# RELATIONSHIP BETWEEN JASMONATES AND ETHYLENE IN REGULATION OF SOME PHYSIOLOGICAL PROCESSES IN PLANTS UNDER STRESS CONDITIONS

### Marian Saniewski<sup>1</sup>, Junichi Ueda<sup>2</sup>, Kensuke Miyamoto<sup>2</sup>, Henryk Urbanek<sup>3</sup>

- <sup>1</sup> Research Institute of Pomology and Floriculture, Skierniewice, Poland
- <sup>2</sup> College of Integrated Arts and Sciences, Osaka Prefecture University, Sakai, Osaka, Japan
- <sup>3</sup> Department of Plant Physiology and Biochemistry, University of Łódź, Łódź, Poland

### Introduction

Jasmonic acid (JA), methyl jasmonate (JA-Me) and their related compounds which are designated as jasmonates, are widely distributed in the plant kingdom and show various important biological activities in the regulation of plant growth and development, resulting in a consideration that they are putative new plant hormones [UEDA, KATO 1980; KODA 1992; SEMBDNER, PARTHIER 1993; CREELMAN, MULLET 1995, 1997; MUROFUSHI et al. 1999; SANIEWSKI, CZAPSKI 1999]. Endogenous levels of jasmonates, mainly JA, increase rapidly and transiently in plants or their organs under both abiotic stress conditions, such as mechanical wounding, osmotic stress, water deficit, dessication stress, heavy metals (Cu++, Cd++, Ag+) and touching, as well as biotic stress ones, such as pathogen infection and an insect attack, and by different natural elicitors-systemin, oligogalacturonide fragments of pectic polysaccharides (i.e. cell wall of yeast or pathogens), chitosan and chitin [CREELMAN, MULLET 1995; SANIEWSKI 1997]. Jasmonates also consist of an integral part of the signal transduction chain between stress signal(s) and stress response(s). Cooperative cross-talk among jasmonates and various hormonal signals, especially ethylene occurs in regulation of growth and development and in defense responses against a wide variety of abiotic and biotic agents. In this paper, we focused on and reviewed interactions of jasmonates with ethylene under stress conditions.

#### Possible role of jasmonates in ethylene biosynthesis in intact plants

Jasmonates have been reported to control ethylene biosynthesis in intact plants and their organs by stimulating activities of ACC synthase and ACC oxidase [SANIEWSKI 1995, 1997; SANIEWSKI et al. 1995]. Exogenously applied JA-Me stimulates ethylene production in tomatoes at different stages of ripening and in preclimacteric apples. FAN et al. [1998] suggested that jasmonates together with ethylene are involved in regulating ripening at an early growth stage of climacteric fruits such as tomatoes and apples.

Treatment of JA-Me at the concentration of 1 mM to broccoli (*Brassica* oleracea L. cv. Italica) florets significantly promoted ethylene production and ACC oxidase activity during senescence. These results suggest that jasmonates are substantially associated with promoting senescence by enhancing ethylenc production [WATANABE et al. 2000]. GARCIA-PINEDA and LOZOYA-GLORIA [1999] showed that JA-Me induced gene expression of 1-aminocyclopropanc-1-carboxylic acid (ACC) oxidase in pepper (*Capsicum annuum*).

On the other hand, we have recently suggested that ethylene exogenously applied affects endogenous levels of jasmonates and/or their sensitivities in plant tissues [SANIEWSKI et al. 1999]. As shown in Fig. 1, similar considerations have been reported in relation to gene expressions after pathogen infection. We would like to described the details in what follows.

### Jasmonates and ethylene biosynthesis, as well as reactive oxygen species (ROS) generation under stress conditions

Plants are always exposed to biotic and abiotic environmental stresses that influence their growth and productivity.

Mechanical wounding and other abiotic and biotic stresses (pathogen infection and insect invasion) are well known to be common factors inducing biosynthesis of ethylene and jasmonates in plants. It is still open question what is a role of JA in controlling ethylene biosynthesis in mechanically wounded tissues, and plants infected and attacked by pathogens and insects, respectively [SANIEWSKI 1997].

Adaptation to abiotic and biotic stresses is crucial for growth and survival of plants. Reactive oxygen species (ROS) generation, including singlet oxygen, superoxide anion radical  $(O_2^{-})$ , hydrogen peroxide and hydroxyl radical, is usually a rapid plant response to pathogen infection and/or herbivore attack and to other abiotic and environmental stresses as well. Recent evidence indicates two very distinct roles for ROS during pathogen attacks and abiotic stresses: they can either serve as destructive molecules or as defensive factors [KuźNIAK, URBANEK 2000; DAT et al. 2000; OROZCO-CÁRDENAS et al. 2001].

ROS are unavoidable, transient products of normal cellular metabolisms. Under normal environmental conditions they are rapidly metabolized. However, when plants are subjected to environmental stresses, excess ROS are generated. Unless these ROS are efficiently metabolized, they oxidize and damage membrane lipids, proteins and nucleic acids. That leads to cellular disfunction and can ultimately cause cell death. So, their generation is frequently considered deleterious and harmful. To protect themselves against toxic ROS, plants employ the antioxidant enzymes and metabolites that catalyze scavenging ROS, in which superoxide dismutase, catalase, ascorbate peroxidase, glutathione peroxidase, ascorbate, glutathione and tocopherols are involved.

