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Atmospheric change and induced plant secondary metabolites — are we reshaping the building blocks of multi-trophic interactions?

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At least for the foreseeable future, atmospheric concentrations of greenhouse gases — particularly carbon dioxide (CO_2) and ozone (O_3) — are projected to rise inexorably. Recent studies have begun to unveil the complex nature of how these gases modulate the expression of plant signaling hormones, the defensive chemistries produced, and the responses of the myriad trophic interactions involving plant pathogens as well as insect herbivores and their natural enemies. Given the ubiquity of complex trophic interactions in both natural and managed systems, it is crucial that we understand how CO_2 and O_3 interact with defense signaling hormones of plants and their consequences for their trophic associates if we are to adapt to, and even mitigate, the effects of climate change.

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Introduction

Anthropogenic climate change is the result of increased atmospheric levels of greenhouse gases (most significantly, carbon dioxide (CO_2), nitrous oxide (N_2O), nitrogen oxides (esp. NO, NO_2), methane (CH_4), and ozone (O_3)) that absorb solar radiation, increasing the surface temperature of the Earth [1]. These increases have been caused by human activities, including fossil fuel combustion and forest destruction (CO_2), agricultural fertilizer use (N_2O), farming of ruminant livestock (CH_4), and the formation of O_3 from the oxidation of hydrocarbons and nitrogen oxides originating from industrial and vehicular emissions. Increased concentrations of greenhouse gases cause average global atmospheric temperatures to rise and create increasingly variable weather patterns. To date, most studies of the biological effects of climate change have focused on the effects of climate change drivers (e.g. elevated concentrations of atmospheric CO_2 (e[CO_2]) and O_3 (e[O_3])) and climate variables themselves (drought and temperature) on plant primary productivity, with a smaller number investigating the consequences for plant relationships with pathogens, herbivores, and natural enemies of herbivores [2,3••,4]. While plant productivity and nutritional quality are fundamentally important to insect herbivores, plant secondary metabolites (PSMs) also play major roles in herbivore behavior and performance with implications extending to their natural enemies [5,6].

