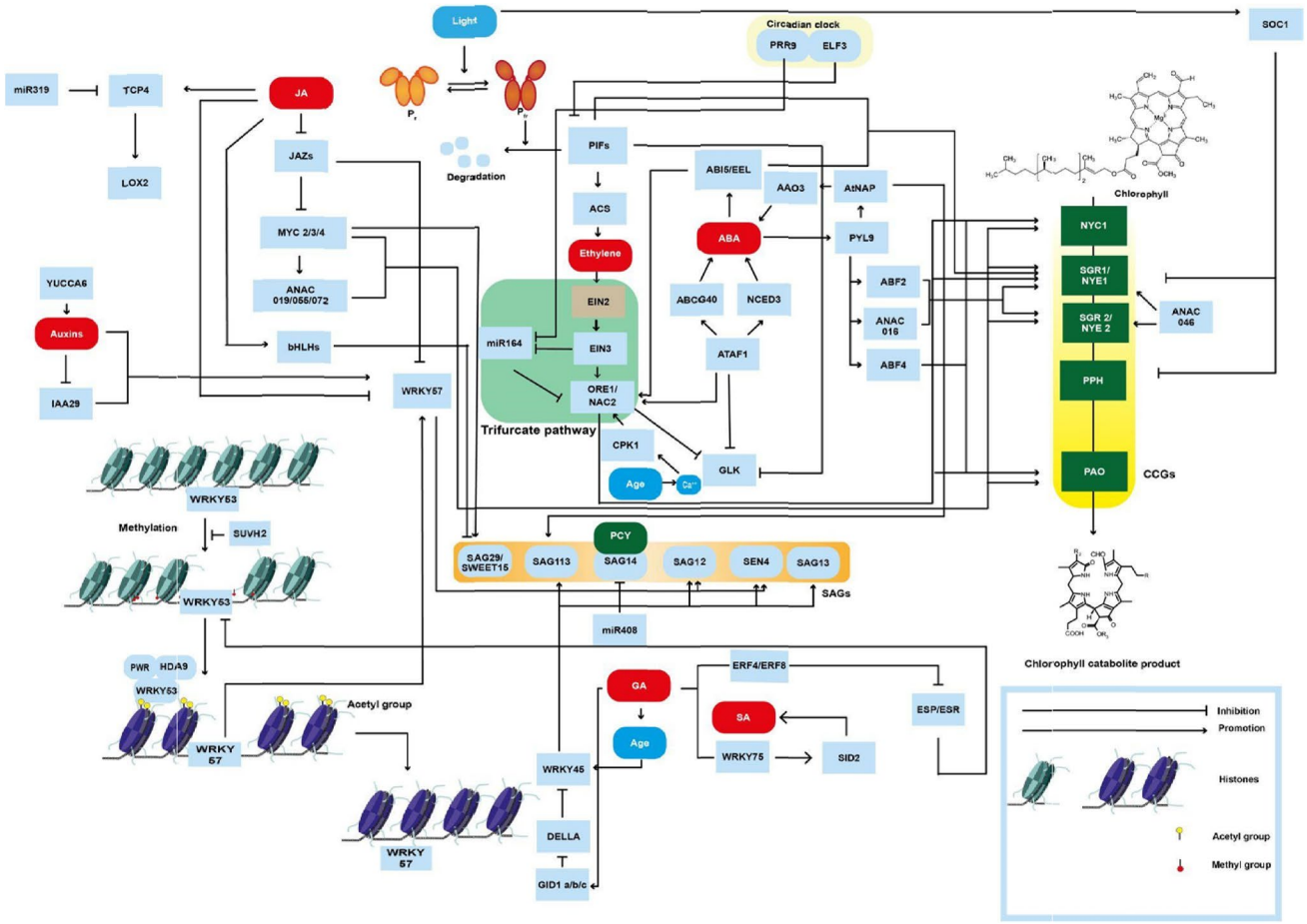
the influence of several internal and external factors during senescence to improve crop yield, an ultimate research goal (Schippers et al. 2015). Understanding these regulatory pathways during senescence could open up new avenues in senescence research, which can further facilitate agronomic applications for breeding new crop cultivars with stable and improved yield. This review compiles the information available on the transcriptional, post-transcriptional, epigenetic, and hormonal level regulation of leaf senescence which may further aid in devising strategies for genetic enhancement of multiple crop species.

Gene Programming Changes During Leaf Senescence

Several experimental approaches, including microarray, mRNA sequencing, Northern blot analyses, and differential screening studies using subtractive hybridisation techniques have been employed to study the gene expression changes during senescence in various plant species. All these studies demonstrated that extensive changes in the expression of specific genes, commonly referred to as SAGs, accompany the senescence programme (Lim et al. 2003). The initial differential screening studies in *Arabidopsis thaliana* have identified approximately 800 cDNA clones representing SAGs; of which, 130 were found to be non-redundant genes and 70 as new SAGs, which exhibit either leaf senescence-specific expression pattern or upregulation during the process (Gepstein et al. 2003). Efforts have been made to provide up-to-date information on the significant classes of SAGs (Table 1) that have been demonstrated to act as regulators of leaf senescence in model plant systems Arabidopsis and rice.

