Volatile isoprenoid emissions from plastid to planet

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Summary

Approximately 1–2% of net primary production by land plants is re-emitted to the atmosphere as isoprene and monoterpenes. These emissions play major roles in atmospheric chemistry and air pollution–climate interactions. Phenomenological models have been developed to predict their emission rates, but limited understanding of the function and regulation of these emissions has led to large uncertainties in model projections of air quality and greenhouse gas concentrations. We synthesize recent advances in diverse fields, from cell physiology to atmospheric remote sensing, and use this information to propose a simple conceptual model of volatile isoprenoid emission based on regulation of metabolism in the chloroplast. This may provide a robust foundation for scaling up emissions from the cellular to the global scale.

Introduction

Land vegetation is the principal non-industrial source of biogenic volatile organic compounds (BVOCs) released to the global atmosphere (Denman et al., 2007). Of a total BVOC emission of c. 1 Pg C a−1, isoprene and monoterpenes emitted by leaves represent by far the largest fraction (Arnth et al., 2008). These reactive compounds play a fundamental role in determining the atmospheric content of greenhouse gases and pollutants, especially tropospheric ozone, methane, and secondary organic aerosol (Arnth et al., 2010; Carslaw et al., 2010). Because high temperatures stimulate emissions, future projections of changes in atmospheric composition and air quality depend on how these emissions will change.

Modelling of BVOC emissions has generally centred on the quantification of leaf emission capacities (Ec), the emission rates obtained under standard light and temperature conditions (Guenther et al., 1995). Temporal and spatial variation of emissions has been derived by modifying Ec using empirical equations describing observed short-term controls by temperature and light, and long-term controls by antecedent weather conditions and environmental and biotic stresses (e.g. Guenther et al., 2006). Emission capacities were initially considered to be species-specific constants. Several lines of evidence now show that Ec acclimates seasonally and over environmental gradients (Niinemets et al., 2010), but global emission models still set fixed values for plant or vegetation functional types that determine maximum emission rates (e.g. Guenther et al., 2006; Arneth et al., 2007a; Heald et al., 2009; Paciﬁco et al., 2011). The ever-increasing complexity of these models largely reﬂects attempts to cope empirically with variations in Ec (Monson et al., 2012).

Monson et al. (2012) argue that the use of empirical functions to describe the relationships between emission rates and
environmental variables is unsatisfactory, and particularly the use of serial multipliers based on single factor relationships to account for co-variation of environmental variables. They make a strong case for the need to base modelling on a fundamental understanding of plant biology. Here we argue that progress in understanding the biological foundations of isoprenoid emissions is sufficient to propose a simple conceptual model of isoprene emission that, we believe, will allow construction of a process-based model that will not require a proliferation of empirical specifications.

The emerging new understanding of volatile isoprenoid emissions originates in disparate fields including molecular biology, plant physiology, chemical ecology and atmospheric science. By combining evidence on the regulation of isoprene and monoterpen production with current understanding of their function in plants, we can explore the controls of emissions in a more fundamental way. Well-established findings include the ubiquity of the gene encoding the monoterpene synthase enzyme (Tholl et al., 2011), in contrast with isoprene synthase which is found in a more limited number of higher plant clades (Sharkey & Yeh, 2001); the high short-term sensitivity of isoprene emission to temperature (reviewed in Arneth et al., 2010 and Niinemets et al., 2011); and the inhibitory effect of high CO₂ concentrations on isoprene emission (e.g. Calfapietra et al., 2008; Wilkinson et al., 2009; Possell & Hewitt, 2011). Recent discoveries (reviewed in Loreto & Schnitzler, 2010) include confirmation of the long-hypothesized protective function of isoprene emission (against high-temperature stress and reactive oxygen species (ROS)), established through genetic manipulation experiments (Behnke et al., 2007; Velikova et al., 2011), and studies of atmospheric column concentrations of formaldehyde (HCHO) – a major isoprene oxidation product – which show the dominant role of temperature variations in determining BVOC emissions at the ecosystem level. Based on current understanding of the physiological and environmental controls on isoprenoid synthesis, we introduce key elements of a new quantitative modelling approach that, while being parsimonious, is firmly based on experimental plant biology. We consider regulation of emissions at the biochemical level first, then the function and control of emissions at the whole-plant level, and finally the emergent behaviour of emissions in ecosystems as seen ‘top-down’ from the atmosphere.

