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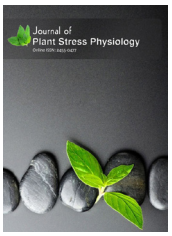
Review Article



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An overview on Azelaic Acid: Biosynthesis, signalling and the action under stress conditions in plants

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ABSTRACT

Plants are exposed to various biotic and abiotic stress factors throughout their lives. For this reason, they have developed some defense mechanisms. They can induce systemic acquired resistance (SAR), which provides long-lasting protection against diverse pathogen attacks. In recent years, several chemical inducers (salicylic acid, glyceraldehyde-3-phosphate, azelaic acid, pipecolic acid, and dehydroabietic acid) have been determined to play roles in this mechanism. The transfer of these signal molecules from infected tissue to non-infected tissues through phloem provides potent defence communication. Azelaic acid is a well-known molecule that triggers salicylic acid accumulation under biotic stress as a priming factor to induce SAR, although little is known about its role under abiotic stress. Here, this review aims to call attention to the effects of AzA under abiotic stress conditions as well as biosynthesis, transport and signalling.

KEYWORDS: Dicarboxylic acid, Oleic acid, Systemic acquired resistance, Oxidative stress

INTRODUCTION

In recent years, stress factors (abiotic and biotic) resulting from a combination of both natural and human-induced causes have negatively affected the growth and development of plants. These environmental factors arise from climatic changes and the deterioration of natural ecosystems in the modern world (Labudda *et al.*, 2022). Biotic stress in plants can be caused by one or multiple pathogens, including fungi, viruses and bacteria that cause crop diseases. Plants have improved a compliant immune system involving proteins, genes, hormones and chemicals that can enable the defence mechanism in local and noninfected regions to cope with the invasion of microbial pathogens (Jones & Takemoto, 2004). Pathogen infection is a type of local defence mechanism; there are two types of immunity PTI (PAMP-triggered immunity) triggered by the molecular model related to the pathogen and immunity ETI (effector-triggered immunity) triggered by the effector (Shah & Zeier, 2013). Furthermore, both PTI (pattern-triggered immunity) and ETI (effector-triggered immunity) can start the production of long-distance signalling molecules for stimulation.

The systemic acquired resistance (SAR) mechanism by developed signal formation in intact plant tissues becomes a pathogen effect and improves a defence mechanism (Winter *et al.*, 2014). It consists of the transport of signal molecules that are formed in leaves during pathogen infection and is related to the SAR mechanism by excitation in distant tissues to other tissues via the phloem (Shah *et al.*, 2014). SAR provides long-term endurance in plants and improves immunity against pathogens such as viruses, bacteria and fungi through its wide spectrum. In addition, ISR (induced systemic resistance) is generally induced in response to root colonization by beneficial bacteria (Van Wees *et al.*, 2008).

Among the structures constituting the signal transduction components of the systemic acquired resistance mechanism, methyl salicylate (MeSA) (Manosalva *et al.*, 2010) and dehydroabietinal (DA), which are called lipid transfer proteins (LTP) (Carella *et al.*, 2015) and provide long-distance signal transmission, are in the first group (Chaturvedi *et al.*, 2012). Salicylic acid plays a pivotal role in SAR. The biosynthesis of SA occurs through chorismic acid in the shikimic acid pathway. After infection, the SA concentration is increased in both infected sites and distal uninfected tissues. In the second group, azelaic acid (AzA) (Jung *et al.*, 2009; Yu *et al.*, 2013), which provides faster and stronger accumulation of salicylic acid (SA) in response to pathogenic infection, glycerol-3-phosphate dependent factor (G3P) (Chanda *et al.*, 2011) and pipecolic acid (Pip) (Návarová *et al.*, 2012; Hartmann *et al.*, 2017), is a product of lysine catabolism. These signalling molecules lead to the systemic expression of antimicrobial PR (pathogenesis-related) genes in uninoculated distal tissue to protect the rest

of the plant from secondary infection. Kinkema *et al.* (2000) described SAR as comprising a broad-spectrum set of PR genes, and the key regulator of signal transduction leading to SAR is NPR1. The NPR1 mutant fails to induce PR genes during systemic resistance, and nuclear localization of NPR1 is essential for the induction of PR genes. In the literature, some important findings have been reported with these molecules (SA, DA, AzA, Pip, G3P) and their interaction in important reviews about the SAR mechanism, but there is no study related only to AzA under stress. This review was focused on emphasizing the lack of information about the role of AzA in protecting plants from abiotic stress damage as well as its other functions in biotic stress.