The transcriptional control mechanism is crucial in coordinating the senescence process through massive reprogramming of gene expression, including transcription factors (TFs) that regulate gene expression by binding to the specific cis-regulatory elements and triggering their activation or suppression. Microarray analysis of senescing leaves revealed that 100 putative TFs belonging to approximately 20 different families, especially NAC, WRKY, C2H2 type zinc finger, AP2/EREBP, bZIP, CCAAT binding, Leu zipper, MADS-box, HSFs, kinases and MYB proteins family were upregulated at least three-fold in *Arabidopsis* (Wollaston et al. 2005; Balazadeh et al. 2008). A similar expression pattern of several of these TFs was also observed in the transcriptomic studies of leaf senescence conducted in other plant species such as maize, cotton and rice (Woo et al. 2019). A survey of available literature revealed a huge amount of information on the TFs associated with leaf senescence, which is beyond the scope of this review. A brief enumeration of the selected class of TFs that are key regulators of leaf senescence is given below.

NAC (NAM, ATAF and CUC) proteins constitute one of the most prominent families of plant TFs that play important regulatory roles in the development, senescence and stress responses of various plant species (Broda et al. 2021; Nie et al. 2021). More than 30 NAC genes are upregulated during leaf senescence in *Arabidopsis* (Breeze et al. 2011). Manipulation in the expression of NAC genes resulted in the alteration of leaf senescence. AtNAP or ANAC029 in particular regulates leaf senescence by binding to the promoter of *SAG113*, a negative regulator of the ABA pathway, resulting into inhibition of stomatal closure and the eventual initiation of leaf senescence (Zhang and Gan 2012). ORE1 or ANAC092 is another positive regulator of leaf senescence in *Arabidopsis,* which regulates at least 170 genes, including the 78 known SAGs and is induced by EIN2 (ETHYLENE INSENSITIVE 2 (Balazadeh et al. 2010). It is believed that with progression in ageing, ORE1 expression elevates and in turn, physically sequesters GLKs (chloroplast activity maintainer) resulting in their reduced transcriptional activity with an ultimate effect on chloroplasts (Rauf et al. 2013). The ORE1, therefore, plays a dual role in controlling leaf senescence: firstly, by triggering the transcription of *SAG*s and secondly, by physically interacting with other senescence-associated TFs, thereby modulating their activity (Fig. 2). A novel regulator mode constituted by NAC075, CATALASE (CAT) and ROS has been shown to actively govern the initiation and progression of leaf senescence (Kan et al. 2021). The studies on transgenic Arabidopsis plants with altered expression of *NAC075* reported its negative regulatory role in senescence as NAC075 promotes the expression of CAT2 by directly binding to the promoter region, thereby reducing the reactive oxygen species levels in the system and delaying the senescence process. Similarly, other NAC transcription factors which are positive regulators of senescence have also been reported in *Arabidopsis* (ATAF2, SNAC-As, ANAC017, ORE1/ANAC092), maize (ZmNAC126) and rice (OsNAC2 and ONAC011) (Mao et al. 2017; Guo et al. 2021; Zhang et al. 2021). A positive regulator of leaf senescence, ANAC046 directly binds to the promoter regions of the genes involved in the breakdown pathway of chlorophyll [*NON-YELLOW COLORING1, Stay-Green 1, Stay-Green 2,* and *PHEOPHORBIDE α OXYGENASE*] (Yamamizo et al. 2016). In *Arabidopsis*, the ANAC019/055/072 and NAC016 activate the chlorophyll degradation during leaf senescence by directly binding to the promoter region of the chlorophyll catabolic gene *SGR1* (Zhang et al. 2021). Apart from the positive regulatory role, few of the NAC TFs are also reported to function as negative regulators in the senescence process. For example, DRL1 was shown to negatively regulate plant senescence in grapevine by fine-tuning the ABA biosynthesis pathway (Zhu et al. 2019). JUNGBRUNNEN 1 (JUB1 or ANAC042), which is induced by H_2O_2, activates the expression of *DREB2A* and several ROS-responsive



genes. Indeed, it is also believed to lower the cellular H_2O_2 levels and reduce the effects of positive regulators of senescence (Wu et al. 2012). It is conceivable that JUB1 is a possible negative regulator of leaf senescence. On the other hand, overexpression of a NAC TF, *TaNAC-S*, resulted in the delay in senescence with a concomitant increase in grain yield and grain protein concentration in wheat indicating its role as a negative regulator of senescence (Zhao et al. 2015).

