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REVIEW PAPER

Deciphering the ozone-induced changes in cellular processes: a prerequisite for ozone risk assessment at the tree and forest levels

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Abstract

• ***Key message*** **Ozone, one of the major atmospheric pollutants, alters tree growth, mainly by decreasing carbon assimilation and allocation to stems and roots. To date, the mechanisms of O_3 impact at the cellular level have been investigated mainly on young trees grown in controlled or semi-controlled conditions. In the context of climate change, it is necessary to introduce a valuable defence parameter in the models that currently predict O_3 impact on mature trees and the carbon sequestration capacity of forest ecosystems.**

• ***Context*** Air pollution is an important factor that affects negatively forest ecosystems. Among oxidative air pollutants, ozone is considered as the most toxic in terms of impact on vegetation.

• ***Aims*** This paper focuses on the negative impacts of ozone on trees in controlled conditions or in their natural environment. The current knowledge of the responses at cell level is presented and ways to improve their use for ozone risk assessment of forest stands are discussed.

• ***Methods*** Information was collected from original papers or reviews, providing an overview of the research conducted over the last 60 years.

• ***Results*** The negative effects of ozone on carbon assimilation and tree biomass production were reviewed and discussed, with a focus on effects on cell processes implied in cell defence, including stomatal regulation, detoxification, signalling, and biosynthesis of wood compound.

• ***Conclusion*** In the context of increasing significance of O_3 flux approach, this review intends to shed light into the black box of defence processes, which are playing a crucial part within the effective O_3 dose modelling. Today, it is recognized that tropospheric ozone inhibits tree growth and its role on the future carbon sink of the forest ecosystem is discussed along with the combination of other environmental factors like elevated temperature,

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water, and nitrogen supply, likely to be modified in the context of climate change.

Keywords Ozone impact · Defence · Carbon assimilation · Carbon allocation · Stomata · Detoxification · Signalling · Cell wall

1 Introduction

As a result of human activities, Earth climate is assumed to change (IPCC 2013) as we enter a new geologic era, the Anthropocene (Barnosky et al. 2012; Steffen et al. 2011). It is now well established that the release of greenhouse gases (GHGs) affects climate on a global scale, since these gases modify radiative transfer and thus change the Earth's energy balance (IPCC 2013). Tropospheric ozone (O_3), one of these GHGs and an important component of air pollution, is predicted to spread over large parts of the globe in the coming decades (Dentener et al. 2005; Fig. 1). In addition, this pollutant is thought to impact negatively forest productivity (Ainsworth et al. 2012), although species composition can modulate this effect (Wang et al. 2016). Since a large part of global forest areas is predicted to be exposed to O_3 in the future (Fowler et al. 1999), carbon sequestration by forests may be reduced (Sitch et al. 2007; Subramanian et al. 2015). Historically, the phytotoxic effect of photooxidants, including O_3, was first discovered in the 1950s in mixed conifer forests from the Los Angeles basin (Haagen-Smit et al. 1952). High concentrations of tropospheric O_3 are an urban problem linked to car traffic and NO_x formation, but O_3 or its precursors are easily airborne and the pollutant can damage forest trees far from the source of emission. In the 1980s, several research groups showed that visible symptoms of injury on tree leaves in different regions of the USA were clearly related to the effect of photooxidants (Miller et al. 1997; Skelly et al. 1997). In Europe, in the middle of the 1980s, the German foresters were the first to draw attention to visible damages observed on coniferous trees, incriminating air pollution (Krause et al. 1986). The same observation was also made in Eastern France, leading to the development of a bilateral cooperation for exploring the causes of this problem between the French DEFORPA programme (1984–1991) and the German partners, followed with the common EUREKA programme EUROSILVA (1992–1994). In the 1990s, extended European cooperation (eight countries) started on the effects of ground-level O_3 on trees and on the reduction of air pollutants, linked to the European Framework Programme for Research (e.g. STEP) and within the United Nations Economic Commission for Europe (UNECE).

The first experiments operated by these research programmes were conducted on trees in the field, e.g. in Germany (Weidmann et al. 1990) and in Austria (Wieser and Havranek 1993). Subsequently, experiments in controlled conditions (open-top chambers and phytotronic chambers) were set up to decipher the mechanisms of O_3 impact on leaves of young trees (Gerosa et al. 2009; Sandermann et al. 1997). The trees display a series of defence responses to O_3 which, when overwhelmed, leads to different types of damages including leaf necrosis and growth reduction. The obtained results proved that, before visible symptoms appear, O_3 affects the leaf metabolism by damaging the photosynthetic machinery and by increasing carbon use, further

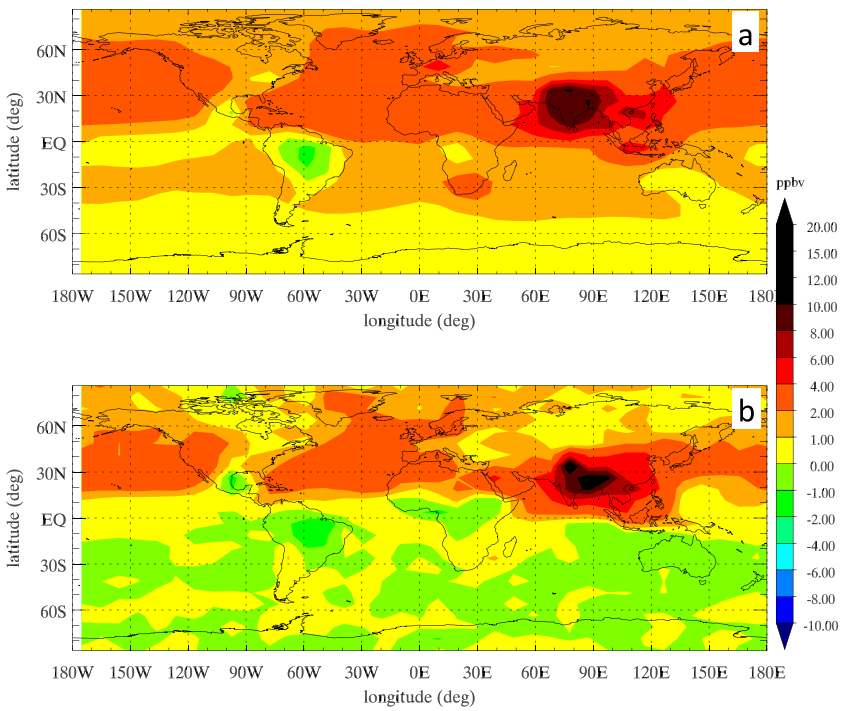

Fig. 1 Predicted differences in decadal averaged surface O_3 concentrations (ppbv) comparing the 2020s and the 1990s for two global chemistry-transport models (from Dentener et al. 2005). **a** TM3 CLE Eulerian global chemistry-transport model using the current legislation (CLE) scenario; **b** STOCHEM CLE Lagrangian tropospheric chemistry-transport model using the current legislation (CLE) scenario

leading to reduced growth and productivity (Dizengremel 2001; Heath and Taylor 1997). The development of free-air CO_2 enrichment (FACE) projects provided a nice opportunity to study forest ecosystem responses to the increase of tropospheric O_3 combined or not with high CO_2 exposure (King et al. 2005). The gap between the two approaches (field versus controlled conditions) has recently been partly reduced through the German CASIROZ programme by developing a large set of ecophysiological and biochemical analyses on mature beech and spruce trees submitted to free-air O_3 fumigation (Matyssek et al. 2010b), on birches in Finland (Oksanen et al. 2007) and on conifers in Austria (Wieser et al. 2013). However, the degree of sensitivity to ozone is highly variable between tree species (Reich 1987; Wittig et al. 2009), raising the question of the underlying physiological processes (Matyssek et al. 2012).