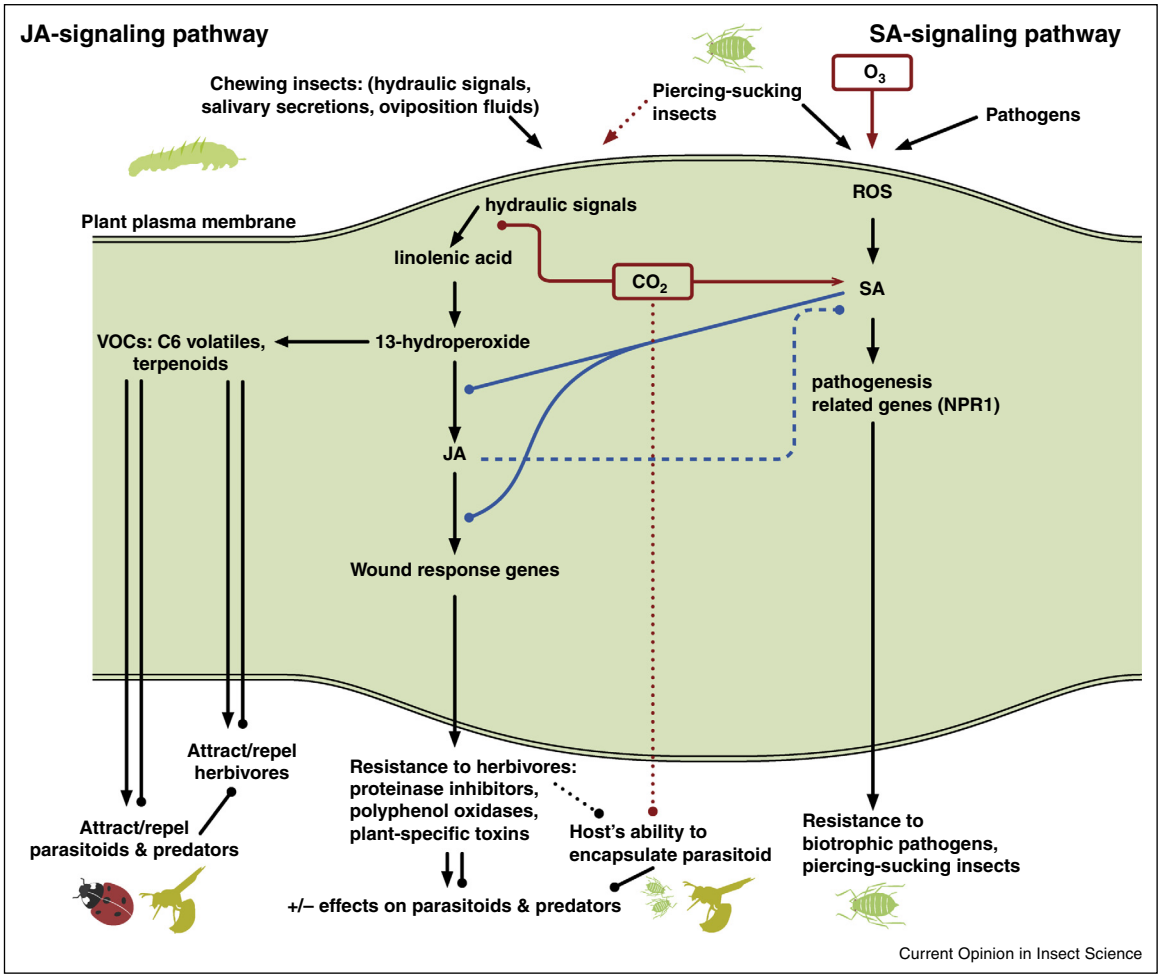
Changes in the atmospheric concentrations of CO_2 and O_3 can modulate the expression of complex plant hormone signaling pathways responsible for induced defenses against pathogens and herbivores, defenses which include a staggering diversity of PSMs. Here, we argue that in order to predict the responses of trophic associates of plants to atmospheric change, we must understand how plant defense signaling pathways are influenced by e[CO_2] and e[O_3]. We recognize that other aspects of climate change also affect plant secondary metabolism, but concentrate here on the relatively well-studied effects of e[CO_2] and e[O_3] on induced plant defenses.

Plant signaling pathways — the precursors of PSMs

Plants possess a diverse array of physical, chemical, and biochemical defenses against herbivores and pathogens. Defenses can be expressed constitutively or can be rapidly induced in response to actual or imminent attack by herbivores. Synthesis of plant defenses against these threats is predominately induced via two plant hormone-mediated signaling pathways, namely the jasmonic acid (JA) pathway and the salicylic acid (SA) pathway (Figure 1).

When a plant is attacked by a chewing herbivore, JA and its volatile methyl ester, methyl jasmonate (MeJA), immediately begin to be synthesized from α-linolenic acid at the site of the wound, increasing in concentration exponentially to amplify the attack signal. Conjugates of JA, particularly jasmonoyl-isoleucine (JA-Ile), subsequently propagate the signal via JAZ proteins and other molecular machinery to upregulate the expression of very large numbers of defense-related genes (e.g. >700 mRNAs affected in *Arabidopsis* [7]). The JA pathway, in combination with the ethylene

Figure 1



Effects of e[CO_2] and e[O_3] (both in red) on the JA-signaling and SA-signaling pathways. Lines ending in an arrow represent stimulatory or positive effects; lines ending in a circle represent suppressive or negative effects. Cross-talk between the JA-signaling and SA-signaling pathways is represented with blue lines. The blue dashed line indicates that the suppression of SA-signaling pathway by JA is relatively weak compared to the suppression of JA-signaling pathway by SA. Interactions represented in this figure comes from several sources (e.g. [28••,77,99–101]).

(ET) pathway can induce most plant physical and chemical defenses, including toxins, antinutrients, digestive enzyme inhibitors, and volatile compounds that can deter subsequent herbivores as well as attract herbivores' natural enemies [8]. The least well-understood aspect of the JA pathway is how wounding initiates JA synthesis, although roles have been identified for degraded endogenous plant molecules or molecules located outside their original compartments [9•] and endogenous elicitor peptides [10•] as well as herbivore-specific recognition factors [11]. Volatile organic compounds including monoterpenes and green leaf volatiles (C_6 molecules originating from the oxylipin pathway, in common with JA) also play important roles in rapid long-distance signal transmission, priming and induction of defense responses, within and between plants.

