Chapter 12
Leaf-Level Models of Constitutive and Stress-Driven Volatile Organic Compound Emissions

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Abstract This chapter provides a review of past and contemporary leaf-level emission algorithms that have been and currently are in use for modelling the emissions of biogenic volatile organic compounds (BVOCs) from plants. The chapter starts with a brief overview about historical efforts and elaborates on processes that describe the direct emission responses to environmental factors such as temperature and light. These phenomenological descriptions have been widely and successfully used in emission models at scales ranging from the leaf to the globe. However, while the models provide tractable mathematical functions that link environmental drivers and emission rates, and as such can be easily incorporated in higher scale predictive models, they do not provide the mechanistic context required to describe interactions among drivers and indirect influences on interactions such as those due to acclimation, accumulated stress and ontogeny. Following a discussion of these issues and the limitations they impose on the current state of model-based prognoses of BVOC emissions, we describe in some detail the knowledge gaps that need to be filled in order to move BVOC emission models into forms that are more directly coupled to physiological processes.

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12.1 Introduction

A diverse range of plant species has the capacity to emit biogenic volatile organic compounds (BVOCs), in particular volatile isoprenoids, in a constitutive manner. These emissions can either come from specialized storage structures, reflecting slow vapourization and diffusion of volatiles synthesized days, weeks and months prior to emission ("storage" emissions such as those from storage structures that play an important role in direct plant defence), or from continuous physiological processes ("persistent physiological emissions" such as methanol emissions from expanding leaves). The key characteristic of constitutive emissions is that they are not dependent on induction by external triggering factors, such as herbivory or abiotic stress. In contrast, induced emissions result from an upregulation of key metabolic pathways in response to external cues, thereby leading to elicitation of BVOC emissions even in species lacking constitutive emissions. Induction of emissions, in particular, includes the upregulation of gene expression to produce additional enzymatic activity, e.g., the induction of genes for terpene synthases in response to insect attack, (Litvak and Monson 1998; Arimura et al. 2000; Li et al. 2002; Babst et al. 2009). In the case of constitutively emitted compounds, such as isoprene and monoterpenes, environmental cues typically alter the level of expression of key controlling enzymes (Wiberley et al. 2008, 2009), complicating separation of stress-triggered and constitutive emission responses. The various timescales across which emissions are influenced, and the interactions of multiple cellular processes in determining the capacity for emissions has created challenges to describing BVOC emissions in mathematical representations, and thus, in producing prognostic models that reflect metabolic and physiological first principles.

In some of the processes that govern emissions we have, in fact, made progress in linking emissions to true physico-chemical theory. For example, chemical properties of volatiles responsible for stomatal control of emissions have been identified (Niinemets et al. 2004; Niinemets and Reichstein 2003), and temperature dependence of many BVOC emissions has been described in terms of fundamental kinetic theory (see Grote and Niinemets 2008; Monson et al. 2012). However, the mechanistic basis for alteration of BVOC emissions by growth in different light or temperature regimes, the influence of drought stress, in either the short- or long-term, and the influence of leaf ontogeny remains largely unresolved (see Monson 2013 in this volume). Furthermore, induction of emissions following biotic or abiotic stress events has not yet been included in emission models, although cellular signalling models have been developed that simulate alterations in gene expression with relatively good success (Vu and Vohradsky 2007; Yip et al. 2010; Muraro et al. 2012).

Apart from the importance of detailed understanding of the emission mechanisms that determine the emissions in the timescale of seconds to minutes, the pathway flux also depends on longer-term emission controls associated with the changes in the capacity of the volatile synthesis pathway. Modelling seasonality has been an especially difficult task, because seasonal variations also involve variations in light and temperature, effects which are hard to disentangle from acclimation responses.

Furthermore, seasonal studies conducted in natural environments inevitably also incorporate long-term stress effects such as soil drought. Embedded within these entanglements are the summed effects of cell birth and death, both of which are controlled by intrinsic 'clocks' as well as programmed responses to stress. We know these synergies exist; we just don't know how to represent them accurately in mathematical representations. The mismatch between our general knowledge of the phenomenology of the processes, and our knowledge about the stoichiometries that determine process rates and feedbacks, has left us with little on which to base our models. In this knowledge gap, and facing pressure from agencies and governments to produce actionable projections of future changes in our environment, we have produced models that work well for replicating observed emissions patterns and dynamics. However, we also know they have limited power to be used in truly prognostic mode, especially if the emission projections have to be made to future conditions and to areas with limited information of species emission characteristics and physiological performance.

In this chapter, we analyse the origin and development of both empirical and semi-mechanistic emission model algorithms to simulate volatile emission responses to immediate variations in empirical drivers. We try to emphasize the gaps in knowledge that cause our projections of BVOC emissions to be bracketed with relatively large uncertainties. Then we consider the knowledge needed to fill some of these gaps, and incorporate especially the long-term influences, such as acclimation mechanisms, and seasonality, in emission models. Finally, we suggest ways to improve our representation of induced emissions in our models – that is, how to design the models to respond to episodic forcing elements in the environment. In assessing the conflicting states of existing knowledge and the need for reliable projections, we end up concluding that the expansion of models to include interactions such as acclimation and ontogeny is a valuable step forward, as it allows for the establishment of a recognized framework within which we must cast our projections. However, we also argue that the limitations of this approach must be broadcast in a more amplified form. It is a dangerous situation to assume that because a model includes a scheme for acclimation or induction, it is in a form capable of more accurate prognosis. We emphasize that new approaches must be developed for assessing the uncertainties created by the gap between knowledge about the existence and basic operation of a certain process, and the exact controls by which the model links emission rates to physiological and environmental drivers.

12.2 Modelling Environmental Dependencies

12.2.1 Brief History of Leaf-Level Emission Models

The complexity of direct emission control represented a big challenge for modelers. Although Sanadze and Kalandaze (1966) reported the dependency of isoprene emission rate on temperature and light long ago, it took more than a decade to

produce the first mathematical relation to describe this dependency (Tingey 1979; Tingey et al. 1981). In developing this first mathematical model, it was noted that isoprene emission in studied broad-leaved species responded to temperature as well as to light, while monoterpene emissions in studied conifers only responded to temperature (Tingey et al. 1980). Thus, monoterpene emission was assumed to originate solely from storage pools within the plant (e.g., oleoresin) that provide an unconstrained source, at least in the case of emissions over minutes to hours to days. Accordingly, most of the control over the short-term monoterpene emission rates was relegated to diffusive resistances. The mechanism of isoprene production was clarified through the studies of Monson and Fall (1989) who highlighted the relationship to photosynthesis (see also Loreto and Sharkey 1990; Monson et al. 1992, 1994). Recognition of this relationship allowed Guenther et al. (1991, 1993) to develop models for leaf-scale isoprene and monoterpene emissions based on the shapes of the light and temperature response curves previously used in photosynthesis models (e.g., Farquhar and von Caemmerer 1982; Tenhunen et al. 1976). Both photosynthesis as well as isoprene emission show an optimum relationship to temperature and a saturation response to increasing light. The temperature optimum of BVOC emission is, however, high compared to most physiological processes (Fig. 12.1a, b). In contrast, the light dependency of isoprene emission is similar to that of photosynthesis (Fig. 12.2a, b). Thus, while the metabolic linkages between photosynthesis and isoprene emission were clear, there was also evidence that isoprene biosynthesis has a unique set of controlling processes that could not be ignored. Later, it was recognized that some species emit monoterpenes that are tightly coupled to their biosynthesis in the chloroplast, and thus, the monoterpene emission mechanisms in these species are similar to isoprene emission mechanisms (Schürmann et al. 1993; Staudt and Seufert 1995). Shortly after the first comprehensive emission models were presented, the main biosynthetic pathway of volatile isoprenoid production in plant plastids, 2-C-methyl-D-erythritol 4-phosphate/1-deoxy-D-xylulose 5-phosphate pathway (MEP/DOXP pathway), was discovered (Lichtenthaler et al. 1997; Rohmer et al. 1993; Eisenreich et al. 2001). Given the central role of the MEP/DOXP pathway, the following efforts of mechanistic and semi-mechanistic BVOC emission model development have primarily focused on understanding linkages to photosynthesis and controls over kinetics within this pathway (Niinemets et al. 1999; Martin et al. 2000; Zimmer et al. 2000).

Shortly after the first leaf-scale models were published, global-scale modelers began to incorporate some of the schemes into projections at scales with considerably longer time and greater space intervals (e.g., Guenther et al. 1995). These projections were inherently constrained by the bottom-up approach, because in this framework there was little potential to validate model predictions. Even within the context of atmospheric chemistry, large gaps in our knowledge, for example of the degree of molecular oxidation of isoprene and the deposition of oxidation products, precluded validation of projected global emission rates. Despite acknowledgement of large uncertainties, the models continued to be expanded in terms of the processes they included (e.g., Fuentes and Wang 1999; Guenther et al.

