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Mini Review

Jasmonates: Hormonal Regulators or Stress Factors in Leaf Senescence?

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Abstract. Specific cyclopentanone compounds such as (−)-jasmonic acid (JA) and its methyl ester (JA-Me) or (+)-7-iso-jasmonic acid are considered putative plant growth regulators for a number of reasons, including their ubiquitous occurrence in the plant kingdom, structural specificity in physiological responses, and interaction with other phytohormones in the biological activities of jasmonates. In this respect leaf senescence promotion is of particular preponderance. Recent progress in the mode of jasmonate actions in the barley leaf segment senescence model system demonstrates two effects at the level of gene expression: the induction by exogenously applied jasmonates of abundant specific proteins and their mRNAs (transcription control), and the cessation of synthesis of normal leaf proteins but not their respective mRNAs (translation control). These effects resemble cellular responses to well-known stress factors. The arguments for and against hormonal or stressor-like actions of jasmonates in leaf senescence are discussed in this review.

(−)-Jasmonic acid (JA) and its derivatives have been postulated to represent putative plant growth regulators (Meyer et al. 1984; Ueda and Kato 1980, 1982a; Ueda et al. 1981; Parthier 1988; Sembdner and Klose 1985). This proposal is based on a variety of criteria valid for the "classic" phytohormone groups. Some criteria such as universal occurrence in plants, biological activity at low concentrations, interaction with other hormones, etc., supporting the phytohormone concept are reviewed in the following, although certain observations suggest to compare jasmonates with stress factors or stress modulators.

Occurrence and Distribution of Jasmonates

(−)-Jasmonic acid methyl ester (JA-Me) (Fig. 1) was the first compound of the jasmonate group (cyclopentanone derivatives) isolated from the essential oil of *Jasminum grandiflorum* (Demole et al. 1962) and *Rosmarinum officinalis* (Crabalona 1967). Ueda and Kato (1980) found methyl(1R,2R)-3-oxo-2-(2′-*cis*-pentenyl)-cyclopentane-1-acetate in the leaves of *Artemisia absinthium* and several other species (Yamane et al. 1981a). Endogenous compounds such as JA or (+)-7-iso-jasmonic acid (old nomenclature: (+)-2-iso-jasmonic acid), and their methyl esters were isolated from *Vicia faba* fruits (Dathe et al. 1981; Miersch et al. 1986) with high activities in several bioassay systems (see reviews by Sembdner and Klose 1985; Sembdner and Gross 1986). Amino acid (Tyr, Ile, Trp) conjugates of jasmonates were likewise isolated in biologically active forms (Brückner et al. 1986; Herrmann et al. 1987).

In an extensive screening program using a radioimmunoassay with JA-specific antisera (Knöfel et al. 1984), as well as physical methods, jasmonates were detected in 206 plant species representing 150 families including ferns, mosses, and fungi (Meyer et al. 1984; Sembdner and Gross 1986). These substances seem to be ubiquitously distributed throughout the plant kingdom.

Studies on the occurrence of jasmonates in the various organs of *Vicia faba* provided evidence for a marked accumulation in flowers, young leaves, and fruits (10–30 $\mu g \times g^{-1}$ fresh weight), but low amounts, if any, were noted for roots, shoots, and mature or old leaves (Knöfel et al. 1984; Meyer et al. 1984). In soybean fruit, the highest jasmonate levels were observed in the pericarp (particularly in its vascular bundles), hilum, and testa; the lowest

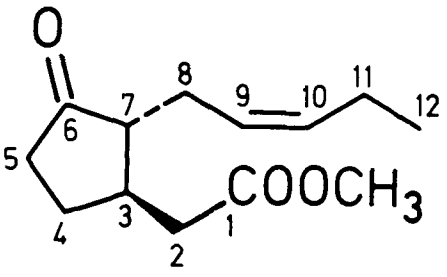

Fig. 1. Structure formula of (−)-jasmonic acid methyl ester (new enumeration). According to biosynthetic studies (Vick and Zimmermann 1986), the naturally occurring substance has been elucidated as a *cis* configuration of the side chains with respect to the plane of the ring. Since this structure is easily isomerized into the more stable *trans* stereoisomer, natural (−)-jasmonates may usually exist as a mixture of the two isomeric forms.