OVERVIEW OF AZELAIC ACID

In the past, azelaic acid, a dicarboxylic acid with nine carbons, was first discovered as a protector of acne therapy in human health and began to be used in cosmetic and pharmacological areas (Figure 1). Later, it was mentioned that in the plant cycle, these compounds survive under biotic stress. First, Jung *et al.* (2009) found a significant enrichment of AzA in petiole exudates collected from SAR-induced Arabidopsis leaves, and this report was a milestone to join plant life for AzA (Table 1). Currently, this molecule has become remarkable as a priming molecule in the SAR mechanism of plants. Little is known about the biosynthesis of AzA, but it was discovered that it could stimulate the release of free fatty acids from membrane lipids, and these can be used in AzA production under pathogen infection (Figure 2). In recent years, it was reported that dicarboxylic acids prepared from oleo-chemicals have been commercialized as azelaic acid, which is produced by ozonolysis of oleic acid (Ackman *et al.*, 1961). Later, azelaic acid was extensively employed in the synthesis of new generation biodegradable copolymers (Brydson *et al.*, 1999).

It was determined that AzA could accumulate in roots (Mukhtarova *et al.*, 2011). Additionally, Yu *et al.* (2013) showed that the accumulation of unsaturated fatty acids with 18 carbon;

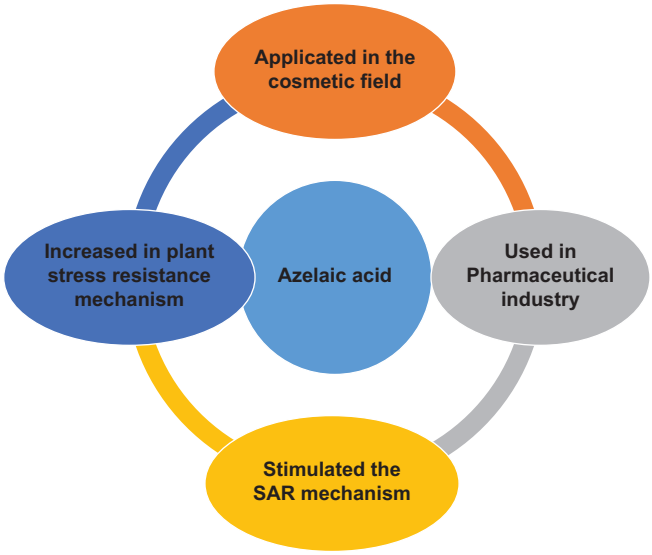

Figure 1: General view of AzA

(18:1) oleic acid, (18:2) linoleic acid (18:3) linolenic acid during pathogen infection is the precursor of AzA (Zoeller *et al.*, 2012). Recently, it was reported that when AzA is exposed to plant roots, it cannot move to leaves but plays a role in inducing systemic disease resistance (Cecchini *et al.*, 2019).

AZA IN SAR MECHANISM

AzA has a stimulant effect on the SAR mechanism via salicylic acid in plants, and it promotes glycerol-3-phosphate (G3P) accumulation and induces SAR. *AZI1* (azelaic acid induced1; putative lipid transfer protein coding gene) was determined, and studies with the *Azi1* mutant showed that it was necessary to induce the SAR mechanism in plants. Microarray studies showed that *AZI1* could be upregulated by cold, wounding, brassinolide/H_3BO_3, zeatin, ethylene, and infection with *Pseudomonas syringae*, *Phytophthora infestans* and nematodes. Additionally, it was determined that *AZI1* was induced in leaves with AzA treatment (Yu *et al.*, 2013). After this, Cecchini *et al.* (2019) documented that the *EARLY ARABIDOPSIS ALUMINIUM-INDUCED GENE1 (EARL1)* gene is the nearest homologue of *AZI1*, which is responsible for AzA, and its movement plays a role in membrane stabilization and localization in the regulation of lipid change/move. AZI and *EARLI1* work together, and they are necessary for AzA priming induction and moving to distant tissues from where Aza is produced.