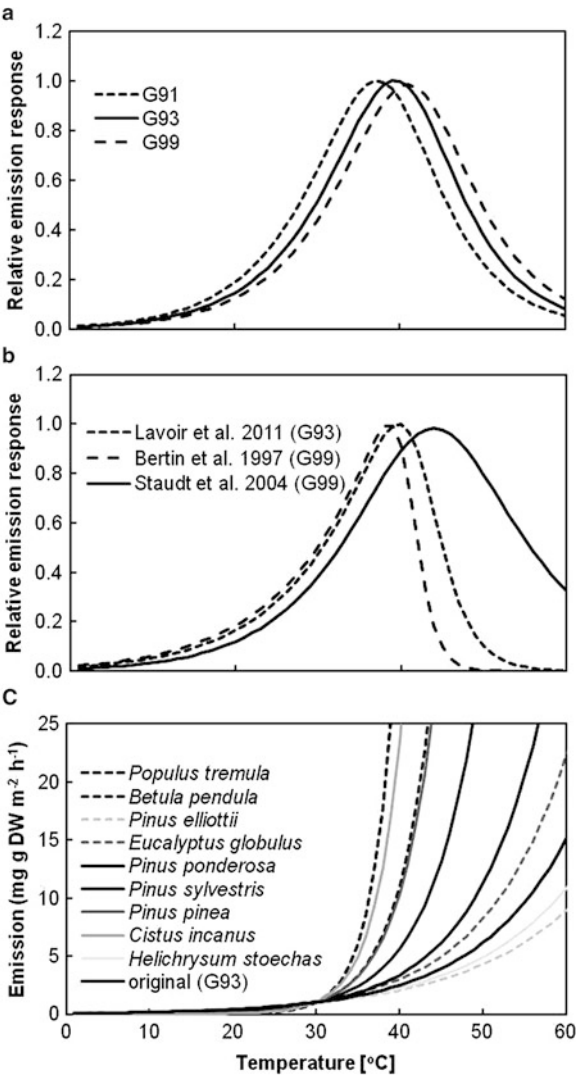

Fig. 12.1 Comparison of shapes of temperature responses of isoprene and monoterpene emissions in species lacking specialized storage structures (**a**, **b**), and monoterpene emissions in species with terpene storage structures (**c**). Panel (**a**) highlights the differences between the various versions of the Guenther isoprene emission algorithm presented in 1991, 1993, and 1999 (G91, G93, and G99), (**b**) compares different monoterpene emission parameterizations in the broad-leaved evergreen sclerophyll (*Quercus ilex*), while (**c**) compares the suggested temperature responses among species (Tingey et al. 1980; Guenther et al. 1991; Holzinger et al. 2006; Ruuskanen et al. 2005; Hakola et al. 1998, 2003; Owen et al. 1997). The *broken lines* correspond to Eq. 12.2 and continuous lines to Eq. 12.3

2000; Müller et al. 2008; Lavoir et al. 2011; Ashworth et al. 2013; Guenther 2013). For example, empirical relationships linking isoprene emission rate to atmospheric CO_2 concentrations (Rosenstiel et al. 2003; Wilkinson et al. 2009) were included in existing global emission models (Arneth et al. 2007; Heald et al. 2009). The 'Model of Emissions of Gases and Aerosols from Nature' (MEGAN) (Guenther et al. 2006, 2012) is currently the most widely used model for projecting global trends in BVOC emissions. MEGAN includes some of the more difficult long-term influences on emissions, such as temperature acclimation, response to drought and

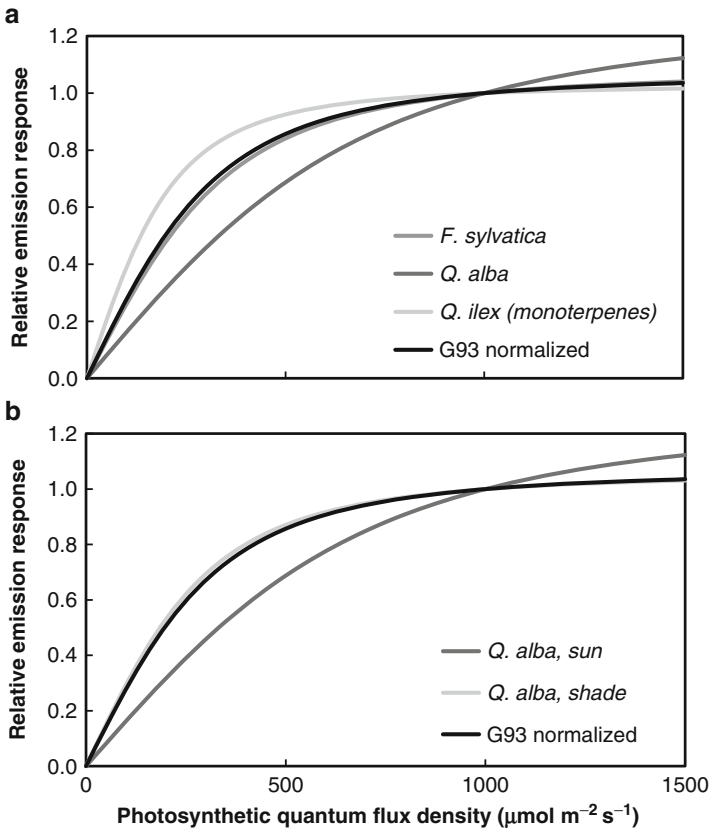

Fig. 12.2 Shapes of light responses of the emissions of isoprene and monoterpenes. Panel (**a**) compares the light response for temperate broad-leaved deciduous trees *Fagus sylvatica* (Schuh et al. 1997), and *Quercus alba* (sun foliage, Harley et al. 2007), and Mediterranean evergreen sclerophyll *Q. ilex* (Lavoir et al. 2011) along with the G93 (Guenther et al. 1993) estimate, while (**b**) shows the light responses for different canopy layers in *Q. alba* from Harley et al. (1997)

ontogeny. However, these schemes are largely non-validated, except for a few case studies. Not yet considered in BVOC emission models are responses to herbivory and pathogen infection, oxidative air pollution stress and soil infertility (Loreto and Schnitzler 2010) and priming of emissions by consecutive and simultaneous stresses or mild stress episode(s) preceding a more severe stress (Niinemets 2010a, b).

One of the more promising approaches to emerge in the past decade with regard to validating model projections and reducing uncertainties is the fusion of satellite remote sensing of formaldehyde and glyoxyl (oxidative products of terpene BVOCs; see Abbot et al. 2003; Palmer et al. 2003, 2006; Ashworth et al. 2013) with emissions models. Inverse modelling of formaldehyde and glyoxyl columns to reveal the locations and magnitudes of BVOC sources and sinks has the potential

to provide new insight into time-dependent interactions between emissions and environmental change, especially at the scales needed to integrate processes from single leaves to regional ecosystems.

In the following sections, we discuss how different environmental drivers are represented in contemporary emission models. Most of these models follow the general idea that there is a certain capacity for the emission of a given compound that depends on the overall diffusion resistance ("storage" emissions) or by the rate of volatile formation ("instantaneous" emissions). The emission capacity can be defined as the maximum emission rate that is physiologically possible (E_{MAX}). For "instantaneous" emissions, this is typically observed at saturating light and a temperature of about 40 °C (see Fig. 12.1a, b; Fig. 12.2). However, no such apparent physiological limitation exists for storage emissions, which are driven solely by volatilization and diffusive resistance. Thus, in emission studies, one often uses a standardized emission rate, E_S (also called the emission factor) that is defined as the emission rate under certain arbitrarily selected environmental conditions (Guenther et al. 1991; Niinemets et al. 2010c). The standard conditions are typically set as the leaf temperature of 30 °C (303.16 K), incident quantum flux density of 1,000 μmol m^{-2} s^{-1} (Guenther et al. 1991, 1993) and ambient CO_2 concentration of 400 μmol mol^{-1} (Wilkinson et al. 2009). Following Guenther et al. (1991), the general form of this approach to estimate the emission rate of a specific compound or compound group, E, can be expressed as:

$$E = E_S f(I_1) f(I_2) \ldots f(I_n), \tag{12.1}$$

where $f(I_i)$ represents different response functions ($i = 1..n$) scaling E_S to environmental conditions other than the standard conditions. These functions are addressed separately in the following paragraphs. Implicit in Eq. 12.1 is that different environmental factors affect the emission rate independently. This is not necessarily valid (e.g., Sun et al. 2012) and will be addressed afterward together with alternative ways of estimating E without the need to specify E_S.

12.2.2 Temperature Dependency

BVOC emissions can originate from specific storages such as resin ducts, or from organelles that are not specifically formed to hold BVOCs (e.g., isoprene from chloroplasts, methanol from cell walls or sesquiterpenes from the cytosol). In some cases, the temperature dependency results from the pathway producing the given compound being sensitive to temperature (here, referring to short-term dynamics in temperature). In other cases, it is the temperature dependency of compound volatility that most determines the emission rate. The first type of temperature dependency is exemplified by the emission of isoprene, methylbutenol, and light-dependent monoterpenes. The second type of temperature dependency is exemplified by the emissions of stored compounds, like the monoterpenes emitted from the resin systems of many conifers. Thus, two separate modelling strategies are developed to represent both types of emission.

12.2.2.1 Temperature Effects on Storage Emissions

These emissions have been described by Tingey et al. (1980) by fitting emissions on a log scale to temperature using a linear relationship:

$$f(T) = \exp\left[p_1 \cdot T + p_2\right] \tag{12.2}$$

with T representing air temperature and p_1 and p_2 being empirical parameters. Guenther et al. (1993) used the following exponential relationship to temperature:

$$f(T) = \exp\left[\beta\left(T_\mathrm{L} - T_\mathrm{S}\right)\right], \tag{12.3}$$

where T_L is the leaf temperature and T_S is the reference temperature (set to 303.16 K = 30 °C) and β is an empirical coefficient. Guenther et al. (1993) set β to 0.09 (K^{-1}) based on observations using 28 species. It should be noted that species-specific estimates ranged from 0.057 to 0.148 in the original Guenther et al. (1993) study, a range that has since been exceeded in other measurements. For example Pokorska et al. (2012) estimated β to be 0.24 for *Abies alba* trees in late summer and Owen et al. (1997) found values larger than 0.3 for *Cistus incanus* plants (see also Fig. 12.1c). In addition, Helmig et al. (2007) showed that β also changes within the canopy. Equations 12.2 and 12.3 have been widely used to describe emissions from storage pools, including those for monoterpenes and sesquiterpenes (e.g., Ormeño et al. 2010).

The implicit assumption in Eq. 12.3 is that the resistance between different types of storage systems and the air is constant. This implies that the storage size is large relative to the emission rate and is not depleted during the emission period – an assumption that has been challenged by the work of Schurgers et al. (2009) who stated that storage emissions in a *Pinus ponderosa* forest could best be described considering a dynamic change in leaf monoterpene concentration during the year.

