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# Emission and Function of Volatile Organic Compounds in Response to Abiotic Stress

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## 1. Introduction

Plants accumulate a diverse array of natural products, which are thought to be involved in their interactions with the environment. These chemicals function in plant communications with microbes, animals, and even other plants, as well as protecting the plant from ultraviolet radiation and oxidants. Some compounds may attract beneficial insects or microbes, whereas others kill or repel herbivorous. Many of these compounds have been referred to as “secondary metabolites” to distinguish them from the “primary metabolites” required for the growth of all plants (Theis & Lerdau, 2003). These secondary metabolites, however, are likely to be essential for successful competition or reproduction.

More than 100,000 chemical products are known to be produced by plants and at least 1,700 of these are known to be volatiles (Dicke & Loreto, 2010). Some of the volatiles considered in this chapter are shown in figure 1. **Volatile organic compounds (VOCs)** are defined as any organic compound with vapor pressures high enough under normal conditions to be vaporized into the atmosphere (Dicke & Loreto, 2010). The importance of these compounds can be deduced by the considerable amount of photoassimilated carbon released back into the atmosphere as VOCs (Holopainen, 2004). In fact, it has been estimated that the emission of VOCs by terrestrial plants accounts for the 36% of the whole photosynthates (Kesselmeier et al., 2002). This emission, therefore, substantially reduces the amount of available carbon and consequently affects plant physiology and productivity. Why plants, under stress conditions, where carbon availability is a crucial limiting resource, lose such a relevant amount of assimilated carbon?

VOCs are involved in a range of ecological functions, including indirect plant defense against insects, pollinator attraction, plant-plant communication, plant-pathogen interactions,

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### Note:

This chapter has been written in cooperation with the students of the course “Ecosystems and Environmental Stress Physiology” of the ERASMUS MUNDUS - International Master in Horticultural Sciences (IMaHS: [www.imahs.unibo.it](http://www.imahs.unibo.it)). Dr. Francesco Spinelli, the tenured professor of the course, was responsible for the whole chapter structure and for the direction and supervision of the manuscript. Dr. Antonio Cellini contributed to the section on nitric oxide. Livia Marchetti (role of light stress on VOCs induction), Chiara Piovene (jasmonates) and Karthik Mudigere Nagesh (drought driven emission of VOCs) were all students of this master course.

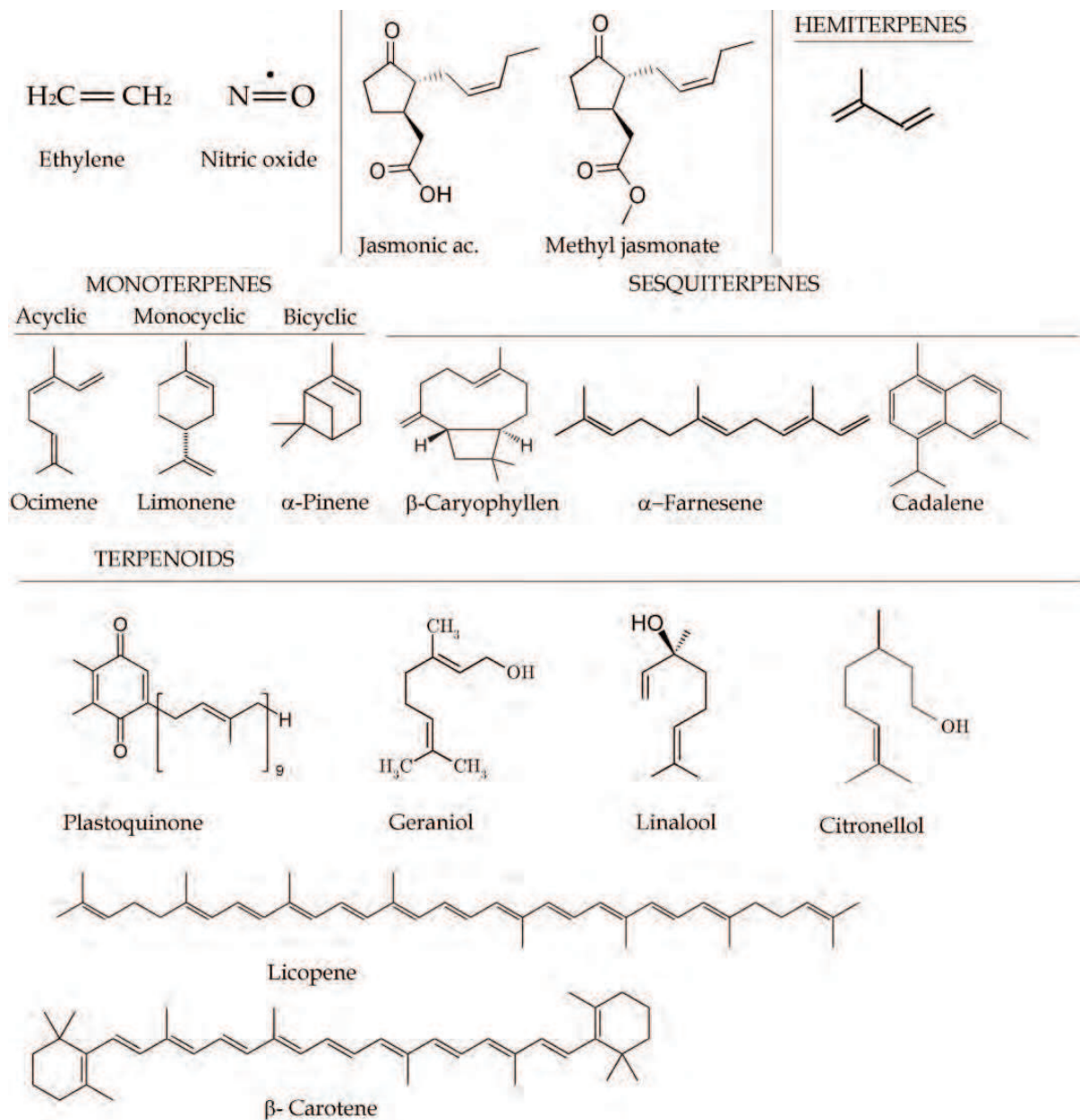


Fig 1. Chemical diversity of the different VOCs, and related compounds, present in the plant. The low molecular weight compounds (i.e. NO, ET, JA, MJA, ISOPRENE) usually act as stress signals. Isoprene, NO and the majority of the other compounds may also directly act as antioxidants.

reactive oxygen species removal, thermo-tolerance and environmental stress adaptation. Their evolution is quite complex and it is affected by interactions of plants with biotic and abiotic factors in constantly changing environments, at local and global level.

**Stored VOCs** may be volatilized into the atmosphere by healthy unwounded plants depending on their concentration and physiochemical properties (Niinemets et al., 2004). While many plants contain large amounts of stored VOCs, others do not synthesize and emit them until an environmental *stimulus* is perceived (Paré et al., 2005). **Induced VOCs** (IVOCs) may be emitted hours or days after a stress, both from the stressed sites as well as systemically from undamaged plant leaves (Paré & Tumlinson, 1997). Most of the

constitutive VOCs normally released from healthy intact plants become inducible volatiles after foliar damage (Wiens, 1991; Vuorinen, 2004). In contrast to the constitutive VOCs, the novel IVOCs are produced only after biotic and abiotic inductions. The advantage of novel IVOCs is that they are *de novo* synthesized only when needed and therefore they optimize carbon usage and do not reduce plant fitness (Dicke, 2000).

The VOCs are important infochemicals and their role in shaping the biotic interactions is well known. However, inducible VOCs are also emitted in response to abiotic stress perception and they may play a role in stress adaptation or response. Environmental stress such as physical damage, nutrient deficiency, salinity, drought and ozone exposure hamper the IVOCs emission. **Stress in plants could be defined as any change in growth conditions that disrupts metabolic homeostasis and requires an adjustment of metabolic pathways in a process that is usually referred to as acclimation (Shulaev et al., 2008).**

Plants have an extraordinarily diverse suite of protective mechanisms against abiotic stresses, since they must be capable of coping with a variety of changes in light intensity, temperature, moisture and other abiotic factors in their environments. When these factors shift out of a certain range, plants are subjected to stress; this can lead to decreased growth rate, reduced reproduction and even death (Vickers et al., 2009). Moreover, these stresses are rarely experienced singularly, but they often occur in combination.

The most common response to stresses is the production of excess reactive oxygen species (ROS), substances continuously produced in plants in normal conditions. Abiotic stress factors can perturb the equilibrium between production and scavenging of ROS, causing an excessive production of these components that lead to a direct damage to plant cells through oxidation of biological components (nucleic acids, proteins and lipids) and can instigate chain reactions resulting in accumulation of more ROS and initiation of programmed cell death (Apel & Hirt, 2004). Well known example of environmental stresses leading to direct ROS accumulation are light excess, which causes photoinhibition and damages to photosynthetic reaction centers, and high temperature stress, which denatures proteins and causes lipid peroxidation. In addition, these two stresses are often coincident thus amplifying one the effect of the other. In this chapter, it will be exposed how the ROS accumulation is the common mechanism underlying the volatile emission in response to both biotic and abiotic stresses.