On the other hand, recent studies have demonstrated that ROS may be implicated in plant defense systems. Evidence for defensive roles of ROS comes from the observation: 1) Different environmental stress factors such as herbi-

vores, pathogens, ozone, cold, high light intensity, mechanical wounding, salinity, drought and UV radiation stimulate the production of ROS; 2) Several factors stimulating oxidative burst and ROS generation induce gene expressions of the same defense proteins such as glutathione transferase, glutathione peroxidase, ascorbate peroxidase and some PR-proteins induced by  $O_2$ <sup>••</sup> or  $H_2O_2$  treatment alone as well. As defensive factors, they can directly inhibit pathogen growth, limit the spread of pathogen infection by strengthening plant cell wall, activate defense genes acting as signaling molecules and be involved in hypersensitive response (HR) [HEATH 2000]. The HR, a well-known resistance mechanism, is characterized by rapid, localized cell death around the site of infection and so it can play efficacious role in limiting the spread of pathogen. HR also has been suggested to release signals that activate variety of defense-related genes and systemic resistance. Some data suggest that HR may function more by releasing signaling molecules involved in the induction of defense genes than as a direct defense mechanism. KENTON et al. [1999] documented the accumulation of JA in hypersensitive response tissues of tobacco plants. ROS have been implicated as a part of the signal systems responsible for inducing the responses of plants to both biotic and abiotic stresses. Especially H<sub>2</sub>O<sub>2</sub> and O<sub>2</sub><sup>--</sup> have been suggested to be diffusible signals that activate genes in local and systemic acquired resistance.

The defensive role of ROS requires their cellular concentration to be carefully controlled. Preventing the high, lethal concentration of ROS generated in response to stress is a promoting way to counteract their damaging action. To avoid deleterious accumulation of ROS, they are kept under control by various antioxidant enzymes and metabolites.

Evidence coming from various observations shows that JA and JA-Me are believed to play an important role in influencing plant resistance to biotic and abiotic stresses [REYMOND, FARMER 1998]. Environmental stresses increase endogenous levels of JA, and exogenous application of JA stimulates the expression of defensive responses. A burst of ROS generation may be involved in membrane depolarization which activates octadecanoid pathway, leading to JA biosynthesis. Generated or applied JA further enhances ROS accumulation in various plant species. There is evidence that  $H_2O_2$  production in wound responses requires a functional JA-mediated signaling pathway.

Under various stresses jasmonates as signaling molecules have been reported to induce genes encoding antimicrobial secondary metabolites – phytoalexin, terpenoids, phenylpropanoids, alkaloids; antimicrobial proteins – thionins, defensins, osmotin; proteinase inhibitors; antioxidant enzymes – glutathione transferase, ascorbate peroxidase, chalcone synthase, phenylalanine ammonia lyase and other PR-proteins [RAKWAL et al. 1999; OROZCO-CÁRDENAS et al. 2001].

Jasmonates have also been implicated as intermediate signals in elicitorinduced secondary metabolite accumulation and other defense reactions against pathogen infections and insect attacks. Exogenously applied JA enhances elicitorinduced accumulation of proteins related to pathogenesis [MEMELINK et al. 2001]. Colonization of the roots of several plant species by selected strains of nonpathogenic *Pseudomonas fluorescens*, similarly to elicitors treatment, has been demonstrated to induce resistance in uninfected plant parts towards a broad spectrum of pathogens [VAN WEES et al. 2000]. Systemic resistance induced by this kind of colonization requires JA and is based on the enhance of sensitivity to JA rather than on an increase of its production.

In studies of the responses of higher plants to wounding, the relationships among gene expression of wound-inducible ACC synthase, the effects of JA and the roles of ROS have not been clarified yet.

Gene expression of wound-inducible ACC synthase (*CM-ACS1*) in *Cucurbita maxima* induced by wounding and the accumulation of the *CM-ACS1* transcript enhanced by wounding have been shown. Initially these were induced by ROS generating in disks upon wounding. Cooperative mechanisms by ROS and JA were then introduced, in which the level of JA increased at a later time after wounding [WATANABE, SAKAI 1998; WATANABE et al. 2001a].

Recently, WATANABE et al. [2001b] isolated further two new cDNA fragments (*CM-ACS3* and *CM-ACS4*) that encode ACC synthases from mesocarp tissue of *Cucurbita maxima* fruit. The accumulation of specific transcripts for *CM-ACS1*, *CM-ACS3* and *CM-ACS4* in tissue disks of *C. maxima* was induced by mechanical wounding. They also demonstrated that the expression of the three ACC synthase genes in wounded mesocarp tissue of *C. maxima* are differentially regulated by ethylene and JA, and ROS stimulates the accumulation of all three transcripts (Table 1).

Table 1; Tabela 1

The effect of jasmonic acid (JA), ethylene and an inhibitor of the generation of ROS, on the expression of three genes for wound-inducible 1-aminocyclopropane-1-carboxylate (ACC) synthase in winter squash (*Cucurbita maxima*) (Table prepared on the basis of data of WATANABE et al. [2001b])

Wpływ kwasu jasmonowego (JA), etylenu i inhibitora wytwarzania wolnych rodników (ROS), na ekspresję trzech genów syntazy kwasu 1-aminocyklopropano-1-karboksylowego, indukowanych po uszkodzeniu *Cucurbita maxima* (tabela opracowana na podstawie danych z pracy WATANABE i in. [2001b])

Gene encoded ACC synthase Geny kodujące syntazę ACC	Gene expression; Ekspresja genów			
	wounding uszkodzenie	wounding +JA uszkodzenie + JA	wounding + ethylene uszkodzenie + etylen	wounding + DPI* uszkodzenie + DPI
CM-ACS1	induction	stimulation	inhibition	inhibition
	indukcja	stymulacja	hamowanie	hamowanie
СМ-АСЅЗ	induction	without effect	stimulation	inhibition
	indukcja	bez wpływu	stymulacja	hamowanie
CM-ACS4	induction	inhibition	without effect	inhibition
	indukcja	hamowanie	bez wpływu	hamowanie

DPI, diphenylene iodonium – an inhibitor of the generation of ROS by NAD(P)H oxidase; inhibitor wytwarzania wolnych rodników (ROS) przez oksydazę NAD(P)H

On the other hand, the important role of ROS and JA as mediators in early responses to wounding or attack by pathogens has also been reviewed by LOW and MERIDA [1996], WASTERNACK and PARTHIER [1997] and KUŹNIAK and URBANEK [2000].