12.2.2.2 Influence of Temperature on Immediate Emissions

The emissions that are tightly coupled to production, increase with increasing temperature in an exponential fashion up to a maximum rate, thereafter the emissions decrease rapidly, reflecting enzymatic degradation or substrate limitations (e.g., Monson and Fall 1989; Loreto and Sharkey 1990; Monson et al. 1992; Rasulov et al. 2010, 2011). Based on earlier work that relied on chemical kinetics theory such a relation has been postulated by Johnson et al. (1942) to take the form:

$$f(T) = \frac{\exp\left[c_\mathrm{T} - \frac{\Delta H_\mathrm{A}}{R T_\mathrm{L}}\right]}{1 + \exp\left[\frac{\Delta S}{R} - \frac{\Delta H_\mathrm{D}}{R T_\mathrm{L}}\right]} \tag{12.4}$$

where H_A is enthalpy of activation in J mol^{-1}, H_D is the enthalpy of de-activation in J mol^{-1}, S is an entropy term and R is the universal gas constant both in J K^{-1} mol^{-1}, and c_T is a scaling constant. Following the form of this relationship, but substituting the enthalpy and entropy terms with empirically derived parameters, Guenther et al. (1991) rewrote the equation as:

$$f(T) = \frac{\exp\left[\frac{c_{T1}(T_L - T_S)}{RT_L T_S}\right]}{1 + \exp\left[\frac{c_{T2}(T_L - T_M)}{RT_L\ T_S}\right]} \tag{12.5}$$

where c_{T1} (J mol^{-1}), c_{T2} (J mol^{-1}) and T_M (K) are 'tunable' coefficients. Guenther et al. (1999) modified the form of Eq. 12.5 to refer directly to the temperature optimum T_{opt} (K) rather than to T_S:

$$f(T) = c_{T3}\frac{\exp\left(c_{T4}\ x\right)}{c_{T3} - c_{T4}\left(1 - \exp\left(c_{T3}\ x\right)\right)} \tag{12.6}$$

$$\text{where } x = \frac{\left(1/T_{opt}\right) - \left(1/T_L\right)}{R}$$

The parameters c_{T3} and c_{T4} (both in J mol^{-1}) are again empirically determined coefficients. Mathematically, Eqs. 12.5 and 12.6 are equivalent to Eq. 12.4, considering that some differences are absorbed within the coefficients (see Monson et al. 2012).

Equation 12.4 was also applied in the models of Niinemets et al. (1999) and Martin et al. (2000) for describing the temperature response of isoprene synthase and Niinemets et al. (2002c) for describing the temperature response of monoterpene synthases. Zimmer et al. (2000) used it to characterize the temperature dependency of isoprene formation from precursor substances. In later work, the Zimmer et al. (2000) model was applied to other isoprenoids using the same temperature response, but with parameters determined independently for various processes within the MEP pathway (Grote et al. 2006). In their studies, these parameters were derived through an inverse modelling approach whereby the 'best-fit' parameter values were determined after assimilating enzyme temperature dependencies into the model. The temperature dependence of isoprene synthase activity was based on in vitro estimations of synthase activity in crude leaf extracts of *Populus tremuloides* (Monson et al. 1992) and *Quercus robur* (Lehning et al. 1999), and for monoterpene synthase extracts of *Quercus ilex* (Fischbach et al. 2000).

12.2.3 Light Dependency

The original Guenther et al. (1991) model was developed on the basis of numerous studies of Sanadze (1964), Tingey et al. (1981), Monson and Fall (1989) and Loreto and Sharkey (1990) that indicated a functional linkage between photosynthetic CO_2 assimilation and the formation of some BVOCs, especially isoprene. It had

been apparent that absorbed photon flux density is the principal driver for this similarity, suggesting that both processes occurred in the chloroplast, both processes exhibited similar shapes in their light-response curves, and both processes required the input of reductant from the photosynthetic electron transport system. The light dependency of the thylakoid electron transport rate (J, μmol m^{-2} s^{-1}) can be described mathematically as:

$$J = 0.5\, a\, I\, (1 - b) \tag{12.7}$$

where a is the fraction of incident photosynthetic photon flux density (I in μmol m^{-2} s^{-1}) absorbed by the leaf, and b is the fraction of the absorbed photon flux diverted to processes other than photosynthetic electron transport. Implicit in Eq. 12.7 is that J is not saturated by the absorbed I. As electron transport starts to become light-saturated with increasing light intensity, the dependence of J on I will begin to exhibit a curvilinear shape. Recognizing that J is influenced by an upper limit (J_{max}, μmol m^{-2} s^{-1}), and recognizing that the influence of J_{max} on J increases as I increases, the following quadratic equation has been developed for photosynthesis:

$$0 = J^2 - [0.5\, a\, I\, (1 - b) + J_{max} + \Theta]\, J + 0.5\, a\, I\, J_{max}\, (1 - b) \tag{12.8}$$

where Θ is a tunable 'curvature factor' that theoretically can vary from 0 (rectangular hyperbola) to 1 (Blackman type response) with a default value of 0.85 corresponding to moderate curvature. Equation 12.8 describes a rectangular hyperbola in which a continuous transition occurs from $J = 0$ at $I = 0$ to $J = J_{max}$ at saturating I (Farquhar and Wong 1984).

Guenther et al. (1991) used Eq. 12.8 to develop an analogue model to describe the light dependency of isoprene emission (as a multiplication factor for Eq. 12.1):

$$f(I) = \frac{x - \sqrt{x^2 - 4\, b\, aI\, c_{L1}}}{2\, c_{L1}} \tag{12.9}$$

where $x = b\, aI + c_{L1} + c_{L2}$.

The parameters c_{L1} and c_{L2} are tunable coefficients that account for the differences in molar stoichiometry of electron transport between isoprene formation and CO_2 and reflect the curvature coefficient (Θ) and the upper limit of the function formerly defined by J_{max}. Note that the meaning of the coefficients a and b is also different for the isoprene light response than for the light response of J. In later publications, Guenther et al. (1993) used a new form for J, aligning it with a mathematical expression of the so-called Smith's (Smith 1937) equation of photosynthesis (see Tenhunen et al. 1976; Harley et al. 1992):

$$J = \frac{\alpha\, I}{\sqrt{\left(1 + \frac{\alpha^2 I^2}{J_{max}{}^2}\right)}} \tag{12.10}$$

where α is the initial slope of the response carrying the units of mol mol^{-1} photons incident to the leaf (the apparent quantum yield). Equation 12.10 defines the shape of a rectangular hyperbola that approaches an asymptote at relatively high values of I. Adopted from this expression Guenther et al. (1993) described the light dependency of BVOC (isoprene) emission by removing J_{max} and adjusting the function with an additional parameter (c_{L3}). However, as Monson et al. (2012) pointed out, there was a mathematical violation in the denominator in that the square root quantity contains a sum that mixes a unitless constant (1.0) with the product of two terms (α and I) that carry units. Thus the equation should be written as:

$$f(I) = \frac{\alpha_{ISO}\, c_{L3}\, I}{\sqrt{\left(1 + \frac{\alpha_{ISO}^2 I^2}{c_x^2}\right)}} \tag{12.11}$$

where c_{L3} is now in m^2 s μmol^{-1}, and α_{ISO} now carries units mol mol^{-1} photons incident to the leaf, while c_x is in μmol m^{-2} s^{-1}. It can be set to 1.0 to represent the same response as before. The coefficients for α_{ISO} and c_{L3} in Eq. 12.11 were assumed to be constant in the Guenther et al. (1993) analysis, but it was later realized that they can vary within the canopy (Fig. 12.2), partly reflecting the explicit connection between the emission capacity and α_{ISO} (Monson et al. 2012), and partly reflecting acclimation within the canopy (Sect. 12.3.2).

Niinemets et al. (1999) followed a different path and related the emission rate to light using an explicit connection with J. They have therefore used an expression of J limited by ribulose-1.5-bisphosphate (RuBP) regeneration related to net CO_2 assimilation, A, μmol m^{-2} s^{-1}:

$$J = \frac{(A + R_D)\,(4\,C_i + 8\,\Gamma^*)}{C_i - \Gamma^*} \tag{12.12}$$

where C_i is the CO_2 mole fraction in the intercellular air spaces of the leaf, R_D is the rate of non-photorespiratory respiration rate in light (μmol m^{-2} s^{-1}) (mostly mitochondrial respiration), and Γ^* is the hypothetical CO_2 compensation point in the absence of R_D that depends on Rubisco kinetic characteristics. Using this relation, Niinemets et al. (1999) linked the emission rate (E) to photosynthetic electron transport rate. However, expressing J from Eq. 12.12 only yields the rate of photosynthetic electron transport that is needed to reduce CO_2 to the level of immediate photosynthetic product, sugars, and that needed for photorespiration. As isoprene is a more reduced molecule than sugars, additional reductive equivalents are needed to synthesize isoprene. Including this additional electron transport rate, the rate of isoprene emission, E_{iso}, was expressed as (Niinemets et al. 1999):

$$E_{iso} = \varepsilon\, J_T \frac{(C_i - \Gamma^*)}{6\,(4.67\,C_i + 9.33\Gamma^*)} \tag{12.13}$$

where J_T is the total electron transport rate, i.e., that used for CO_2 fixation and photorespiration plus that needed for additional reduction of sugars to the level of

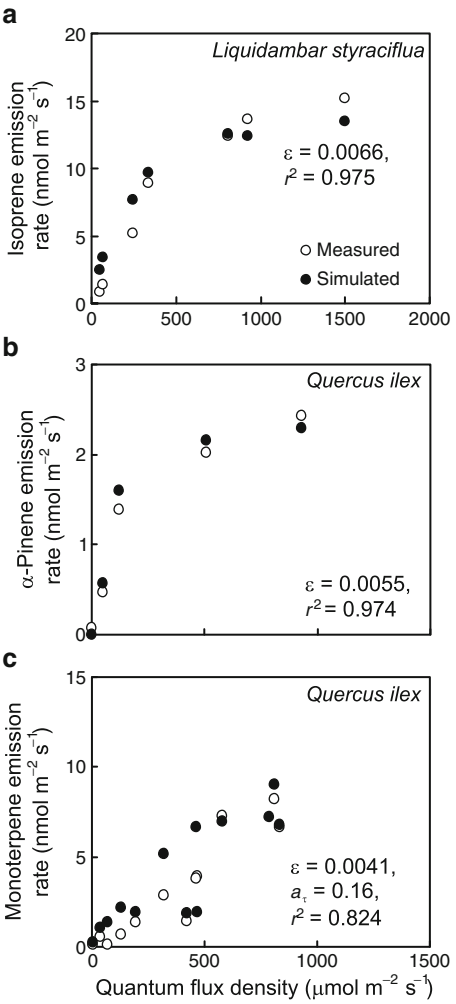

Fig. 12.3 Comparison of measured and simulated light dependencies of isoprene emission rate (**a**), α-pinene (**b**) and total monoterpene emission rate (**c**) in broad-leaved deciduous tree *Liquidambar styraciflua* (**a**), and in evergreen sclerophyll *Quercus ilex* (**b**, **c**). In (**a**), the measurements were conducted at a constant leaf temperature of 25 °C (Data from Niinemets et al. 1999), in (**b**) at 30 °C (Data from Loreto et al. 1996), and in (**c**), the data were filtered from daily time-courses of monoterpene emission measured between Aug. 3–5, 1994, for a temperature range of 25–35 °C (Niinemets et al. 2002c). The data were fitted by Niinemets et al. (1999, 2002c) isoprenoid emission model (Eq. 12.14). Only one leaf-dependent coefficient, ε, the fraction of electrons in isoprenoid synthesis, was used in (**a**), and (**b**), while the dependence of ε on leaf temperature was considered in (**c**) using an exponential scaling coefficient (a_τ (Niinemets et al. 1999, 2002c)