In 2000, to meet the request from policy makers to the scientific community for quantitative information about O_3 effects, a specific European programme for the assessment, validation and mapping of visible O_3 injury on the vegetation was set up, based on the ICP Forests monitoring network. The concept of a critical level for O_3, introduced by UNECE (LRTAP convention 2004), was originally based on the accumulated exposure over a threshold concentration of 40 ppb (AOT40). For forest trees, exceeding an AOT40 value of 5 ppm.h accumulated over one growing season would cause growth reduction (LRTAP convention 2004). More recently, a flux-based concept was developed in order to take into account the actual O_3 flux in the leaf through the stomata (Emberson et al. 2000; Grünhage et al. 2004; Karlsson et al. 2004). Indicators such as AFstY (*a*ccumulated stomatal *f*lux above a threshold of *Y* nmol m^{-2} s^{-1}) or POD_Y (*p*hytotoxic *o*zone *d*ose above a threshold flux of *Y* nmol m^{-2} s^{-1}) appear more representative of the impact of O_3 on vegetation as, being based on the Jarvis multiplicative model of stomatal conductance (Jarvis 1976), they take into account temperature, water vapor pressure deficit (VPD), light, soil water potential, concentration of O_3 and the plant phenology. These indices simulate the uptake of O_3 in leaves and represent the hourly average flux accumulated above a threshold *Y* by a leaf during the plant growth. Hoshika et al. (2014) also proposed to take into account a stomatal O_3 flux per net photosynthesis rate rather than stomatal O_3 flux only. All these flux-based methodologies rely on empirically derived relationships, linking stomatal O_3 flux to tree biomass loss through a series of models at leaf, tree and forest levels. These relationships thus provide estimates of the effective O_3 dose, i.e. the fraction of O_3 flux exceeding the plant detoxification capacity, under consideration of the environmental conditions (Matyssek et al. 2004; Musselmann et al. 2006; Dizengremel et al. 2008; Heath et al. 2009; Buker et al. 2015). The importance of the detoxification capacity of the tree was recently emphasized (de Temmerman et al. 2002; Dizengremel et al. 2013; Dizengremel et al. 2008) and was previously raised to explain the differences in O_3 sensitivity of tree species (Pell et al. 1999). Detoxification capacity depends on a network of molecular and physiological processes which needs to be deciphered in order to identify a reliable parameter, integrated in models and allowing a more accurate risk assessment. In brief, the integration in field models of a pertinent ozone-damaging factor, identified at the cellular level, could improve risk assessment and help policy makers with related socio-economic decisions.

Since 60 years, O_3 research has covered a large spectrum of interest, including detailed studies of the cellular events induced by acute doses. In this review, acute doses correspond to observed peak concentrations with values above 120 ppb for several days, while chronic exposure refers to long-term exposures (weeks, months, years) to ozone concentrations below 120 ppb. The increasing interest to study the effects of this gas is partly due to the fact that the exposure to high O_3 concentrations elicits a strong oxidative stress at the tissue level. Moreover, with hourly peak O_3 concentrations at peri-urban regions reaching 200 ppb (Feng et al. 2014), acute conditions are de facto validated in natural conditions inducing serious damage to plants. O_3 research has also known a progressive extent in the study of chronic exposure with values not higher than 100 ppb over the growth period. However, experiments in controlled conditions with slightly higher O_3 exposure (up to 120 ppb) applied every day during a shorter time (no more longer than 3 weeks/1 month) were also frequently used to mimic the effect of these ambient long-lasting O_3 exposures. Even though the increase in tropospheric O_3 concentrations has recently flattened in mid-latitudes of the Northern Hemisphere (Oltmans et al. 2013), assessing the effects of O_3 on forest trees remains a timely question considering that the threshold for a negative impact on growth has been already reached. In this context, we present a survey of works on the effects of this oxidative pollutant in a relative short term on tree physiology (Fig. 2). These information are necessary to extrapolate on a longer term the O_3 effect at the forest ecosystem scale, which have also to integrate with the complex inter-relationships among the environmental factors occurring over the life span of a tree.

2 Formation, transport, deposition of O_3, and leaf damage

2.1 Formation

In the troposphere, under the action of sunlight, primary pollutants like nitrogen oxides and hydrocarbons are able to form photochemical air pollutants, referred to as secondary pollutants, namely peroxyacetyl nitrate (PAN), O_3 and to a lower extent aldehydes and ketones (Becker et al. 1985). The future of PAN is closely related to atmospheric

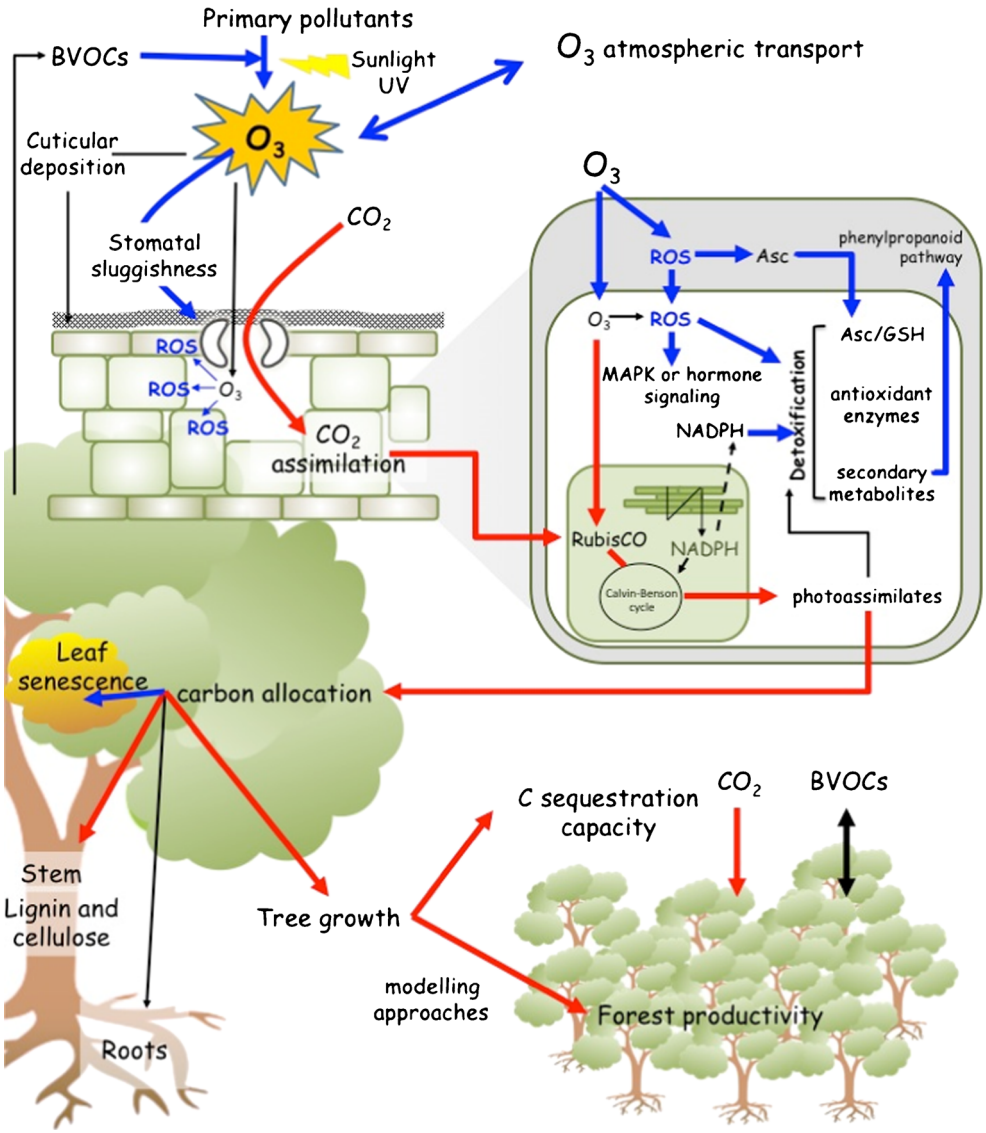

Fig. 2 Overview of O_3 effects on trees, from cell metabolism to forest ecosystem scale, highlighting (i) the perception of the pollutant at the leaf scale, (ii) the cellular responses implying detoxification and CO_2 assimilation and (iii) the carbon allocation to the various plant organs and the consequences on tree growth and on carbon sequestration at the forest level. Where *arrows* are present, *red* and *blue* indicate an O_3-driven inhibition and stimulation, respectively. *BVOCs* biogenic VOCs