levels were reported for cotyledons and embryo axes (Lopez et al. 1987). There are preliminary data available (G. Sembdner, personal communication) that this type of jasmonate distribution is likewise true for other plants, namely, monocotyledons.

Biological Activities

Earlier research studied the biological activities with endogenous jasmonates (Ueda and Kato 1980, 1981; Yamane et al. 1981a, b), but most experiments were done with the racemates of JA or JA-Me, as they are easily available from the perfume industry. Jasmonates exert both stimulatory and inhibitory effects in different biological test systems. They inhibit the growth of rice or wheat seedlings in bioassays (Dathe et al. 1981; Yamane et al. 1981a, b). Dose–response and structure–response relations indicate that JA and JA-Me are very effective substances. Three important structural moieties are necessary for seedling growth inhibition (and probably for any biological activity): the acetyl side chain, the *n*-pentenyl chain inserted at C-7, and a keto or hydroxy group at C-6 (Yamane et al. 1980; see Fig. 1).

The inhibitory activity of JA-Me (+)-enantiomer was found to be lower than that of the naturally occurring (−)-form, although the (+)-form is still fairly potent (Yamane et al. 1981b). Growth inhibition occurs in both shoots and roots at 10^{-5} M or higher concentrations (Sembdner and Klose 1985). Soybean callus growth and cytokinin-dependent cell divisions were strongly inhibited by JA, less so by JA-Me, and minimally by ABA. The reverse was observed in the radish cotyledon growth test (Ueda and Kato 1982a). Another inhibitory response to JA treatment was reported for pollen germination (Yamane et al. 1981a, 1982). Seed germination appears not to be affected by JA treatment.

The ability of jasmonates to promote leaf senescence was observed previously (Ueda and Kato 1980, 1981) and recently studied in much more detail. It is not surprising that multiple effects were described as an expression of the complexity of the senescence syndrome. Most studies have dealt with a marked loss of chlorophyll resulting in the yellowing of leaf tissue (Ueda and Kato 1980, 1981, 1982a; Satler and Thimann 1981; Weidhase et al. 1987b), because carotenoids are less affected by jasmonate treatments. In ripening tomato fruits JA-Me prevents lycopene accumulation and stimulates β-carotene synthesis (Saniewski and Czapski 1983). The treated fruit tissues remain in a yellow, unripe stage of development. In addition, JA-Me stimulates ethylene biosynthesis but inhibits or does not affect the activity or synthesis of polygalacturonase, the key enzyme in fruit softening (Saniewski et al. 1987a, b).

Other typical senescence symptoms, such as cellular respiration and proteolytic (Satler and Thimann 1981) as well as peroxidase activities (Weidhase et al. 1987b), increase in JA-Me-treated leaf segments. Structural damaging of chloroplasts (U. zur Nieden, unpublished results) and, in particular, the reduction of photosynthetic activity as markers for normal leaf senescence are also observed after JA treatment (Popova et al. 1988). Concurrently, a rapid decline of activity and protein degradation of ribulose-1,5-bisphosphate carboxylase (RuBPCase) is observed (Parthier et al. 1987b; Weidhase et al. 1987b) as well as the cessation of synthesis (Popova and Vaklinova 1988; Weidhase et al. 1987a). RuBPCase is markedly more affected by JA-Me treatment than the other soluble cell proteins. The senescence symptoms caused by jasmonate can be restored by cytokinins (Ueda et al. 1981; Ueda and Kato 1982a; Weidhase et al. 1987b). These phenomena resemble or seem to be identical with symptoms of natural senescence (Thimann 1980; 1985; Thomas and Stoddart 1980; Parthier 1988; Woolhouse 1984). However, functional damage and subsequent dismantling of chloroplasts are regarded to represent late stages in the leaf senescence syndrome; dissimilarities could exist earlier between the natural aging process and jasmonate-induced senescence.