The WRKY family of TFs is comprised of many members implicated in multiple plant processes, including developmental and stress responses (Zhang et al. 2021). WRKY proteins usually interact with other proteins, including regulatory factors, to constitute an essential component of the kinases signalling cascade. Notably, a complex regulatory network involving combinatorial interactions of WRKY TF family members fine-tune the leaf senescence process. For instance, the EPITHIOSPECIFYING SENESCENCE REGULATOR (ESR) inhibits the DNA-binding activity of WRKY53 and acts as a negative regulator of senescence (Woo et al. 2019). Similarly, WRKY13-A, a partial functional homolog of AtWRKY53, positively regulates both dark-induced and natural leaf senescence by promoting JA biosynthesis in wheat (Qiao et al. 2021). WRKY54 and WRKY70 both regulate leaf senescence and the positive regulator WRKY53 possibly interacts with WRKY30 and targets WRKY22. Together they constitute a major part of the regulatory network that integrates with internal and external signals to regulate the initiation and progression of leaf senescence (Jiang et al. 2014). A model that depicts the tripartite amplification loop involving WRKY75, SA, and ROS has been proposed by Guo et al. (2017) in *Arabidopsis*. The positive regulator of senescence, WRKY75, induces the transcription of *SA INDUCTION-DEFICIENT2 (SID2)* and promotes SA production during senescence. At the same time, WRKY75 suppresses the H_2O_2 scavenging by repressing *CAT2* expression. Auxin and JA are shown to act antagonistically on WRKY57, a negative regulator of JA-induced leaf senescence (Jiang et al. 2014). The JAZ4/8 and IAA29 function as negative and positive regulators of JA-induced leaf senescence, respectively, and interact competitively with the zinc-finger domain of WRKY57. The WRKY57 binds directly to the promoters of *SEN4* and *SAG12* and suppress their transcription. WRKY45 positively regulates leaf senescence by activating the transcription of *SAG12, 13, 113* and *SEN4*. RGL1 (a repressor of the GA signalling pathway) interacts with WRKY45, resulting in the loss

◂**Fig. 2** An overview of multi-tiered natural leaf senescence regulatory network. The signalling pathways of phytohormones JA, ethylene, ABA, SA, and GA initiate and promote leaf senescence in leaves. By binding to the promoters of important chlorophyll (Chl) catabolic genes (*NYE1, NYC1* and *PAO*), MYC proteins regulate JA-induced Chl degradation downstream of JAZs in the JA signalling pathway. Moreover, MYCs indirectly regulate Chl degradation through the transcription factors ANAC019/055/072, which can trigger the activation production of the same Chl catabolic genes (CCGs). AtNAP positively regulates leaf senescence by promoting ABA production and SAG113 expression. ATAF1 contributes to ABA-induced senescence by activating the expression of genes involved in ABA biosynthesis and transport (NCED3 and ABCG40). ATAF1 enhances and suppresses the expression of ORE1 and GLK1, respectively, by directly binding to their promoter region. As a result, the expression of GLK target genes is hindered, resulting in an age-dependent drop in the expression of GLKs, whilst the expression of ORE1 target genes is increased, triggering senescence. ABA-induced senescence is mediated by the action of ABFs downstream of ABA signaling modules. EIN3, which is activated by EIN2, represses miR164 transcription by binding directly to the its promoter region which results in elevation of *ORE1* transcript levels, thereby promoting leaf senescence as ORE1 activates the transcription of *SAG29*, *SINA1* and *SWEET15* and represses the expression of GLKs. PRR9 activates ORE1 and suppresses miR164 indirectly during leaf senescence. PIFs, whose expression is inhibited by ELF3, promote chloroplast deterioration by suppressing GLKs. Age and GA-induced WRKY45 promotes the expression of a number of SAGs. WRKY75 is involved in a tripartite amplification loop where it promotes SA synthesizing gene SID2. JA-induced TF, TCP4, positively regulates leaf senescence by enhancing the expression of LOX2. Whereas bHLHs, negatively regulate leaf senescence by repressing SAG29 expression. Leaf senescence is also regulated at epigenetic level. Expression of WRKY53 is partially mediated by methylation of histones via SUVH2. WRKY53, in turn along with PWR and HDA9, removes the acetylation marks from the histones of WRKY57 thereby leading to its suppression which influences the antagonistic regulation of leaf senescence by auxin and JA. Expression of WRKY57 protein level is positively mediated by auxin, whereas JA represses its expression at transcript level. The age-induced Ca^{2+} levels promote leaf senescence by activating the Ca-dependent protein kinase (CPK1), which in turn phosphorylates and activates the master regulator of senescence, ORE. The blue boxes represent the cytoplasmic/nuclear regulatory component of senescence; green boxes represent chloroplastic regulatory components; red boxes represent phytohormones associated with senescence. LOX: Lipoxygenase; IAA: INDOLE-3-ACETIC ACID INDUCIBLE; JAZ: JASMONATE ZIM-DOMAIN protein; ANAC: Arabidopsis NAC transcription factor; PIF: Phytochrome interacting factor; ACS: 1-aminocyclopropane-1-carboxylate synthase; EIN: Ethylene insensitive; ORE: Oresara; CPK: calcium dependent protein kinase; GLK: Golden2-like transcription factor; ATAF: Arabidopsis thaliana ACTIVATING FACTOR; ABCG40: ARABIDOPSIS THALIANA ATP-BINDING CASSETTE G40; NCED: 9-cis-epoxycarotenoid dioxygenase; ABA: Abscisic acid; AAO: Arabidopsis aldehyde oxidase; ABI: ABSCISIC ACID INSENSITIVE; PRR: PSEUDO-RESPONSE REGULATOR; ELF: early-flowering; PYL: pyrabactin resistance-like; ABF: ABRE-binding factors; NYC: NON-YELLOW COLORING; SGR: STAY GREEN; PPH: PHEOPHYTINASE; PAO: pheide a oxygenase; SOC: SUPPRESSOR OF OVEREXPRESSION OF CO; SAG: Senescence-associated gene: PCY: Plastocyanin; SUVH: SU(VAR)3–9 homolog; HDA: histone deacetylase; GID: GA INSENSITIVE DWARF; SID: SALICYLIC ACID INDUCTION DEFICIENT; ESP: Epithiospecifier protein; ERF: Ethylene-responsive element binding factors; JA: Jasmonic acid; GA: Gibberellic acid; SA: Salicylic acid