isoprene, and ε is the fraction of J_T required to synthesize isoprene. The dependence of J on I was modelled using Eq. 12.10 and the resultant value of J was inserted into Eq. 12.13. In this equation, the connection between isoprene emission and photosynthetic electron transport results from the assumption that NADPH availability controls the rate of isoprene biosynthesis, although an analogous dependence on ATP availability has also been formulated (Niinemets et al. 1999). Thus, the light dependence of isoprene emission could be explained by only one isoprene synthesis-specific coefficient, ε (Fig. 12.3a). In further model development (Niinemets et al. 2002c; Niinemets 2004), the equation was generalized as:

$$E = \varepsilon\, J_T \frac{(C_i - \Gamma^*)}{\varsigma\,(4C_i + 8\Gamma^*) + 2\,(C_i - \Gamma^*)\,(\vartheta - 2\varsigma)}, \qquad (12.14)$$

where ς is the carbon cost of isoprenoid emission (6 mol mol^{-1} for isoprene and 12 mol mol^{-1} for monoterpenes), and ϑ is the NADPH cost of specific isoprenoid compounds (mol mol^{-1}). Differently from the initial model formulation (Niinemets et al. 1999) where the extra electron transport was assumed to originate from mitochondrial catabolism of the photosynthetically fixed carbon, Eq. 12.14 assumes that the rate of photosynthetic electron transport in photosynthezising leaves is larger than can be predicted by Eq. 12.12, and thus, the extra reductive equivalents rely on this "excess" electron transport (Niinemets et al. 2002c; Niinemets 2004). Overall, the model fit to monoterpene emissions was good (Fig. 12.3b, c), although it was realized that less volatile terpenoids can be non-specifically stored in the leaf liquid and lipid phases, resulting in delays between biosynthesis and emission (Niinemets et al. 2002a, c; Niinemets and Reichstein 2002).

Zimmer et al. (2000) and Grote et al. (2006) modelled BVOC emission on the basis of changes in metabolite pools. Their numerical model is based on reaction rates of various enzymes in the production pathway that are described based on Michaelis-Menten kinetics. Dynamics in the concentration of photosynthates that also serve as primary emission precursors, pyruvate and glyceraldehyde 3-phosphate, are directly related to photosynthesis and then enter the isoprenoid synthesis pathway as substrates. Thus, in this form of model logic, the light dependencies of isoprene and light-dependent monoterpene emission are introduced by the photosynthesis model used to produce emission substrates. Thus, the light relationship is ultimately driven by the same dependence of J on I that was reflected in the Guenther et al. (1991) and Niinemets et al. (1999) models.

As a further simplification, Niinemets et al. (2013 in this volume) directly linked isoprene emission to photosynthesis. In the so-called C-ratio model, isoprenoid emission was calculated as the product of the gross assimilation rate and monoterpene emission to assimilation rate ratio, r_C (Niinemets et al. 2013 in this volume). While being the simplest model, r_C was shown to depend on light and temperature, and thus, required somewhat greater parameterization effort than linking emissions to electron transport rate (Eq. 12.14). Nevertheless, comparison of different model approaches (Eqs. 12.11 and 12.14 and C-ratio model) at canopy level indicated that once correctly parameterized, all models performed similarly (Niinemets et al. 2013 in this volume).

As Monson et al. (2012) pointed out, all these approaches share a common 'quasi-mechanistic' basis in their relation to photosynthesis, with 'quasi-mechanistic' meaning that the dependence is not yet fully understood so that uncertainties remain. Although the overall flux of electrons going into volatile isoprenoid synthesis is small, there is evidence of control of the MEP pathway flux by ATP and/or NADPH status of chloroplasts (Loreto and Sharkey 1993; Rasulov et al. 2009, 2011; Li and Sharkey 2013a, b). This might suggest that the effective Michaelis-Menten constant of MEP pathway for ATP and/or NADPH controls the pathway flux. Given that daytime production of reductant and chemical energy in the chloroplast is driven by J, linking isoprenoid biosynthesis to the assimilation of CO_2 and to the supplies of precursor molecules into the MEP pathway constitutes still a promising way to simultaneously model CO_2 exchange and volatile isoprenoid emission.

12.2.4 CO_2 Responses

The negative relationship between isoprene emission and the concentration of atmospheric CO_2 was first reported in Sanadze (1964). The response of emission rate to changes in intercellular CO_2 concentration (C_i) follows a pattern with an optimum at a C_i of 150–200 μmol mol^{-1} ($C_{i,opt}$) (Loreto and Sharkey 1990; Rasulov et al. 2009; Sun et al. 2012). It has been demonstrated that the CO_2 dependence of isoprene emission is determined by the immediate precursor, dimethylallyl diphosphate (DMADP) pool size over the whole CO_2 range (Rasulov et al. 2009; Sun et al. 2012), but there is still a debate as to why DMADP pool size varies with CO_2 concentration.

Most of the modelling efforts have focused on understanding the decline in isoprene emission at CO_2 concentrations exceeding $C_{i,opt}$. Sanadze (2004) developed a biochemical hypothesis to explain this decline by postulating a competitive partitioning of chloroplast reductant and energy between the reductive pentose phosphate pathway and the MEP pathway. According to the hypothesis, the partitioning in turn depended on the demand for reductant and energy by the reductive pentose phosphate pathway. If this is low, these compounds are more readily available for isoprenoid production, implying that the pathway under these circumstances acts as a kind of excretion system. Conversely, under conditions of high Rubisco activity (e.g., high C_i), the reductant and energy will be diverted predominantly to synthesize and process sugars from photosynthesis.

This logic was to some degree already captured in the model developed by Niinemets et al. (1999) which is based on photosynthetic electron transport rate with isoprene biosynthesis rate defined by the fraction of J that is partitioned to the MEP pathway (Eq. 12.13). In subsequent work, therefore, based on observations, Arneth et al. (2007) introduced an additional empirical relation into Eq. 12.13 characterizing the partitioning as a hyperbolic function of C_i. Following this same concept, the model produced by Martin et al. (2000) represented isoprene emission as driven by competitive partitioning of chemical energy. In this model, as C_i increased, a negative feedback loop was imposed on emission by the decreasing availability of ATP.

On the other hand, experiments by Rosenstiel and others (Rosenstiel et al. 2003, 2004; Loreto et al. 2007) have suggested that the CO_2 sensitivity of isoprene emission can also be explained by competition for carbon substrate between cytosolic and chloroplastic processes. Wilkinson et al. (2009) model is based on this proposed mechanism and follows the assumptions that (1) at low C_i, the availability of recently-produced photosynthates limits isoprene production, although carbohydrate reserves may allow for some emission, and (2) that at increasing C_i, enzyme activity limits isoprene biosynthesis, while carbon precursors are getting more adequate. The overall response to C_i can then be expressed with an inverse sigmoidal function:

$$f(C) = 1 - \left[\frac{E_{MAX} \, (C_i)^{cC1}}{(C^*)^{cC1} + (C_i)^{cC1}} \right] \tag{12.15}$$

C^* is a reference C_i and c_{C1} is a unitless scaling coefficient that forces the right-hand term to be reduced exponentially at low C_i and increase exponentially at high C_i. A similar model that is based on the concentration of dimethylallyl diphosphate, [DMADP] in the chloroplast rather than C_i has been proposed by Possell and Hewitt (2011). Because [DMADP] decreases as C_i increases, in those cases where a negative CO_2 response has been observed, the model takes the following form:

$$f(C) = \frac{V_{MAX}\,[\mathrm{DMADP}]^{c_{C2}}}{K_M{}^{c_{C2}} + [\mathrm{DMADP}]^{c_{C2}}} \tag{12.16}$$

where V_{MAX} and K_M are the Michaelis-Menten constants for isoprene synthase, and c_{C2} is a unitless scaling coefficient. This model was shown to provide good descriptions of the CO_2 response for several species. However, we note that no model is currently able to mechanistically capture the reduction of isoprene emission at CO_2 concentrations below $C_{i,opt}$ of ca. 150–200 μmol mol^{-1} (Loreto and Sharkey 1990; Rasulov et al. 2009; Sun et al. 2012), and empirical fits best describing the entire CO_2 response curve of isoprene emission have been suggested (Sun et al. 2012).

Very recently, Harrison et al. (2013) proposed another relation of isoprene emission to photosynthesis, assuming that isoprene emission depends on excess reducing power, which is increased by the electron transport supply (J), and reduced by the electron transport requirements for the dark reactions of photosynthesis. 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 that is determined by C_i, J and maximum carboxylase activity of Rubisco ($V_{c,max}$). The isoprene emission rate is thus given by:

$$E_{iso} = p_3 J - p_4 V_{c,max} \frac{[C_i + 2\Gamma^*]}{[C_i + K_{c,M}]} \tag{12.17}$$

where p_3 and p_4 are empirical parameters that represent the 'baseline' fraction of the total photosynthetic electron flux used for isoprene synthesis (p_3), and the fraction of 'excess' electron flux (i.e., electrons not used in photosynthetic carbon fixation) used for isoprene synthesis (p_4), and $K_{c,M}$ is the effective Michaelis-Menten constant for Rubisco carboxylase activity. The approach is attractive, combining CO_2 and all other direct effects on photosynthesis, but it remains to be validated by mechanistic knowledge concerning the relation of J to E_{iso}, and it does not fully address the combination of both substrate availability and isoprene synthase activity as controls over E_{iso}.