JA-Me treatment of isolated chloroplasts has no effect on chlorophyll content or RuBPCase activities (Parthier et al. 1987a). This indicates an indirect effect on chloroplasts by jasmonates, which should act primarily on nonchloroplast cellular constituents. The senescence symptoms of chloroplasts in situ might be caused by imported molecules from

the surrounding cytoplasm, as suggested by Choe and Thimann (1975). These symptoms are probably connected with proteolytic or lipolytic activities. This concurs with our observations (Mueller-Uri et al. 1988; Weidhase et al. 1987a; and unpublished results) that amino acid incorporation into both types of subunits (LS and SS) of RuBPCase is stopped soon after JA-Me treatment of leaf tissues, followed by a continuous loss of subunit proteins, whereas (preexisting) mRNA remains stable. Thus, transcription and translation of plastid proteins are affected differently by JA-Me treatment of barley leaf tissues.

Presently, we can only speculate about the physiological role and mode of action of jasmonates in leaf senescence. One idea pointed out by Leshem (1987) is based on the fact that jasmonates are synthesized from free linolenic acid (Vick and Zimmermann 1986), probably due to membrane lipid peroxidation. In a cascade process, sufficient amounts of endogenous jasmonate could be produced for triggering visible senescence symptoms. Another explanation might be referred to JA-induced changes in the expression of specific genes resulting in the synthesis of novel polypeptides, as described below.

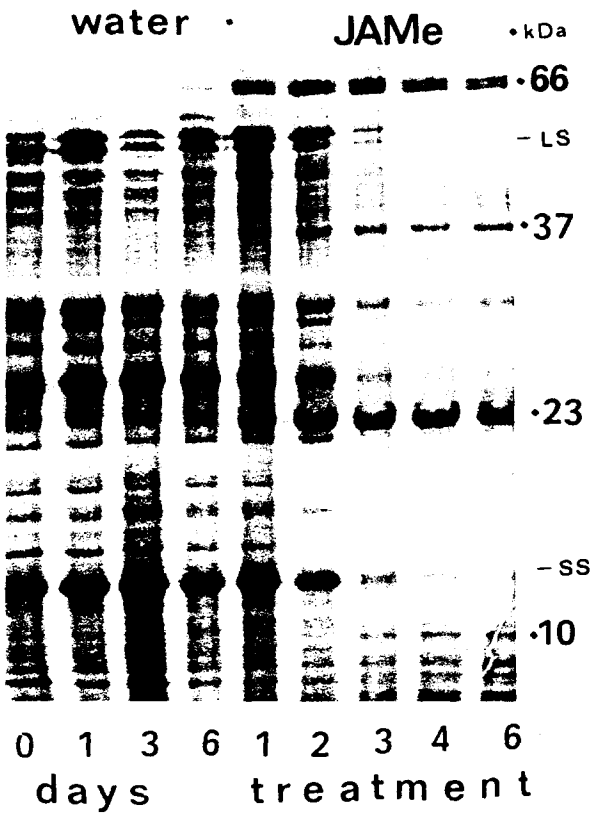

Fig. 2. Coomassie-stained soluble proteins of 5×10^{-5} M JA-Me-treated and control (water) barley leaf segments after SDS gel electrophoresis. Abundant JA-Me-induced protein classes are indicated as relative molecular masses in kilodaltons (kDa) at the right ordinate.