Inoculation of a virulent pathogen triggers is not dependent on signalling events that lead to the accumulation of SA and NO (nitric oxide). NO triggers the synthesis of reactive oxygen species (ROS), which act in an additive manner to catalyse the oxidation of free C18 unsaturated fatty acids (FAs) that are released from membrane lipids (Yu *et al.*, 2013). NO and ROS operate in a feedback loop. A stress effect releases free FAs from membrane lipids that are hydrolysed by ROS to generate AzA. Plant cell wall lipids digalactosyldiacylglycerol (DGDG) and monogalactosyldiacylglycerol (MGDG) contain C18 FAs that are catalysed by different ROS species (Gao *et al.*, 2014). Oxidation of C18 FAs generates AzA, which triggers biosynthesis of glyceraldehyde-3-phosphate (G3P) via upregulation of genes encoding G3P biosynthetic enzymes. *DIR1* (defective in induced resistance) and *AZI1* operate in a feedback loop and depend on each other for their stability. AzA and G3P are imported via the symplast through plasmodesmata (PD). DIR1 and AZI1, both of which depend on G3P for stability, are needed for G3P-mediated SAR signalling (Figure 3). G3P is either transformed to glycerol by G3P phosphatase or used to synthesize membrane lipids (glycerolipids) and triacylglycerol (TAG). The source of DHA phosphate (DHAP) is glyceraldehyde-3-phosphate originilates, which are derived from glycolysis and the Calvin cycle. Cellular NO levels are regulated via their storage in *S*-nitrosoglutathione (GSNO), which can be reduced to glutathione disulfide (GSSG) and NH3 by *S*-nitrosoglutathione reductase (GSNOR) (Wang *et al.*, 2014). In the SA branch, Enhanced Disease Susceptibility 1 (EDS1) regulates both SA and AzA levels. NPR1 is a key downstream component in the SA branch that is nitrosylated by NO (Gao *et al.*, 2015). NPR1 activation by SA leads to the expression of defence genes that contribute to SAR. Finally, it was

Table 1: Studies from past to present with AzA in years

Findings	References
AzA has been shown to penetrate tumoral cells and acne, abnormal melanocytes, lentigo malign at a higher level than normal cells.	Nazzaro-Porro, 1987
The beneficial effect of AzA on various forms of acne (comedogenic, papulopustular, nodulocystic) has been clearly demonstrated.	Gollnick, 1990
AzA functions were showed as a skin whitening agent by initiating the inhibition of tyrosinase activity.	Lemic-Stojcevic *et al.*, 1995
The main pharmacological effect of AzA in Rosacea is an anti-inflammatory action obtained by reducing reactive oxygen species.	Gollnick & Layto, 2008
AzA is imported critical role in the plant defensive response by serving as a signal molecule to accumulate salicylic acid to defence against infection.	Jung *et al.*, 2009
The effects of AzA on the inflammatory response of normal human keratinocytes to ultraviolet B light, which is a photosensitizer agent in Rosacea was determined.	Mastrofrancesco *et al.*, 2010
AzA application and its overexpression can lead to improved freezing tolerance in Arabidopsis.	Xu *et al.*, 2011
The biosynthese of AzA under the patogen infection was reported.	Zoeller *et al.*, 2012
AzA could be generated by the hydrolyse of fatty acids.	Yu *et al.*, 2013
Preparation of antibacterial TiO_2 particles by hybridization with AzA for applications in cosmetics was reported.	Leong & Oh, 2018
AzA promotes adaption to low temperature in *C. elegans* via shift- ing fatty acid profile to unsaturated long-chain fatty acids.	Bai *et al.*, 2020
AzA was determined naturally in wheat and barley.	Brenna *et al.*, 2020
AzA improved salt stress tolerance in *Lycopersicum esculentum*	Haghighi & Sheibanirad, 2018
AzA induced antioxidant enzymes under pathogen enfection in soybean plants	Rodrigues *et al.*, 2023