temperatures and could contribute to O_3 formation in the warmer lower atmosphere (Singh 1987). The presence of O_3 in the troposphere was initially considered as the result of a stratospheric O_3 transfer, which only accounts for up to 25 % of tropospheric O_3 (LRTAP convention 2010). In fact, O_3 formation mainly results from complex processes already detailed in several reviews (Becker et al. 1985; Royal Society 2008; Stockwell et al. 1997). Briefly, once nitrogen dioxide (NO_2) is formed, it endures a photodissociation caused by short radiations (between 280 and 430 nm) producing nitrogen monoxide (NO) and free oxygen atoms. The free oxygen atom presents a high excitation level leading to a reaction with O_2 to form O_3. The subsequent reaction of O_3 with NO can lead to the destruction of O_3 in a non-polluted area, where the NO_2/NO ratio is low. However, one parameter susceptible to unbalance these reactions is the presence of volatile organic compounds (VOCs) including CH_4 (Royal Society 2008). VOCs are able to oxidize NO, increasing the NO_2/NO ratio and shifting the reactions towards O_3 accumulation. Finally, the production of O_3 in the troposphere is linked to changing precursor concentrations, a relationship that highlights the non-linearity of the O_3-VOC-NO_x system (Monks et al. 2015). Vegetation and particularly forests are natural VOC producers (Sharkey et al. 2008). In a context of warming climate, VOC emissions are projected to increase and to contribute to the occurrence of O_3 peaks or the increase of the tropospheric O_3 background level. Finally, the photolysis of O_3 leads to additional radicals that can react with carbon monoxide and organic species, leading to additional O_3 production (Royal Society 2008).

2.2 Transport of the pollutants in the troposphere

Most of air pollutant emissions, including O_3 precursors, originate from regions within the mid-latitudes, where long-range

transport of air masses is dominated by westerly winds. These winds convey emissions from source regions to downwind regions at interregional and even intercontinental scales (Stohl and Eckhardt 2004). The lifetime of O_3 in the free troposphere varies from weeks to months, which is compatible with long-range transport that occurs on timescales of days to weeks (LRTAP convention 2010). Due to the stronger winds at high altitudes, O_3 formed or transported into the mid- and upper troposphere travels further and faster than O_3 remaining in the lower troposphere, below 3 km in altitude (LRTAP convention 2010). O_3 formation can also occur at distance from precursor source regions, when polluted air masses arrive at a downwind region and meet conditions that promote O_3 formation (Lin et al. 2012).

When confined within the atmospheric boundary layer, O_3 has a relatively short lifetime (hours to days) due to dry deposition at terrestrial surfaces (Wesely and Hicks 2000). Dry deposition occurs when O_3 is taken up or absorbed onto surfaces (vegetation, soil, materials) that provide a chemical sink for O_3 decomposition (Cape et al. 2009). Surface removal represents an important control on the near-surface O_3 concentrations and constitutes a major term in the global mass balance of tropospheric O_3 (Fowler et al. 2009). While molecular processes become important very close to surfaces (less than 1 mm), turbulent transfer represents the main driver of gas exchange between vegetation and the atmosphere. Forests being aerodynamically rough surfaces, the rates of turbulent exchange between the atmosphere and forests exceed by an order of magnitude or more those over crops or grasslands (Fowler et al. 1999). As a consequence, forests represent a major sink for O_3 dry deposition.

2.3 O_3 deposition to soil and vegetation

The foliage of forest trees acts as the dominant O_3 sink in the atmosphere-forest interaction, and the canopy structure has a noticeable effect on its uptake (Zhang et al. 2006). Forest ecosystems have the capacity to remove O_3, through both stomatal and non-stomatal mechanisms (Fig. 2) (Dizengremel et al. 2013; Fares et al. 2013b). The stomata are the main entry point of ozone into the leaves, and the non-stomatal mechanisms of ozone deposition include cuticular deposition, deposition at the soil surface and destruction by chemical reactions (NO_x, biogenic VOC). Most studies of vertical O_3 concentration gradients show that only minor variations occur throughout forest canopies during daytime, mainly due to convective mixing caused by solar radiation (Jaggi et al. 2006). Overall, during daytime, the O_3 concentrations below the canopy of various forest types are 0 to 15 % lower than those measured at the top of the canopy (Andreae et al. 2002; Fontan et al. 1992; Joss and Graber 1996). However, stronger gradients appear at night because of a greater air stability, which limits the exchange between the canopy and the atmosphere above, and due to radiative cooling inducing strong temperature inversions near the ground (Skelly et al. 1996). As a consequence, the O_3 concentrations near the forest floor are lower than those measured in the canopy or in the atmosphere above, especially at night (Fontan et al. 1992). By combining O_3 concentration measurements and stomatal conductance estimations, it is possible to get a rather good knowledge of the dose absorbed by the plant. However, the determination of the O_3 stomatal conductance is far from trivial. Whatever the method used, the values are subject to uncertainties. Many measurements and modelling studies of O_3 flux for various canopies and different seasons exist (Massman 2004; Padro 1996; Wesely and Hicks 2000). However, there are still large doubts concerning the processes controlling O_3 deposition to plant surfaces (Ashmore et al. 2007), and therefore in the partitioning of the O_3 flux between stomatal and non-stomatal uptakes, whose relative contributions vary with canopy type and with the season of the year (Tuovinen et al. 2009; Zhang et al. 2006). Many studies have been dedicated to this question, and most of them show an enhancement of O_3 deposition with increased surface wetness.

Significant (20–80 % of total) non-stomatal O_3 fluxes have been observed in different forests in southern European conditions (Cieslik 2009), which often limit the gas flux through stomata. A 10-year-long measurement in a boreal Scots pine forest in Finland showed that the non-stomatal O_3 deposition in the daytime during the growing season varied within 26–44 % of total deposition (Rannik et al. 2012). Another decade-long dataset collected in a mixed temperate forest in Belgium showed larger non-stomatal fractions, exceeding 60 % even during the daytime in summer (Neirynck et al. 2012).

The correct quantification of the different components of the deposition, including the stomatal fraction, is also required when assessing the possible feedbacks between O_3 uptake rates and plant injury or damage, photosynthesis and plant defences. Scaling functional processes of forest trees from leaves and compartments (soil, canopy) to stands, ecosystems and, finally, the landscape level (Wieser et al. 2008) is fundamental for understanding the capacity of forest ecosystems to mitigate air pollution effects and to adapt to changing environmental conditions (Matyssek et al. 2012).

2.4 Leaf damage

As a strong oxidant, O_3 causes several types of visible injury, including chlorosis and necrosis (http://hermes.wsl.ch/didado/ozoniwww.page0?sprache=E). These symptoms, well characterized in controlled conditions, could be also observed on leaf trees in rural areas and mountains, downwind from cities (Dalstein et al. 2002; Feng et al. 2014 ; Miller et al. 1994) or in forested areas exposed to ambient O_3 concentrations high enough to produce phytotoxic effects (de Vries et al. 2014). In this latter case, it is obvious that the

interpretation is more doubtful, conditioned by the complex interactions between O_3 and environmental factors inside the canopy of adult trees or linked to water and mineral availability of the soil (Bussotti and Ferretti 2009; Manning 2005). O_3 can also induce an accelerated senescence and leaf abscission (Gielen et al. 2007; Karnosky et al. 2005; Ribas et al. 2005). Finally, although visible symptoms can be useful for detecting an O_3 impact, their relationship with growth reduction is not always found (de Vries et al. 2014).

3 Cellular and molecular mechanisms impacted by O_3

3.1 Impact on guard cells

Ainsworth et al. (2012) reported that a reduction in stomatal conductance in plants exposed to chronic elevated O_3 (range to 80–120 ppb) could be attributed to a direct effect of O_3 on photosynthesis and to a resultant increase in internal CO_2 concentration. However, alternative reactions might explain this response (Pell et al. 1992). In fact, studies reported that stomata are impaired by chronic O_3 exposure in their ability to close rapidly in response to environmental stimuli (McAinsh et al. 2002; Reich et al. 1984). More recently, it was shown that stomata open and close more slowly in response to changing light conditions, VPD or CO_2 concentrations as a result of 120 ppb O_3 exposure (Dumont et al. 2013) or with 1.5- to 2-fold O_3 ambient level (Paoletti 2005; Paoletti and Grulke 2010). Some molecular aspects were studied in order to explain how O_3 modifies the signals involved in opening or closure processes (Vahisalu et al. 2010). Dumont et al. (2014) showed on poplar genotypes that modification of stomatal responses by an exposure to 120 ppb of O_3, such as stomatal sluggishness (Fig. 2), does not result from ultrastructural changes but from a disturbance of ion fluxes and a regulation of the gene expression involved in signal transduction. The expression of a majority of the studied genes coding for plasma membrane and vacuolar channels was inhibited by O_3, especially the expression of genes coding for the plasma membrane proton ATPase (AHA11) and the vacuolar calcium channels (CAX1 and CAX3) (Dumont et al. 2014).