Among various air pollutants, the tropospheric ozone causes the greatest damage to both natural and cultivated plants [RAO et al. 2000; SCHRAUDNER et al.

1997]. Damage symptoms in plants range from inhibition of photosynthesis and associated growth inhibition, and yield loss to premature senescence and tissue necrosis. Ozone enters the leaves via stomata, diffuses through intercellular air spaces, and in apoplast it may react with water or some cell wall constituents and is immediately converted to ROS. Then  $O_3$  or ROS alters the physicochemical properties of plasmalemma by its depolarization and initiate lipid peroxidation. The plasmalemma depolarization activates octadecanoid pathways leading to jasmonate biosynthesis. These signaling molecules, in turn, enhance production of ROS, induce cell death and influence expression of defense genes. The phytotoxicity of  $O_3$  is likely a result of deleterious actions associated with ROS. Active ROS production during  $O_3$  exposure has been detected [RAO et al. 2000]. Plant antioxidant systems are thought to be potential factors in ozone tolerance. Correlations between higher activities of antixidant enzymes and tolerance to ozone were established.

It has been demonstrated that ozone exposure activates HR-like cell death pathways in plants. A potential role of JA as a central molecule modulating stress responses is shown by the observation that its application prior to  $O_3$  exposure reduces  $O_3$ -induced concentration of  $H_2O_2$  and abolishes  $O_3$ -induced cell death in tobacco [ORVAR et al. 1997], poplar [OVERMYER et al. 2000] and high  $O_3$ sensitive *Arabidopsis* ecotype [RAO et al. 2000]. Additional evidence implicating the role of JA in modulating the magnitude of  $O_3$  induced reactions is connected to harpin, a proteinaccous elicitor of defense gene expression, isolated from different pathogenic bacteria. It has been reported that the JA-Me pretreatment inhibits the harpin induced defense responses such as  $H_2O_2$  generation, HR and gene expression encoding phenylalanine ammonia lyase in tobacco cell suspension cultures [ANDI et al. 2001]. It is possible that jasmonates as key signal molecules controlling defense responses moderate the range of plant reactions to stresses depending on physiological situation of plant tissues exposed to detrimental factors.

A recent survey of various plant species indicated a relationship between  $O_3$  sensitivity and  $O_3$ -induced stress ethylene production.  $O_3$ -induced ethylene biosynthesis is believed to induce premature senescence and/or react with ethylene to form damaging ROS [RAO et al. 2000]. It is well known that inhibition of ethylene biosynthesis reduced  $O_3$ -induced damage in tomato [TUOMAINEN et al. 1997] and treatment of plants with norbornadiene, an inhibitor of ethylene perception, blocked  $O_3$ -induced cell death [BAE et al. 1996]. Preliminary results indicate that ethylene is a major regulator of  $O_3$ -induced defense responses and cell death and that ethylene signaling pathways interact with both salicylic acid and JA-dependent signaling pathways [RAO et al. 2000].

### Interaction of jasmonates with ethylene in regulation of some physiological processes in plants under stress conditions

#### a) Synergistic interaction

Jasmonates have been well known to induce strongly gene expressions of wound- or defence-related ones when plants have some stresses from mechanical injury and pathogen infection or insect invasion, resulting in the accumulation of proteinase inhibitors (PI) and other pathogenesis-related proteins [O`DONNELL et al. 1996; KOIWA et al. 1977; SEO et al. 1997]. The interaction of jasmonates with ethylene has been reported, in which these compounds acted together to regulate PI gene expression during wound response. Gene expressions of these proteins were inhibited by inhibitors of JA and ethylene biosynthesis [O`DONNELL et al. 1996]. Wounding has also been reported to be responsible for the regulation of a wide variety of other genes: cathepsin D inhibitor, chloramphenicol acetyltransferase, vegetative storage protein, lipoxygenase, chalcone synthase and hydroperoxide lyase. Jasmonates have been reported to increase an accumulation of mRNA encoding these enzymes [DE WALD et al. 1994; ROJO et al. 1998].

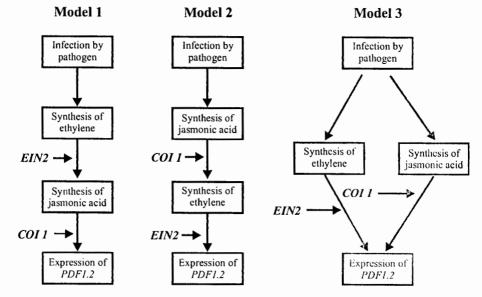
Combinations of jasmonates and ethylene caused synergistic induction of pathogenesis-related gene expression osmotin mRNA in tobacco seedlings [XU et al. 1994]. It remains to be demonstrated whether ethylene acts upstream or downstream in concert with jasmonates.