Altered Gene Expression and Jasmonate-Induced Proteins

Jasmonate-treated barley leaf segments respond with a marked accumulation of novel abundant proteins (Fig. 2), which indicates a dramatic alteration in gene expression of these tissues. The first report on jasmonate-induced proteins (JIPs) was presented on February 1986 at the UCLA Conference on Molecular Biology of Plant Growth Control (Parthier et al. 1987b). JIPs occur in isolated barley tissues 1 day after treatment with 5×10^{-5} M JA-Me or JA. They are synthesized de novo as demonstrated by both labeling and inhibitor experiments (Weidhase et al. 1987a). Cycloheximide prevents JIP accumulation, whereas chloramphenicol does not, suggesting cytosolic polyribosomes as the sites of synthesis. On the other hand, immunological and electron microscopic studies indicate that at least certain species of JIPs may be located in cell organelles, e.g. nuclei (unpublished results).

We have found jasmonate-induced proteins accumulated in the leaf tissues of a large number of monocotyledonous and dicotyledonous plant species. Jasmonate-induced proteins have not been detected in JA-Me-treated roots or nontreated senescent leaves attached to mature barley plants (Herrmann et al. 1989). There is a great variability in the number and relative molecular masses of JIPs in various plant species, and even barley cultivars can differ in JIP patterns (Herrmann et al. 1989). Cell suspension cultures of soybean also contain JA-Me-induced proteins (Anderson 1988), the major ones with molecular masses of 31 and 39 kDa, of which the former is a glucoprotein.

The barley leaf JIP ensemble consists of a number of isomeric proteins, of which the most abundant ones correspond with M_rs of 66,000, 37,000, 30,000, 23,000, and 10/12,000 (JIP 66, JIP 37, JIP 30, JIP 23, JIP 10) (Mueller-Uri et al. 1988; Weidhase et al. 1987a). Immunologically, they are unrelated to one another, to RuBPCase polypeptides (Herrmann et al. 1989), or to heat-shock proteins (Mueller-Uri et al. 1988). Salt stress (0.5 M NaCl), ethylene-treatment, or anaerobic conditions do not induce JIPs or immunologically related proteins (unpublished observations).

Kinetics of in vivo ^{35}S-methionine-labeling experiments, as well as in vitro translation of JIP mRNAs, indicate a molecular response within 3–5 h

after JA-Me treatment of barley leaf segments (Mueller-Uri et al. 1988), that is, JIPs are detectable much earlier than any of the above-mentioned senescence symptoms. Thus, JIP formation should be considered a cause rather than a consequence of senescence, provided a causal relationship exists at all.

Of special interest is the observation that ABA and JA induce apparently the same abundant proteins in barley leaf segments (Weidhase et al. 1987a). Respective proteins with the same M_r are immunologically cross-reactive, and the population of translatable mRNAs do not differ in JA- or ABA-treated barley segments (Lehmann et al., unpublished observations).

On the one hand, the kinetics and pattern of JIP mRNA translation suggest (positive) transcription control by jasmonates, but on the other hand, a (negative) translation control. The latter is indicated by maintaining almost all translatable RNAs for normal leaf proteins in JA-Me-treated tissues, compared with a lack of in vivo synthesis of these proteins after jasmonate treatment (Mueller-Uri et al. 1988). In conclusion, jasmonates cause a massive reprogramming of the gene expression program of treated leaf segments independent of their developmental stage. These phenomena are typical for stress responses in living matter (see reviews by Nover 1984; Sachs and Ho 1986).

Arguments For and Against a Unified Concept

Taking into consideration the preceding observations about jasmonate-induced senescence symptoms in isolated leaf tissues, it seems reasonable to compare JA or JA-Me with other stress factors which elicit, directly or indirectly, leaf senescence by induction or altered gene expression programs (Parthier 1988; Thomas and Stoddart 1980). Ethylene and ABA, both well-recognized phytohormones, belong to such substances. In recent years, there has been a greater focus on the genetic level for both hormone (Parthier 1989) and stress responses (Nover 1984; Sachs and Ho 1986); it may be stimulating to assume a unifying view with respect to a common signal transduction to the genetic level. This would agree with the idea of elicitor-induced syntheses of a multiplicity of specific proteins ("stress proteins").