Biochemical controls and trade-offs
Isoprene and monoterpenes are synthesized via the plastidic 2-C-methyl-D-erythritol 4-phosphate (MEP) pathway (Lichtenthaler, 1999), which is also the beginning of the synthesis route for essential metabolites including abscisic acid, photoprotective compounds (carotenoids and tocopherol) and the phytol side-chain of chlorophyll. Demand for the various downstream products of the MEP pathway can be a significant drain on photoassimilates, energy supply and reducing power (Loreto & Sharkey, 2004; Owen & Peñuelas, 2005; Li & Sharkey, 2012). When the production of different metabolites is viewed in terms of the overall allocation of carbon and energy supplies, trade-offs between the attainment of optimal photosynthetic rates, components of the photosynthetic apparatus (e.g. light-harvesting pigments) and secondary metabolites (e.g. volatile isoprenoids) are seen to be inevitable.

The regulatory network components of the MEP and associated pathways are illustrated in Fig. 1(a) (see also Supporting Information Notes S1). In the accompanying conceptual model (Fig. 1b), we distinguish short (seconds to minutes, A), medium (hours to days, B) and long (weeks to years, C) time-scales of regulation. On short time-scales, constitutive emission levels are linked to substrate availability. Isoprene emission, in particular, is strongly determined by the pool size of one of its immediate precursors, dimethylallyl pyrophosphate (DMAPP) (Rasulov et al., 2009a). Substrate-induced surges through the MEP pathway under non-steady-state conditions are constrained by key reactions such as the conversion of methylerythritol cyclodiphosphate to hydroxymethylbutenyl diphosphate (Li & Sharkey, 2012). On medium time-scales, transcription rates of enzymes come into play (Mayrhofer et al., 2005). Most genes in the MEP pathway have light-regulated circadian elements (Cordoba et al., 2009) along with putative heat-shock promoter elements upstream of their initiator codon (Sharkey et al., 2008). Hence, it is likely that transcriptional regulation of isoprenoid emission occurs during transient periods of heat and oxidative stress. On long time-scales, activation of genes from other pathways (e.g. carotenoid biosynthesis) needs to be considered as linked to volatile isoprenoid emission (Owen & Peñuelas, 2005).

The adaptive significance of isoprenoid emissions
Isoprene is thought to fulfil a protective role in isoprene emitters, either by quenching ROS that are produced in response to high temperatures and excessive light intensities as well as to externally imposed oxidative stress (e.g. high ozone concentration) (Vickers et al., 2009a; Jardine et al., 2011) or by stabilizing thylakoid membranes (Velikova et al., 2011). Recent experimental evidence suggests that stabilization of thylakoid membranes by isoprene reduces the formation of ROS (Velikova et al., 2012). Monoterpenes are known to have a wider variety of functions, including signalling and deterrence of herbivory (Duce & Baldwin, 2010). Some species store monoterpenes in specialized organs, releasing them by evaporation in response to warming or after mechanical stresses. But many other species emit monoterpenes constitutively in a similar light- and temperature-dependent way to isoprene. It has been proposed that the emission of monoterpenes in these species has a similar function to that of isoprene. Monoterpene emission is elicited by stresses (Loreto et al., 2004). Even in normally non-emitting species, emissions of monoterpenes can be induced by stress (Niinemets et al., 2010), and stress may induce monoterpene emissions instead of isoprene emission (Brilli et al., 2009).

Thus, an emerging view is that volatile isoprenoids in general are important agents in cellular protection from ROS generated during stress events that impair optimal coupling of light and dark reactions within the chloroplast (Loreto & Schnitzler, 2010; Velikova et al., 2012). Although a wide range of strategies to cope
with oxidative stress has evolved in plants, the induction of volatile isoprenoids is important because it can be activated rapidly.