of its transcriptional activation potential (Woo et al. 2019). WRKY93 has been shown to play a dual role in regulating flag leaf senescence and response to fungal pathogen infection in rice (Li et al. 2021b).

Besides NAC and WRKY TFs, another TF family associated with the regulation of leaf senescence is MYB. Overexpression of R-R type *MYB-like transcription factor* (*MYBL)* in *Arabidopsis* displayed an enhanced senescence phenotype (Zhang et al. 2021). The OsMYB102 demonstrated a negative role in plant senescence via regulating the ABA accumulation and the signalling cascades by activating and repressing the ABA catabolic enzyme ABSCISIC ACID 8′-HYDROXYLASE and ABA-responsive genes (*OsABF4* and *OsNAP*), respectively (Guo et al. 2021). In *Arabidopsis,* RAV1, a related to ABI3/VP1 (RAV) TF family member was found to act as a positive regulator of leaf senescence (Woo et al. 2010). Several research groups have also reported the involvement of various basic helix-loop-helix (bHLH) TFs, such as MYC2, MYC3, MYC4, to antagonistically interact with bHLH03, bHLH13, bHLH14 and bHLH17 and activate *SAG29* expression for initiating JA-triggered leaf senescence (Woo et al. 2019). The phytochrome-interacting bHLH TFs, such as PIF4 and PIF5, positively regulate the dark-induced and natural leaf senescence in *Arabidopsis* (Li et al. 2021a). PIF4 binds to the promoters of *NYE1* (a chlorophyll degradation regulatory gene) and *GLK2*, which eventually initiates chlorophyll degradation machinery (Woo et al. 2019).

Several genes encoding HSFs have also shown significant expression changes during leaf senescence in *Arabidopsis* and rice (Raxwal et al. 2012). Particularly, the AtHSFB1 and AtHSFA6a were upregulated during leaf senescence in *Arabidopsis* (Balazadeh et al. 2008). As a proof, the *hsfB1* mutants exhibited early leaf senescence and significant upregulation of several SAGs, including *SAG12, WRKY* and peroxidase gene. The transcriptomic studies in maize, cotton, rice, petunia and bermudagrass also showed differential expression of several HSFs during age-dependent leaf senescence (Lin et al. 2015; Wang et al. 2018; Fan et al. 2019). All these studies demonstrate the complexity of signaling networks associated with leaf senescence and demand the identification of more TFs to gain a better understanding of transcriptional regulation during the last stage of leaf development process.

Epigenetic Control of Senescence

Epigenetic regulation involves modifications in gene expression without any change in the genomic sequence. This involves chemical alterations of DNA such as methylation, changes in chromatin modelling, post-translational modifications of histones, and involvement of non-coding RNAs (Guo et al. 2021). The developmental switches associated with the transition from cell survival to cell death in

leaves are also tightly controlled by epigenetic and genetic mechanisms, which ultimately regulate the changes in SAGs expression. Here, we summarize the epigenetic mechanisms that regulate leaf senescence in plants.

DNA methylation

DNA methylation ensures the silencing of transposons, DNA repeats, and gene body, and thereby controls the gene expression and protects the genome from superfluous mutations. The methylation of cytosine nucleotides resulting in the formation of 5-methylcytosine is the most frequent phenomenon in plants (Law and Jacobsen 2011). In *Arabidopsis,* the CG and CHG type methylations are controlled by MET1 (METHYLTRANSFERASE 1) and CHROMOMETHYLASE 3 (CMT3), respectively, while the DOMAINS REARRANGED METHYLTRANSFERASE 2 (DRM2) catalyzes the symmetric and asymmetric DNA methylation (Feng et al. 2010). Mutations in these methyltransferases (*MET1*, *DRM2* and *CMT3*) trigger pleiotropic developmental abnormalities due to genome-wide hypomethylation (Moritoh et al. 2012). Currently, the clarity on senescence-specific DNA methylation changes in plants, however, is lacking, but few reports indicate that the status of DNA methylation alters as the plant ages (Zhang et al. 2021). During senescence, the retrotransposon controlling epigenetic mechanisms ceases to function which results in the demethylation of transposable elements (TEs) and thereby facilitating their transcription and transposition (Guo et al. 2021). A study by He et al. (2018) identified a retrotransposon, NMR19 (naturally occurring DNA methylation variation region 19), whose methylation level and genomic location varied among different accessions of *Arabidopsis thaliana*. It was thus concluded that NMR19-4 is a novel naturally occurring epiallele that regulates leaf senescence by controlling the expression of *PHEOPHYTIN PHEOPHORBIDE HYDROLASE* (*PPH*) and eventually tuning the levels of chlorophyll. Henceforth, it would be interesting to investigate the role of transposable elements (TEs) in plant senescence. Ogneva et al. (2016) demonstrated a decline in the transcription of DNA methyltransferase genes such as *CMT3* and *MET1* during ageing, while the expression of demethylase genes like *ROS1*, *DME*, *DML2* and *DML3* were elevated at certain stages of development in *Arabidopsis*. Yuan et al. (2020) also revealed an epigenetic regulatory mechanism controlling leaf senescence in *Arabidopsis*. The *DML3* expression level increases exponentially and activates the *SAG*s by demethylating their promoters, gene body and 3' UTRs (upstream regulatory regions) during senescence. Arellano et al. (2020) showed that genes mediating chromatin silencing were down-regulated during dark-induced senescence, which disrupted the silencing of TEs and finally led to the reactivation of young TEs. All these studies demonstrate the relevance of DNA methylation in epigenetic reprogramming during the later phases of plant development, including leaf senescence**.**