There is also more recent evidence that stomatal conductance is not universally reduced by elevated O_3 concentration, but that leaf age and tree developmental stage can alter the degree to which O_3 affects stomatal conductance (Uddling et al. 2009). Danielsson et al. (2003) and Pleijel et al. (2002) added the effect of O_3 to the phenology function (related to the reduction of the stomatal conductance in senescing leaves) of the stomatal conductance model in potato and wheat. That new function is being increasingly used, but more knowledge is needed to determine if the effects of O3 on trees should really be integrated in the same way as to link the O3 function to another function than phenology function so far. Further research is needed (i) to characterize O_3 impacts on stomatal function as well as the interaction with other abiotic stresses like drought and (ii) to improve stomatal conductance models like the DO3SE model (Büker et al. 2012). In these models, we need to improve the estimation of the start and end of the growing season today and in the future.

3.2 Detoxification

Once O_3 enters the sub-stomatal chamber, it rapidly induces the formation of reactive oxygen species (ROS) like hydrogen peroxide, singlet oxygen and hydroxyl radicals, increasing the oxidative load to the apoplastic fluid (Fig. 2). The primary effect of ROS is the alteration of membrane and enzyme proteins, e.g. Rubisco (Dizengremel 2001; Heath 2008; Pell et al. 1992). The ability to limit the occurrence of ROS in the apoplast could confer the O_3 tolerance of aspen clones (Oksanen et al. 2004). Apoplastic ascorbate (Asc) is considered as the first line of defence against O_3, as observed in herbaceous plants (Conklin and Barth 2004) but also in beech (Haberer et al. 2007; Luwe and Heber 1995), silver birch (Padu et al. 2005) or spruce and pine needles (Polle et al. 1995). However, the Asc levels in the apoplast are not sufficient to explain the different degrees of O_3 sensitivity in poplar clones (Ranieri et al. 1999). The lack of any direct relation between species sensitivity and apoplastic Asc levels was attributed (i) to the high level of Asc oxidation in this compartment, (ii) to its low concentration relative to the total cell content and (iii) to the occurrence of other antioxidants (Castagna and Ranieri 2009; Dizengremel et al. 2013).

At the whole-leaf level, changes in Asc content increased or decreased according to the exposure level and the duration of O_3 treatment, the leaf age, the growth stage or the position of the leaf in the canopy (Haberer et al. 2007; Strohm et al. 2002; Tausz et al. 2004; Wellburn et al. 1996). Thus, the higher O_3 sensitivity of young beech in phytotrons compared to adult forest trees in the field has been partly attributed to differences in detoxification capacity and notably total ascorbate concentration (Nunn et al. 2005). From a series of studies conducted on poplar exposed to O_3 in controlled conditions or in the field, it is difficult to establish a clear relation between the constitutive level of Asc and the genotype sensitivity (Di Baccio et al. 2008; Dumont et al. 2014; Yun and Laurence 1999). However, a higher level of dehydroascorbate (DHA) in a sensitive poplar genotype (Dumont et al. 2014) exposed to 120 ppb O_3 may express a lower capacity of the genotype to regenerate the reduced form (AsA), a hypothesis supported by a lower NADPH content in the leaves of this genotype (Dghim et al. 2013a).

In the absence of glutathione in the apoplasm, the Asc regeneration process implies the transport of DHA in the

cytosol followed by the functioning of the intra-cellular ascorbate-glutathione cycle (Noctor 2006). This cycle sustains AsA regeneration in the cytosol. Glutathione is known as an antioxidant, able to directly react with ROS, sometimes functioning in a compensatory manner to Asc but also with specific functions (Noctor et al. 2012). Total glutathione and/or reduced glutathione content generally increased in leaves of tree species under O_3 fumigation (100 to 120 ppb) (Dumont et al. 2014; Wellburn et al. 1996). In adult beech trees, the glutathione level is affected by canopy position, but O_3 exposure (twofold ambient concentration) involved higher content in both shade and sun leaves (Herbinger et al. 2005). In some works, the differences in the constitutive levels of glutathione between poplar genotypes appeared to contribute to the higher tolerance to chronic O_3 exposure (Di Baccio et al. 2008; Dumont et al. 2014). However, attempts to increase glutathione content and glutathione reductase activity in transgenic poplar were unsuccessful in increasing tolerance, at least to acute (300 ppb) O_3 exposure (Strohm et al. 2002).

Phenolic compounds are also recognized in leaf like important metabolites to cope with elevated or chronic O_3 exposure of trees (Fares et al. 2010; Kontunen-Soppela et al. 2007; Peltonen et al. 2005; Yamaji et al. 2003). For some phenolics and condensed tannins, a potential role in the O_3 tolerance has been claimed (Haikio et al. 2009; Kontunen-Soppela et al. 2007). Furthermore, in accordance with an increased level in phenolics, the induction of the shikimate and phenylpropanoid pathways shared by the flavonoid, anthocyanin, tannin, stilbene and lignin biosynthesis has been well documented under O_3 exposure (see Cabané et al. 2012 for review). For some tree species that emit large amounts of VOCs, it is also interesting to consider the potential role of these compounds like antioxidants, as found for isoprene in poplar (Loreto et al. 2001) and for monoterpenes in oak (Loreto et al. 2004). However, recent works on poplar showed that the relationship between O_3 tolerance and the ability to emit isoprene is not so clear (Behnke et al. 2009; Calfapietra et al. 2008). The involvement of polyamines like radical scavengers and protectant against O_3 has been also considered although these studies are limited (Ludwikow and Sadowski 2008).

In addition to metabolites, a large panel of antioxidant enzymes are involved in the defence mechanisms to decrease the ROS level or to regenerate the reduced form of some antioxidants (Fig. 2). Firstly, extracellular enzymes like superoxide dismutases (SOD) and peroxidases (with ascorbate or phenolics as preferential electron donors) were identified as significant contributors to the mitigation of ROS generation in the apoplasm of birch (Padu et al. 2005) and poplar leaves (Castagna and Ranieri 2009). When the apoplastic antioxidant capacity is overwhelmed, other isoforms of these enzymes were implied to limit the generation of cytoplasmic ROS. Thus, total SOD and/or peroxidase activities generally increased in O_3-treated leaves of trees (Bernardi et al. 2004; Diara et al. 2005; Sehmer et al. 1998; Tuomainen et al. 1996) even though conflicting results have been mentioned (Heath and Taylor 1997). It has been claimed that the different affinities for ROS of the antioxidant enzymes either may be linked to the regulation of ROS as signalling actors or may be responsible for the removal of excess ROS (Mittler 2002). More generally, maintaining a cellular steady state of the ROS in responses to stresses is assigned to a complex enzyme network with actors like thioredoxin-dependent peroxidases (including peroxiredoxins and glutathione peroxidase), glutaredoxins and glutathione-S transferases (Foyer and Noctor 2011; Mittler et al. 2004; Rouhier and Jacquot 2005). Hence, the regulation of the enzymatic antioxidant system involves a fine redox regulation (Jacquot et al. 2013). For some of these enzymes, their characterization in trees (mainly poplar) is recent (Navrot et al. 2006; Rouhier 2010) but their regulation under O_3 or other oxidative pollutants is still undefined. Moreover, in these stress conditions, the source of reducing power to supply some of the ROS-scavenging systems remains to be elucidated (Dghim et al. 2013b; Dizengremel et al. 2008). Finally, to propose a new index for O_3 risk assessment integrating the plant detoxification capacity, a complex network of metabolites and enzymes has to be taken into account. But for trees, the variations of these parameters along the successive growing seasons as well as the canopy position must also be considered.