The idea of functional ‘substitutability’ between isoprene and monoterpenes is consistent with the marked L-shape pattern shown in Fig. 2, which illustrates that species with moderate to high volatile isoprenoid emissions predominantly (or entirely) emit either isoprene or monoterpenes at any given time (the data in Fig. 2 are from studies in which both were measured; see Notes S2). The L-shape pattern is largely driven by species that do not store monoterpene. In species that emit both isoprene and monoterpenes simultaneously, the trade-off is manifested as an inverse relationship between the two emission capacities (inset, Fig. 2). This inverse relationship may reflect a competition between monoterpenes and isoprene biosynthesis for common precursors and reducing power.

Plants that do have isoprene synthase preferentially emit more isoprene than monoterpenes. This makes the lower affinity of isoprene synthase for its substrate DMAPP (Michaelis–Menten coefficient \( K_m \) of 0.5–2.5 mM; see Notes S1) compared with that of geranyl pyrophosphate synthase (GPS) for isopentenyl pyrophosphate (IPP) and DMAPP (< 0.05 mM) somewhat puzzling. Moreover, the affinity of monoterpenene synthase for geranyl pyrophosphate (GPP) synthesized by GPS is even higher (\( K_m \) of 0.006 mM). The substrate pool size of GPP is estimated to be much larger than that of DMAPP, suggesting that the kinetics of GPP synthase strongly influences GPP availability for

Fig. 1  (a) Summary of the biosynthetic pathways closely linked to plant isoprenoid emission via the 2-C-methyl-D-erythritol 4-phosphate (MEP) pathway. (b) Conceptual representation of diversity in constitutive and inducible emission capacities amongst plant genera juxtaposed with biochemical regulatory elements and their operating time-scales. In (a) and (b), zone A corresponds to constitutive emission controlled by substrate pool limitation; B denotes the transcriptional controls on transient maximum emission; C identifies significant pathway trade-offs influencing, for example, seasonal variability. (See Supporting Information Notes S1 and S5 for references.)
downstream processes. It is unclear how adequate substrate is still available for isoprene synthesis, given the bias towards synthesis of IPP over DMAPP, in emitting species. It is possible that the DMAPP pool co-limits isoprene and GPP synthesis, depending on the diurnal and seasonal variations in the relative $V_{\text{max}}$ (maximum enzyme reaction rate) values for the two enzymes (Fig. 1).

### Isoprenoid emissions and plant ecological strategies

There are broadly predictable ecological trends in the predominance of isoprene vs monoterpane emissions (Fig. 3). The strongest association is with species’ shade tolerance, which is an indicator of successional status. Early successional and light-demanding plants have a greater tendency to produce isoprene (Fig. 3a). This reflects the fact that coping with oxidative stress and/or high temperature is likely to be a greater challenge in higher irradiance or higher-light sites, and that there are few ‘excess’ electrons (and carbon chains) for isoprene production in low light. Isoprene emission is also associated with leaf traits characteristic of species with rapid growth in high-resource (including high-light) environments, including high photosynthetic capacity ($A_{\text{max}}$) (Fig. 3b), short leaf lifespan (Fig. 3c), and high specific leaf area (SLA) (Fig. 3d). Investment in isoprene, being costly because of its high volatility, can be viewed as a fast-response strategy that pays off in an environment where transient stress is frequent or as a component of a fast-growth strategy in nonlimited resource environments. By contrast, investment into monoterpenes could be seen as more efficient under conditions where constitutive emissions are required over long periods, for example to confer protection in longer-lived leaves against herbivory.
Decoupling of isoprenoid emissions and photosynthesis

Although isoprenoid emissions are dependent on photosynthesis for the supply of energy (ATP), reducing power (NADPH) and carbon skeletons, several environmental and ontogenetic factors decouple the two processes. For instance, both high temperatures and soil water deficits reduce photosynthesis, while isoprene emission can continue at a high level (reviewed in Niinemets, 2010). Isoprene emissions from leaves grown at above-ambient CO₂ concentration are inhibited, while emissions from leaves grown at subambient CO₂ concentration are increased (see next section). Light-dependent isoprene emission has been observed from leaves that have been severed from the stem and ceased to photosynthesize (Loreto & Schnitzler, 2010; Brilli et al., 2011). Finally, a lag between photosynthesis and isoprene emission in developing leaves has been reported in several temperate and tropical plants (see Table S1).