Histone modifications

Histones are post-translationally modified at their N-terminal tail through covalent modifications, including methylation, ubiquitination, acetylation, SUMOylation and phosphorylation, which influences the transcriptional activity of chromatin (Guo et al. 2021; Zhang et al. 2021). Of these, histone acetylation and methylation are the two key modifications associated with leaf senescence. Acetylation of lysine residue has been correlated with transcriptional activation of chromatin. The *Arabidopsis* mutants for HISTONE DEACETYLASE19 (HDA19) and HDA6 showed pleiotropic developmental defects, including increased leaf longevity and delayed flowering (Wu et al. 2008). While the expression of jasmonate-responsive genes (*PDF1.2*, *VSP2*, *JIN1* and *ERF1*) and *SAG*s (*SAG12* and *SEN4*) was down-regulated, the transcript level of *FLOWERING LOCUS C (FLC)* was elevated in the *HDA6*-RNAi plants. HDA6 turns off the expression of *FLC,* a floral repressor by deacetylating the histones associated with *FLC* (Wu et al. 2008). AtHDA9 (an RPD3-like histone deacetylase) promotes the initiation of leaf senescence in *Arabidopsis* by interacting with the transcription factor WRKY53 and the SANT domain-containing chromatin-binding protein POWERDRESS (PWR). The genome wide HDA9 occupancy profiling has shown that it directly binds to the promoters of critical negative regulators of senescence with a requirement of PWR (Woo et al. 2019). In addition to histone acetylation, histone methylation also plays a significant role in epigenetic control of gene expression. During the onset of senescence, a higher expression level of *WRKY53* has been observed because of the establishment of active marks of H3K4me3 at *WRKY53*-associated histones (Zhang et al. 2021). A ChIP-seq study of non-senesced and senesced leaves revealed that H3K4me3 is linked to active chromatin, whereas the H3K27me3 mark is associated with repressed chromatin. Moreover, overexpression of histone methylases demonstrated ectopic heterochromatinisation and delayed senescence phenotypes in *Arabidopsis*. JMJ16, a JmjC domain-containing protein, is particularly an H3K4 demethylase that inhibits leaf senescence through its enzymatic activity in *Arabidopsis*. Genetic studies have shown that JMJ16 is a negative regulator of leaf senescence which inhibits the expression of positive regulators, WRKY53 and SAG201. JMJ16 binds to WRKY53 and SAG201 and reduces H3K4me3 levels at these loci leading to suppression of their early expression in mature leaves (Liu et al. 2019). As *Arabidopsis* plants mature, the double-strand breaks (DSBs) increase due to the decline in efficacy of DNA repair mechanisms. A premature senescence phenotype results from the generation of DSBs through the

inducible expression of an intron-encoded endonuclease (I-*PpoI*). The histone lysine methylation regulated by ATM (ATAXIA TELANGIECTASIA MUTATED) represses the DSB-induced expression of senescence-associated genes, including those encoding WRKY *and NAC* TFs, the essential components of the leaf senescence process (Li et al. 2020).

Chromatin alterations

ATP-dependent chromatin remodeling factors can recognise the histone modifications through their histone-binding motifs, bromo or chromodomains, and can non-covalently restructure nucleosomes by disrupting or destabilising their structure (Brusslan et al. 2012). A mutation in *DRD1* (a SWI2/SNF2 chromatin remodeling protein encoding gene) affects the progression of dark-induced leaf senescence given an observed decline in SAGs induction (Zhang et al. 2021). The precise underlying mechanisms by which DRD1 is implicated in leaf senescence, however, remains unknown. AT-hook proteins are among the many ATP-dependent chromatin remodeling factors that are involved in leaf senescence and regulation of chromatin structure. The overexpression and activation-tagged mutants of ORESARA 7 (ORE7), an AT-hook protein exhibited a delayed leaf senescence phenotype in *Arabidopsis* (Lim et al. 2007a). Additionally, a genome-wide transcriptome of senescing *Arabidopsis* leaves showed upregulation of chromatin remodelingfactors, CHR10 and CHR19 indicating theirpossible positive role during the progression of senescence (Breeze et al. 2011).