There was documented synergy between jasmonates and ethylene for the induction of the plant defense gene of *PDF1.2* in *Arabidopsis thaliana* infected by *Alternaria brassicicola* [PENNINCKX et al. 1998]. Conceptually, three different models for the interaction between JA and ethylene arc suggested by PENNINCKX et al. [1998] (Scheme 1).

Scheme 1; Schemat 1

Three models for the interaction between ethylene and jasmonate signals during activation of the *PDF1.2* gene in pathogen-challenged *Arabidopsis* plants [PENNINCKX et al. 1998]

Trzy modele interakcji między etylenem i kwasem jasmonowym w czasie aktywacji genu *PDF1.2* u *Arabidopsis* w wyniku infekcji przez patogena [PENNINCKX i in. 1998]



EIN2 – a component of the ethylene signal transduction pathway; czynnik transdukcji sygnału w szlaku etylenowym

COII – a component of the jasmonate signal transduction pathway; czynnik transdukcji sygnału w szlaku kwasu jasmonowego

Model 1 implies that pathogen infection initially stimulates production of ethylene, which subsequently stimulates production of jasmonates, which in turn activates *PDF1.2*. Model 2 implies that pathogen infection initially leads to enhanced production of jasmonates, which subsequently triggers elevated production of ethylene, which in turn controls *PDF1.2* expression. Model 3 predicts that pathogen infection results in simultaneous production of ethylene and jasmonates, which are both required for induction of *PDF1.2*.

As described above, one of the most rapid events observed during plantpathogen interactions is the production of ROS, which are initially in the form of superoxide anion, by NADPH oxidase complex [MEHDY 1994; LAMB, DIXON 1997]. Activation of *PDF1.2* by superoxide anion-generating paraquat (subherbicidal level) has been shown to require functional ethylene and jasmonates response pathways [PENNINCKX et al. 1998].

Gum induction is the first example for an important interaction of jasmonates and ethylene under stress conditions. Gums are a secretion mainly consisting of polysaccharides exuded onto surface of the fruits or the shoots when some plants have several kind of stresses including physical injury, insect attack and pathogen infection. Gum formation in tulip bulbs or stone fruit trees is induced by ethylene and ethylene-releasing compound, ethephon. It has been shown that jasmonates have a promoting effect on the induction and/or production of gums in stone fruit trees and tulips. It has been found that JA-Me causes a strong induction of gum formation in the bulb, stem and basal part of the leaf in tulips. It should be mentioned that ethylene did not induce gums in stem and leaves of tulips. It has been shown, however, that the simultaneous application of ACC (1-aminocyclopropane-1-carboxylic acid) with JA-Me greatly accelerates gum formation in bulbs, stems and leaves of tulip in comparison with JA-Me treatment alone [SANIEWSKI et al. 1999; SANIEWSKI et al. 2000].

Disease resistance is associated with a plant defense response that involves an integrated set of signal transduction pathways. SCHENK et al. [2000] examined changes in the expression patterns of 2375 selected genes by cDNA microarray analysis in Arabidopsis thaliana after inoculation with incompatible fungal pathogen Alternaria brassicicola or treatment with defense-related signaling molecules: JA-Me, ethylene and salicylic acid (SA). In leaves inoculated with A. brassicicola, the transcript levels of 168 genes were increased whereas those of 39 genes were decreased. Similarly, the transcript abundance of 221 genes for JA-Me, 55 genes for ethylene, and 192 for SA was increased as a result of treatment with these signal molecules. In contrast, transcript abundance of 96, 16 and 131 genes was reduced after treatment with JA-Me, ethylene and SA, respectively. Of 168 genes that were significantly induced by fungal inoculation, 33, 4 and 21 genes (50 genes in total) were also induced by JA-Me, ethylene and salicylic acid, respectively. JA-Me and ethylene jointly regulate transcription of 36 of 2375 genes tested. In addition, 50% gencs induced by ethylene treatment were also induced by JA-Me treatment. These results indicated the existence of a substantial network of regulatory interactions and coordination occurring during plant defense among the different defense signaling pathways [SCHENK et al. 2000].

Synergistic interaction between jasmonates and ethylene was documented in induction of basic chitinase [NORMAN-SETTERBLAD et al. 2000].

Jasmonates are required, alone or in combination with ethylene, for defense against insects and necrotrophic pathogens [McConn et al. 1997; VIJAYAN

et al. 1998; Тномма et al. 2000].

Interaction of JA-Me and ethylene and binding models for elicited biosynthetic steps of paclitaxel in suspension cultures of *Taxus cuspidata* and *T. Canadensis* were elaborated by MIRJALILI and LINDEN [1996], PHISALAPHONG and LINDEN [1999].

HUNG and KAO [1996] suggest that jasmonate-promoted senescence is mediated through an increase in ethylene sensitivity in detached maize leaves.

## b) Ethylene suppresses processes induced by jasmonates

There are many reports that ethylene substantially suppresses some physiological processes induced by jasmonates. In Nicotiana species, jasmonates act as a signal molecule for the herbivore- and wound-induced accumulation of nicotine, an important defense compound in these plants [BALDWIN 1999]. It was documented that ethylene suppresses jasmonate-induced gene expression in nicotine biosynthesis [IMANISHI et al. 1998; SHOJI et al. 2000]. It is significant that feeding experiment using hawkmoth (Manduca sexta) results in a rise in ethylene biosynthesis that reduces JA-induced nicotine biosynthesis in Nicotiana attenuata, thus diminishing plant defenses [KAHL et al. 2000]. In ethylene-insensitive mutants, gene expression of the JA-responsive vegetative storage proteins (VSP) is also reported to increase, suggesting that the signal pathway represses the induction of VSP [Rojo et al. 1999; NORMAN-SETTERBLAND et al. 2000]. Antagonistic interactions between JA and ethylene are also reported to regulate the antifeedant plant lectin GS-II in locally wounded leaves of Griffonia simplicifolia [ZIIU-SALZ-MAN et al. 1998]. When Arabidopsis leaves were damaged, several jasmonateinducible genes were repressed in locally wounded tissue through the production and perception of ethylene [ROJO et al. 1999].