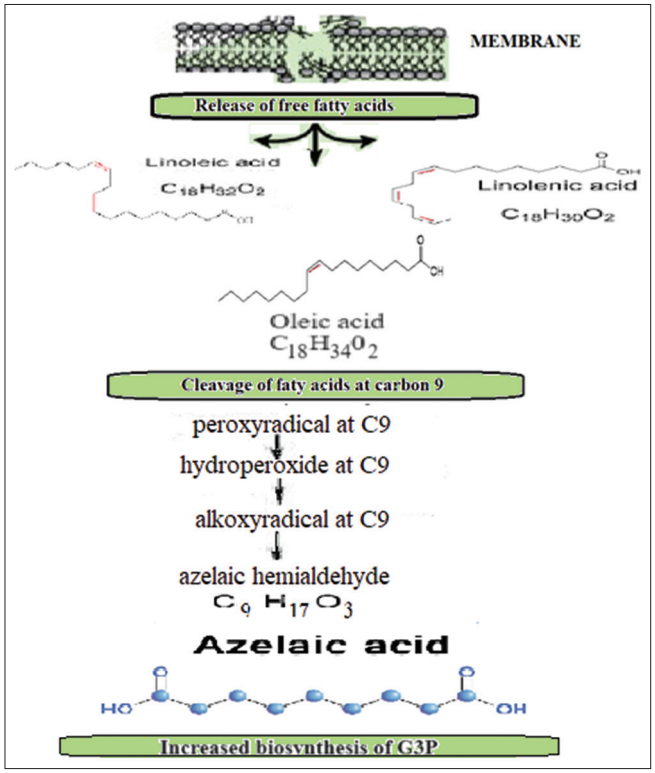

Figure 2: The formation of AzA from oleic acid (modified from Yu *et al.*, 2013)

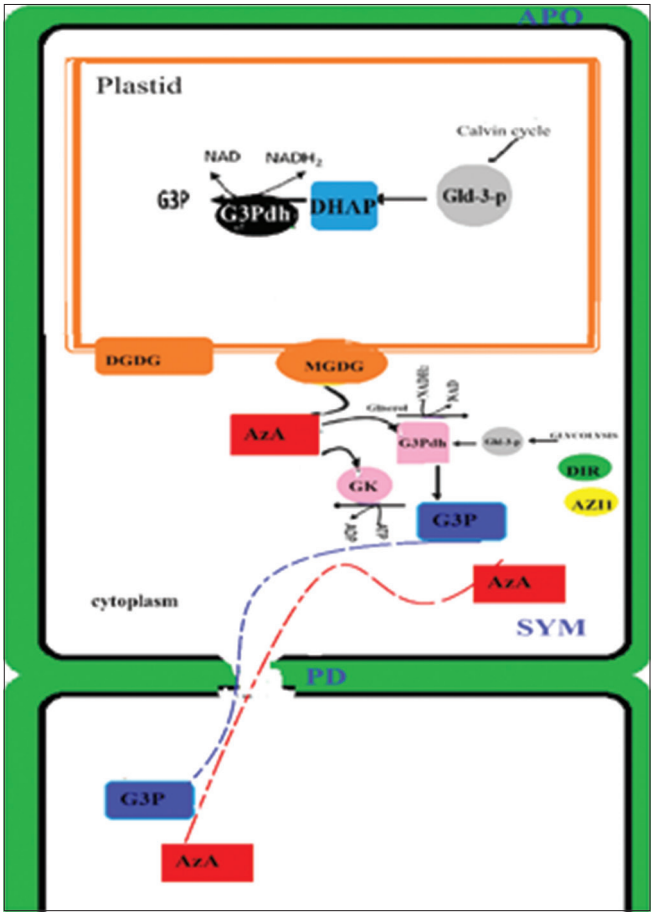

Figure 3: Transport of AzA in plants (modified from Kim & Lim, 2023)

suggested that systemic translocation of AzA is not essential for the establishment of SAR, while when it is translocated, AzA can add to the strength of systemic immunity observed during SAR.