For instance, low molecular weight elicitors originating from cell wall polysaccharides, after their partial digestion by pathogens, induce the synthesis of chitinase, β-1,3-glucanase and various so-called pathogen-related proteins of different functions (Boller et al. 1983; Van Loon 1985). The same effects are observed after exogenous application of ethylene. Another example is the loss of cell membrane integrity observed in plant organs that are distant from mechanically damaged (wounded) or pathogen-attacked tissues. This phenomenon suggests that a systemic signal derived from the cell wall (oligouronide fragments) is rapidly transmitted through the plant (Walker-Simmons et al. 1984). It may trigger gene expression and subsequent accumulation of considerable quantities of proteinase inhibitor proteins as a response to wounding stresses (Cleveland et al. 1987).

Although the primary site(s) of jasmonate action is unknown as yet, one is tempted to speculate that exogenous applications of jasmonates or ABA result in stress responses that involve damaging cell membranes including the liberation of elicitors therefrom. The latter may be transmitted to the genetic level and signalize selective protein (JIP) gene expression. Thus, jasmonate itself must not act as an internal signal. Moreover, this role could be played by cell membrane constituents, preferably polyunsaturated fatty acids liberated from lipids during senescence (Thomas 1986). Similar to ABA, exogenously applied C_{18}-unsaturated fatty acids can promote oat leaf senescence (Ueda and Kato 1982b). Other polyunsaturated fatty acids, arachidonic and eicosapentaenoic acids, are involved in the response to pathogen attack leading to pathogen resistance reactions and including de novo synthesis of proteins (Bostock et al. 1986; Van Loon 1985). As mentioned above, polyunsaturated fatty acids (e.g., linolenic acid) which are probably derived from cell membranes, serve as precursors in the biosynthesis of jasmonates (Vick and Zimmermann 1986) and support the view of being the source for "autocatalytic" production of endogenous JA (Leshem 1987).

Before we reconsider the hormone concept of jasmonate action in leaf senescence and try to combine it with the idea of stress response, we should be aware that first, endogenous hormones and exogenously added respective substances must not a priori cause the same effects. Second, normal senescence of leaves attached to the plant may be discriminated from the yellowing and other senescence symptoms observed with abscised leaf tissues. Keeping in mind these problems, at least three possibilities can be discussed about the manner of jasmonate action in foliar senescence.

1. Both endogenous and exogenous jasmonates promote senescence by actions similar to those observed with other phytohormones (at least ABA); this "hormonal effect" could be true for attached and abscised leaves.

2. Endogenous jasmonates are senescence-promoting hormonal substances (in situ), but exogenously applied jasmonate acts as a stress modulator by enhancing (or suppressing) developmental processes, which are normally less obvious. According to foliar senescence, exogenous jasmonate could accelerate a process that normally proceeds much slower.
3. Endogenous as before, but exogenously applied jasmonate itself plays the role of a stressor that causes typical stress responses, inter alia the induction of stress proteins (JIPs).

We will briefly comment on the above three assumptions.

Case 1

There are some indications that jasmonates share the characteristics of plant growth regulators [physiological responses modulated by interaction with other phytohormones (e.g., synergistic to ABA, antagonistic to cytokinins), low dosage effects, ubiquitous occurrence]. Neither of these arguments is fully convincing but deserves critical remarks (e.g., although well-established plant hormones, ABA as well as ethylene elicit stress responses under certain circumstances; otherwise, they play a protective role as stress metabolites; cytokinins could act as stabilizing agents in general; ubiquitous occurrence is not specific for hormones; exogenous concentration does not reflect the amount at the site of action, etc.). It should be emphasized that not all plant growth regulators can be related with stress responses.