3.3 Carbon assimilation and leaf senescence

One of the first O_3-driven decreases in growth was characterized by Reich (1983) in greenhouse-grown trees and directly correlated to a reduction in net CO_2 assimilation rate (Fig. 2). This correlation was then confirmed in fumigation chamber, open-top chamber and field fumigation systems across several tree species (Reich 1987). In a meta-analytic review, Wittig et al. (2007) showed that the O_3-driven decrease in carbon assimilation reached 14 % for angiosperm trees grown in ambient background O_3 relative to charcoal-filtered air. Differently, gymnosperms were not significantly affected. However when exposed to severe O_3 concentration (85 ppb), net CO_2 assimilation was similarly decreased (up to 19 %) in both angiosperms and gymnosperms. The average decrease in CO_2 assimilation was progressively greater as the O_3 treatment increased (Wittig et al. 2007). O_3 impact on photosynthesis could be threshold dependent, highlighting the importance to better define critical O_3 threshold across species and/or environmental conditions. Differences in photosynthesis response to O_3 may partly be explained by interspecific variability of stomatal response to O_3 and therefore gas flux entering the leaf. Over hundreds of individuals indicate an 11% and 13 % decrease in average in CO_2 uptake and stomatal conductance, respectively (Wittig et al. 2007). Stomatal limitation under O_3 may in part explain

the reduced photosynthetic CO_2 uptake in restricting CO_2 diffusion from the atmosphere to the intercellular space and therefore CO_2 availability at the site of carbon fixation (see section on stomatal regulation above).

In addition, O_3 has been widely shown to affect both the Calvin-Benson cycle and photochemistry activity (Fig. 2) (Saxe 2002). A decrease in Rubisco activity and content was strongly correlated with cumulative O_3 exposure in loblolly pine (Dizengremel et al. 1994). Similarly, in Aleppo pine, both Rubisco and Rubisco activase levels were reduced under O_3 (Pelloux et al. 2001). In angiosperms also, multiple works supported the idea of O_3-driven alteration of Rubisco activity (Gaucher et al. 2003; Lutz et al. 2000; Matyssek et al. 1991; Pell et al. 1992). However, it remains unclear whether the decrease in Rubisco activity is due to enhanced degradation, ROS-mediated protein oxidation, decreased Rubisco activase activity or altered gene expression (Brendley and Pell 1998; Dizengremel 2001; Heath 2008; Pell et al. 1994). It is well documented that chronic as well as acute O_3 exposures impact gene regulation (Ernst 2013; Renaut et al. 2009) even though acute episodes could provoke more important changes than chronic ones (Ainsworth et al. 2012; Ernst 2013). Furthermore, proteomic analysis confirms the downregulation of a large number of proteins involved in the Calvin-Benson cycle in poplar leaves exposed to chronic O_3, as well as proteins involved in chloroplastic electron transport (Bohler et al. 2007). O_3 also decreases chloroplast size and cell starch content (Oksanen et al. 2004). Chlorophyll and carotenoid levels have been shown to decrease under O_3, resulting likely in a less active photochemistry and a slower electron transport rate (Bagard et al. 2008). It is, however, unclear whether the slower electron transport rate under O_3 is due to a lower leaf pigment level or a downregulation of PSII activity in order to avoid photooxidative damage. In addition, photochemistry can also be diminished under a long-lasting reduction of stomatal conductance. Such electron transport slowdown would likely limit NAD(P)H production and availability for anabolic process, detoxification and photosynthetate synthesis. The drastic decrease in chlorophyll and Rubisco contents is correlated with accelerated leaf senescence (Miller et al. 1999). Genes involved in senescence and protein turnover are upregulated in *Populus tremuloides* leaves exposed to O_3 (Gupta et al. 2005). Early senescence would restrict the "return on investment" in leaf buildup and therefore drastically affect plant carbon budget.

The reduced net CO_2 assimilation in O_3-exposed leaves is largely driven by a decrease in gross CO_2 assimilation rate by the Rubisco, but it also results from increased CO_2 losses through an enhanced respiration, as observed in poplar (Bagard et al. 2008; Noormets et al. 2001; Reich 1983), Norway spruce (Küppers and Klumpp 1988), Scots pine (Kellomaki and Wang 1998; Skärby et al. 1987), birch (Matyssek et al. 1997) and beech (Kitao et al. 2009). The increase in CO_2 efflux from respiration is supported by an enhanced glycolysis, pentose-phosphate pathway and TCA cycle activity (Dizengremel 2001). A central enzyme linking these metabolic pathways, the phosphoenolpyruvate carboxylase, is strongly upregulated in O_3-exposed leaves of a wide range of species (Dizengremel 2001; Dizengremel et al. 2012). PEPc activity produces oxaloacetic acid, a precursor for malate and pyruvate synthesis, and can therefore support the rising demand in organic acids for TCA cycle decarboxylation or anaplerotic pathways. PEPc-induced pathways could play a central role in O_3 tolerance in providing additional carbon skeletons and NAD(P)H to detoxification processes (Dizengremel et al. 2009). Additionally, the increase in dark respiration under O_3 is likely supported by a higher contribution of alternative pathways of the mitochondrial electron transport chain (Dizengremel 2001) that may be driven by a higher PEP content and subsequent higher pyruvate level (activator of alternative oxidase). The enhancement of the mitochondrial alternative electron transport would help to maintain a high respiratory rate in avoiding any respiratory control through oxidative phosphorylation and therefore contribute to reducing power availability for detoxification and repair of cellular damage (Dizengremel et al. 2009).

3.4 Cell wall component biosynthesis in leaves and stems

In addition to inducing a diverse range of defence responses, O_3 has been shown to modify the cell wall. Anatomy analysis revealed thickened cell walls with pectinaceous projections in leaves of deciduous trees showing visible leaf symptoms (Gunthardt-Goerg et al. 1997; Gunthardt-Goerg et al. 2000). These observations suggested strong rearrangements in the cell wall organization and in its component biosynthesis in leaves of trees subjected to O_3. However, most studies focused on one component, lignin.

O_3 has been shown to stimulate phenylpropanoid metabolism in leaves of many tree species and under different fumigation protocols involving both acute and chronic exposure (Fig. 2). O_3 increased both enzyme activities and related gene transcript levels involved in lignin biosynthesis in *Pinus sylvestris* (Rosemann and Heller 1991; Zinser et al. 1998), *Picea abies* (Galliano et al. 1993a; Galliano et al. 1993b; Heller et al. 1990), *Populus* spp. (Cabané et al. 2004; Di Baccio et al. 2008; Koch et al. 1998; Wustman et al. 2001), *Betula pendula* (Pääkkönen et al. 1998a; Tuomainen et al. 1996) and *Fagus sylvatica* (Jehnes et al. 2007; Olbrich et al. 2005). The responses of the phenylpropanoid metabolism can be both fast (Koch et al. 1998) and substantial (Cabané et al. 2004), and the induction levels were generally correlated with O_3 concentrations (Cabané et al. 2004; Galliano et al. 1993b; Rosemann et al. 1991). Stimulation of the phenylpropanoid pathway was often maintained during the whole period of O_3 exposure (Cabané et al. 2004; Galliano et al. 1993a) and could

even continue after the end of the treatment (Tuomainen et al. 1996). Most of the above results were obtained from trees growing in controlled conditions or open-top chambers, and the same trend was also observed in more natural conditions such as free-air O_3 fumigation facilities (Betz et al. 2009; Wustman et al. 2001). All these results unambiguously demonstrate that the phenylpropanoid pathway is upregulated in leaves under O_3 exposure and is therefore probably involved in defence and acclimation mechanisms. As a consequence of the phenylpropanoid pathway stimulation, the lignin content must increase as a result of O_3 exposure. Indeed, such results were found in leaves of sugar maple (Boerner and Rebbeck 1995), poplar (Cabané et al. 2004) and beech (Betz et al. 2009; Jehnes et al. 2007; Olbrich et al. 2010b). However, the effects of O_3 fumigation on lignin content in leaves were not so clear. Thus, no modifications of lignin content were recorded in western yellow pine (Tingey et al. 1976), black cherry and yellow poplar (Boerner and Rebbeck 1995), loblolly pine (Booker et al. 1996), birch (Oksanen et al. 2005) and holm oak (Baldantoni et al. 2011). These varying results may be explained by potential error due to different techniques (Klason, LTGA, etc.) used to determine lignin content as well as their relative (in)sensitivity (Dence 1992), especially in the case of weak variations between control and treated samples. Another explanation could be species-specific differences in response to O_3 treatment. For example, conifers never showed increased lignin content. Stimulation of the phenylpropanoid pathway could in such cases be associated with a modification of the pool of soluble phenolic and not necessarily lead to increased lignification (Tingey et al. 1976). Nevertheless, the newly synthesized lignin in leaves displayed changes in its structure (Betz et al. 2009; Cabané et al. 2004). Lignin was enriched in carbon-carbon interunit bonds and in H-units indicating the production of a more condensed lignin than usual. Moreover, lignified cells were observed in the mesophyll or epidermis near the necrotic lesions (Cabané et al. 2004) where ROS (H_2O_2) were also shown to be accumulated (Pellinen et al. 2002). These results support the idea that stress lignins are synthesized in response to and in defence against O_3 or ROS excess. Due to its scavenging properties, lignin may act as an antioxidant (Blokhina et al. 2003; Dizhbite et al. 2004).