There is a broad diversity of known IVOCs, including alkenes, alkanes, carboxylic acids, nitrogen-containing compounds and alcohols, but the dominating compounds tend to be isoprene, terpenes and C6 green leaf volatiles (GLVs) (Holopainen & Gershenzon, 2010). The emission of VOCs from plants varies extensively depending on the species, organs, development stage and environmental conditions (Holopainen & Gershenzon, 2010). GLVs are produced *via* the lipoxygenase (LOX) pathway, and they can account for more 50% of the emissions from damaged plant parts. Chemically, GLVs are mostly saturated or monounsaturated aldehydes, alcohols and esters, and they can have different configurational isomers with different sensory properties (Ruther, 2000). GLVs are typically released only from damaged plant organelles within 1–2 seconds of the mechanical damage occurring (Fall et al., 1999), but some GLVs are released from younger undamaged leaves of herbivore damaged plants, indicating that the LOX pathway can be activated in intact leaves.

The high diversity of IVOCs suggests that plants are capable of disseminating information to their environment by using IVOCs and that plants can actively change the growth conditions using reactive IVOCs (reviewed by Holopainen, 2004).

The present chapter will review the current knowledge on the emission and function of IVOCs in response to abiotic stress. The review will also focus on the role of ethylene, nitric oxide, jasmonic acid and derivatives (i.e. methyl jasmonate), isoprene and terpenes.

## 2. Ethylene

Ethylene is the first gaseous hormone discovered in nature (Bleeker & Kende, 2000). Ethylene is a gaseous alkene and it is the simplest in structure among all the plant hormones. This 2-carbon olefin is a powerful elicitor of morphological changes during all stages of the plant life cycle from development, to fruit ripening and senescence. **In addition, a variety of stresses such as wounding, pathogen attack, flooding, drought, hypoxia, temperature shifts, physical loads and noxious chemicals (i.e. ozone and sulfur dioxide) induce ethylene production** (Yang & Hoffman 1984; Abeles et al., 1992; Bleeker & Kende 2000, Tschardt et al., 2001, Overmyer et al., 2003; Vahala et al., 2003). Furthermore, there is extensive crosstalk between the ethylene response pathway and other signaling networks (Johnson & Ecker 1998) (Fig. 2).

This complex and multifaceted regulatory network has not yet been completely elucidated. Nonetheless it is well known that ET induces diverse effects in plants throughout their life cycle from seed germination, floral differentiation to senescence. In order to achieve the myriad of effects it elicits in numerous physiological process, ET response must be precisely regulated at multiple levels, from hormone synthesis and perception to signal transduction and transcriptional regulation. However, even if ET has a central role in many fundamental processes, some evidences suggest that ET acts more as a potentiator or enhancer and it is not strictly required for survival. Failure to perceive ethylene is apparently not essential for survival in the laboratory setting, but ethylene signaling undoubtedly contributes to the hardiness of plants in the wild (Johnson & Ecker 1998).

**Ethylene also gave an example of plant-to-plant signalling**, probably unrelated to a role in defence: normally, the leaves of wild-type tobacco plants tend to stop growing as they approach neighbouring tobacco plants, this may stop them wasting energy producing leaves that would be shaded from useful light.

Plants in the vegetative growth phase, flowers, and immature fruits produce barely detectable levels of ethylene until they are subjected to stress or undergo maturation events, after which ethylene production accelerates in a spatially and temporally specific pattern (Johnson & Ecker 1998). All vascular plants analyzed to date synthesize ethylene via the Yang cycle, wherein S-adenosyl methionine is diverted from alternative fates to make the ethylene precursor 1-aminocyclopropane-1-carboxylic acid (ACC) (Yang & Hoffman, 1984) (fig. 3).

Expression of the enzyme catalyzing this reaction, ACC synthase, is induced by stimuli that lead to increased ethylene production, suggesting that ACC synthase activity is rate-limiting for ethylene synthesis (fig. 3). ACC can be readily converted to ethylene, CO<sub>2</sub>, and HCN by ACC oxidase, or it can be conjugated to a malonyl or glutamyl group to limit its availability for ethylene production. ACS is encoded by a multigene family, and different members show distinct patterns of expression during growth and development, and in response to various external cues. In addition to this transcriptional control, the stability of the ACS protein is also highly regulated (Argueso et al., 2007). Although control of ethylene production is largely attributed to ACC synthase, the differential expression patterns of ACC oxidase (ACO) genes suggest that oxidases contribute to regulation of ethylene production as well. Periods of ACO induction correlate with ethylene-regulated events in several instances, including senescence, fruit ripening, and wounding.

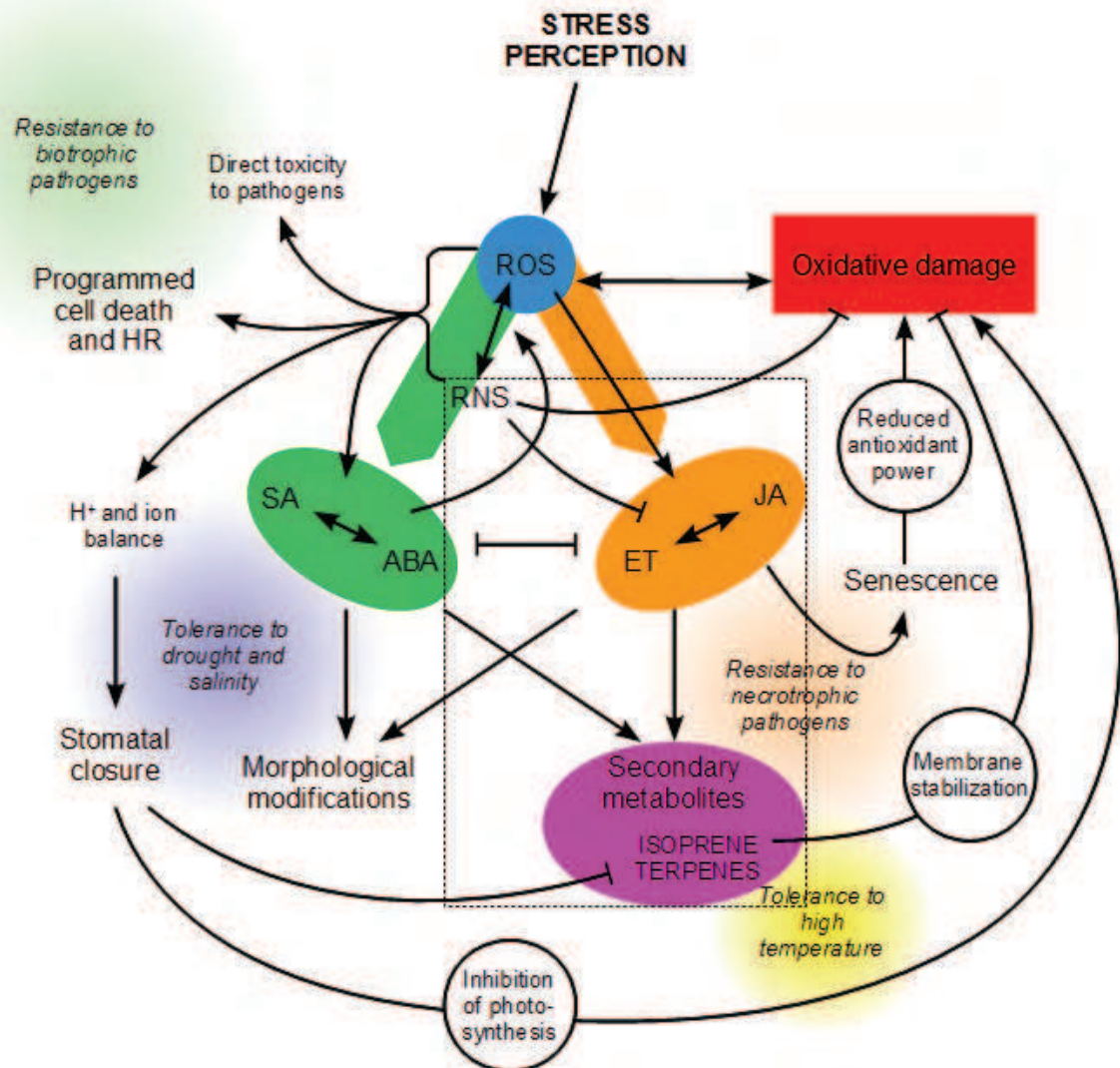


Fig. 2. Overview of the signalling events and reactions following the perception of stress. As a primary consequence, an oxidative burst occurs, due to the production of Reactive Oxygen Species (ROS). This early event causes oxidative damage and trigger the signal cascades leading to stress tolerance. The plant reacts to the ROS accumulation through two possible ways. On one side (green path), the interplay of ROS and Reactive Nitrogen Species (RNS) allows the induction of programmed cell death, the modulation of ion fluxes (including  $\text{Ca}^{2+}$ ), and the direct killing of noncompatible pathogens. Salicylic (SA) and abscisic acid (ABA) are long-ranged hormones mediating these responses in feedback with ROS and RNS. Several stressing agents, both abiotic and biotic, stimulate this pathway. A distinct and partially antagonistic signal cascade (orange path) involves the production of ethylene (ET) and jasmonates (JA). These hormones are required for the resistance to necrotrophic pathogens. Notably, the production of isoprene and terpenoids is stimulated by JA, and contributes to thermotolerance and stress mitigation. Apart from their role as signal molecules, isoprenoids can also act as quenching molecules of ROS. The volatile messengers in the described processed, namely NO (the precursor of RNS), ET, JA, isoprene and terpenoids are evidenced in the dashed frame.