The lag between photosynthetic competency and measurable isoprene emission is not simply a function of leaf expansion. It varies from a few days to several weeks, depending on the growth temperature (see Notes S3 and Table S2) and appears to be under transcriptional control, that is, there is delayed expression of the gene encoding isoprene synthase (Wiberley et al., 2005; Sharkey et al., 2008). The adaptive significance of this genetically programmed delay is uncertain. We suggest that it is linked to the priority for synthesis of more essential isoprenoids during early leaf development. The picture is less consistent in ageing leaves: some studies suggest that the biochemical capacity to produce isoprene is unaffected by senescence, some that isoprene declines before photosynthesis, and some that measurable isoprene emission persists in senescing leaves even after the cessation of photosynthesis (Table S2). In poplars (Populus × euramericana) grown at elevated CO₂, isoprene emission was sustained for longer periods in senescing leaves, while the decline in photosynthesis was accelerated (Centritto et al., 2004). Under these conditions, Tallis et al. (2010) showed increased expression of genes involved in glycolysis, suggesting that PEP (phosphoenolpyruvate) from glycolysis, translocated to the chloroplast, may provide the substrate for sustained isoprenoid emission in senescing leaves (Fig. 1; Loreto et al., 2007).

The delayed onset of volatile isoprenoid emissions is reflected in large-scale observations over vegetation with a strong component of seasonally deciduous trees (see Notes S4). Remotely sensed HCHO concentration can be used as a proxy for regional volatile isoprenoid emission (e.g. Barkley et al., 2009). In temperate latitudes (Fig. 4), HCHO lags the increase of remotely sensed leaf area index (LAI) in spring. There is no such clear signal in the autumn: the decline in HCHO occurred before the decline in LAI in 2007 and after the decline in LAI in 2006. The lags differed between the 2 years analysed, suggesting a possible route to investigate the environmental cues involved from a ‘top-down’, ecosystem-level perspective. The interpretation of the HCHO signal in the tropics is complicated by the substantial contribution of pyrogenic emissions (Gonzi et al., 2011). Nevertheless, disregarding situations when the peak in HCHO occurs in the dry season when the trees are leafless, tropical deciduous forests and savannas show a quite different pattern from temperate deciduous forests (Fig. 4). Low isoprenoid emissions occur in the wet season when air and canopy temperatures are at a minimum.

Fig. 4 The relationship between normalized leaf area index (LAI), 2-m air temperature in degrees Celsius (2m T AIR) and volatile isoprenoid emissions indexed by the normalized remotely sensed atmospheric column concentration of formaldehyde (HCHO). Data are from a temperate (a, b) and tropical (c, d) broad-leaved deciduous forest during 2006 and 2007. The second HCHO peak in the tropical forest (c, d) is attributed to biomass-burning emissions, as shown by the remotely sensed fire counts (FIRE) (see Supporting Information Notes S4).
Thus, the seasonal cycles of LAI and HCHO are not correlated in the tropics, and HCHO concentration tracks temperature rather than LAI.

### Isoprene responses to CO₂ and drought

The experimentally observed responses of isoprene emissions to CO₂ and water stress are fundamental in the context of global climate change and need to be accounted for in models (Arneth et al., 2007b; Heald et al., 2009; Young et al., 2009; Paciﬁco et al., 2012). The effect of high CO₂ concentration in suppressing isoprene emission is sufﬁcient to strongly reduce or even offset the increase in isoprene emission resulting from high temperatures, which in turn signiﬁcantly affects ozone projections (Young et al., 2009; Paciﬁco et al., 2012).