Since the response to O_3 has been extensively studied in leaves, few studies have analysed the O_3 response of stems. This is understandable since leaves show clear O_3-induced damage and a fast response while stems react much later (Richet et al. 2012). An increase in lignin content was observed in stems of poplar and birch fumigated for 3 years in a free-air fumigation experiment (Kaakinen et al. 2004), but this observation was not maintained after 5 years (Kostiainen et al. 2008). In a recent study, O_3 was observed to repress the phenylpropanoid pathway in poplar wood (Richet et al. 2011), probably as a result of reduced cambial growth. However, the relative cell wall lignin content increased due to an O_3-induced reduction in cellulose biosynthesis, thereby modifying the cellulose to lignin ratio (Fig. 2). The stem response seems to correspond to a metabolic adjustment due to the reorientation of the metabolism to stress acclimation in leaves, rather than to a specific defence mechanism. O_3 would not impact directly the stem organ (Richet et al. 2012). It was hypothesized that the modification of the cellulose to lignin ratio in the stem could allow the tree to maintain radial and height growth while minimizing carbon cost (Richet et al. 2011). More detailed analyses are needed to draw definite conclusions.

3.5 O_3-induced signalling in trees

O_3-signalling events are very quickly initiated during O_3 exposure, leading to plant survival or acclimation. Acclimation first implies O_3 perception and concomitant signalling cascades, ultimately succeeding in re-programming tree metabolism (Fig. 3). In order to elicit and decipher signalling processes, trees were exposed to acute O_3 dose (>150–200 ppb) for few hours. In these conditions, some signalling actors, as calcium or protein kinases, were well characterized in herbaceous plants (Baier et al. 2005; Vainonen and Kangasjarvi 2014; Vaultier and Jolivet 2015). However, O_3-induced signalling is by far less documented in trees. Direct extrapolation from model herbaceous plants to trees is not always possible, as woody plants may possess their own defence signalling systems (Dizengremel 2001; Koch et al. 2000). In beech (55–60 years old) exposed to an experimental enhanced free-air O_3 setting (up to 150 ppb), the expression level of genes connected with signalling, i.e. ethylene (ET) biosynthesis-related genes *ACC* (1-aminocyclopropane-1-carboxylic acid) *synthase* or *oxidase*, was increased (Jehnes et al. 2007; Olbrich et al. 2010a). Besides, an environmental genomic study performed on 5-year-old trembling aspen exposed to 1.5 times ambient O_3 showed that many genes involved in signal transduction were upregulated, e.g. ET biosynthesis-related genes such as *ACC oxidase* or a gene coding for a mitogen-activated protein kinase (MAPK) (Gupta et al. 2005). MAPK cascades are well-known components of stress-induced signalling pathways, and several studies revealed their activation in response to O_3 exposure in herbaceous plants (Ahlfors et al. 2004; Samuel et al. 2000). In trees, Hamel et al. (2005) showed in hybrid poplar cell suspensions and leaf tissue that O_3 (500 ppb) induced a rapid and transient activation of at least two MAPKs, independently or upstream of both salicylic acid (SA) and jasmonic acid (JA) signalling. However, to date, involvement of the O_3-activated MAPKs in regulating O_3 sensitivity in poplar is still to be investigated.

Trees respond to O_3 exposure by producing other signalling molecules as ROS (Diara et al. 2005; Moura et al. 2014; Pellinen et al. 1999). The O_3-induced ROS production and subsequently the formation of necrosis are part of the

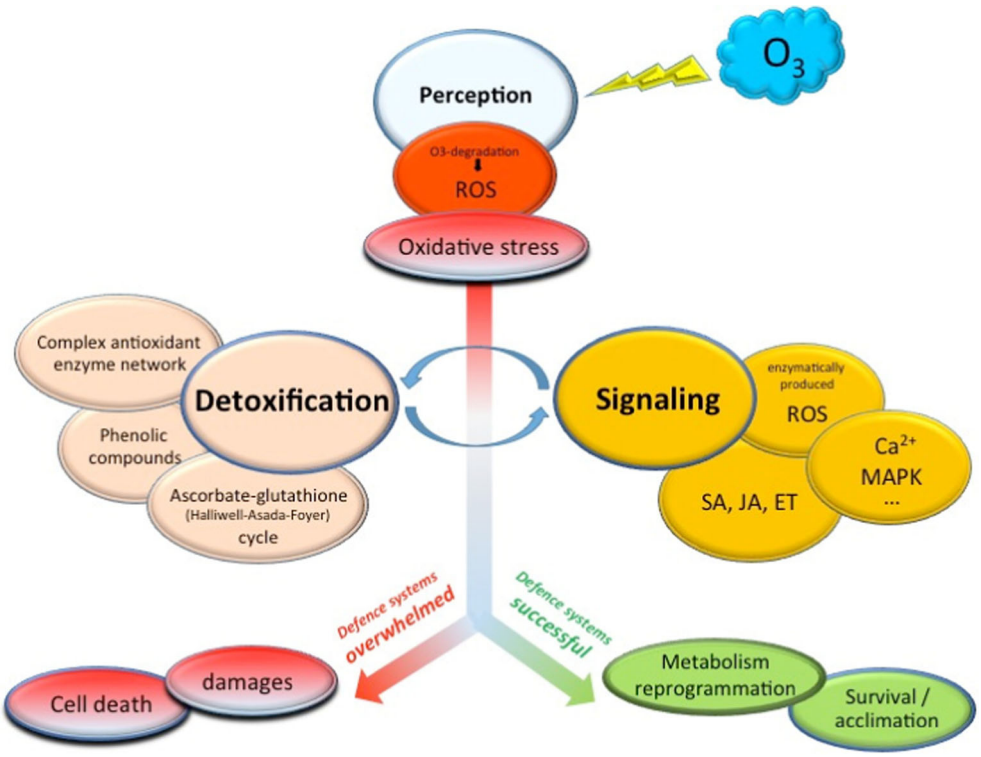

Fig. 3 Schematic representation of interactions between signalling and detoxification processes in the cell of plants exposed to ozone. The acclimation of the plants to this pollutant is upon a tight control of these two processes. *ET* ethylene; *JA* jasmonic acid; *MAPK* mitogen-activated protein kinase; *SA* salicylic acid

similarities shared with early senescence and hypersensitive response elicited by the incompatible plant-pathogen interaction (Moura et al. 2014; Pellinen et al. 1999; Tuomainen et al. 1996). ROS generated either (i) directly from O_3 degradation or (ii) actively enzymatically produced could be crucial components of O_3-induced signalling (Vainonen and Kangasjarvi 2014).