Sensitivity of BVOC emission to the atmospheric CO_2 concentration has, to this point, been described only for isoprene. Yet, we know that the substrate constraints and mechanisms that affect the MEP pathway should affect the production of other terpenoid compounds as well. Thus, in species without specialized terpene storage structures, analogous CO_2-responsiveness of monoterpene emissions is expected. Indeed, a decrease in the rate of monoterpene emissions with increasing CO_2

concentration has been found in *Quercus ilex* (Loreto et al. 2001) and (to a smaller degree) in *Betula pendula* (Vuorinen et al. 2005). In other studies, no effects (Baraldi et al. 2004; Paoletti et al. 2007) or even an increase of monoterpene emission (Staudt et al. 2001; Himanen et al. 2009) have been observed. Clearly more work is needed to gain insight into CO_2 effects on monoterpene emissions, and it remains to be tested if and under which conditions the described models are applicable for direct emissions other than isoprene. Given the large number of terpene synthases and highlighted differences in regulation for some of these synthases (Rajabi Memari et al. 2013; Rosenkranz and Schnitzler 2013), simulating monoterpene emissions under future conditions is currently bound to large uncertainties.

12.2.5 Needs for Future Developments

Implicit in constructing the isoprene emission model as a product of multiplicative type equations (Eq. 12.1), is that environmental drivers such as light, temperature and CO_2 independently affect isoprene emission, i.e., any response function does not depend on other response functions. Recent progress in determining the mechanistic underpinnings of isoprene emission defines DMADP concentration and kinetic controls over isoprene synthase activity as basic determinants. DMADP concentration depends on energy/reductant availability, as well as on temperature and photosynthetic precursors, while the kinetic controls over isoprene synthase activity depend on temperature and DMADP concentration (Monson 2013). However, mixed control by both factors has not yet been fully reflected in models. For example, recent research indicates that the temperature response of isoprene emission depends on DMADP concentrations only at temperatures greater than 30 °C (Magel et al. 2006; Rasulov et al. 2010; Li et al. 2011). The situation is analogous with the CO_2 response. Given that DMADP availability ultimately controls the whole CO_2 response of isoprene emission, and DMADP level is also affected by light availability (Rasulov et al. 2009), CO_2 responses can vary in their dependence on the instantaneous photosynthetic photon flux density. Such a modification in the shape of CO_2-response curve by light has been recently demonstrated by Sun et al. (2012). The interactive effects of key environmental drivers suggests that models based on DMADP pool size may be more accurate for simulating isoprene emissions under co-varying light, temperature and CO_2 conditions.

The models that have been based on cytosol-chloroplast competition for substrate have not been able to explain one aspect of the CO_2 response – the steep reduction toward zero of the isoprene emission rate at a critically low value of C_i (Loreto and Sharkey 1990; Rasulov et al. 2009, 2011; Sun et al. 2012). Typically, this value is 150–200 μmol mol^{-1} that may be occasionally reached under drought in leaves in their native environments. Furthermore, the declining part of the CO_2 response curve below this critical threshold can provide fundamental information on the mechanism(s) responsible for the overall CO_2 dependence of isoprene emission, and clearly, this is an issue in need of further study.

Rasulov et al. (2009) used observations of the response of E_{iso} and DMADP pool size as a function of C_i to argue that the CO_2 effect on E_{iso} is due to variations in chloroplastic ATP supply, not variations in the channeling of PEP from the cytosol to the chloroplast. Both hypotheses rely on the fundamental observation that plastidic DMADP pool size decreases as C_i increases; the debate posed by Rasulov et al. (2009), as a counterpoint to the perspective of Rosenstiel et al. (2004), is focused on the cause of that decrease. Most of the evidence underlying both perspectives is correlative – positive correlations between ATP availability and E_{iso} have been observed (Loreto and Sharkey 1993) and negative correlations between PEP carboxylase activity and E_{iso} have been observed (Rosenstiel et al. 2003, 2004; Loreto et al. 2007; Possell and Hewitt 2011). In a recent study by Trowbridge et al. (2012), proton-transfer reaction mass spectrometry was used to detect the differential kinetics of ^{13}C incorporation into fragments of isoprene presumed to come from cytosolic versus chloroplastic sources. The results during periods of low versus high C_i suggested slower labelling in the fragment originating from cytosolic sources, and this fragment was more highly labeled in the presence of low CO_2, compared to that derived directly from glyceraldehyde 3-phosphate (GAP). These latter results might be used as support for the Rosenstiel et al. (2003) perspective. Once again, this is an issue that needs more study before a definitive model for C_i can be identified.

12.3 Modelling Acclimation and Seasonality

Seasonal dynamics of physiological pre-conditioning have long been either neglected (particularly when only short periods have been investigated) or have been empirically adjusted to time series measurements (e.g., Staudt et al. 2000, 2002). However, instantaneous emission responses to environmental drivers and maximum emission rates depend on the weather conditions days to weeks prior to the emission measurements and on ontogenetic changes in foliage emission capacity. That is why recent weather as an important driver of isoprenoid emission rate is now increasingly included in emission models (Guenther et al. 2006; Keenan et al. 2009; Niinemets et al. 2010a).

12.3.1 Seasonal Changes, Leaf Age Effects and Temperature Acclimation

12.3.1.1 Empirical Dependencies

After establishing that plants emit isoprenoid compounds instantaneously in a manner that is dependent on light and temperature, it was recognized that these dependencies change during the season (Ohta 1986). This has been noted to

lead to considerable biases in total emission inventories and has been related to temperature degree sums in past studies, similar to the metric used to describe phenological development in plants (Monson et al. 1994). Nevertheless, most early attempts on seasonal adjustment related the emission capacity to the day of the year (D). Schnitzler et al. (1997) proposed an asymmetric equation to define the seasonal factor, $f(S)$, which was intended as an additional multiplier in Eq. 12.1, and was described by an equation analogous to those used for enzyme activity modelling:

$$f(S) = \frac{\exp\left(c_{S1}\ D + c_{S2}\right)}{1 + \exp\left(c_{S3} D + c_{S4}\right)} \tag{12.18}$$

where c_{S1-n} are curve fitting coefficients. Pier and McDuffie (1997) used a second-order polynomial with three parameters to describe symmetric seasonal variation of isoprene emission potential observed in white oak (*Quercus alba*):

$$f(S) = c_{S5} + c_{S6} D + c_{S7} D^2 \tag{12.19}$$

Another equation that included parameters with physical meaning was proposed by Staudt et al. (2000) describing a Gaussian (bell-shaped) response with an offset:

$$f(S) = 1 - \rho\left[1 - \exp\left(-(D - D_0)^2/\tau\right)\right] \tag{12.20}$$

with ρ representing the relative annual amplitude of the maximum possible seasonal emission rate (between 0 and 1.0), D_0 the day at which the emission capacity reaches a maximum, and τ the breadth (kurtosis) of the seasonal amplitude in days. Additional asymmetric functions have been used by Lavoir et al. (2011), Keenan et al. (2009) and Niinemets et al. (2013 in this volume). Keenan et al. (2009) compared the seasonality function shapes, asymmetric vs. symmetric, and concluded that an asymmetric function better adheres to the data and is recommended for simulation of seasonal variations in isoprenoid emission.

12.3.1.2 Dependencies Imposing Genetic and Environmental Controls

Early in the history of isoprene emissions studies, it was hypothesized that it is not the day of the year, but the previous integrated environmental conditions that determine seasonal shifts in the isoprene emission rate (Monson et al. 1994). As a consequence, it has been proposed that leaf developmental processes, controlled by genetic-environment interactions, underlie expression of the genes for emission synthases. Two modelling approaches were suggested: (1) isoprene synthase development follows leaf phenology, assuming that only fully-grown and active leaves are able to emit BVOCs at potential rates; (2) synthase activity is subject to continuous

but slow formation and decay processes that depend on environment. Thus, previous environmental conditions are important determinants of E_{max}. Lehning et al. (2001) followed this concept explicitly. The Seasonal Isoprenoid synthase Model (SIM) is split into a description of leaf development and senescence, and an equation that calculates dynamics of enzyme activity. The first mechanism represents the building and decline of emission capacity assuming a linear relation to leaf development (or more precisely, relative canopy leaf area). It has been elaborated to be applicable for evergreen species by Grote (2007) who described leaf development for each leaf age class separately. The second impact is a description of synthase turnover:

$$f(S) = S_0 + [g(S_F) + h(S_D)]\Delta t \tag{12.21}$$

where S_0 is the previous (or initial) state of the seasonality function, $g(S_F)$ is a function that describes the rate of protein synthesis in dependence on past light and temperature conditions and phenological state of the leaves, and $h(S_D)$ is a function that describes the rate of protein degradation (for details see Grote et al. 2010). In contrast to the previous approaches, this model introduces some mechanistic cause-effect relationships by considering the increase of enzyme activity as dependent on absorbed radiation and its decay as a function of temperature.

Another approach has been presented with the Model of Emissions of Gases and Aerosols from Nature (MEGAN) (Guenther et al. 2006). In this model, age effects are described by separating the foliage among new, young, and recently matured leaves. The seasonality aspect was described by adjustment of E_S (Eq. 12.1) independent of phenology in dependence on the temperature of the previous days:

$$f(S) = c_{S8} \exp\left[c_{S9}\left(T_{24} - T_{REF}\right)\right] \exp\left[c_{S9}\left(T_{240} - T_{REF}\right)\right] \tag{12.22}$$

where c_{S8} and c_{S9} are empirical parameters, T_{REF} is a reference temperature (297 K), and T_{24} and T_{240} are average temperatures for the previous 24 and 240 h, respectively.

In MEGAN, the overall response represents a sine function while, the SIM approach follows the general pattern of an exponential response, which generally provides a better fit to data. We present some of the approaches that have been used for seasonal adjustment of E_{MAX} in Fig. 12.4. The shapes of the seasonal responses and their maxima near day of year of 200 are generally conserved. However, the slopes of the responses for the ascending and descending trajectories on either side of the maxima differ, and this is where model-dependent differences are likely to be greatest.

Overall, we note that modelling seasonality remains a challenging task. As acclimation and age effects cannot be deconvoluted, it is important to be aware that the seasonality and age models may partly include acclimation effects. This understanding is relevant especially when trying to incorporate various acclimation, stress, and seasonal controls in multivariate models (Eq. 12.1) to avoid "double-counting" of various factors, thereby over-parameterizing the model (Niinemets et al. 2010a).