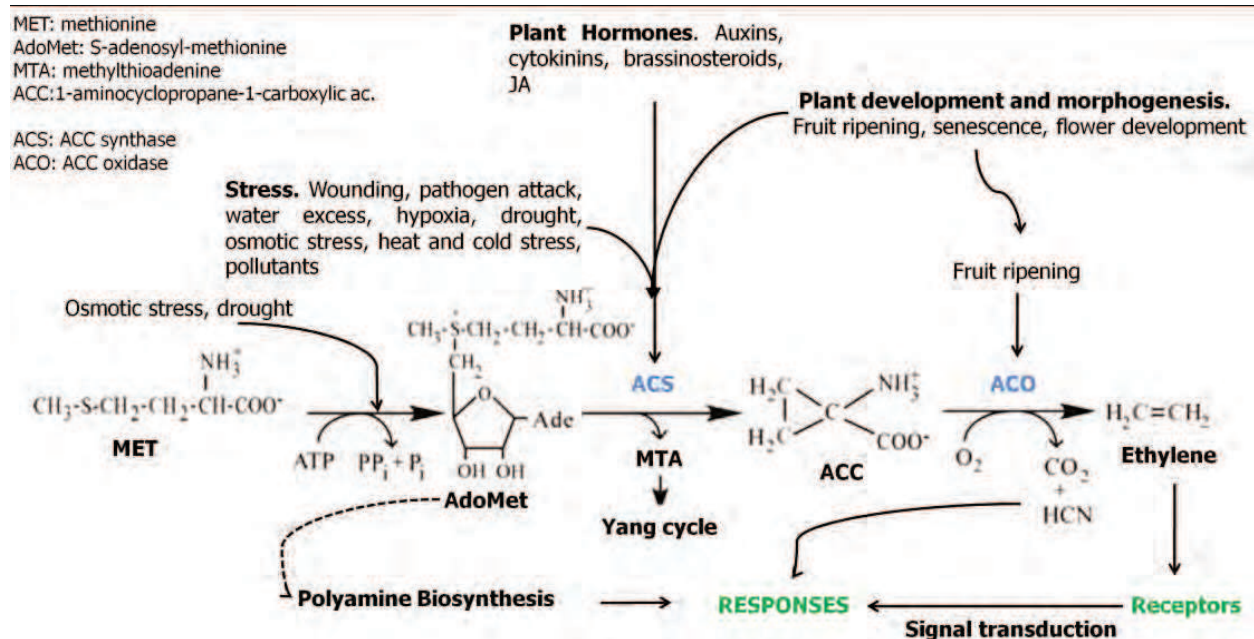


Fig. 3. The ethylene biosynthetic pathway and signalling showing the different enzymes involved in the process. The diverse *stimuli* promoting ET synthesis are also reported. In some plants, not only ACC and ACO are affected, but also AdoMet synthase. Finally, stress ethylene may also relate to stress symptoms through hydrogen cyanide, a by-product of ACC oxidase. Polyamine biosynthesis that start from AdoMet may interact with ethylene biosynthesis and responses to stress (After Argueso et al., 2007)

**Ethylene emission increases under drought stress** (McKeon et al. 1982). In this kind of stress ethylene may act as a signal molecule, but its possible protective role is still unclear. The literature collectively suggests that the production of ethylene in water-stressed plants depends on the rapidity of the decrease of plant water potential, the duration of the stress and the recovery conditions. In addition, plants that have previously experienced mild drought, can be hardened by former stress episodes (Morgan & Drew, 1997). One of the first reactions to water deficit is the stomata closure that is regulated by the hormone abscisic acid (ABA). Water stress induces the root synthesis of ABA which in turn induces ACC synthesis, and conversion of the ACC to ethylene in leaves (Morgan & Drew, 1997). Stomata closure prevent further lack of water from the plant thus avoiding wilting and more severe damages. Ethylene seems to antagonize this effect. In fact, ACC application or the use of the *Arabidopsis* ethylene overproducing mutant *eto1* leads to decreased stomata closure after ABA application, indicating that ethylene inhibits ABA-induced response (Tanaka et al., 2006). In addition, inhibition of ethylene synthesis in wheat inhibits chlorophyll loss associated with drought-induced senescence (Beltrano et al., 1999).

**Ethylene is also induced in osmotic stress**, and, again, its role is not well understood. ACS activity in tomato cells is increased after osmotic shock (Felix et al., 2000). Transgenic tobacco plants overexpressing an ethylene receptor show increased salt sensitivity compared to wild-type plants (Cao et al., 2006). On the other hand, overexpression of the ethylene-responsive transcription factor increased salt tolerance (Huang et al., 2004 - Reviewed by Argueso et al., 2007).

**Drought stress is often accompanied by high temperature which can additionally promote ethylene production.** Very high temperatures often occur at the edge of tree canopy under the sun. The temperature of an individual plant cell can change much more rapidly than other

factors that cause stress (e.g. water levels or salt levels). Thus, plants have evolved strategies for preventing damage caused by rapid changes in temperature and for repairing what damage is unavoidable. **Heat stress results in the production of specific families of proteins known as heat shock proteins (HSPs)** (Howarth & Ougham, 1993). Despite the ubiquitous nature of the heat shock response, little is known about how the plant senses an increase in temperature or the signaling pathways resulting in HSPs. **There is considerable evidence that oxidative stress induces pathways resulting in accumulation of some HSPs** (Dat et al., 1998). Even under optimal conditions, dangerous active oxygen species are synthesized at very high rates from electron transport chains involved in respiration and photosynthesis (Noctor & Foyer, 1998). Once damage is done to the photosystems by extreme temperature and/or light conditions, the production of these potentially damaging molecules increases, and these are the likely cause of the light-dependent, heat-induced oxidative damage (Noctor & Foyer, 1998). Given that high temperature, light excess and drought often occur simultaneously, their damages to the plants are usually cumulative. **High temperature can induce ethylene emission, up to a limit (about 35°C) after which production is inhibited** (Abeles et al, 1992). In *Arabidopsis*, calcium, abscisic acid (ABA), ethylene, and salicylic acid are involved in the protection against heat-induced oxidative damage. In fact, exogenous applications of SA, ACC and ABA protect plants from heat-induced oxidative damage. In addition, the ethylene-insensitive mutant *etr-1*, the ABA-insensitive mutant *abi-1*, and a transgenic line expressing *nahG* (consequently inhibited in SA production) showed increased susceptibility to heat (Larkindale & Knight, 2002).

**Ethylene may also be involved in the regulation of plant responses to low oxygen conditions, or hypoxia.** Hypoxia primarily occurs when the soil is flooded or water logged. Ethylene is highly induced when roots face a low oxygen environment. In these conditions, in *Arabidopsis*, the mRNA levels of a number of genes of the ACS family (i.e. ACS2, ACS6, ACS7, and ACS9) are upregulated (Peng et al., 2005). Under conditions of oxygen deficiency, ethylene is implicated in the triggering of a large number of responses that assist plants in avoiding stress. Among these responses are the accelerated elongation of submerged stems and leaf petioles, promotion of adventitious rooting on stems at or above the water line, leaf epinasty and hypertrophy of lenticels on stems and woody roots. One of the effects of hypoxia is the formation of aerenchymatic tissues. This kind of tissue is produced by programmed cell death that leave empty spaces inside the roots facilitating the gas diffusion. Ethylene may work as a signal regulating programmed cell death and lysigenous aerenchyma formation. A possible explanation reside on a production of cyanide. This toxic compound is produced in the ethylene biosynthetic pathway in stoichiometrically equivalent amounts to ethylene. It has been suggested that HCN may have a role in cell death in a variety of circumstances where ethylene synthesis is strongly stimulated. Induction of aerenchyma formation by ethylene is closely associated with increases in activity of cellulase (He et al, 1994), and also with expression of a putative cell wall metabolism gene, a xyloglucan endo-transglycosylase (XET) (Saab & Sachs 1996). It seems likely that cellulase, XET, and other potentially wall-degrading enzymes contribute to the rapid dissolution of dead cortical cells to give rise to aerenchyma.

**Pollutants such as heavy metals or O<sub>3</sub> also induce ethylene emission.** For example, lithium ion induces ACS gene expression and activity in various plant species (Tsuchisaka & Theologis 2004).

Ozone (O<sub>3</sub>) is presently a major phytotoxic air pollutant, and **exposure of plants to O<sub>3</sub>, or to other pollutants such as SO, or HF, or NO<sub>x</sub>, leads to stress ethylene production.** It has been recognized for many years that there is a broad correlation between stress ethylene formation and sensitivity of different species and varieties to O<sub>3</sub> (see Wang et al, 1990).