Small non-coding RNAs

Small RNAs are known to regulate the senescence process through fine-tuning the expression of genes involved in signalling pathways, including TFs, phytohormone metabolism and kinases (Xu et al. 2014). For instance, a decline in the level of miR164 has been confirmed, while there is up-regulation in the *ORE1* expression during leaf ageing. The ORE1/AtNAC2 positively controls ageing-induced cell death by enhancing the expression of SAGs in the *Arabidopsis* leaves (Kim et al. 2009). Elaborately, ORE1 is negatively regulated by miR164 during the initial stages of senescence, but this inhibition is released by EIN2 (ETHYLENE INSENSITIVE 2), which governs miR164 levels during later stages of senescence. A trifurcate feed-forward pathway was thus proposed with ORE1, miR164 and EIN2 for fine regulation of leaf senescence in an ethylene-dependent manner (Kim et al. 2009). Later studies by Kim et al. (2018) showed that PSEUDO-RESPONSE REGULATOR (PRR9), a vital component in the circadian clock of plants, directly activates the transcription of *ORE1* and indirectly suppresses miR164, thereby establishing the link between ageing and circadian clocks in plants (Fig. 2). In *Arabidopsis,* miR319 regulates the expression of a TF, TCP (TEOSINTE BRANCHED 1, CYCLOIDEA, PCF1), which in turn, controls *LOX2,* a lipoxygenase that controls JA biosynthesis (Schommer et al. 2008). miR319 is also known to target another positive regulator of senescence, *WRKY53* (Guo et al. 2021). ARF2, a positive regulator of senescence, targets tasiRNAs (trans-acting siRNAs) generated from TAS3 precursor transcript, whose cleavage is mediated by miR390 (Lin and Wu 2004). The miR390-TAS3-ARF2 node is, therefore, hypothesized to play a significant role in regulating leaf longevity. In *Arabidopsis*, the overlapping 3'-UTR of PPR (Pentatricopeptide repeat-containing protein) and WHY3 genes possess a miRNA gene locus MIR840, and both of these genes happen to be the predicted targets of miR840 and miR840*. Ren et al. (2022) reported that an increased accumulation of pre-MIR840 transcripts correlated with a decrease in PPR transcripts, but not with that of WHY3 transcript levels as its protein levels were significantly reduced at the onset of senescence. Overall, the miR840*–PPR and miR840–WHY3 pair modulate the expression of some SAGs with an effect on senescence. Moreover, the overexpression of *Sly*miR208, which targets two cytokinin biosynthesis genes (*Slipt2 and Slipt4*) results in reduced cytokinin levels and accelerated leaf senescence (Zhang et al. 2021). The overexpression and short tandem target mimic (STTM) lines of miR408 also exhibited contrasting dark-induced senescence phenotypes via modulating PCY-SAG14 module and copper reallocation in the chloroplast (Hao et al. 2022). The advent of high-throughput next-generation sequencing (NGS) has further enabled the identification of several small RNAs associated with senescence. For example, six miRNA families, osa-miR159, osa-miR160, osa-miR164, osa-miR167, osa-miR172 and osa-miR1848 were found to be involved in leaf senescence possibly by regulating the phytohormone signalling in rice (Xu et al. 2014). Exploration of miRNAs in stay-green and early leaf senescence lines of maize revealed differential expression of 16 senescence-associated miRNAs (SA-miRNAs) which mainly target TFs and chlorophyll degradation pathway genes (Wu et al. 2016). Sasi et al. (2019) provided the repository of differentially expressed miRNAs during rice flag leaf senescence, wherein 116 novel and 21 known miRNAs were differentially expressed, and their predicted targets encoded for TFs and phytohormone homeostasis pathway components.

Hormonal Regulation of Senescence

Phytohormones, in combination with both developmental and environmental signals, play a significant role in fine-tuning the leaf senescence process. A few of these act as positive regulators while some are negative regulators, and

together they comprise a complex network of signalling pathways. Some of these found to control the senescence process are mentioned below:

Oxylipins, including jasmonic acid and its derivatives, are known as jasmonates (JAs) and are vital signalling molecules critical for the development and stress responses in plants (Guo et al. 2021). COI1 (CORONATINE INSENSITIVE 1; a JA receptor) triggers JA signalling pathway by activating ubiquitin-mediated proteasome degradation of JAZ proteins, a repressor of JA response (Woo et al. 2019). Several studies demonstrated a higher level of JA accumulation due to the induction of JA biosynthesis genes during leaf senescence. Research on *Arabidopsis* revealed that TCP TFs control JA biosynthesis under the tight control of miR319 (Guo et al. 2021). Studies on knock-out mutant of rice *COI1 (oscoi1b-1)* demonstrated a stay-green phenotype and down-regulation of *SAG*s suggestive of a crosstalk between ethylene and JA signalling pathways during senescence (Lee et al. 2015). Antagonistic regulation of JA-induced senescence pathway by bHLH subgroup IIIe (MYC2, MYC3, and MYC4) and IIId (bHLH03, bHLH13, bHLH14, and bHLH17) factors have also been reported (Qi et al. 2015). The MYC2/MYC3/MYC4 proteins reportedly activate the NAC transcription factors (ANAC019/055/072), which in turn trigger chlorophyll catabolic genes (NYC1 and NYE1/SGR1) expression. Very recently, studies on the knockout mutants of *JAZ7* revealed that it represses the expression of *MYC2* leading to the suppression of JA-induced leaf senescence pathway (Guo et al. 2021). JA also regulates the level of H_2O_2 by repressing CAT2 expression and thus, triggering leaf senescence.