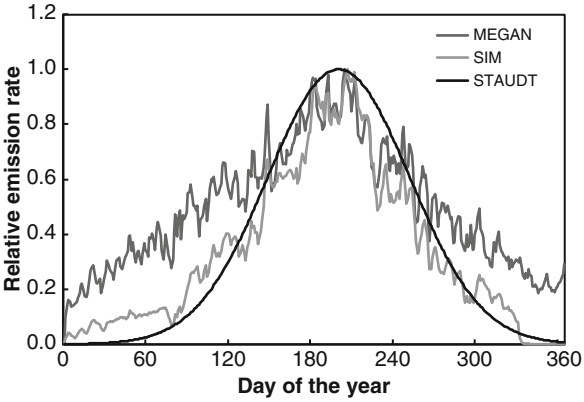

Fig. 12.4 Seasonal adjustment of maximum emission rate in broad-leaved sclerophyll *Quercus ilex* according to an empirical fit to data from Staudt et al. (2000), and the emission capacity predicted according to weather-dependent MEGAN (Guenther et al. 2006) and SIM (Lehning et al. 2001) approaches using the weather conditions at Montpellier, France for 2006 growing season (Modified from Grote et al. 2010). The emission rates were normalized to the highest observed value

12.3.2 Acclimation to Variations in Light Environment

Several studies have demonstrated that both isoprene emission capacity (Harley et al. 1996, 1997; Geron et al. 1997; Hanson and Sharkey 2001; Funk et al. 2006; Niinemets et al. 2010a, b) and monoterpene emission capacity in "non-storage" species (Lenz et al. 1997; Niinemets et al. 2002a; Staudt et al. 2003) increases with increasing long-term light availability. In particular, extensive within-canopy variation in isoprene emission rate of 3-27-fold has been recently demonstrated in broad-leaved deciduous trees (Fig. 12.5). Depending on within-canopy plasticity in isoprene emission potential, model estimates of whole canopy isoprene emissions using a constant emission factor are biased by −8 to +68 % (Niinemets et al. 2010b). Guenther et al. (1999) linked such within-canopy variations at the level of coefficient c_{L3} of the light response function of Eq. 12.11. Thus, the coefficient c_{L3} essentially functions as a scaling factor. As no long-term light measurements were available, c_{L3} was linked to cumulative leaf area index (L_{cum}) as (Guenther et al. 1999):

$$c_{L3} = 1.42 \exp(-0.3 L_{cum}) . \tag{12.23}$$

However, foliage acclimates to long-term quantum flux density, Q_{int}, rather than to L_{cum}, and Q_{int} corresponding to a given value of L_{cum} may vary in dependence on foliage angular distribution and spatial aggregation (Cescatti and Niinemets 2004). Despite species-specific variations in the within-canopy variability of emission capacity, Niinemets et al. (2010b) demonstrated that when all data across the species

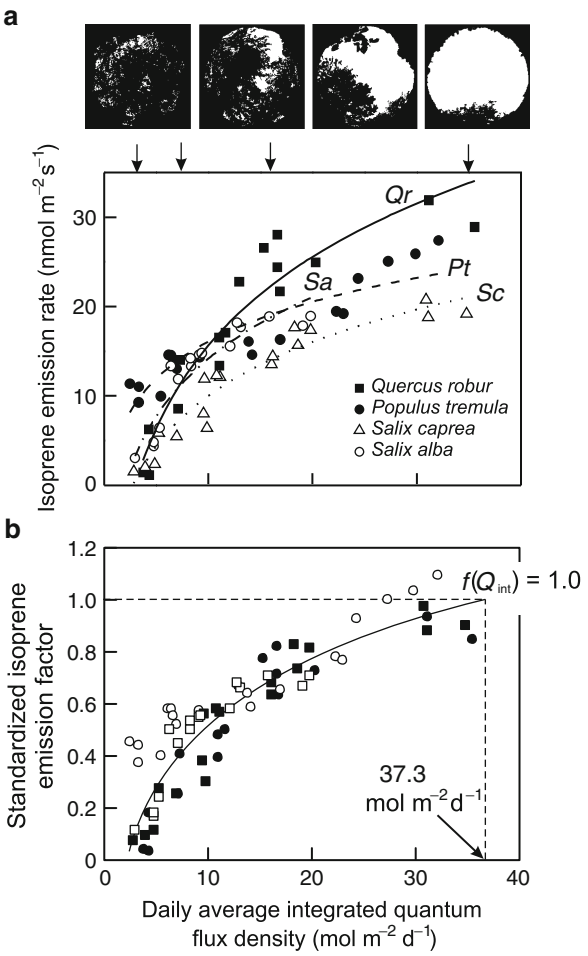

Fig. 12.5 Variation of isoprene emission rate at standard conditions of leaf temperature of 25 °C and incident quantum flux density of 1,000 μmol m^{-2} s^{-1} (emission factor) with seasonal average integrated quantum flux density (Q_{int}) in four temperate deciduous species (**a**), and the emission factor standardized to the above-canopy (average seasonal maximum) quantum flux density of 37.3 mol m^{-2} day^{-1} (modified from Niinemets et al. 2010b). Data in (**b**) are fitted by the so-called canopy function of isoprene emission ($f(C)$, Eq. 12.24). Representative hemispheric photographs demonstrating different light environments within the canopy are also shown (*arrows* denote the Q_{int} values corresponding to the four hemispheric photographs)

were standardized to above-canopy seasonal average Q_{int} (37.3 mol m^{-2} day^{-1} in their study), the variation decreased (Fig. 12.5) and all data could be fitted by a single canopy function, $f(C)$:

$$f(C) = 0.843\mathrm{Log}\,(0.411\,Q_{int})\,. \tag{12.24}$$

The bias of using Eq. 12.24 in estimating whole canopy isoprene emission flux relative to the use of species-specific variation patterns was only −11 % to +6 % (Niinemets et al. 2010b). Thus it would be more accurate to use Eq. 12.24 instead Eq. 12.23 in future emission models.

The other parameter, susceptible to acclimation is the quantum yield (α in Eq. 12.11) used to define the instantaneous light response of immediate emission. This parameter can vary within the canopy (Fig. 12.2b) due to changes in leaf

chlorophyll content (Niinemets 2007). For isoprene and monoterpenes, studies have suggested an increasing apparent quantum yield with canopy depth (Harley et al. 1996, 1997; Staudt et al. 2003), and the apparent quantum yield has thus been expressed in dependence on L_{cum} similar to c_{L3} (Guenther et al. 1999):

$$\alpha_{app} = 0.001 + 0.00085 L_{cum} \tag{12.25}$$

However, we note that there is an explicit connection between α_{app} and c_{L3} as indicated in Sect. 12.2.3 and in Monson et al. (2012). In fact, Harley et al. (1997) fit the light response curves of isoprene emission using Eq. 12.10, and observed only minor within-canopy variation in the true quantum yield. Thus, it remains to be tested to what extent the true quantum yield for isoprene emission does indeed vary in plant canopies.

12.3.3 Needs for Future Developments

Representing acclimation processes of foliage emission at the ecosystem scale is a difficult task since seasonal development of cell-to-leaf level states are not only directly affected by environmental conditions, but are also indirectly influenced by phenological, ontogenetic and structural properties of the emitting plants. This is most obvious for foliage amount which varies during the year due to leaf flushing and senescence, and these effects are particularly obvious in deciduous species. Additionally, ontogenetic changes affect isoprenoid emission potentials (maximum rate under standardized conditions) (e.g., Grinspoon et al. 1991; Kuzma and Fall 1993; Fuentes and Wang 1999). The capacity to emit isoprenoids generally develops gradually during leaf development and reaches a maximum only after full leaf expansion; following maximum leaf expansion, and the emission potentials further gradually decrease with increasing leaf age (Fischbach et al. 2002). With respect to evergreen species, it is thus important that functional activity continuously decreases with increasing leaf age (Niinemets et al. 2006, 2013; Grote 2007). Finally, canopy structure determines microclimatic conditions that affect short- and long-term impacts on emission processes throughout the canopy (Keenan et al. 2011). All of these vegetation processes develop dynamically and simultaneously in response to changes in the seasonal environment. It is difficult to disentangle the direct and indirect seasonal influences on emission potential and to define species-specific differences in acclimation capacity, principally due to the lack of empirical information. For example, surprisingly little information is available on emission potentials in older leaves. Other physiological developments such as seasonal dynamics in isoprenoid storage pools, which are not yet considered in any model (Schurgers et al. 2009) add to these uncertainties. Finally, emissions related specifically to bud, flower, and fruit development are not addressed in models, although modified emission patterns – qualitatively and quantitatively – have been reported for the period of bud burst (e.g., Kuhn et al. 2004).

12.4 Incorporating Stress in Models of Constitutive Emission

Stress can have several effects on volatile emissions. First, in constitutively emitting species, stress may modify the emission capacity and/or the shape of emission responses to environmental drivers. Second, stress can lead to induction of volatile emissions in both emitting and (otherwise) non-emitting species. As natural vegetation is often under stress, even suffering frequently from co-occurring and sequential stress episodes (Loreto and Schnitzler 2010; Niinemets 2010a, b), our ability to predict stress responses on volatile emissions is urgently needed for reliable prediction of emission time series. In this section, we analyse how stress effects on constitutive emissions can be incorporated in emission models focusing on the influences of altered transfer conductances and biochemical modifications as exemplified by drought responses. For pollutant effects on constitutive emissions we refer to Calfapietra et al. (2013) in this volume.

12.4.1 Impacts on Conductances

12.4.1.1 Stomatal Controls

Constitutive emissions are controlled by temperature and the diffusive resistances between storage pools and the atmosphere. Several past studies have focused on stomata as the primary resistance to emission from internal storage pools. In the early studies of foliage isoprene emission, it was recognized that the steady-state isoprene emission rate is independent of stomatal conductance (G_s) (Monson and Fall 1989; Fall and Monson 1992). Fall and Monson (1992) hypothesized that steady-state reductions in G_s were compensated by increases in Δp, the difference in isoprene partial pressure between the intercellular air spaces of the leaf (p_i) and the ambient atmosphere (p_a). Thus, $E = G_s(\Delta p/P)$, where P is the air pressure. The theory underlying this relation and its application to a range of emitted BVOCs requires that for compounds which have relatively high Henry's law constants (gas/liquid phase partition coefficients), perturbations in G_s should result in rapid (within seconds) establishment of a new diffusion steady state (Niinemets and Reichstein 2003). This would not be true for BVOCs with lower Henry's law coefficients (e.g., oxygenated isoprenoids, organic acids or methanol). Niinemets and Reichstein (2003) formalized the theory on these relations by stating:

$$E = G_s \frac{(p_i - p_a)}{P} = G_L \frac{(H\ C_w - p_i)}{P} \tag{12.26}$$

where H is the Henry's law constant for the particular BVOC (Pa m^3 mol^{-1}), C_w is its concentration in the liquid (water) phase of the cell or cell wall (mol m^{-3}), and G_L is the gas-phase equivalent of liquid phase conductance from the site of compound synthesis to the outer surface of cell wall. Implicit in Eq. 12.26 is that compounds

with low H support a lower vapour pressure for given liquid phase concentration, and accordingly, the diffusion gradient, Δp, increases slowly such that changes in stomatal conductance can transiently limit volatile emissions (Niinemets and Reichstein 2003; Harley 2013).