Experimental application of high levels of ozone to plants induces a burst of ethylene (Mehlhorn & Wellburn 1987), promoting ozone-induced cell death (Overmyer et al., 2003) through a mechanism that might involve the biosynthesis and accumulation of salicylic acid (SA) (Ogawa et al., 2005) and suppression of the cell-protective action of JA (Tuominen et al., 2004). In *Arabidopsis*, ozone treatment elevates the steady-state level of ACS6 gene, one of the numerous genes of the ACS family (Vahala et al., 1998). Suppression of ACS activity in plants increases tolerance to oxidative stress and diminishes the damage caused by ozone treatment (Sinn et al., 2004).

**Finally, ethylene is stimulated by cold damages.** However, it is still not clear whether additional ethylene production is only a symptom of injury, or it has special significance in relation to acclimation or tolerance of chilling stress. As a generalization, greater stress ethylene production is indicative of greater chilling sensitivity (Morgan & Drew, 1997).

Ethylene seems to have a role as a switch by reducing the production of constitutive defence compounds after herbivore damage and stimulating the production of jasmonic acid and IVOCs (Kahl et al., 2000). Intriguingly, JA has been found to be conjugated to ACC in *Arabidopsis* plants, suggesting that JA-ACC conjugates could be involved in the co-regulation and crosstalk between JA- and ethylene-dependent pathways in plants (Staswick & Tiriyaki 2004). Three endogenous plant signalling molecules: salicylic acid (SA), jasmonic acid (JA) and ethylene regulate plant defences in response to microbial attack (Dong, 1998). There is a growing body of literature that reports that the NO, JA, SA and ET defence signalling pathways do not work independently but rather influence each other through a complex network of regulatory interactions (fig.2).

A greater understanding of the NO, SA, JA and ET signalling pathways and their reciprocal modulation should provide insight into the mechanisms underlying the activation and regulation of plant responses to biotic and abiotic stress. While the SA and JA signalling pathways are mutually antagonistic, several studies provide evidence for positive interactions between the JA and ET signalling pathways. Both JA and ET signalling are required for the expression of the defence-related genes even when applied exogenously. This regulatory cross-talk may have evolved to allow plants to fine-tune the induction of their defences in response to different plant pathogens (reviewed by Kunkel & Brooks, 2002). NO generally counteracts the effects of ethylene by delaying senescence, flowering and ripening (Leshem & Wills, 1998). Methionine adenosyltransferase and S-adenosyl homocysteine hydrolase, required for ethylene biosynthesis, are inactivated by S-nitrosylation and nitration, respectively (Lindermayr *et al*, 2006). It is reported in a wide variety of fruits, both climacteric and non climacteric, that NO emission is higher before ripening, whereas it drops along with the ethylene increase during maturation or senescence; nitrous oxide (N<sub>2</sub>O) fumigations were successfully employed to extend the postharvest life of crops (Leshem & Wills, 1998).

### 3. Nitric oxide (NO)

**Nitrogen monoxide (more commonly named nitric oxide, NO) is one of the chemical signals shared by all the kingdoms**, and it can therefore be involved in interorganism communications. However, NO is also abundantly originated by antropic activities, such as fossil fuel combustion, nitrogen fertilization and it has long been considered only as a pollutant. The formation of photochemical smog involves NO, that drives the synthesis of ozone, either photochemically in presence of O<sub>2</sub>, or by oxidizing volatile compounds (Pinto et al., 2010; Lindroth, 2010). This

process increases the amount of ozone in the troposphere thus affecting the composition of cuticular waxes and volatile emissions and impacting on the regulation of ecological relations (insects and host plants, predators and parasitoids) (Lindroth, 2010).

**Since early 1990s, NO has been characterized as an actively produced physiological regulator in plants.** Nonetheless, its metabolism is not easy to study, because of several, uncommon biochemical features of the NO molecule. In fact, unlike most signalling molecules, NO is a small, ubiquitous, unstable radical gas, freely diffusible both in aqueous and lipidic media. The unpaired electron is delocalized between the N and O atoms in an anti-bond  $\pi$  molecular orbital. This fact grants the NO molecule a relatively high stability, and in the same time explains its typical reactivity. Other compounds biochemically related to NO, and possibly mediating its effects, are collectively named reactive nitrogen species (RNS) and include  $N_2O$ ,  $NO_2$ ,  $N_2O_3$  and peroxyxynitrite.

As a radical molecule, NO is highly reactive to other radicals. Due to its lipophilic nature, NO is supposed to quench effectively the lipid peroxidation chain reaction. The reaction with superoxide ( $O_2^{\cdot-}$ ) is nearly diffusion-limited, and yields peroxyxynitrite ( $ONOO^{\cdot-}$ ). Peroxyxynitrite deserves a mention for its high reactivity and its powerful oxidizing (nitrating) activity toward phenolic compounds, such as tyrosine. In physiological conditions, very little superoxide is produced and not removed by superoxide dismutase: therefore, the occurrence of tyrosine nitration is proposed as a stress marker. Protonation of peroxyxynitrite yields the highly reactive nitrogen dioxide and hydroxyl radicals, further contributing to oxidative stress and disruption of cell structures. An overview of NO chemistry is shown in fig. 4.

NO is produced in plant in a number of possible ways such as the non-enzymatic reduction of  $NO_2^-$  to NO and the dismutation of nitrous acid to NO (Stöhr & Ullrich, 2002; Bethke et al., 2004), the NO synthase-like activity ("NOS-like activity") (del Río et al., 2004) and the enzymatic biosynthesis mediated by nitrate reductase (Yamasaki, 2005). Many other biochemical routes leading to production of NO have been described. Polyamines, salicylhydroxamic acid, hydroxylamine, hydroxyurea and hydroxyarginine were shown to act as NO precursors, possibly in association with reactive oxygen species (Arasimowicz-Jelonek et al., 2009; Rümer et al., 2009; Tun et al., 2006);  $NO_2$  can be photooxidized to NO by carotenoids. Among the enzymatic candidates for NO production, heme-proteins like peroxidases, cytochrome P450, hemoglobin and catalase, have been proposed. Xanthine oxidase, according to  $O_2$  availability, is reported to produce either  $O_2^{\cdot-}$  or NO (del Río et al., 2004).

Considering the role of **reactive oxygen species** in signalling, and how NO reacts with them, **NO can be considered a mediator of ROS signalling.** Plant cell death, for example induced by salicylic acid in the hypersensitive response, requires definite rates between  $H_2O_2$  and NO (Delledonne et al., 2001). If superoxide is not converted to  $H_2O_2$ , peroxyxynitrite will be formed, which has no effect in plant cell death, but may be toxic for an invading pathogen. In general, the causal relations may not be clear, since NO is reported both to promote ROS synthesis (stimulating the plasma membrane NADPH oxidase) (Zhang et al., 2007) and to act downstreams of  $H_2O_2$  (e. g. in ABA stomata closure signal) (Bright et al., 2006).

The effects of  $H_2O_2$  and NO are partially overlapping, for what concerns the regulation of ion channels (notably  $Ca^{2+}$ ), the activation of  $H^+$ :ATPase and  $H^+$ :pyrophosphatase, and second messenger cascade of the phospholipase products.

**Most of the NO-modulated genes in *Arabidopsis thaliana* respond to abiotic or biotic stress conditions, and are involved in signal transduction, cell death, defence,**