The effect of CO₂ concentration on isoprene production is in the opposite sense to the CO₂ effect on photosynthesis. Plants grown at high atmospheric CO₂ concentrations emit less isoprene than those grown at lower concentrations (Sharkey et al., 1991; Centritto et al., 2004; Scholeﬁeld et al., 2004). It has been shown that isoprene emission responds to the leaf-internal CO₂ concentration (cᵢ), with lower emission rates associated with higher cᵢ (Monson et al., 2007; Wilkinson et al., 2009; Guidolotti et al., 2011; Possell & Hewitt, 2011). The effect has been found in many plant species, and is persistent – it applies to plants grown in different CO₂ concentrations, as well as in short-term experiments that manipulate ambient CO₂ concentration in order to alter cᵢ and to genotypes with different cᵢ grown under the same CO₂ concentration. Variation of the pool size of DMAPP is the most likely explanation for the CO₂ dependence of isoprene synthesis as seen in short-term experiments (Rasulov et al., 2009b). Although not explicitly identiﬁed, this regulation could occur because of changes in DOXP (1-deoxy-D-xylulose-5-phosphate)/MEP pathway enzymes upstream of isoprene synthase, changes in the energy status of chloroplasts, or competition for DMADP between other key metabolic pathways (Sun et al., 2012). When plants are grown under different CO₂ concentrations, it is likely that transcriptional regulation of isoprene synthase activity is also involved (Scholeﬁeld et al., 2004; Possell & Hewitt, 2011).

We propose a unifying mechanistic hypothesis, based on the requirement of energy and reducing power for isoprene biosynthesis. NADPH is needed in order to produce DMAPP (Rasulov et al., 2011). When photosynthesis is electron transport-limited (at high cᵢ and/or low light), the shortfall of ATP and NADPH for CO₂ assimilation will cause a deﬁcit of reducing power to transform carbohydrates into DMAPP. When photosynthesis is Rubisco-limited (at low cᵢ and/or high light), the plant will use a part of the excess of ATP and NADPH (resulting from the excess of electrons produced by photochemical reactions) to reduce carbohydrates to DMAPP. This hypothesis predicts that isoprene emission will increase in response not only to excessively high temperatures but also, more generally, to any situation where light availability exceeds photosynthetic capacity. The fraction of the carbon assimilation allocated to isoprene production increases with light intensity, even when photosynthesis is light-saturated (Sharkey & Loreto, 1993), consistent with this hypothesis. An important corollary is that stomatal closure in response to dry conditions will increase isoprene emission by lowering cᵢ and thereby increasing the supply of electrons relative to the carbon ﬁxation rate. Therefore, this hypothesis simultaneously accounts for observed responses of isoprene emissions to CO₂, and to drought – because under drought conditions cᵢ is reduced, and photosynthetic carbon ﬁxation is reduced, while electron transport is maintained.

The simple model proposed here is based on the Farquhar & Von Caemmerer (1982) model of leaf photosynthesis, which assumes that photosynthesis is limited by either the electron transport supply or the rate of carbon ﬁxation by Rubisco. The excess or deﬁcit of electrons produced by photochemical reactions during photosynthesis can be calculated as the difference between the total photosynthetic electron ﬂux and the total ﬂux of electrons used for carbon assimilation (Fig. 5a). We argue that the rate of isoprene emission (i; nmol m⁻² s⁻¹) depends on excess reducing power, which is increased by the electron-transport supply (J; µmol e⁻ m⁻² s⁻¹), and reduced by the electron-transport requirement of the dark reactions of photosynthesis, calculated as

\[
V_{\text{max}} \frac{C_i + 2\Gamma^*}{C_i + K_m} \quad \text{Eqn 1}
\]

where \( V_{\text{max}} \) is the maximum rate of Rubisco activity, \( C_i \) is the intercellular CO₂ concentration, \( \Gamma^* \) is the CO₂ compensation point in the absence of dark respiration, and \( K_m \) is the Michaelis–Menten coefficient.

The isoprene emission rate is thus given by

\[
i = aJ - bV_{\text{max}} \frac{C_i + 2\Gamma^*}{C_i + K_m} \quad \text{Eqn 2}
\]

where \( a \) and \( b \) are empirical parameters: \( a \) represents a maximum fraction of the total photosynthetic electron ﬂux used for isoprene synthesis, while \( b \) represents the rate at which isoprene synthesis is diminished in proportion to the demand set up by the dark reactions of photosynthesis. Preliminary comparisons with isoprene emissions from two experimental species shows that this modelling approach reproduces observed responses to temperature (Fig. 5b), light intensity (Fig. 5c) and CO₂ (Fig. 5d). This approach is attractively parsimonious, and consistent with the idea that isoprene production is not tightly linked to carbon assimilation. It requires further testing (e.g. for the response to simultaneous perturbations of different factors). Whether it is also applicable to monoterpenes emissions, especially in species that emit these compounds in an isoprene-like fashion, also needs to be examined.