AZA UNDER STRESS CONDITIONS IN PLANTS

Most of the studies are related to the effect of AzA under normal conditions in plants (Jung *et al.*, 2009; Xu *et al.*, 2011; Yu *et al.*, 2013). On the other hand, some biotic stress applications with AzA have been tested in plants (Wubben *et al.*, 2008; Parker, 2009; Chaturvedi *et al.*, 2012; Cecchini *et al.*, 2019), but studies with abiotic stress are limited. Atkinson *et al.* (2013) reported that the overexpression of the *AZI1* gene in Arabidopsis transgenic plants was sensitive to drought stress, but *AZI1* increased chilling

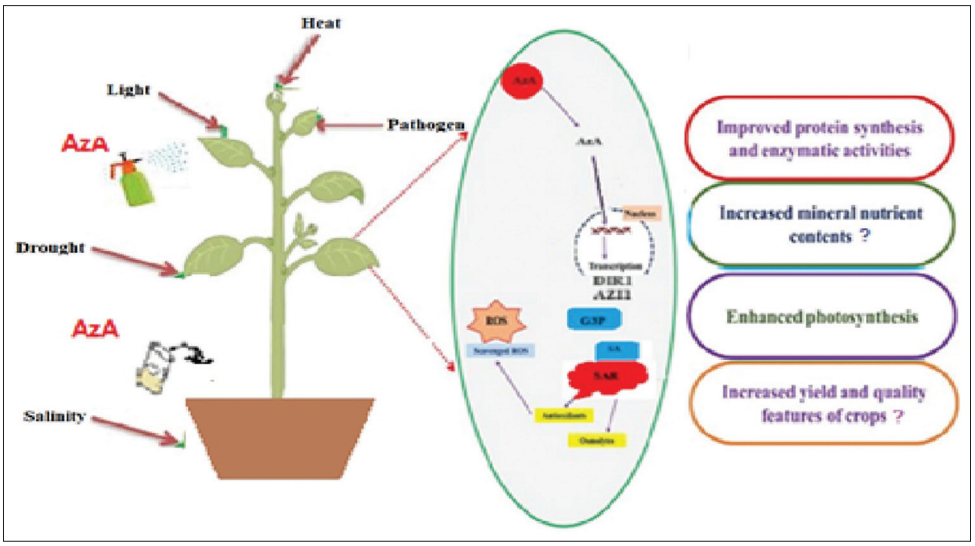

Figure 4: The effects of AzA on plants under stress

and salt tolerance via abscisic acid (ABA) (Xu *et al.*, 2011; Pitzschke *et al.*, 2014). In addition, it was reported that *AZI1* plays a role in the phosphorylation of the mutagenic protein kinaz 3 (MAPK3) (Pitzschke *et al.*, 2014). In recent years, exogenous treatment of AzA was applied to *Lycopersicum esculentum*, and it was determined that AzA improved chlorophyll content, phenolic compounds, stomata and photosynthetic conductance under salinity (Haghighi & Sheibanirad, 2018). Last, a study reported that 1 mM AzA sprayed on soybean plants could enhance some of the antioxidant enzymes under pathogen infection (Rodrigues *et al.*, 2023). In summary, the effects of AzA on protein synthesis, antioxidant enzyme activity, and photosynthesis efficiency in plants have been studied, while osmolyte accumulation, mineral nutrient content, yield and quality, respiratory mechanism and secondary metabolites have not yet been reported (Figure 4).

CONCLUSION

Azelaic acid is an interesting molecule in the plant immune system. Previous studies showed that AzA plays a role as a chemical inducer in the SAR mechanism and protects plants from biotic stress. Although most papers are related to the action of AzA under biotic stress (pathogen attack), few of them are connected with abiotic stress (drought, salt and chilling). Currently, more detailed research should be performed on AzA, especially its movement under abiotic stress to increase plant stress resistance against extreme environmental conditions.

AUTHOR CONTRIBUTION

BSD collected literature, compiled information and drafted manuscript. HC surveyed literature and revised the Tables and Figures. All authors read and approved the final manuscript.

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