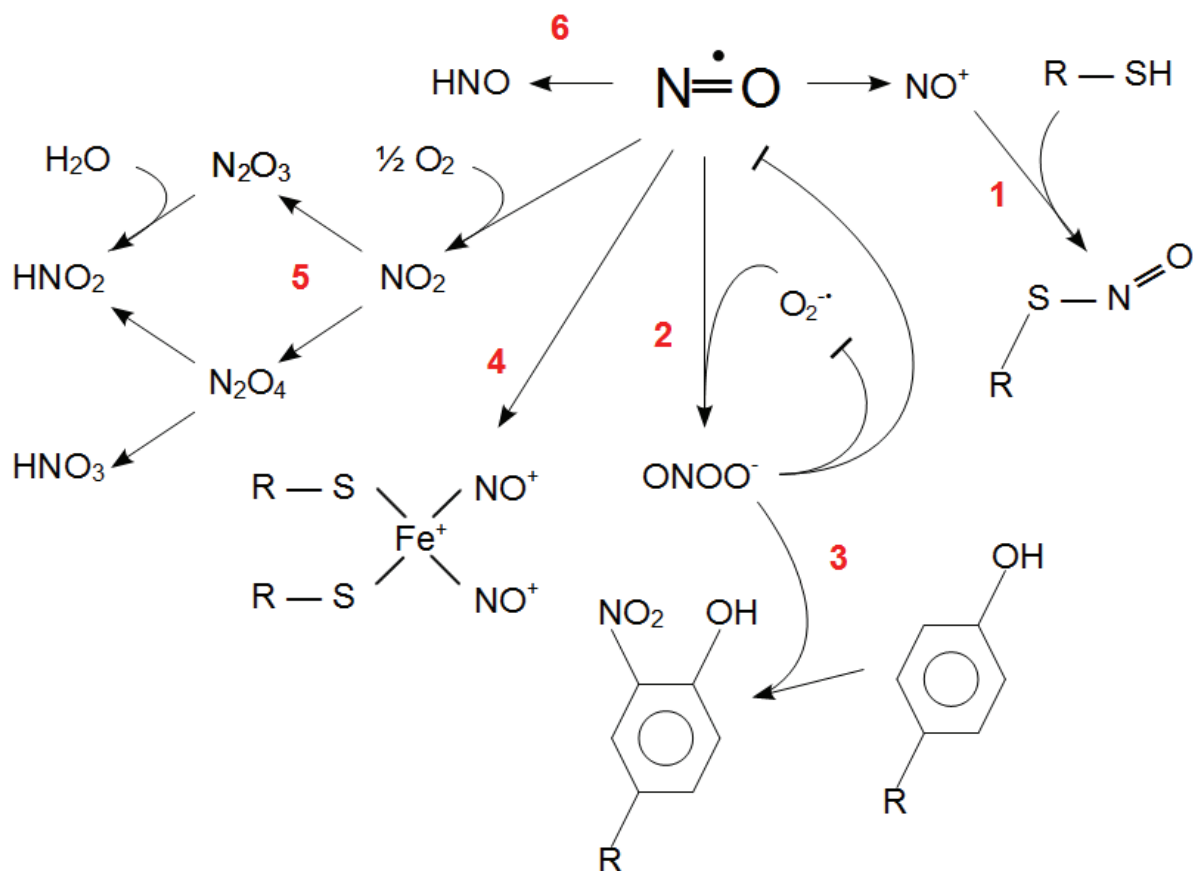


Fig. 4. Chemistry of NO. (1) Nitrosation of thiols (R-SH) by nitrosonium. (2) Production of peroxynitrite. (3) Nitration of a phenolic group. (4) Formation of a dinitrosyl-iron compound. (5) Oxidation reactions to nitric and nitrous acid. (6) Reduction to nitroxy.

**generation or detoxification of ROS, photosynthetic processes, intracellular trafficking, and basic metabolism;** only a few among the classified ones can be directly linked to stress defence, but a dose-dependence to NO emerges for many genes, pointing to a form of signal specificity.

Apart from these functions, similarly to the traditional plant hormones, **NO has also a variety of effects on plant developments and morphogenesis.** For example, NO, acting downstream to auxins, mediates their effects on root architecture, promoting the formation of lateral roots against the elongation of the primary root (Correa-Aragunde et al., 2004) and root hair development (Lombardo et al., 2006). NO acts as a mediator in photoperiod sensing, delaying the floral transition (He et al., 2004). Along with other light-dependent responses, such as de-etiolation and internodal growth, NO affects seed germination overcoming light requirement, similarly to gibberellic acid, and possibly sharing the same cGMP signalling pathway (Beligni & Lamattina, 2000). While ABA is essential for the establishment and maintenance of dormancy, a NO donor renders arabidopsis seeds insensitive to ABA (Bethke et al., 2006).

**NO plays also a crucial role in the abiotic stress signaling and tolerance.** NO function in abiotic stress tolerance may be carried out by a signal cascade, or by direct detoxification. Plants improve their health status in response to many kinds of environmental stress, when exposed to low NO concentrations. Thus, **NO could be considered a generalized stress signal.** To prove this, several works were carried out adopting different species and tissues

and an array of stressing conditions (Corpas et al., 2008; Gould et al., 2003; Huang et al., 2004). As a result, increases in NO contents were observed in some, but not all the treatments, although its occurrence varies among species, and possibly on the basis of tissue-specific mechanisms. For example, **NO has a role in the plant response to drought and salinity stress.** One of the first responses to these stresses is the ABA-mediated stomata closure. A simplified model for the process, not accounting for self-amplification and feedback effects, is the cascade  $H_2O_2 \rightarrow NO \rightarrow cGMP-cADPR \rightarrow$  release of  $Ca^{2+}$ . The  $K^+$ -intaking channels are inactivated by  $Ca^{2+}$ , whereas the outwards  $K^+$  channels open in response to  $H_2O_2$ . As a result, the rise in water potential causes the guard cell to collapse and close the stoma (Bright et al., 2006). Salinity and osmotic stress share a common basis, so it is reasonable that plant adopt similar mechanisms to cope with them, consisting in the regulation of the water loss by transpiration, and of  $Na^+$  uptake, transport and redistribution. Proton concentration provides energy for  $Na^+$  sequestration in the apoplast or in the vacuole, carried out by a  $H^+Na^+$  antiport; NO activates the  $H^+ATPase$  and  $H^+$ :pyrophosphatase activities, in concert with  $H_2O_2$  and  $Ca^{2+}$  (Tanou et al., 2009; Zhang et al., 2006; Zhang et al., 2007; Zhao et al., 2007).

**NO is involved also in the response to hypoxia.** Seeds and roots face oxygen deprivation mainly when the soil is saturated with water. In such conditions, nitrite accumulation and NO production are often reported. NO lowers  $O_2$  consumption by repressing cytochrome C oxidase (COX), and shifts the metabolism toward fermentation, as a mechanism for hypoxic stress avoidance (Borisjuk et al., 2007). A class of plant stress-induced hemoglobins might help the de-repression of COX, or prevent PCD in the short term, allowing the development of adventitious root primordia (Dordas et al., 2003).

**The reaction to the metal ions excess also involves NO.** Metal ions, including heavy metal pollutants, can induce plant stress in several ways, such as the Fenton reaction (in which highly oxidizing ROS are produced), substitution of physiological metal cofactors, and effects on nucleus activity, hormonal signalling and photosynthetic apparatus. The oxidative stress symptoms can be reverted by NO (Laspina et al., 2005), or are associated to a reduced NO content (Rodríguez-Serrano et al., 2006; Tian et al., 2007). **NO might act by chelation of the metal ions, preventing them to react with sensitive targets.**

**Finally, NO has an important function also in the ecological interactions such as symbiosis and pathogenesis.** For example, NO regulated genes are crucial for the establishment of symbiotic root interactions between plants and host-specific bacteria, such as legumes/rhizobia or *Alnus firma*/*Frankia spp.* (Nagata et al., 2008; Perazzolli et al., 2006; Sasakura et al., 2006).

In addition, NO interplays with salicylic acid (SA), ethylene and jasmonate are known to act in the plant responses to invading pathogens. The reaction to avirulent pathogens required NO for the induction of phenylpropanoid metabolism genes, through the cGMP-cADPR pathway (Durner et al., 1998), and of hypersensitive response, together with  $H_2O_2$ . Furthermore, the production of peroxynitrite is suggested as a protective reaction aimed to killing the invader (Delledonne et al., 2001). The recognition of a potential pathogen causes a NO and ROS burst, which stimulates the synthesis of salicylic acid. In turn, SA promotes NO- and ROS-mediated redox signalling, in a positive feedback. Direct consequences of NO are the release of  $Ca^{2+}$  in the cytosol and the activation of protein-kinase cascades (Zottini et al., 2007). The programmed cell death is triggered by definite rates of NO and  $H_2O_2$ , whereas the absence of either one, or other RNS different from NO give no effect on cell viability (Delledonne et al., 2001).

#### 4. Jasmonates

Jasmonates group includes **jasmonic acid** (JA) and its esters, such as **methyl** and **n-propyl jasmonate** (respectively, MJ and PJ). The fragrant compound methyl jasmonate has been mostly studied for its role as a volatile signal in the regulation of pathogenesis-related gene expression (Farmer & Ryan, 1990; Farmer et al., 1992; Schweizer et al., 1997) and up-regulation of VOCs (Martin et al., 2003). **The role of JA in priming plant defences against pests and pathogens and its interactions with other signals and hormones (e.g. ET, SA, NO, IAA, ABA) has been widely studied** (fig. 2). The accumulation of jasmonic acid is caused by wounding, and it is followed by induction of a number of pathogenesis related genes (Schweizer et al., 1998).

Since jasmonates are derived from polyunsaturated C18 fatty acids, the corresponding signalling pathway has been referred to as the octadecanoid pathway (Sembdner & Parthier, 1993) which has been identified as one of the major signalling pathways in plant defence (Farmer & Ryan, 1990).

**The major functions of JA and its various metabolites is regulating plant responses to abiotic and biotic stresses as well as plant growth and development.**

Jasmonates have been shown to stimulate growth and development, including flower development, tuber formation, tendril coiling, nyctinastic movements, trichome formation and senescence, and it inhibits root and leaf growth and seed germination (Onkokesung 2010). Moreover, **JA levels in plants rapidly and transiently increase in response to wounding, water deficit, mechanical stimulation, elicitors and it also mediates some of the UV-induced defense responses** (Xiang 1998).

**Interestingly, the JA-dependent plant defenses which are activated in response to herbivores or pathogens are also triggered by abiotic stresses such as wounding, UV irradiation or O<sub>3</sub>. This observation suggests that the JA signal activates common, unspecific stress-defensive mechanisms. One of this mechanisms is the increase of glutathione (GSH) level** (Xiang 1998). GSH is an essential component of the glutathione-ascorbate cycle, which is the major metabolic pathway responsible for reduction and detoxification of hydrogen peroxide (H<sub>2</sub>O<sub>2</sub>) (Noctor & Foyer, 1998). In addition, oligomers of GSH form the phytochelatin that chelate toxic heavy metals (Ha, 1999). Finally, GSH is used as substrate by glutaredoxins which are oxidoreductases involved in flower development, salicylic acid metabolism and plant defense signaling (Rouhier et al., 2008).