## c) Jasmonates suppresses processes induced by ethylene

Contrary to this it has been shown that jasmonates substantially suppress the formation of apical hook induced by ethylene in *Arabidopsis thaliana* [ELLIS, TURNER 2001].

## JA carboxyl methyltransferase – a key enzyme for jasmonate-regulated plant responses

SEO et al. [2001] suggested that the S-adenosyl-L-methionine: JA carboxyl methyltransferase (JMT) is a kcy enzyme for jasmonate-regulated plant responses. Activation of JMT expression leads to production of JA-Me. JMT was expressed differentially in different organs at particular developmental stages and induced by wounding, and was activated when *Arabidopsis* tissues were treated with exogenous JA-Me. Thus, JA-Me can amplify JMT expression induced at the developmental stages and by external stimuli including wounding. JA-Me can act as an intracellular regulator, a diffusible intercellular signal transducer, or an airborne signal mediating intra- and interplant communications. Some signals generated during an early event of developmental processes or defense responses may activate JMT that can self-amplify, stimulate, or regulate its own expression,

propagating the JA-Me-mediated cellular responses throughout whole plants [SEO et al. 2001].

It is interesting that ethylene inhibited JMT expression [SEO et al. 2001]. The result suggests that the synergistic effect exerted by ethylene on the activation of *PDF1.2* is not by the enhancement of JA-Me synthesis but rather by a cross-talk between the independent signaling pathways derived from them.

### References

ANDI S., TAGUCHI F., TOYODA K., SHIRAISHI T., ICHINOSE Y. 2001. Effect of methyl jasmonate on harpin-induced hypersensitive cell death, generation of hydrogen peroxide and expression of PAL mRNA in tobacco suspension cultured BY-2 cells. Plant Cell Physiol. 42: 446–449.

**BAE G.Y., NAKAJIMA N., ISHIZUKA K., KONDO N. 1996.** The role in ozone phytotoxicity of the evolution of ethylene upon induction of *l*-aminocyclopropane-*l*-carboxylic acid synthase by ozone fumigation in tomato plants. Plant Cell Physiol. 37: 129–134.

**BALDWIN I.T. 1999.** Inducible nicotine production in native Nicotiana as an example of adaptive phenotypic plasticity. J. Chem. Ecol. 25: 3–30.

**CREELMAN R.A., MULLET J.E. 1995.** Jasmonic acid distribution and action in plants: Regulation during development and response to biotic and abiotic stress. Proc. Natl. Acad. Sci. USA 92: 4114–4119.

CREELMAN R.A., MULLET J.E. 1997. Biosynthesis and action of jasmonates in plants. Annu. Rev. Plant Physiol. Plant Mol. Biol. 48: 355–381.

DAT J., VANDENABEELE S., VRANOVÁ E., VAN MONTAGU M., INZÉ D., VAN BREUSEGEM F. 2000. Dual action of the active oxygen species during plant stress responses. Cell Mol. Life Sci. 57: 779-795.

DE WALD D.B., SADKA. A., MULLET J.E. 1994. Sucrose modulation of soybean VSP gene expression is inhibited by auxin. Plant Physiol. 104: 439–444.

ELLIS C., TURNER J.G. 2001. The Arabidopsis mutant cev1 has constitutively active jasmonate and ethylene signal pathways and enhanced resistance to pathogens. Plant Cell 13: 1025–1033.

FAN X., MATTHEIS J.P., FELLMAN J.K. 1998. A role for jasmonates in climacteric fruit ripening. Planta 204: 444–449.

GARCIA-PINEDA E., LOZOYA-GLORIA E. 1999. Induced gene expression of 1-aminocyclopropane-l-carboxylic acid (ACC oxidase) in pepper (Capsicum annuum L.) by arachidonic acid. Plant Science 145: 11–21.

HEATH M.C. 2000. Hypersensitive response-related death. Plant Mol. Biol. 44: 321–334.

HUNG K.T., KAO C.H. 1996. Promotive effect of jasmonates on the senescence of detached maize leaves. Plant Growth Regul. 19: 77-83.

IMANISHI S., HASHIZUME K., NAKAKITA M., KOJIMA H., MATSUBAYASHI Y., HASHIMOTO T., SAKAGAMI Y., YAMADA Y., NAKAMURA K. 1998. Differential induction by methyl jasmonate of genes encoding ornithine decarboxylase and other enzymes involved in nicotine biosynthesis in tobacco cell cultures. Plant Mol. Biol. 38: 1101–1111.

KAHL J., SIEMENS D.H., AERTS R.J., GABLER R., KUHNEMANN F., PRESTON C.A., BALDWIN I.T. 2000. Herbivore-induced ethylene suppresses a direct defense but not a putative indirect defense against an adapted herbivore. Planta 210: 336-342.

KENTON P., MUR L.A.J., ATZORN R., WASTERNACK C., DRAPER J. 1999. (-)-Jasmonic acid accumulation in tobacco hypersensitive response lesions. Mol. Plant-Microbe Interac. 12: 74-78.