The dilemma in addressing bioactive substances either as hormonal regulators or stress factors lies not only in the "sensitivity" and "competence" of the target organs and cells, which are connected with receptors and binding molecules. It concerns likewise—already by hormone definition—that the site(s) of synthesis or accumulation differ from the site(s) of action. Therefore, the assumption that jasmonate accumulation in the pericarp reflects a sink function for assimilates (Lopez et al. 1987) is an interesting but unproved aspect. By contrast, it would make biological sense if mature, more or less jasmonate-free leaves represent sinks for endogenous jasmonates from young organs. Mature leaves then reach the threshold to become senescent not only as a result of organ competition for nutrients but also by basipetal transport of a "senescence signal" (Noodén 1984): Perhaps endogenous jasmonates originating from generative organs or young leaves? Evidence for experimentally proven directed transport of jasmonates would fulfill a decisive criterion for their hormonal regulator function.

In context with the latter role of jasmonates in senescence, one has to consider that the enzymes of their biosynthesis in plants show the highest activity in young tissues (Vick and Zimmermann 1986). These findings do not abandon the source–sink relationship just mentioned for possible physiological functions in plant senescence (if developing pericarp is the main source for endogenous jasmonates); however, other subtle functions of these substances in plant cell metabolisms cannot be excluded at present.

Case 2

Jasmonates could exert stress modulation, that is, promoting or enhancing senescence in attached and detached leaves. The latter in particular is stressed by abscission from the plant and thus sensitive to exogenously applied specific substances like ABA or jasmonates, but other membrane-active substances inducing jasmonate biosynthesis might be predicted (see above). The point is whether or not they promote senescence *and* the formation of stress proteins. A separation of the two processes should help to discriminate the induction of senescence from stress responses. In the barley leaf segment model system, JIP formation is most probably connected with stress response, as can be deduced from kinetic experiments (Mueller-Uri et al. 1988), but we have insufficient markers for early senescent responses. Another argument against the "hormonal" action of exogenous jasmonates is our observation that JIPs were not observed in naturally senescent barley leaves (Herrmann et al. 1989), but these leaves respond with JIP accumulation after JA-Me treatment in situ.

Case 3

Continuing this idea, exogenous jasmonates as well as ABA applied to excised leaf tissues of barley, as a model, cause effects which resemble stressor-induced responses. Most evident is the synthesis of novel abundant proteins and the cessation of synthesis of normal ("control") proteins. The obvious identity of ABA- and JA-induced proteins deserves attention, since ABA is not only involved in dessication stresses as a stress metabolite but induces the synthesis of novel specific proteins (Gomez et al. 1988; Mundy and Chua 1988), which might be important in acquired stress tolerance.

However, the stressor hypothesis for exogenous

jasmonates includes high specificity in respect to the substances. There is a lack of JIP-like protein formation in otherwise stressed barley leaf segments (salt, anaerobiosis, wounding). A high sensitivity is shown by dose–response relations: JIPs are already accumulated after treatment with low ("hormonal") JA-Me concentrations connected with specific response in respect to the stereochemical structure of jasmonates; and the naturally occurring (−)-enantiomer induces JIPs in the barley leaf segment model system at concentrations of 10^{-8} M to 10^{-5} M, but the (+)-form fails even at 10^{-5} M to 10^{-4} M (Herrmann et al., unpublished observations). This observation is interesting compared with the data of Ueda et al. (1981), who found the (+)-form also less active than the (−)-form of JA-Me in the oat leaf senescence bioassay, but still fairly potent.

In spite of the model character of the abscised leaf tissue system and the discrepancies discussed in this review, we think that exogenous jasmonates can also be helpful tools for studying certain steps (gene expression) in foliar senescence.

Acknowledgment. This mini review is in homage to Professor Kenneth V. Thimann, the nestor of plant senescence research, on the occasion of his 85th birthday.

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