If JA is involved in the tolerance to the oxidative stress, how is this stress perceived and how does it stimulate the JA-mediated reaction? Oxidative species and O<sub>3</sub> react primarily with the plasma membrane, causing alterations in lipid composition and increasing the production of linoleic acid, which is the precursor of JA biosynthesis.

Thus, it is possible that reactive oxygen species (ROS) and, especially, O<sub>3</sub> directly trigger JA biosynthesis and that JA function as a signal in the cascade of plant reaction to oxidative stress-generating stimuli. In addition, JA synthesis and signaling are interlinked by a positive feedback loop whereby jasmonates stimulate their own synthesis (Sasaki et al., 2001; Acosta & Farmer, 2010).

Jasmonic acid is also involved in the response to wounding. JA biosynthesis is initiated by a wound-mediated release of  $\alpha$ -linolenic acid from chloroplastic membranes, followed by the activity of several chloroplast-located enzymes, including 13-lipoxygenase. The combination of JA deficiency and ET insensitivity resulted in a novel growth phenotype characterized by massive cell expansion around wounds, suggesting that both JA and ET may repress local growth after wounding and/or herbivore attack (Onkokesung 2010).

These evidences suggest that both JA and ET, while mediating inducible defenses, may also function as major switches between growth and defenses and the associated changes in resource allocations. Given that wounding and herbivory reduce leaf growth, both cell division and cell expansion could be possible targets of JA and ET action in preventing additional growth, putatively enhancing the accumulation of various defense metabolites used against herbivores. Exogenously applied JA inhibits tobacco BY-2 cell proliferation by arresting the cells in the G1 phase, thus suggesting that the JA and ET effects in primarily on cell division. Increasing endogenous JA levels *in vivo* caused by continuous wounding of *Arabidopsis* plants reduced their growth by suppressing mitosis in the young leaves (Zhang & Turner, 2008).

However, the leaf growth inhibition is also associated with a smaller leaf size as well as reduced fresh mass, but not dry mass, of leaves, suggesting that a decrease in cell expansion is additionally responsible for the observed growth differences (Onkokesung 2010).

**Finally, jasmonates also influence the emission of other stress-related IVOCS.** The biosynthesis and emission of the volatiles is, like many other chemical defenses, under the control of the octadecanoid signalling pathway, and is effectively stimulated by free jasmonic acid (Krumma et al., 1995). Treatment of plants with exogenous JA or MJ has been reported to induce volatile emission similar to herbivore induction, extrafloral nectar production, increased levels of endogenous secondary metabolites, increased VOCs-mediated attraction of predators and parasitoids, and enhanced parasitism rates of herbivores for a wide variety of plant species (Bruinsma et al., 2009).

The role of jasmonate in regulating other VOCs emission is confirmed in the tomato mutant *def-1*, which is deficient in inducing jasmonic acid accumulation upon wounding or herbivory. The application of exogenous JA restored the emission of volatile **methyl salicylate (MeSA)** and volatile terpenes by spider mite-infested *def-1* mutants increased significantly after the plants had been treated with exogenous JA (Ament et al., 2004).

## 5. Isoprene and terpenes<sup>1</sup>

The functional diversity of chemicals within plants is best demonstrated by terpenoids (Theis & Lerdaу, 2003). More than 30,000 terpenoids have been identified (Buckingham 1998). Terpenoids, also known as isoprenoids, are functionally diverse, comprising both primary and secondary metabolites. These compounds include hormones, such as gibberellins and abscisic acid, electron carriers, such as plastoquinone and ubiquinone, terpene-derived compounds that form structural parts of membranes such as phytosterols, photosynthetic and photoprotective pigments such as carotenoids (Theis & Lerdaу, 2003), the phytyl side chain of chlorophyll (Paiva, 2000), toxins, membrane-bound sugar carriers and heat stress resistance compounds (Sharkey & Singaas, 1995). Volatile terpenoids are important compounds for plant biology and atmospheric chemistry because of their role in plant protection (e.g. in the protection of photosynthesis against thermal and oxidative stresses) and in direct and indirect defense against herbivores. Although the majority of volatiles emitted by damaged plants are GLVs, when the plants is intact, the emission of volatile isoprenoids is estimated to account for more than half of

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<sup>1</sup> Terpenes are derived biosynthetically from the polymerization isoprene units, whereas, terpenoids derive from chemical modification (e.g. oxidation, rearrangement) of the basic terpenes structure. In this chapter, a broad definition of terpenes which includes also terpenoids is used.

the total emission of VOCs and is constitutively ten times higher than other emissions (Loreto & Schnitzler, 2010).

All **isoprenoids** are produced from the precursor dimethylallyl diphosphate (DMAPP) and its isomer isopentenyl diphosphate (IPP), which are synthesized by the deoxyxylulose-5-phosphate (DXP) pathway in the chloroplasts and by the mevalonate pathway in the cytoplasm (Lichtenthaler, 1999). Isoprenoids carbon skeletons are composed of five-carbon building blocks that may be assembled in a variety of formations and contain many different modifications. The five-carbon building blocks are added together to make 10-carbon, 15-carbon, and even 2000–500000 carbon chains, as in the case of rubber.

On the other hand, in the case of isoprene (2-methyl 1,3-butadiene), the five-carbon molecules are formed by the elimination of a phosphate group from dimethylallyl pyrophosphate (DMAPP), either in an acid-catalyzed or an enzyme-catalyzed reaction (Harley et al., 1996).

IPP can condense with one, two, or three molecules of DMAPP to form geranyl pyrophosphate (GPP) the 10-carbon precursor of **monoterpenes**, farnesyl pyrophosphate (FPP) the 15-carbon precursor of **sesquiterpenes**, or geranylgeranyl pyrophosphate (GGPP) the precursor of hundreds of **diterpenes**, including the growth hormone **gibberellic acid**. IPP or DMAPP can also serve as the source of the prenyl side of a number of natural products, including **cytokinins** (Paiva, 2000).

Two routes to IPP have been demonstrated in plants: the mevalonate (MVA) pathways and the recently discovered 2-deoxyxylulose 5-phosphate/2-methylerythritol 4-phosphate (MEP) pathway (Rohmer, 1993, Theis & Lerda, 2003; Degenhardt & Lincoln, 2006). The mevalonate pathway appears to function in the cytoplasm, whereas MEP pathway is responsible for most, and probably all, isoprenoids made in plastids (Lichtenthaler et al., 1997; Paiva, 2000; Dudareva et al., 2004), although studies are still in progress to localize the enzymes involved conclusively.

Terpenoids induce the expression of a number of **defence genes** (Arimura et al., 2000). Terpenes also act as band aids by sealing a **plant wound**. Once plant tissue is exposed to the air, the volatile terpene portion evaporates leaving a semi-hardened mass (Phillips & Croteau 1999). Concentrations of terpenoids are generally higher in reproductive structures and typically highest in the foliage during, and immediately following, flowering (Rapparini et al., 2001). Young leaves are more important to a plant than older leaves, and thus the highest levels of terpenoids are found in the young organs (Fischbach et al., 2002).

Finally, **terpenes emission is strongly increased by temperature increase**. This effect is due to the immediate stimulation of temperature on the activity of the enzymes that catalyze the synthesis of many VOCs (Loreto & Schnitzler, 2010).

## 6. Monoterpenes

Monoterpenes include many volatile flavours and aroma components (Sharkey & Yeh, 2001). Some compounds play a major role in plant defence being involved in plant-insect interactions as toxins and deterrents. Representative monoterpenes include the mildly antimicrobial menthol, aroma components citral and geraniol, insect repellent citronellal, parasitoid wasp attractant linalool, and allelopathic camphor (Paiva, 2000). Monoterpenes non-storing species synthesize and emit these compounds in a light, temperature and damage-dependent way. Whereas, the storing species have a special structure for monoterpenes accumulation such as glandular cells on the leaf surface (Paiva, 2000;

Holopainen, 2004). In these species, emission of monoterpenes generally originates from pools of hydrocarbon stored in resin ducts, glands, or trichomes (Loreto et al., 1996). Monoterpenes emission is therefore dependent in large measure on their volatility (Lerdau, 1991) and on damage of leaves (Tingey et al., 1980; Tingey et al., 1991; Litvak et al., 1998). Only in a few cases, trees emit some monoterpenes as a result of *de novo* synthesis (Sharkey & Yeh, 2001): for example linalool is synthesized after herbivore damage and released systemically from the whole plant (Holopainen, 2004).

Monoterpenes, such as eucalyptol, linalool, camphor,  $\alpha$ -pinene,  $\beta$ -pinene,  $\alpha$ -terpineol, borneol and many others, are the principal components of plant volatile oils (Dorman & Deans, 2000, Candan et al., 2003). These are principally present in the aromatic plants (storing species). **Several works observed that volatile essential oils are involved in antimicrobial and antioxidant activity** (Candan et al 2003; Cimanga et al., 2002ab; Kuhnt et al., 1995; Setzer et al., 1999; Dorman & Deans, 2000).