KODA Y. 1992. The role of jasmonic acid and related compounds in the regulation of plant development. Inter. Rev. Cytol. 135: 155–198.

KOIWA H, BRESSAN R.A., HASEGAWA P.M. 1997. Regulation of protease inhibitors and plant defence. Trends in Plant Science 2: 379–383.

KUŹNIAK E., URBANEK H. 2000. The involvement of hydrogen peroxide in plant responses to stresses. Acta Physiol. Plant. 22: 195–203.

LAMB C., DIXON R.A. 1997. The oxidative burst in plant disease resistance. Ann. Rev. Plant Physiol. Plant Mol. Biol. 48: 251–275.

Low P.S., MERIDA J.R. 1996. The oxidative burst in plant defense function and signal transduction. Plant Mol. Biol. 21: 985–992.

MCCONN M., CREELMAN R.A., BELL E., MULLET J.E., BROWSE J. 1997. Jasmonate is essential for insect defense. Proc. Natl. Acad. Sci. USA 94: 5473-5477.

**MEHDY M.C. 1994.** Active oxygen species in plant defense against pathogens. Plant Physiol. 105: 467–472.

MEMELINK J., VERPOORTE R., KIJNE J.W. 2001. ORCAnization of jasmonate-responsive gene expression in alkaloid metabolism. Trends in Plant Science 6: 212–219.

MIRJALILI N., LINDEN J.C. 1996. Methyl jasmonate induced production of taxol in suspension cultures of Taxus cuspidata: ethylene interaction and induction models. Biotechnol. Prog. 12: 110–118.

MUROFUSHI N., YAMANE H., SAKAGAMI Y., IMASEKI H., KAMIYA Y., IWAMURA H., HIRAI N., TSUJI H., YOKOTA T., UEDA J. 1999. *Plant Hormones*, in: *Comprehensive Natural Products Chemistry*. Editor-in-Chief: Sir Derek Barton, Koji Nakanishi, Executive Editor: Otto Meth-Cohn. Vol 8, Miscellaneous Natural Products including Marine, Natural Products, Pheromones, Plant Hormones, and Aspects of Ecology (Volume Editor: Kenji Mori), Elsevier, Amsterdam: 19–136.

NORMAN-SETTERBLAD C., VIDAL S., PALVA E.T. 2000. Interacting signal pathways control defense gene expression in Arabidopsis in response to cell wall-degrading enzymes from Erwinia carotovora. Mol. Plant-Microbe Interac. 4: 430–438.

O'DONNELL P.J., CALVERT C., ATZORN R., WASTERNACK C., LEYSER H.M.O., BOWLES D.J. 1996. Ethylene as a signal mediating the wound response of tomato plants. Science 274: 1914–1917.

OROZCO-CÁRDENAS M.L., NARVÁEZ-VÁSQUEZ J., RYAN C.A. 2001. Hydrogen peroxide acts as a second messenger for the induction of defense genes in tomato plants in response to wounding, systemin, and methyl jasmonate. Plant Cell 13: 179–191.

**ORVAR B.L., McPHERSON J., ELLIS B.E. 1997.** Pre-activation wounding response in tobacco prior to high-level ozone exposure prevents necrotic injury. Plant J. 11: 203–212.

OVERMYER K., TUOMINEN H., KETTUNEN R., BETZ C., LANGEBARTELS C., SANDERMANN H., KANGASJÄRVI J. 2000. Ozone-sensitive Arabidopsis rcd1 mutant reveals opposite

roles for ethylene and jasmonate signaling pathways in regulating superoxide-dependent cell death. Plant Cell 12: 1849–1862.

**PENNINCKX I.A.M.A., THOMMA B.P.H.J., BUCHALA A., METRAUX J.-P., BROEKAERT W.F. 1998.** Concomitant activation of jasmonate and ethylene response pathways is required for induction of a plant defensin gene in Arabidopsis. Plant Cell 10: 2103–2113.

**PHISALAPHONG M., LINDEN J.C. 1999.** Ethylene and methyl jasmonate interaction and binding models for elicited biosynthetic steps of paclitaxel in suspension cultures of Taxus canadensis, in: Biology and Biotechnology of the Plant Hormone Ethylene II. A.K. Kanellis, C. Chang, H. Klee, A.B. Bleecker.J.C. Pech, D. Grierson (eds.), Kluwer Academic Publishers, Dordrecht, Boston: 85–94.

**RAKWAL R., AGRAWAL G.K., YONEKURA M. 1999.** Separation of proteins from stressed rice (Oryza sativa L.) leaf tissues by two-dimensional polyacrylamide gel electrophoresis: Induction of pathogenesis-related and cellular protectant proteins by jasmonic acid, UV irradiation and copper chloride. Electrophoresis 20: 3472–3478.

RAO M.V., KOCH J.R., DAVIS K.R. 2000. Ozone: a tool for probing programmed cell death in plants. Plant Mol. Biol. 44: 345–358.

**REYMOND P., FARMER E.E. 1998.** Jasmonate and salicylate as global signals for defense gene expression. Current Opinion in Plant Biology 1: 404–411.

**ROJO E., LEON J., SANCHEZ-SERRANO J.J. 1999.** Cross-talk between wound signalling pathways determines local versus systemic gene expression in Arabidopsis thaliana. Plant J. 20: 135–142.

ROJO E., TITARENKO E., LEON J., BERGER S., VANCANNEYT G., SANCHEZ-SERRANO J.J. 1998. Reversible protein phosphorylation regulates jasmonic acid dependent and independent wound signal transduction pathways in Arabidopsis thaliana. Plant J. 12: 153–165.