### Towards greater scientific integration

The processes involved in regulating the emission of volatile isoprenoids operate on many time and space scales, and are consequently studied within several disciplines that do not habitually communicate. Integration across disciplinary boundaries is
necessary to develop a more comprehensive understanding of these processes. In particular, better integration of modelling, remote sensing, and experimental plant biology is important to overcome the current paradoxical situation whereby models linking emissions, atmospheric chemistry, and climate are strongly data-driven, yet hampered by the lack of data on both the short time-scales needed for derivation of environmental response curves and the longer time-scales needed to characterize the response to environmental change.

The concept of trade-offs at the biochemical level provides a useful framework for explaining and modelling isoprenoid emissions. But more, and more systematic, observations are needed. There are too few measurements of enzyme activities and substrate pools across emitting and non-emitting species to allow cost–benefit analysis for the different ‘flavours’ of isoprenoid emission. There has been insufficient analysis of the relative costs of alternative stress-protection strategies. Gaps also remain in process understanding at the cellular level. We still do not know, for example, what controls the transport of substrates between the cytosol and the chloroplast.

Plant trait databases that include geo-located records (e.g. Katte et al., 2011) make it possible to explore the relationships between emissions and other plant characteristics. Such investigations could help to clarify further the interaction between ecological strategies and emissions, or to analyse trade-offs at the species level – for example, what strategies to deal with oxidative stress are used by non-emitters – and to discern macro-scale relationships between emissions and environment.

In response to the need to develop a more biologically robust way of simulating plant emissions (Monson et al., 2012), we have proposed a simple concept for modelling the emission of volatile isoprenoids that is consistent with current understanding of physiological mechanisms, including dependence on reducing power (Li & Sharkey, 2012), and with a diverse set of observations. Although some earlier models have simulated isoprenoid emissions based on assigning a fraction of the total electron transport to the process (e.g. Niinemets et al., 1999; Arneth et al., 2007a), they required empirical modifications to represent CO2 and drought effects (Monson et al., 2012). While our model appears to capture the expected responses to temperature, light and CO2, it remains to be shown whether it can reproduce features such as the large diurnal range in emissions, seasonality induced by temperature and water stress variations, short- and long-term responses to CO2 concentration, and global patterns emerging from satellite observations of HCHO and other BVOC oxidation products. Nevertheless, application and extension of this framework should provide more robust estimates of isoprenoid emission in a changing environment.

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Fig. 5 Characteristic responses of isoprene emission to temperature, incident photosynthetically active radiation (PAR) and intercellular CO2 concentration captured by a simple model of ‘excess’ reducing power within the chloroplast. (a) At low temperatures, photosynthesis is limited by electron transport availability (dashed line), whereas at high temperature photosynthesis is limited by Rubisco activity (solid line), resulting in (b) a steep increase in excess reducing power and isoprene emission with temperature (data show measured photosynthesis and isoprene emission in tobacco (Nicotiana tabacum) (Vickers et al., 2009a); error bars represent 5E). (c) Increase in isoprene emission with PAR can be attributed to the increase in reducing power (data from Vickers et al., 2009b). (d) Decrease in isoprene emission with increasing internal CO2 concentration can be attributed to an increase in photosynthetic rate which consumes reducing power (data from Possell & Hewitt, 2011).
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References


Supporting Information

Additional supporting information may be found in the online version of this article.

Table S1 Information on $K_{m}$ values

Table S2 Observational or experimental evidence for lag in emissions

Notes S1 Source of $K_{m}$ data.

Notes S2 Trait and isoprene database.

Notes S3 Experimental and field evidence for seasonal leads and lags between isoprene emission and photosynthesis.

Notes S4 Analysis of isoprene emissions at a regional scale.

Notes S5 References.

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