Ethylene is a gaseous plant growth regulator. Its accumulation has been reported during leaf senescence (Woo et al. 2019). Transcriptomic studies identified many ethylene response factors (ERFs) controlling leaf senescence in many plant species (Zhang et al. 2021). Plants deficient in *ACC synthase* showed a delayed senescence phenotype, which suggests a positive role of ethylene during leaf senescence (Wang et al. 2002). In Arabidopsis, *EIN2* positively regulate leaf senescence by controlling the expression of *ORE1/NAC2* (Kim et al. 2009). Further, the over-expression of *EIN3* caused early senescence, whereas its silencing resulted in a delayed senescence phenotype (Li et al. 2013). Henceforth, *EIN3* was said to act downstream of *EIN2* and control the *ORE1/NAC2* transcript levels in *Arabidopsis.* The *ORE1/NAC2* is an established positive regulator of leaf senescence given it activates a large set of SAGs.

Cytokinin is a negative regulator of senescence. It is also involved in various plant developmental processes such as apical dominance, shoot and root branching, leaf expansion, the growth of lateral buds, photosynthesis, seed germination, floral transition, and leaf senescence (Lim et al. 2007b). In *Nicotiana,* the expression of *IPT* under the control of a senescence-inducible promoter significantly enhanced the leaf longevity, while under a stress-inducible promoter, it delayed drought-induced senescence (Rivero et al. 2007). Higher accumulation of extracellular invertase (*CIN1*) and hexose transporters were moreover reported upon cytokinin treatments. The cytokinin treatment-based delayed senescence is usually attributable to the consequence of enhanced sink strength of the tissue (Schippers et al. 2015).

Auxin is considered as a senescence-delaying hormone. Its accumulation transiently increases during the onset of senescence. Jiang et al. (2014) demonstrated the repressive role of auxin on *SAG12* expression. In auxin treated *Arabidopsis*, WRKY57 was shown to accumulate and negatively regulate the expression of *SAG12* by directly binding to its promoter. Further, WRKY57 was found to competitively bind to JA and auxin pathway repressors JAZ4/8 and IAA29, respectively. These results indicate that the JA-induced senescence process is antagonised by auxin via WRKY57 (Jiang et al. 2014). An elevated level of auxin response pathway repressor ARF2 has also been observed during senescence. Mutants deficient in *ARF2* exhibited delayed senescence phenotype, indicating its possible positive role in senescence (Lim et al. 2010). Asides from this, ARF7 and ARF19 have also been reported to regulate the onset of leaf senescence in *Arabidopsis* (Ellis et al. 2005).

Abscisic acid is most crucial for plant development and responses to various environmental stresses (Guo et al. 2021). ABA has been reported to be an enhancer of senescence. It functions by promoting the expression of SAGs that are mainly related to chloroplast degradation. These include *NON-YELLOW COLORING1 (NYC1), STAY-GREEN (SGR), PHEOPHYTINASE (PPH)* and *PHEIDE A OXYGENASE (PAO)* (Pruzinska et al. 2005). Additionally, the expression of many NAC TF family members, including *VND-INTERACTING2 (VNI2), A SUBFAMILY OF STRESS-RESPONSIVE NAC (SNAC-A), ORE1, Oryza sativa NAC-LIKE, ACTIVATED BY APETALA3/PISTILLATA (OsNAP)* and *OsNAC2* were also found to be enhanced upon ABA treatment in *Arabidopsis* and rice (Woo et al. 2019). A study by Mao et al. (2017) in rice demonstrated that *OsNAC2* was actively involved in leaf senescence by altering the expression of chlorophyll degradation genes (*OsSGR* and *OsNYC3*). Also, the overexpression of *OsNAC2* resulted in the upregulation of ABA biosynthesis genes and down-regulation of ABA catabolic genes. A positive ABA-mediated regulatory role of *ATAF1* has also been reported during *Arabidopsis* leaf senescence. The binding of ATAF1 to the promoters of *NAC092* and *GLK1* triggers chlorophyll degradation and leaf senescence. Additionally, ATAF1 regulates the ABA-mediated leaf senescence by maintaining the expression of ABA biosynthesis (*NCED3*) and transport (*ABCG40*) genes, but the operational upstream transcriptional network remains unclear (Garapati et al. 2015).

Salicylic acid is a phenolic compound that functions as a signalling compound with a primary role in plant immune responses. Salicylic acid (SA) has been known to accumulate in leaves during leaf senescence (Breeze et al. 2011). SA signalling mutants, *npr1* and *pad4,* exhibited a delayed senescence phenotype indicating the participation of SA in leaf senescence (Schippers et al. 2015). Transcriptome studies have revealed that the SA hormone response pathway was exclusive to developmental senescence (Wollaston et al. 2005). A study on *senescence-associated ubiquitin ligase1* (*saul1*) mutants with an early senescence phenotype have also shown a higher-level expression of SA response pathway genes under low-light conditions, which was related to PHYTOALEXIN-DEFICIENT4 (PAD4)-dependent SA biosynthetic pathway (Vogelmann et al. 2012).