12.4.1.2 Breakage of Storage Structures

Enhancements of emissions of stored BVOCs occur when leaf tissue is wounded and broken epidermis and cuticle strongly decrease diffusive resistances. These effects are particularly relevant in characterizing the impact of logging operations in forests where terpene-filled tissue is destroyed (e.g., Strömvall and Petersson 1991). Similarly, insect attacks can open plant storages of volatile compounds that often act as a defence and serve to poison or otherwise deter attackers (Loreto et al. 2000; Trowbridge and Stoy 2013). We note that past relationships of terpene content vs. emission rate as shown for some conifers (Lerdau et al. 1994, 1995) may reflect the "rough handling problem", i.e., exposure of internal storage structures to ambient air during measurements.

To date, the effects of rapid changes in diffusion conductance from the site of storage to ambient air have not been considered in emission models. However, BVOC pools have previously been quantified (e.g., Llusià and Peñuelas 1998; Llusià et al. 2010) and their release can be simulated according to Eqs. 12.2 or 12.3 with the additional assumption of a limited (and decreasing) storage pool size (Schurgers et al. 2009). Nevertheless, the models likely need to be more complex than just first order decay functions, because the initial rapid increase in emissions is followed by time-dependent reduction of the emission rates as the wound becomes sealed, e.g., as the result of oxidation and polymerization of oleoresin components (Loreto et al. 2000).

12.4.2 Impacts on Biochemistry

The impact of drought has been studied in several investigations as time-integrated, long-term influence on isoprene emission (e.g., Fang et al. 1996; Brüggemann and Schnitzler 2002; Pegoraro et al. 2004; Brilli et al. 2007). However, until recently, drought has not been considered as a modifier in BVOC emission models. Drought can influence the emissions in three ways. First, reductions in leaf evaporative cooling due to constrained leaf transpiration rates, leading to concomitant increases in T_L. Second, decreases in stomatal conductance result in reduced C_i. Finally, there can be direct effects of drought on metabolic processes.

The first effect can be accommodated in the models by considering the deviations between T_L and air temperature. The second influence can be incorporated through Eqs. 12.15, 12.6 and 12.17 when properly parameterized to consider reduced CO_2 growth regime, especially considering the reduction of emissions below a critical C_i.

Modelling the effects of metabolic modifications is most complex. Drought tends to trigger a cascade of metabolic feedbacks that function to balance metabolism with growth potential. Grote et al. (2009) took advantage of previous studies of changes in the concentrations of certain photosynthetic metabolites to represent drought effects on monoterpene emissions through the availability of BVOC precursors. One premise of this approach is that a tight coupling exists between leaf carbon balance, as influenced by leaf photosynthesis rate, and isoprenoid emission. However, this assumption neglects the shift in resources between different biochemical pathways under stress. Within the MEGAN model, Guenther et al. (2006) introduced a drought scaling factor as a linear relation between relative water availability and E as an additional multiplier in Eq. 12.1. This function was defined as:

$$f(W) = \begin{cases} 1, \text{ if } \theta \geq \theta_1 \\ \dfrac{(\theta - \theta_W)}{\Delta\theta_1}, \text{ if } \theta_W < \theta < \theta_1 \\ 0, \text{ if } \theta \leq \theta_W \end{cases} \tag{12.27}$$

where θ is the extractable water content ($m^3\ m^{-3}$), θ_w is the soil water content at leaf wilting point, i.e., the soil water content that cannot be extracted by plant roots, $\Delta\theta_1$ is an empirically-determined soil water limit that can be expressed as $\theta_1 = \theta_w + \Delta\theta_1$. $\Delta\theta_1$ is commonly set as 0.06 following Pegoraro et al. (2004). One of the difficulties with using this type of model is the determination of θ_w as well as $\Delta\theta_1$. Guenther et al. (2006) used the wilting point database of Chen and Dudhia (2001) for global emission estimation. However, there are no studies to date that have established the wilting point as a conserved and relevant determinant of drought stress on photosynthesis or BVOC emission.

The greatest barrier to progressing in our ability to model drought stress effects on BVOC emission is our incomplete understanding of the metabolic connections among drought, expression of BVOC synthase activities, availability of BVOC substrates, and drought-induced changes in the sensitivities of BVOC formation to light, temperature and intercellular CO_2 concentration. Future studies should focus on these connections, which may allow us to integrate drought-stress models more effectively into BVOC models.

12.5 Simulation of Induced Emissions

12.5.1 General Patterns

Consistent with the theory and evidence that BVOC emissions serve primarily as a protection against abiotic stress and for communication among ecological tropic levels (Holopainen 2004; Sharkey et al. 2008). BVOC emissions can be induced by practically any stress factor in species emitting and non-emitting volatiles constitutively (Heiden et al. 2003). The emission of stress volatiles reflects

elicitation of defence pathways, side-products of intermediates of which are volatile, and synthesis of volatile products with known or yet unknown functions in direct and indirect defence (Pare and Tumlinson 1999, Kessler and Baldwin 2001; Peñuelas and Llusià 2003; Owen and Peñuelas 2005, 2006; Niinemets 2010a, b). Induction of volatile emissions has been demonstrated in response to both biotic stresses such as insect herbivory (e.g., Priemé et al. 2000; Miller et al. 2005; Dicke et al. 2009; Copolovici et al. 2011; Blande et al. 2009), and fungal pathogens (Steindel et al. 2005; Toome et al. 2010) and abiotic stress such as UV radiation (e.g., Blande et al. 2009), ozone (e.g., Beauchamp et al. 2005; Blande et al. 2007), heat and frost (Loreto et al. 2006; Copolovici et al. 2012), flooding (Copolovici and Niinemets 2010; Kreuzwieser and Rennenberg 2013), and mechanical wounding (Fall et al. 1999; Banchio et al. 2005; Loreto et al. 2006).

Emissions of early stress volatiles during and immediately after stress reflect activation of signalling at the level of membranes and cell walls and are associated with the release of methanol (Beauchamp et al. 2005; Loreto et al. 2006; von Dahl et al. 2006; Copolovici and Niinemets 2010) and green leaf volatiles (various C_6 aldehydes) (Priemé et al. 2000; Loreto et al. 2006; Copolovici and Niinemets 2010; Copolovici et al. 2011, 2012; Blande et al. 2007, 2009; Kirstine and Galbally 2004; Loreto et al. 2006; Davison et al. 2008; Brilli et al. 2012). These emissions are followed by activation of gene expression and emissions of specific volatile isoprenoids from stressed foliage (Dicke 1994; Paré and Tumlinson 1997; Beauchamp et al. 2005; Toome et al. 2010; Copolovici et al. 2011; Blande et al. 2007, 2009). Furthermore, release of volatiles and synthesis of non-volatile phytohormones in stressed leaves can elicit systemic response in neighboring non-stressed leaves of the same plant and in neighboring different plants, resulting in volatile emissions of apparently healthy leaves (Dicke 1994; Röse et al. 1996; Paré and Tumlinson 1998; Staudt and Lhoutellier 2007; Holopainen et al. 2013; Trowbridge and Stoy 2013).

Characteristic stress-induced volatile isoprenoids are monoterpenes linalool and ocimenes, homoterpenes DMNT and TMTT and various sesquiterpenes (Loivamäki et al. 2004; Herde et al. 2008; Dicke et al. 2009; Toome et al. 2010), and thus, the composition of elicited isoprenoids typically differs from the volatiles released in non-stressed conditions (Loreto and Schnitzler 2010; Niinemets et al. 2010c; Schnitzler et al. 2010). As noted above, in constitutively emitting species, biotic or abiotic stress may result in suppression of constitutive emission rates (Anderson et al. 2000; Copolovici and Niinemets 2010; Toome et al. 2010), but not always (Calfapietra et al. 2007, 2008; Copolovici and Niinemets 2010). Yet, in constitutively non-emitting species, volatile emissions generally increase from low background level by several orders of magnitude even above the level observed in constitutively emitting species (Niinemets et al. 2010c for a review). For instance, temperate deciduous broad-leaved birch (*Betula*) species have been observed to emit mono- and sesquiterpenes at a low level of only 0.1–0.4 $\mu g\ g^{-1}\ h^{-1}$ in some studies and during certain periods during the growing season (Fig. 12.6, König et al. 1995; Hakola et al. 1998, 2001). However, under stress conditions, they have been found to be relatively strong emitters of monoterpenes linalool and ocimenes, and sesquiterpenes, with standardized emission rates (leaf temperature of

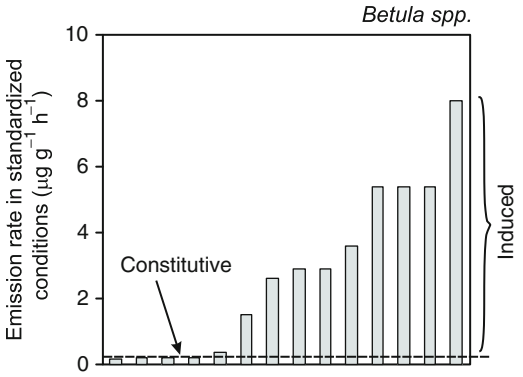

Fig. 12.6 Variation in standardized monoterpene emission factor (leaf temperature of 30 °C, incident quantum flux density of 1,000 μmol m^{-2} s^{-1}) in temperate deciduous birch (*Betula*) species (Data from König et al. 1995; Steinbrecher et al. 1997; Hakola et al. 1998; Hakola et al. 2001; Owen et al. 2003). Sustained emissions under non-stressed conditions are defined as constitutive emissions, while emissions elicited under certain stress periods are defined as induced emissions

30 °C and incident quantum flux density of 1,000 μmol m^{-2} s^{-1}) between 1.5 and 8 μg g^{-1} h^{-1} (Fig. 12.6, König et al. 1995; Hakola et al. 1998, 2001; Steinbrecher et al. 1999; Owen et al. 2003). Analogously, in a constitutive-emitter Mediterranean evergreen conifer *Pinus pinea*, total emissions from stressed plants are several-fold greater than the constitutive emissions from non-stressed plants (Staudt et al. 1997, 2000; Niinemets et al. 2002b, c).