Concerning hormones, Koch et al. (1998) suggested the role of SA in mediating some O_3-induced responses in trees. O_3 can induce lesion formation via the activation of programmed cell death, and SA perception is required for the activation of a hypersensitive cell death pathway (Koch et al. 2000). Poplar has higher constitutive levels of free SA compared with herbaceous plant species such as tobacco and *Arabidopsis* (Diara et al. 2005; Koch et al. 2000). In birch and in hybrid aspen, free SA accumulated in response to acute O_3 conditions (Vahala et al. 2003a; Vahala et al. 2003b). Similarly, a significant increase in the conjugated pool of SA was observed in poplar during acute O_3 fumigation (150 ppb for 5 h) (Diara et al. 2005). Optimal SA concentration is required to fine-tune the plant response in order to achieve the maximum stimulation of defence responses with minimal induction of cell death (Diara et al. 2005). JA was also evidenced as an important O_3-induced signal molecule in trees as acute O_3 exposure increases endogenous JA levels in poplar or in birch (Koch et al. 2000; Koch et al. 1998; Vahala et al. 2003b). JA has at least two different roles in O_3 responses: one in lesion formation and the other in lesion containment (Kangasjarvi et al. 2005). Activation of SA- and JA-mediated signalling pathways, which may be important in triggering defence responses against oxidative stress, leads to O_3 tolerance (Koch et al. 1998). Lesion propagation and containment in O_3 damage are under the control of ET (Kangasjarvi et al. 2005). O_3 exposure leads to ET release from leaves, in poplar clones (Diara et al. 2005; Kargiolaki et al. 1991), in birch (Vahala et al. 2003b) and in pine needles (Telewski 1992). In aspen, O_3 caused a clear concentration-dependent response in ET evolution (Vahala et al. 2003a). Marked increases in the pool of free ACC, precursor of ET, and in ACC synthase transcripts were also detected in poplar (Diara et al. 2005). The role of ET under chronic (75 ppb) and acute O_3 (up to 200 ppb) was investigated in aspen and silver birch by Vahala et al. (2003a; 2003b). Comparing results obtained on different species, herbaceous or not, Diara et al. (2005) hypothesized a "pro-survival" role for ET and the existence of a threshold below which ET would not trigger lesion development. ET can serve as a mediator of either survival or cell death, depending on the magnitude of synthesis and its temporal pattern (Vahala et al. 2003b). In hybrid aspen, ET accelerated leaf senescence under low O_3, but under acute O_3 elevation, ET signalling seemed to be required for protection from necrotic cell death (Vahala et al. 2003a).

Of course, complex interactions between hormones are involved in tree response to O_3 exposure. All the three hormonal signalling pathways: SA, JA and ET, were involved in cell death induced by a short exposure to high O_3 concentration (200 ppb for 8 h) in birch (Vahala et al. 2003b). Early high ET production may antagonize the late SA accumulation, and

conversely, increased SA production may downregulate ET accumulation and thus prevent the ET-dependent cell death (Vahala et al. 2003b). In poplar, difference in O_3 sensitivity would depend on differences in the modulation of signal transduction pathways, i.e. the timing and magnitude of SA and ET production, as well as on cross-talking with other signalling molecules (Diara et al. 2005). Further investigations are really needed in trees to unravel this puzzling network triggered by O_3, particularly under realistic chronic O_3 doses. In these conditions, not only the signalling pathways at the onset of an O_3 episode in natural conditions must be considered but also the impacts, some days or weeks later, when the cellular defence mechanisms are overwhelmed, followed with the beginning of cell death and the occurrence of necrosis (Fig. 3). Finally, it is still necessary to decipher the steps by which defence reactions like the ascorbate-glutathione cycle are under the control of signalling (Fig. 3).

4 Impact of O_3 on tree growth, forest productivity and carbon sequestration

4.1 Carbon allocation and tree growth

A meta-analysis by Wittig et al. (2007) concluded that significant decreases in both photosynthesis and stomatal conductance of trees under O_3 may negatively affect both carbon sequestration and transpiration. A limitation of extrapolating these data to mature forests is that the estimates are largely based on individual juvenile trees growing in a non-competitive environment, and extrapolation of results from seedlings may not be appropriate for predicting the response of mature trees and forests to O_3 (Chappelka and Samuelson 1998; Ollinger et al. 1997). But recently, Matyssek et al. (2010a) concluded that adult and juvenile trees of pioneer and climax tree species show similar growth sensitivity to chronic O_3 stress, although the underlying response mechanisms may differ. Tree growth and productivity are expected to decrease under O_3 considering aforementioned effects of this pollutant (Fig. 2). Indeed, lower growth and diameter have been observed in a wide range of tree species after long-term exposure to O_3 (Booker et al. 2009; Karnosky et al. 2005; King et al. 2005; McLaughlin et al. 2007; Pretzsch et al. 2010; Tjoelker et al. 1994). Summarizing hundreds of studies, Wittig et al. (2009) reported a decrease in total biomass, leaf area, root to shoot ratio, height and diameter in trees exposed to chronic O_3 concentration (in the range 40–100 ppb) relative to charcoal-filtered atmosphere. However, the intensity of O_3 effects on tree carbon uptake and growth differs depending on tree age, the loss in biomass production following O_3 exposure being greater in young compared to older trees (Herbinger et al. 2005). Since O_3 reduces carbon gain by limiting stomatal diffusion, lowering Rubisco activity, inducing early senescence and increasing carbon cost for tissue repair and antioxidant synthesis, it drastically decreases source strength and carbon availability for export to sink tissues. Additionally, increased soluble sugar content and carbohydrate retention have been observed in source tissue exposed to chronic O_3 concentration (from 0 to 110 ppb) (Friend and Tomlinson 1992; Grantz and Farrar 1999; Grantz and Yang 2000), suggesting a decrease in carbon export under O_3. A lower leaf sucrose export and higher carbohydrate level would lead to feedback regulation of photosynthesis and therefore partly explain the reduction in carbon assimilation. Several works have shown a decrease in allocation to roots and root to shoot biomass ratio in response to O_3 (Gorissen et al. 1994; Grantz and Farrar 2000; Grantz and Yang 2000; Rennenberg et al. 1996; Spence et al. 1990). Given that mature leaves preferentially allocate carbon resources to stems and roots (Gordon and Larson 1970; Matyssek et al. 2010b; Rangnekar and Forward 1969), it appears logical that the O_3-induced early senescence would primarily affect root growth. Such modification may therefore have drastic impacts on tree surrounding rhizosphere and tree survival to environmental constraints such as drought (Agathokleous et al. 2016).

4.2 Ozone impact on forest productivity and carbon sequestration: results from free-air fumigation experiments and modelling studies

Fowler et al. (1999) used the 3-D chemistry-transport model STOCHEM (Collins et al. 1997) to simulate the global distribution of tropospheric O_3 from 1860 to 2100. The results indicate that the area covered by forests exposed to >60 ppb increased from 0 in 1860 to 8.3 million km^2 in 1990, i.e. 24 % of global forest area. According to this study, this area could reach 17 million km^2 in 2100, that is half of the projected global forest area, if precursor emission rates remain constant (Fowler et al. 1999). In order to advance from exposure assessment to impact prediction, subsequent modelling studies implemented the linear empirical model of O_3 impact on tree biomass production developed from experimental data in the pioneer work of Reich (1987). The results indicated that biomass production of forest ecosystems would be reduced by 3–22 % in the northeastern USA as an effect of tropospheric O_3 concentrations recorded during the period 1987–1992 (Ollinger et al. 1997). With a similar approach, Subramanian et al. (2015) found that biomass growth of forest trees in Sweden could be reduced annually by 4.3–15.5 % for conifers and 1.4–4.3 % for birch by current O_3 when compared to prehistoric O_3. Proietti et al. (2016), by combining satellite productivity estimates, O_3 measurement data and impact functions, found that current O_3 concentrations could reduce gross primary productivity of European forests by 0.4–30 %

along a North-West-South-East transect. An alternative, flux-based methodology emerged from the International Cooperative Programme on the effects of air pollution on vegetation (Mills et al. 2013) under the UNECE Convention on Long-Range Transboundary Air Pollution (LRTAP). In this approach, the European Monitoring and Evaluation Program (EMEP) and Rossby Centre Regional Climate (RCA3) models provided O_3 and meteorological input data that fed the Deposition of Ozone for Stomatal Exchange (DO_3SE) model, which simulates O_3 dry deposition (Büker et al. 2012) and subsequently O_3 stomatal fluxes and phytotoxic O_3 doses (POD_Y) as described in the LRTAP convention manual (2010). O_3 flux-response relationships were applied to calculate biomass and carbon losses for tree species groups and representative species. The data were combined to land cover data and overlain to EMEP and RCA3 resolved grids. Finally, European forest inventory and carbon sequestration datasets were used to calculate absolute carbon losses due to O_3 in Europe. Applying a generic parameterization for deciduous and conifer trees, the authors estimated a reduction of carbon sequestration in the living biomass of trees by 12 % (EMEP input data) to 16 % (RCA3 input data) for the year 2000 as compared to pre-industrial O_3 levels (Mills et al. 2013).