Strigolactone (SL) is a recently identified phytohormone involved in many developmental and stress response processes (Guo et al. 2021). The SL-deficient mutants exhibited a 'stay-green phenotype upon dark treatment. The application of GR24 (a synthetic SL analogue) and ethylene drastically induced SAGs expression and enhanced senescence indicating a positive regulatory role of SL during leaf senescence. The double mutants of *max1* and *ein2* exhibited an enhanced stay-green phenotype which suggests that SL moderately augments leaf senescence in an ethylene-independent manner (Ueda and Kusaba 2015).

Other Regulators of Senescence

Nitric oxide (NO) is a gaseous free radical and an essential signalling molecule in plants. Transcriptome studies of plants over-expressing *NO degrading dioxygenase (NOD)* showed a massive gene-expression change, including the down-regulation of photosynthetic genes and up-regulation of several SAGs and ethylene biosynthesis genes. Literature suggests that ethylene and NO control leaf senescence antagonistically. The early senescence phenotype is moreover attenuated when NOD over-expression plants are subjected to NO treatment. Additionally, an induced expression of *NOD* results in the accumulation of SA in *Arabidopsis* (Mishina et al. 2007). The gene expression studies with NO-deficient *Arabidopsis* mutants also revealed the profound upregulation of chlorophyll catabolic pathway genes resulting in an early senescence phenotype. Also, NO was shown to be essential for the stability of thylakoid membranes (Liu and Guo 2013). Reportedly, an altered expression of the amino acid transporter gene, *Os*AAP3, results in a high-level accumulation of amino acids (arginine and lysine), which facilitates change in NO signalling pathways leading to the formation of lesions and senescence in rice (Wei et al. 2021).

Polyamines (PAs) are ubiquitous polycationic compounds with multiple developmental and physiological functions. The major PAs include putrescine (Put), spermidine (Spd), spermine (Spm), and thermo-Spm (t-Spm). PAs were shown to inhibit senescence in oat, barley and petunia (Nowicka 2017). This inhibition has been demonstrated to be mediated by the reduced activity of RNase, chlorophyll degradation and LHCII protein degradation. Dark-induced senescence results in a higher accumulation of Put, Spd and Spm at the initial stages, but their level declines during the later stage of senescence (Nowicka 2017). The association of PA accumulation with ROS scavenging capacity at the onset of the senescence process is yet unknown.

Melatonin is one of the primitive biomolecules, ubiquitously found from photosynthetically autotrophic bacteria to mammals and higher plants and has been well characterized as an anti-ageing agent in animals since its discovery. In recent years, melatonin has been linked to delayed leaf senescence (Zhao et al. 2021). The emerging research in melatonin biosynthesis indicates its inhibitory role in leaf senescence. The delayed leaf senescence phenotype obtained upon melatonin application is attributed to the cuticular structure maintenance, balancing of redox homeostasis and prevention of chlorophyll catabolism (Zhao et al. 2021). However, the genetic and molecular basis of melatonin treatment-based delayed leaf senescence is yet to be explored.

Conclusions and future perspectives

The terminal phase of plant development, including leaf senescence or aging, ensures nutrient recycling, plant fitness and reproductive success. This is an enormously intricate, yet highly orchestrated and regulated process. Decades of research encompassing several physiological and biochemical studies, application of genomics and other multi-omics approaches, etc., have answered some of the fundamental questions on the components associated with leaf senescence. These include the contribution of internal and external factors and multi-layered regulation occurring along the progression of senescence. The identified commonalities and interactions among the molecular components and hormones regulating various stress responses and leaf senescence highlight the crosstalk between development and several stress-responsive pathways. However, limited information is available on how plants or their organs coordinate these complex diverse processes together to decide when and how to die. Most of the studies have been carried out at the organ level, which is a culmination of coordinated cell development. Nevertheless, it is pertinent to investigate senescence at the cellular level. Further, the development of new assay systems and monitoring protocols for validating the molecular basis of senescence across different plant systems is also imperative. Few new regulators of senescence have been identified such as melatonin, polyamines

and nitric oxide, however, the genetic and molecular basis of their action needs to be explored. In this regard, systems biology-based approaches would be instrumental in obtaining an accurate and detailed knowledge of the molecular networks associated with the regulation of leaf senescence. All this information would benefit us in designing strategies for fine-tuning senescence in crops species to improve their quality and productivity.

Acknowledgements Research fundings from PURSE (Promotion of University Research and Scientific Excellence) and CRG (Core Research Grant) grants of Department of Science and Technology (DST), Government of India; DU-IoE (Delhi University-Institute of Eminence, IoE/FRP/LS/2020/27), Delhi, India; UGC (University Grants Commission, 41-512/2021(SR)) and DBT (Department of Biotechnology), Government of India are acknowledged.

Declarations

Conflict of interest No competing interest.

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