Many SESQUITERPENES are typical fragrances emitted from flowers (Chen et al., 2003). A considerable amount of sesquiterpenes is also emitted from the herbivore-damaged foliage (Vuorinen et al., 2004), whereas in intact plants, the emission is lower (Wiens et al., 1991). **High levels of sesquiterpenes are produced after O<sub>3</sub> exposure.** The speed of sesquiterpenes release is related to the resistance of the plant thus demonstrating their role in O<sub>3</sub>-defence (Heiden et al., 1999). Sesquiterpenes are among the most studied class of **phytoalexins**. In the case of the phytoalexins and other toxins, the plant product is not only toxic to the microbial pathogen, but also potentially toxic to the plant cells. Many of the phytoalexin biosynthetic genes are not expressed until the plant senses the presence of the pathogen, when complex signal transduction mechanisms activate transcription of each of the biosynthetic steps (Paiva, 2000).

**ISOPRENE** (C<sub>5</sub>H<sub>8</sub>, 2-methyl 1,3-butadiene) is a natural product of many organisms (Sharkey, 1996). Sanadze and Kursunov discovered isoprene emission from plants in the 1950s (Sanadze & Kursanov, 1966).

**The global isoprene emission is now estimated to be about 500 Tg C yr<sup>-1</sup>, making it the dominant hydrocarbon that moves from plants to the air, roughly equal to the flux of methane to the atmosphere** (Guenther et al., 1995; Wang & Shallcross, 2000). Isoprene drains a considerable percentage of the carbon fixed through photosynthesis out of the pathway forming structural and storage sugars, especially in stressed leaves: in major emitting plant species such as oak and aspen its synthesis is typically 2% of photosynthesis at 30°C (Sharkey & Yeh, 2001).

Isoprene is a hemiterpene produced from DMAPP by the isoprene synthase (Silver & Fall, 1991). This enzyme has a relatively high pH optimum and a requirement for Mg<sup>2+</sup> (Schnitzler et al., 1996; Silver & Fall, 1995), consistent with its location inside chloroplasts (Mgaloblishvili et al., 1979; Wildermuth & Fall, 1996; Wildermuth & Fall, 1998). The energy cost of isoprene emission using the MVA pathway is 9 carbon atoms, 24 ATP, and 14 NADPH. However, the MEP pathway is more efficient than the MVA pathway in photosynthetic organisms, and isoprene emission based on the MEP pathway costs only 6 carbon atoms, 20 ATP, and 14 NADPH.

Therefore, the cost of isoprene production is substantial, and any benefits ascribed to its emission will have to be weighed against this cost in terms of carbon and energy. Isoprene emission is a common but not universal plant trait (Kesselmeier & Staudt, 1999). Nearly all plant species emit very low levels of isoprene, but only about one third of angiosperm



species tested emit isoprene at substantial rates (Hanson et al., 1999). Given the cost, plants that do not emit would out-compete those that do emit, unless isoprene emission provides a benefit that exceeds the cost of emission. What benefit, if any, do plants derive from isoprene emission? In other words, why do plants make isoprene? Some of the reasons suggested for isoprene emission include a leaf thermoprotector, a flowering hormone (Terry et al., 1995), an antioxidant (Zeidler et al., 1997), and a metabolite overflow to get rid of excess carbon (Logan et al., 2000; Wagner et al., 1999).

Although the isoprenoids are very well known, much less is known about biological isoprene production because isoprene is not an intermediate in isoprenoid production. Unlike monoterpenes, it is not stored within the leaf, but emitted through the stomata immediately upon its production. Thus, isoprene emission requires *de novo* synthesis (Fall & Monson, 1992; Loreto et al., 1998). For this reason, in plants, **isoprene synthesis is dependent on photosynthesis**. Fall and Wildermuth (1998) reported that changes in pH and  $Mg^{2+}$  that normally occur in thylakoids in response to light can cause an 11-fold stimulation in isoprene synthase activity. Therefore, the factors affecting photosynthesis, such as nitrogen deficiency, water stress, light and temperature excesses usually influence also isoprene emission.

Isoprene emission is very sensitive to temperature, although the temperature dependence of isoprene emission is different from that of photosynthesis. In *Quercus spp.*, an increase of temperature from  $\pm 30\text{ }^{\circ}\text{C}$  to  $\pm 40\text{ }^{\circ}\text{C}$  does not affect photosynthesis or slightly reduce it, whilst, it increases isoprene emission from typically 2% to 15% of carbon fixed by photosynthesis (Sharkey et al., 1996). On the contrary, photosynthesis is highly sensitive to  $\text{CO}_2$  but isoprene is relatively insensitive. Isoprene emission in  $\text{CO}_2$ -free air can be 50% of maximal rates; but if oxygen is also removed, isoprene emission stops. The interpretation of this phenomenon is that isoprene emission requires photosynthetic activity (Loreto & Sharkey, 1990).

**Isoprene emission can be also affected by nitrogen nutrition** (Harley et al., 1994; Litvak et al., 1996). Trees with low nitrogen availability had lower rates of isoprene emission than did trees with higher nitrogen nutrition. This effect interacted with light. Trees grown in sun or shade but with low nitrogen availability had similar, low rates of isoprene emission; but trees with high nitrogen availability emitted substantially more isoprene when grown in the sun than when grown in the shade. **On the other hand, isoprene emission seem to be only marginally effected by water-stress**. Drought directly affects stomatal conductance and produce diffusive and biochemical limitations of photosynthesis (Fall et al., 1999). Both the reduction of photosynthesis and the stomatal closure are expected to negatively impact on biogenic volatile organic compounds emission by altering the carbon supply into the MEP pathway and by increasing resistance to their emission (Fall. et al., 1999). However, isoprene emission changed little, even when photosynthesis is inactivated by a total and prolonged stomata closure (Tingey et al., 1981). In addition, upon rewatering, isoprene emission increased several fold above the pre-stress rate and stayed high for several weeks (Sharkey & Loreto, 1993). Nonetheless, the effect of drought, like other stressors on plant VOC emissions, can depend on the level of stress or damage caused to the plant by drought. Thus, severe drought might largely decrease emissions, whereas mild drought stress might increase emissions (reviewed by Peñuelas & Staudt, 2010). Similarly to isoprene, also terpenes and terpenoids are negatively affected by severe drought (Lerdau et al., 1994).

The role of isoprene in **thermotolerance** has been extensively studied. Since the 90s, several experiments indicated that isoprene has some effect on the temperature tolerance of the

photosynthesis (Sharkey & Singsaas, 1995; Seemann et al., 1984; Singsaas et al., 1997). Isoprene resulted to have a role mainly in the protection against short high-temperature episodes (Singsaas & Sharkey, 1998). As leaves can be subjected to dozens of high-temperature episodes each day, the increased recovery from each episode allowed by isoprene could become very important to the plant (Singsaas & Sharkey, 1998). The mechanism underlying the increased thermotolerance is still partially unknown and it has been attributed to the stabilization of membrane lipid bilayer by enhancing the hydrophobic interactions (Gounaris et al., 1984). In fact, the lipid membranes are particularly sensitive to exposure to high temperatures and often they get damaged or denatured unless protection mechanisms occur. Isoprene has been supposed to stabilize exclusively the thylakoids membranes of chloroplast in which it is formed (Sharkey, 1996; Sharkey & Yeh, 2001). However, no enhancement of stabilization by isoprene has been observed by using artificial membranes (Logan et al., 1999). Monoterpenes emitted in a light-dependent manner also provide this type of thermoprotection. Repeated cycles of high-temperature stress reduced the recovery in leaves without isoprene or monoterpene, although leaves with isoprene or monoterpene maintain high rates of photosynthesis, especially after repeated periods of high temperature (Loreto et al., 1998; Sharkey, 1996).

The thermotolerance hypothesis largely explains what plants may gain from isoprene emission, and the effect can be large compared to the cost of emission. This hypothesis suggests that plants suffering short high-temperature episodes, but not long term, constant high-temperature exposure, should emit isoprene. Trees often fit this description: the leaves at the tops of the canopy are exposed to full sun and they heat substantially up if the air is still (Ehleringer, 1991). On the other hand, desert plants, which are adapted to prolonged high temperatures, generally, do not emit isoprene. In fact, in desert plants, the leaves are absent or very small leaves so that the boundary layer is small and the leaves cannot heat up much above air temperature. A common mechanism to reduce the leaf temperature is transpiration that is regulated by stomata opening and air relative humidity. However, where the air relative humidity is very high, such as in humid tropical environments, transpiration is highly reduced and other mechanisms should account for the thermoprotection of leaves. In fact, tropical plants usually emit relatively more isoprene than most of the plants in temperate or cool climates (Keller & Lerda, 1999).