Either exposure- or flux-based modelling studies provide congruent estimates of forest biomass production reduction as an effect of current O_3. However, the impact functions used rely on O_3 exposure- or flux-response relationships that were derived for relatively young trees (<10 years of age) exposed to O_3 under semi-natural, non-competitive conditions (Karlsson et al. 2007; Reich 1987). Although epidemiological studies suggest that such functions are applicable to mature trees within forest stands (Braun et al. 2010), whether conclusions on the effects of O_3 on forests can be drawn from the extrapolation of results obtained on seedlings remains a matter of debate (Samuelson and Kelly 2001). In this respect, the free-air concentration enrichment experiment Aspen FACE led in Rhinelander (WI, USA) provided concordant results. In this study, young forest stands were subjected to an O_3-enriched atmosphere (1.5 × ambient) during 11 years from seedling establishment to maturity (Karnosky et al. 2003). The results showed significant reductions in the total biomass of young stands of trembling aspen (−23 %), aspen-sugar maple (−14 %) and aspen-paper birch (−13 %) (King et al. 2005), but these results reflect in main part the impact of O_3 on young trees during their initial, rapid growth stage. Because it was conducted on a mature forest stand composed of 60-year-old trees in a 30-m closed canopy, the free-air O_3 fumigation experiment led in the Kranzberg forest in Germany represents a valuable alternative to FACE systems (Matyssek et al. 2010b). This study showed that stem biomass production of beech trees exposed to elevated O_3 (2× ambient, <150 ppb) during 8 years was reduced by more than 40 %, but also highlighted the strong influence of environmental factors such as drought on tree responses to O_3. The combination of stem growth, sap flow velocity and O_3 measurements has been used to investigate the effect of O_3 on mature trees in a mixed deciduous forest in eastern Tennessee (USA) (McLaughlin et al. 2007). This study revealed that daily events of high O_3 exposure (daily maximum hour ≥100 ppb for 1 day or ≥85 ppb for two consecutive days) could decrease stem growth by up to 30 to 50 % over a season, which suggests that episodes of acute exposure might have consequences on tree biomass production that modelling approaches cannot predict. Combined with climate-controlled branch cuvettes, sap flow measurements could represent a valuable alternative to heavy and expensive free-air fumigation experiments for studying the impact of O_3 on adult forest trees (Wieser et al. 2012).

Meta-analyses (Wittig et al. 2009) as well as modelling studies based on response functions estimate that O_3 has a significant impact (−5 to −30 %) on the net primary productivity of forest ecosystems, which might impair their capacity for carbon sequestration (Ainsworth et al. 2012). However, field experiments on mature forest stands have shown that many factors can modulate tree responses to the pollutant (e.g. environmental conditions, stand dynamics and competition) and remain to be considered in modelling approaches. The main challenge of stomatal deposition models is to accurately predict stomatal conductance in response to environmental drivers (Emberson et al. 2000). Recently, Fares et al. (2013a), Hoshika et al. (2011) and Nunn et al. (2010) applied Jarvis's model parameterized with environmental observations with field data, and they were able to predict well stomatal conductance. The recent study of Wang et al. (2016), which simulated the O_3 impact on forest composition and ecosystem dynamics over 500 years, indicated that elevated O_3 could even lead to an increase in forest productivity due to diversity change and compensatory processes at the community scale.

5 Combination of O_3 and other environmental factors

The interaction of O_3 with other abiotic or biotic factors can first be considered through its combination with other pollutants, which can occur simultaneously or sequentially. A review on tree exposure to pollutant mixtures showed that the observed responses were highly variable according to tree species, age, genotype, composition of rain solution and soil type (Chappelka and Chevone 1992). In a context of global change, a range of constraints, including high CO_2, increased temperature, altered precipitation and drought episodes, will also affect trees exposed to O_3 pollution episodes. A first interaction has been investigated in a context of rising atmospheric CO_2 concentrations, which

generally results in a reduction of stomatal conductance (Ainsworth et al. 2012). Thus, based on simulated stomatal O_3 uptake, the flux of O_3 entering the leaves would be decreased (Klingberg et al. 2011; Sitch et al. 2007). However, the reduced stomatal conductance on a long-term exposure under high CO_2 seems uncertain considering contrasting results from FACE experiments (Uddling et al. 2010). The stage of plant and stand development, as well as the consideration of overstorey/understorey species, would influence the stomatal response. Drought also reduces stomatal conductance, with a potential subsequent restriction of O_3 effects. In fact, the protective effect of drought would only occur in severe drought conditions while under low water restriction the damage caused by O_3 appeared additive (Matyssek et al. 2006; Pääkkönen et al. 1998b). However, there are clear inter- and intra-specific differences in response to the combination of drought and O_3 (Dixon et al. 1998). The protective effect of drought may be the result of stomatal exclusion of O_3 but also the induction of defence reactions (Matyssek et al. 2006). Other works underlined that the combined effects of drought and O_3 could also decrease the antioxidant capacity of leaf cells in a higher extent than with the constraint alone, leading to a higher susceptibility to oxidative stress (Wellburn et al. 1996). Thus, in these conditions of combined stresses, the fine-tuning between O_3 uptake and defence capacity appears crucial (Matyssek et al. 2006). Because of changes in plant metabolism, carbon assimilation and allocation and chemical leaf defences, O_3 may also modify plant responses to biotic factors as insect or plant pathogens. Trees can be weakened by O_3, promoting biotic attack (Chappelka and Chevone 1992; Dowding 1988). However, these studies also underlined that a great number of factors linked to the environment, the host plant or the pathogen may modulate the O_3-host-pathogen interactions and their consequences. Some works attempted to better understand these interactions and identify underlying biochemical and physiological mechanisms. Using Aspen FACE, investigations were carried out on the impact of both increased CO_2 and O_3 concentrations on forest insects in a poplar canopy (Percy et al. 2002). In response to O_3, the production and chemical composition of leaf cuticular waxes and the concentrations of protective compounds in the leaf were modified. These modifications may explain an increase in rust infection under O_3 while a higher abundance of leaf-chewing insects as well as aphids have been observed, an effect alleviated by the combination of CO_2 and O_3. Changes in leaf morphology and composition, including phenolic content, induced by O_3 appeared to be determinant in explaining the larger deleterious effect of an herbivorous insect on O_3-treated aspen trees (Freiwald et al. 2008). Finally, the O_3 impact on carbon allocation must be also considered as a factor that modulates the beneficial interaction of fungi and plants via mycorrhization (Nikolova et al. 2010; Pritsch et al. 2009), an aspect that needs to be clarified.

6 Concluding remarks

It is now widely accepted that O_3 is able to reduce the growth of forest trees. Even though the results could differ in intensity between experiments conducted in phytotrons or in FACE systems, between young and mature trees, the major impact is a decreased carbon assimilation resulting in a reduced carbon sequestration. The precise mechanisms leading to this loss of available carbon are still to be totally deciphered, notably the respective roles of stomatal resistance and detoxification processes, which determine the tree sensitivity. In addition, the mechanisms underlying the O_3 transduction signal begin to be clarified in acute conditions while it remains fragmented in chronic O_3 exposure, particularly for trees. Another consequence of the O_3 effect is the modification of carbon allocation to the different organs of the tree, which can lead to changes in wood quality and quantity. In this context, alterations of the functioning of forest ecosystems need also to be better investigated by taking into account the interactions between O_3 and other abiotic (CO_2, water, temperature, etc.) and biotic stresses. Emphasis should be put on water availability, a factor already mentioned as determinant to scale O_3 effects from seedlings to forest trees (Samuelson and Kelly 2001). Contradictory reports of antagonistic or synergetic effects of O_3 and CO_2 or drought clearly show that additional research effort is needed. The advancement of an integrative knowledge of O_3 impact from the leaf cell to the tree level will allow a significant improvement of the existing models of O_3 risk assessment on ecosystems.

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