12.5.2 Modelling Induced Emissions

Induction of BVOC emissions can reflect activation of enzymes that are already present or increased expression of the genes that encode various BVOC synthases. Given the growing evidence that a considerable fraction of emission responses are related to stress induction, models that describe these processes are beginning to emerge. Iriti and Faoro (2009) have suggested differentiating between primary and secondary metabolic pathways in the induction process with concomitant modifications in carbon fluxes among the pathways. The mechanism of volatile induction is complex, starting with signal perception that triggers the cascade of events leading ultimately to activation of transcription regulators and onset of expression of volatile synthases (Bolwell et al. 2002; Maffei et al. 2007; Mithöfer and Boland 2008; Loreto and Schnitzler 2010; Niinemets 2010a; Arimura et al. 2011). The mechanisms of signal perception and elicitation of gene expression can differ for different stresses, but there is evidence of a uniform stress response elicitation pathway for both biotic and abiotic stresses at the level of oxidative signalling (Bostock 2005; Fujita et al. 2006). Often, there is also a cross-talk between ethylene-, salicylate- and jasmonate-dependent stress response pathways (Thaler et al. 2002; Traw and

Bergelson 2003; Bostock 2005; Fujita et al. 2006; Mithöfer and Boland 2008). Thus, general stress response models can in principle be constructed (Niinemets 2010a).

From an experimental perspective, there is increasingly more evidence that stress severity and plant volatile emission response are quantitatively related, including positive correlations between the severity of ozone (Beauchamp et al. 2005), heat (Karl et al. 2008; Copolovici et al. 2012) and insect herbivory (Copolovici et al. 2011) stresses. Scaling of volatile emission response with the stress severity has been used in predicting methyl salicylate emissions from a walnut (*Juglans californica* × *Juglans regia*) agroforest on the basis of average temperature preceding the measurements (Karl et al. 2008). While such empirical models based on average level of environmental drivers can be useful once the emissions have been triggered, emissions typically are not induced until a certain stress threshold has been exceeded (Beauchamp et al. 2005; Copolovici et al. 2012), except perhaps for wounding and insect herbivory that essentially always trigger emissions. Thus, the key issue in predicting stress induction of volatiles is to determine when a given environmental driver is sensed as a stress by the plant. The stress thresholds depend on a variety of factors including plant tolerance to given type of stress and past stress history such as stress priming (Conrath et al. 2006; Heil and Kost 2006; Heil and Silva Bueno 2007; Niinemets 2010a, b). Thus, a stress of given severity may or may not result in inductions of volatile emissions.

The second difficulty of simple empirical models is what happens after stress. When the stress is relieved, for how long do the triggered emissions continue? There is evidence that after the stress relief, the induced emissions may reach to a background level in a few days (Copolovici et al. 2011). The emissions may also decrease during the stress as plant acclimates to the stress (Copolovici and Niinemets 2010). However, there is also evidence of sustained emissions once elicited (Staudt et al. 1997, 2000; Hakola et al. 2001; Niinemets et al. 2002b; Copolovici and Niinemets 2010).

Stress signalling models are currently being intensively developed (Vu and Vohradsky 2007; Yip et al. 2010; Muraro et al. 2012), but due to limited knowledge of signal transduction and transcription regulators, completely mechanistic models cannot yet be derived. We suggest that for the time being, the dynamic controls on induced BVOC emissions can be simulated based on the theory of recursive action of regulators on the target gene(s) over time (Vu and Vohradsky 2007; Yip et al. 2010). Thus, the target gene activity change over time, dz/dt, is expressed as (Vu and Vohradsky 2007; Yip et al. 2010):

$$\frac{dz}{dt} = \frac{v_{\max}}{1 + \exp\left(-\sum_{j=1}^{j=n} w_j y_j + c\right)} - kz(t), \tag{12.28}$$

where $v_{\max}$ is the maximum rate of expression, k is the rate constant of degradation, $z(t)$ is the gene product amount at time t, n is the number of gene regulators considered, w_j is the weighting factor for a given control function y_j, and b is the delay factor describing the lag in the transcription initiation. Thus, $v_{\max}$

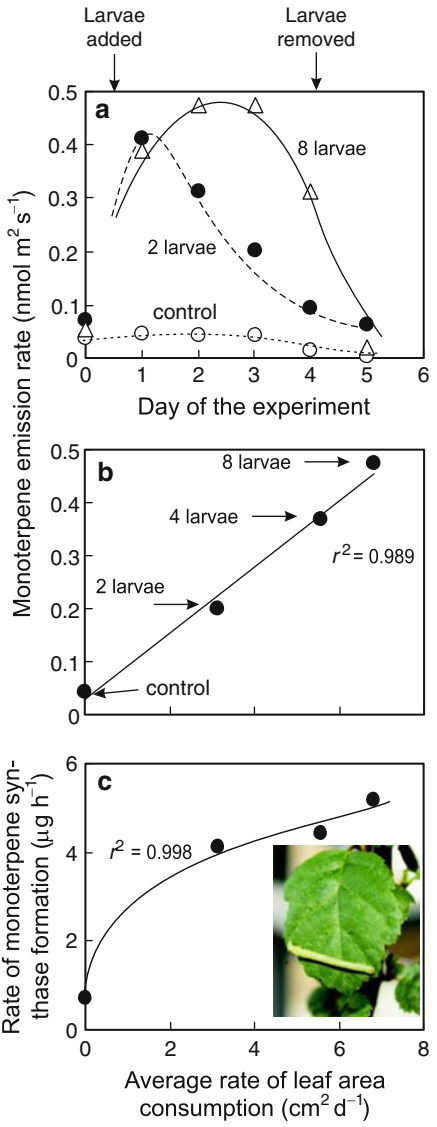

Fig. 12.7 Illustration of elicitation of monoterpene emissions in temperate deciduous tree *Alnus glutinosa* by the common white wave (*Cabera pusaria*) larvae (**a**), and the relationships between the average rate of leaf consumption and monoterpene emission rate (**b**) and the rate of monoterpene synthase formation (**c**) (Data from Copolovici et al. 2011). In the experiment, the plants of *A. glutinosa* were subject to different levels of herbivory by using either 0 (control), 2, 4 or 8 *C. pusaria* larvae per plant. The measurements were conducted at 28 °C. The data in (**a**) were simulated by a model based on dynamic transcriptional control (Eq. 12.28), and the rate of monoterpene synthase formation was found by fitting the data in (**a**) by the model. In calculating the protein formation rate, a specific activity of monoterpene synthase of 94 nmol g^{-1} s^{-1} at 28 °C was used (Niinemets et al. 2002c), and it was further assumed that the induced monoterpene synthases operate in substrate-saturated conditions

and the denumerator determine the onset of gene expression, while kz and $w_j y_i$ functions determine the silencing of the response. This model assumes that the overall regulatory effect on a given gene can be expressed as the combination of all regulators (Vu and Vohradsky 2007; Yip et al. 2010). Highly plastic non-linear transcription control effects can be simulated using various linear or non-linear y_i functions, and it has been demonstrated that Eq. 12.28 provides excellent fits to complex gene expression profiles (Vu and Vohradsky 2007).

Equation 12.28 was applied here to the induction of monoterpene emissions in the temperate deciduous tree black alder (*Alnus glutinosa*) (Fig. 12.7a, Copolovici

et al. 2011). In this study, different levels of herbivory were achieved by using either 0 (control), 2, 4 or 8 larvae of the common white wave (*Cabera pusaria*) on each plant (Copolovici et al. 2011). The rate of leaf biomass consumption scaled positively with the number of feeding larvae, and the rate of induced monoterpene emission was quantitatively related to the rate of foliage consumption (Fig. 12.7a, Copolovici et al. 2011). In the model fit, only one transcriptional control was assumed and the control function was described by a fifth order polynomial.

The model applied here provides excellent fits to the data (Fig. 12.7b), and allows for the estimation of kinetic dynamics in transcription, maximum rates of induced monoterpene synthase formation and rates of monoterpene synthase decay under different herbivory treatments. The maximum rate of monoterpene synthase formation was quantitatively associated with the rate of herbivory (Fig. 12.7c). To our knowledge, this is the first evidence demonstrating that stress signal strength can be quantitatively simulated to project target protein synthesis rate. On the other hand, we also observed differences in the transcription regulation function in different treatments, with the emissions being both elicited and declining earlier in the treatment with two than in the treatment with eight larvae (Fig. 12.7a). Such differences cannot be currently explained, and apart from differences in plant transcription regulation, might reflect differences in the feeding behavior of herbivores in different treatments. Overall, this exercise provides encouraging evidence that models based on dynamic transcription control can be used to simulate induced emissions, and we suggest that simple dynamic regulatory models such as Eq. 12.28 together with quantitative relationships between the severity of stress and maximum plant response have large potential to simulate stress-driven emissions in larger-scale models.

12.6 Conclusions

Considering the different environmental impacts that affect BVOC emission, it has become increasingly apparent that integrated descriptions of processes are beginning to emerge. Such integrated models will permit us to begin examining higher-order interactions between environmental change and ecosystem BVOC emissions, including the feedbacks that control regional- to global-level dynamics in atmospheric chemistry and in the production and lifetime of radiatively-important trace gases such as O_3 and CH_4. In this chapter, we have concentrated on the leaf-scale modelling as the most significant breakthroughs in recent BVOC modelling have been made at this scale. There is now increasing recognition that the mechanistic emphasis that has been in focus at the leaf scale needs to be expanded to consider processes at greater spatial scales and longer temporal scales (Guenther 2013 in this volume). Consideration of the latter, takes us into the need to discover ways of simulating the interactions between environmental cues and gene expression. Simulation of these larger and longer-term processes will allow us to begin tackling some of the regional and global dynamics in air chemistry.

These controls are increasingly recognized as being central components in Earth system models (Ashworth et al. 2013; Kulmala et al. 2013), and we argue that more biological realism needs to be incorporated in these models in near future.

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