SANIEWSKI M. 1995. Methyl jasmonate in relation to ethylene production and other physiological processes in selected horticultural crops. Acta Hortic. 394: 85–98.

SANIEWSKI M. 1997. The role of jasmonates in ethylene biosynthesis, in: Biology and Biotechnology of the Plant Hormone Ethylene. A.K. Kanellis, C. Chang, H. Kende, D. Grierson (eds.), Kluwer Academic Publishers, Dordrecht, Boston, London: 39-45.

SANIEWSKI M., CZAPSKI J. 1999. Jasmoniany i ich funkcja allelopatyczna. Postępy Nauk Rolniczych I: 3–18.

SANIEWSKI M., LANGE E., CZAPSKI J. 1995. Rola estru metylowego kwasu jasmonowego – nowego hormonu roślinnego w biosyntezie etylenu. Postępy Nauk Rolniczych 3: 3–17.

SANIEWSKI M., UEDA J., MIYAMOTO K. 1999. Interaction of ethylene with jasmonates in the regulation of some physiological processes in plants, in: Biology and Biotechnology of the Plant Hormone Ethylene II. A.K. Kanellis, C. Chang, H. Klee, A.B. Bleecker, J.C. Pech, D. Grierson (eds.), Kluwer Academic Publishers, Dordrecht, Boston: 173–180.

SANIEWSKI M., UEDA J., MIYAMOTO K., HORBOWICZ M., PUCHALSKI J. 2000. Methyl jasmonate induces gummosis in plants. Human and Environmental Sciences 9: 93–100.

SCHENK P.M., KAZAN K., WILSON I., ANDERSON J.P., RICHMOND T., SOMERVILLE S.,

MANNERS J.M. 2000. Coordinated plant defense responses in Arabidopsis revealed by microarray analysis. Proc. Natl. Acad. Sci. USA 97: 11655–11660.

SCHRAUDNER M., LANGEBARTELS C., SANDERMANN H. 1997. Changes in the biochemical status of plant cells induced by the environmental pollutant ozone. Physiol. Plant. 100: 274–280.

**SEMBDNER G., PARTHIER B. 1993.** The biochemistry and the physiological and molecular actions of jasmonates. Annu. Rev. Plant Physiol. Plant Mol. Biol. 44: 569–589.

SEO S., SANO H., OHASHI Y. 1997. Jasmonic acid in wound signal transduction pathways. Physiol. Plant. 100: 740-745.

SEO H.S., SONG J.T, CHEONG J.-J., LEE Y.-H, LEE Y.-W., HWANG I., LEE J.S, CHOI Y.D. 2001. Jasmonic acid carboxyl methyltransferase: A key enzyme for jasmonate-regulated plant responses. Proc. Natl. Acad. Sci. USA 98: 4788–4793.

THOMMA B.P.H.J., EGGERMONT K., BROEKAERT W.F., CAMMUE B.P.A. 2000. Disease development of several fungi on Arabidopsis can be reduced by treatment with methyl jasmonate. Plant Physiol. Biochem. 38: 421–427.

SHOJI T., NAKAJIMA K., HASHIMOTO T. 2000. Ethylene suppresses jasmonate-induced gene expression in nicotine biosynthesis. Plant Cell Physiol. 41: 1072–1076.

TUOMAINEN J., BETZ C., KANGASJARVI J., ERNST D., YIN Z.H., LANGEBARTLES C., SANDER-MANN H., Jr. 1997. Ozone induction of ethylene emission in tomato plants: regulation by differential transcript accumulation for the biosynthetic enzymes. Plant J. 33: 1151–1162.

**UEDA J., KATO J. 1980.** Isolation and identification of a senescence-promoting substances from wormwood (Artemisia absinthium L.). Plant Physiol. 66: 246–249.

VAN WEES S.C.M., DE SWART E.A.M., VAN PELT J.A., VAN LOON L.C., PIETERSE C.M.J. 2000. Enhancement of induced disease resistance by simultaneous activation of salicylate- and jasmonate-dependent defense pathways in Arabidopsis thaliana. Proc. Natl. Acad. Sci. USA 97: 8711–8716.

VIJAYAN P., SHOCKEY J., LEVESQUE C.A., COOK R.J., BROWSE J. 1998. A role for jasmonate in pathogen defense of Arabidopsis. Proc. Natl. Acad. Sci. USA 95: 7209-7214.

WASTERNACK C., PARTHIER B. 1997. Jasmonate signalled plant gene expression. Trends in Plant Science 2: 302–307.

WATANABE T., FUJITA H., SAKAI S. 2001b. Effects of jasmonic acid and ethylene on the expression of three genes for wound-inducible 1-aminocyclopropane-1-carboxylate synthase in winter squash (Cucurbita maxima). Plant Science 161: 67–75.

WATANABE K., KAMO T., NISHIKAWA F., HYODO H. 2000. Effect of methyl jasmonate on senescence of broccoli florets. J. Japan. Soc. Hort. Sci. 69: 605-610.

WATANABE T., SAKAI S. 1998. Effects of active oxygen species and methyl jasmonate on expression of the gene for a wound-inducible 1-aminocyclopropane-1-carboxylate synthase in winter squash (Cucurbita maxima). Planta 206: 570–576.