Hanson et al. (1999) speculated that **isoprene emission might have been an important step in the evolution of land plants**. As plant progenitors started to stand up in the air, the low heat capacity of air caused plant temperatures to vary in a greater range than when the organisms were in water. Thus, the isoprene emission may have played a role in land colonization by the first terrestrial plants such as *Bryophyta*. Isoprene emission is, in fact, common in mosses but absent in algae (Hanson et al., 1999, reviewed by Sharkey & Yeh, 2001).

**Isoprene may play an important role also as antioxidant in leaves.** This idea is normally put forward on the basis of the rapid reaction of isoprene with ozone and hydroxyl radicals. Isoprene can dramatically reduce the damage caused by acute and short (3 h, 300 nL L<sup>-1</sup>) or relatively low and long (8h, 100 nL L<sup>-1</sup>) ozone treatments in leaves (Loreto et al., 2001; Loreto & Velikova, 2001). This effect may be perhaps related also to the membrane strengthening action of this compound. However, isoprene may also effectively react with ozone forming hydroxymethyl hydroperoxide and aggravating the ozone induced damage (Salter & Hewitt, 1992).

**Isoprene emission is a sensitive indicator of wound signals that can travel through plants** (Loreto & Sharkey, 1993). In a number of studies, wounding was inflicted by puncturing,

smashing, cutting and burning leaves. By wounding one leaf while monitoring isoprene emission from a different leaf, researchers could show the transmission of a signal. The time between wounding one leaf and a change in rate of isoprene emission of a different leaf was linearly related to the distance between the two leaves, allowing a calculation of travel rate of the signal. The signal travelled about  $2 \text{ mm s}^{-1}$ , which is likely to result from electrical signals travelling through the plant. The calcium chelator EGTA substantially delayed the wound signal effect on isoprene emission, indicating that the electrical signal may have caused calcium fluxes that ultimately affected isoprene emission.

## 7. Conclusions

The development and survival of all living things relies on the ability of organisms to perceive and respond to their environment. Responses to internal and external signals are frequently elicited by hormones, promoting changes in morphology to accommodate an ever-changing habitat. Figure 5 summarizes the VOCs emitted in response to different biotic and abiotic stresses.

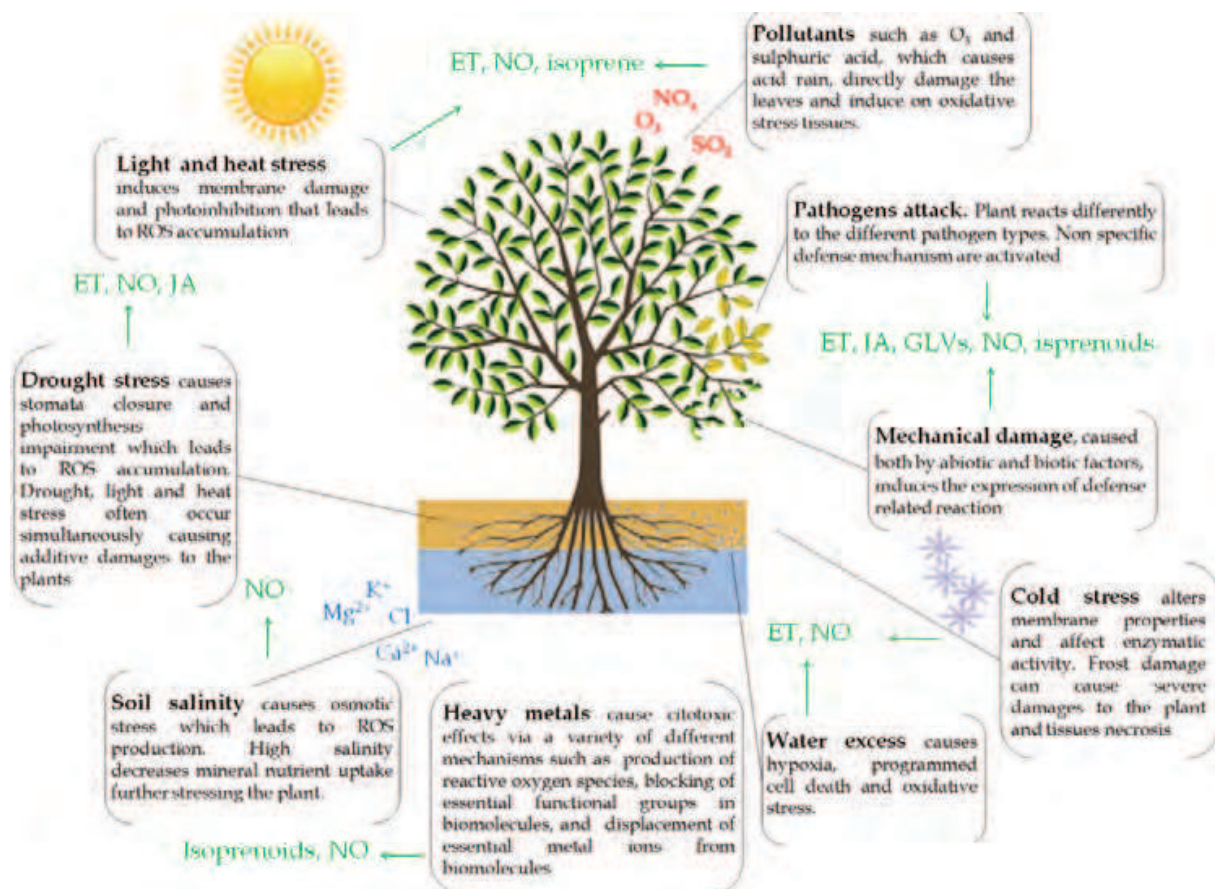


Fig. 5. All the abiotic stresses affecting plant trigger complexes responses aiming to increase the stress tolerance. The responses include the emission of VOCs. The VOCs produced in response to the different stresses are reported in green. A common mechanism links together the different stresses: all causes oxidative stress and hamper the production of reactive oxygen species (see also fig. 2). Excess light and heat, as well as exposure to oxidizing air pollutants, cause direct accumulation of ROS which crucially contributes to for initiate the stress-related signal cascades (see Fig. 2) - (After Vickers et al., 2009)

All kinds of stresses induce in the plants the production of ROS which will result in oxidative stress. Therefore, the complex network of reactions protecting the plant from stress is strictly linked with the response to oxidative stress. Changes in volatile emission under stress conditions supply provide evidence that VOCs are linked with the plant responses to stress. Their emissions often increase under abiotic stress conditions, particularly under leaf damage, water, heat and light stress (Vickers et al., 2009). Some of the volatiles, such as ethylene and NO, are primarily acting as stress messenger that allows the plant to trigger the stress induced defenses. On the other hand, other volatiles, such as isoprene and isoprenoids, and NO as well, play an important role in a direct protection against several of these stresses. The common mechanism underlying the protective effect of these VOCs is their general antioxidant role (Calfapietra et al., 2009).

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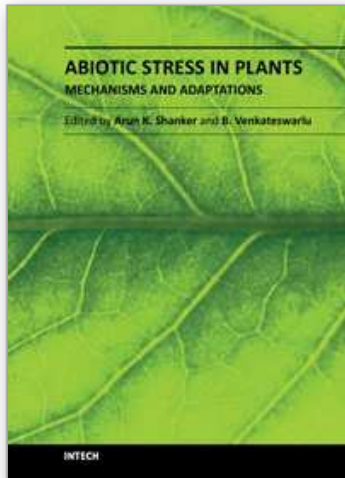
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## **Abiotic Stress in Plants - Mechanisms and Adaptations**

Edited by Prof. Arun Shanker

ISBN 978-953-307-394-1

Hard cover, 428 pages

**Publisher** InTech

**Published online** 22, September, 2011

**Published in print edition** September, 2011

World population is growing at an alarming rate and is anticipated to reach about six billion by the end of year 2050. On the other hand, agricultural productivity is not increasing at a required rate to keep up with the food demand. The reasons for this are water shortages, depleting soil fertility and mainly various abiotic stresses. The fast pace at which developments and novel findings that are recently taking place in the cutting edge areas of molecular biology and basic genetics, have reinforced and augmented the efficiency of science outputs in dealing with plant abiotic stresses. In depth understanding of the stresses and their effects on plants is of paramount importance to evolve effective strategies to counter them. This book is broadly divided into sections on the stresses, their mechanisms and tolerance, genetics and adaptation, and focuses on the mechanic aspects in addition to touching some adaptation features. The chief objective of the book hence is to deliver state of the art information for comprehending the nature of abiotic stress in plants. We attempted here to present a judicious mixture of outlooks in order to interest workers in all areas of plant sciences.

### **How to reference**

In order to correctly reference this scholarly work, feel free to copy and paste the following:

Francesco Spinelli, Antonio Cellini, Livia Marchetti, Karthik Mudigere Nagesh and Chiara Piovene (2011). Emission and Function of Volatile Organic Compounds in Response to Abiotic Stress, *Abiotic Stress in Plants - Mechanisms and Adaptations*, Prof. Arun Shanker (Ed.), ISBN: 978-953-307-394-1, InTech, Available from: <http://www.intechopen.com/books/abiotic-stress-in-plants-mechanisms-and-adaptations/emission-and-function-of-volatile-organic-compounds-in-response-to-abiotic-stress>

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