WATANABE T., SEO S., SAKAI S. 2001a. Wound-induced expression of a gene for 1-aminocyclopropane-1-carboxylate synthase and ethylene production are regulated by both reactive oxygen species and jasmonic acid in Cucurbita maxima. Plant Physiol. Biochem. 39: 121–127. XU Y., CHANG P.-F.L, LIU D., NARASIMHAN M.L., RAGHOTHAMA K.G., HASEGAWA P.M., BRESSAN R.A. 1994. Plant defence genes are synergistically induced by ethylene and methyl jasmonate. Plant Cell 6: 1077–1085.

ZHU-SALZMAN K., SALZMAN R.A., KOIWA H., MURDOCK L.L., BRESSAN R.A., HASEGAWA PM. 1998. Ethylene negatively regulates local expression of plant defense lectin genes. Physiol. Plant. 104: 365–372.

Key words: ethylenc, jasmonates, physiological processes, stress conditions, regulation, reactive oxygen species

#### Summary

A relationship between jasmonates and ethylene in regulation of some physiological processes in plants under stress conditions is presented. Jasmonates are naturally occurring plant hormones showing various important biological activities in the regulation of plant growth development and in defense responses against a wide variety of abiotic and biotic agents. Jasmonates have been reported to control ethylene biosynthesis in intact plants and their organs. Mechanical wounding and other abiotic (osmotic stress, water deficit, dessication stress, heavy metals, touch, ozone) and biotic stresses (pathogen infection and insect invasion) are well known to be common factors inducing ethylene and jasmonates biosynthesis, and reactive oxygen species generation (ROS). Jasmonates have been well known to interact with ethylene in regulation of different processes; various kinds of interactions were documented: 1) synergistic interaction (i.e gene expression of proteinase inhibitors, osmotin, defensin), 2) ethylene suppresses processes induced by jasmonates (i.e. biosynthesis of nicotine, vegetative storage proteins and lectins), 3) jasmonates suppress processes induced by ethylene (i.e. ethylcne-induced apical hook). Jasmonic acid carboxyl methyltransferase (JMT) is a key enzyme for jasmonate-regulated plant responses. Activation of JMT expression leads to production of methyl jasmonate (JA-Me). JA-Me can act as an intracellular regulator, a diffusible intercellular signal transducer, or an airborne signal mediating intra- and interplant communications. Jasmonates represent an integral part of the signal transduction chain between stress signal(s) and stress responses(s), in most cases of the induction of gene expression and the accumulation of defense specific proteins and secondary metabolites.

### ZALEŻNOŚCI MIĘDZY JASMONIANAMI A ETYLENEM W REGULACJI NIEKTÓRYCH PROCESÓW FIZJOLOGICZNYCH U ROŚLIN W WARUNKACH STRESOWYCH

Marian Saniewski<sup>1</sup>, Junichi Ueda<sup>2</sup>, Kensuke Miyamoto<sup>2</sup>, Henryk Urbanek<sup>3</sup> <sup>1</sup> Instytut Sadownictwa i Kwiaciarstwa, Skiernicwice

<sup>2</sup> College of Integrated Arts and Sciences, Osaka Prefecture University, Japonia <sup>3</sup> Katedra Fizjologii i Biochemii Roślin, Uniwersytet Łódzki, Łódź

Słowa kluczowe: etylen, jasmoniany, procesy fizjologiczne, warunki stresowe, regulacja, reaktywne formy tlenu

#### Streszczenie

Zależności między jasmonianami i etylenem w regulacji niektórych procesów fizjologicznych u roślin w warunkach stresowych są przedmiotem tego przeglądu. Jasmoniany są naturalną grupą hormonów roślinnych i wykazują wielc ważnych funkcji fizjologicznych w regulacji wzrostu i rozwoju roślin i w reakcjach obronnych przeciwko różnym czynnikom abiotycznym i biotycznym.

Wykazano, że jasmoniany odgrywają ważną rolę w regulacji biosyntezy etylenu w roślinach nienaruszonych i ich organach. Mechaniczne uszkodzenie i inne czynniki abiotyczne (stres osmotyczny, deficyt wodny, stres desykacyjny, metale ciężkie, dotyk, ozon) i czynniki biotyczne (infekcja przez patogeny i żerowanie owadów) powodują wzmożoną biosyntezę etylenu i jasmonianów oraz generowanie reaktywnych form tlenu (ROS). Jasmoniany współdziałają z etylenem w regulacji różnych procesów, a interakcje te mają różny charakter: 1) synergistyczne współdziałanie (np. ekspresja genów inhibitorów proteinaz, osmotyny, dcfenzyny), 2) etylen hamuje procesy indukowane przez jasmoniany (np. biosynteza nikotyny, wegetatywnych białek zapasowych, lektyn), 3) jasmoniany hamują procesy indukowane przez etylen (np. wygięcia części wierzchołkowych powodowane przez etylen). Metylotransferaza karboksylowa kwasu jasmonowego (JMT) jest kluczowym enzymem w procesach regulowanych przez jasmoniany w roślinach. Aktywacja ekspresji JMT doprowadza do powstawania jasmonianu metylu (JA-Me) z kwasu jasmonowego. JA-Me może działać jako regulator w obrębie komórki i jako międzykomórkowy sygnał transdukcyjny, a jako lotna substancja stanowi przekaźnik informacji między roślinami. Sugeruje się, że cndogenne jasmoniany stanowią przekaźnik informacji między sygnałem stresowym a reakcją stresową, polegającą głównie na indukcji ekspresji genowej i biosyntezie specyficznych białek i metabolitów wtórnych.

Prof. dr hab. Marian Saniewski, czł. koresp. PAN Instytut Sadownictwa i Kwiaciarstwa ul. Pomologiczna 18 96–100 SKIERNIEWICE e-mail: msaniew@insad.pl