The second major plant hormone signaling pathway is the salicylic acid (SA) pathway, which initiates defense against many plant pathogens, including viruses and microbes, as well as against piercing-sucking phloem feeders that largely avoid causing the tissue damage that is required to induce JA (nonetheless, it is JA-induced rather than SA-induced defenses that confer the strongest resistance against aphids [12]). Like JA, SA activates a very large set of defense genes, both locally at the site of attack or infection and systemically, most of which are regulated centrally via the protein NPR1. In the context of defense against chewing and biting insect herbivores however, the SA pathway is most significant for its participation in cross-talk with the JA cascade, where its most common (but not universal) role is suppression of JA responses. Numerous plant hormones in addition to JA and SA are involved in the modulation of defense

responses to different biotic challenges, but ethylene (ET) is particularly significant for its role in prioritizing JA induction over SA induction when multiple attacks occur at once. JA and SA have separate biosynthetic origins and use separate compounds for signal transduction, however cross-talk between these pathways is an ancient phenomenon [13] and occurs at numerous biosynthetic nodes [14••]. Nonetheless, it is unclear whether cross-talk generally benefits either plants or herbivores [13]. Cross-talk is complex and varies among species. For example in *Nicotiana attenuata*, NPR1 downregulates SA production, thus inhibiting the antagonistic effects of SA upon JA and helps this plant avoid potential exploitation of cross-talk by herbivores as has been shown in several other systems [15]. One such instance is that *Spodoptera littoralis* oviposition fluids trigger SA in *Arabidopsis*, which benefits larval growth by countering the JA response [16]. It should be noted, however, that the great majority of studies of JA and SA crosstalk involve direct application of plant hormones to damaged leaf tissue of a few species in the laboratory, followed by analysis of gene expression profiles, and rarely have the implications for defense against herbivores *per se* been directly demonstrated, least of all under field conditions.

General effects of e[CO_2]

CO_2 is the key substrate for photosynthesis; not surprisingly, atmospheric CO_2 levels are strongly correlated with increased photosynthetic rates and net primary productivity [17–19]. Most effects of e[CO_2] on herbivores and their natural enemies are indirect, mediated by CO_2 effects on plant traits. e[CO_2] alters plant nutritive value, especially by decreasing the concentration (and sometimes the digestibility) of plant protein/amino acids and increasing the concentrations of structural and non-structural carbohydrates which are of little nutritional benefit to insect herbivores and which commonly defend cell contents. Here, we principally focus on how e[CO_2] affects allelochemical (defensive chemistry) production by plants [3••,4,20], with a focus on volatile chemical signals released by plants after attack by pathogens and/or herbivores. Responses of plant volatile and non-volatile allelochemical profiles to e[CO_2] are highly system-specific, posing a serious challenge to our ability to predict how ongoing climate change will affect the trophic interactions ubiquitous in all ecosystems [3••,4,21,22]. What is missing from most of these studies is a clear, mechanistic understanding of how e[CO_2] influences the expression of plant defensive chemistry and its subsequent effects on other trophic levels.

While e[CO_2] is generally associated with increases in carbon:nitrogen (C:N) ratios within plant tissues, the production of specific plant defensive chemistries and the performance of herbivorous insects in response to these are far from consistent. Concentrations of some carbon-based compounds such as phenolics (e.g. flavonoids, tannins, some of which have defensive functions against herbivores) generally increase in response to e[CO_2] [3••]; responses of other defensive chemicals to elevated CO_2 are less generalizable. For instance, concentrations of many, but not all, terpenoids (carbon-based compounds synthesized from isoprene units) decrease under e[CO_2] [3••,23]. Concentrations of nitrogen-containing defensive compounds such as glucosinolates either increase or decrease in response to e[CO_2] depending on the specific glucosinolate compound and plant species in question [21,24–26]. Induced glucosinolate production by *Arabidopsis thaliana* in response to herbivory is increased under conditions of e[CO_2] [25]. e[CO_2] is also known to suppress production of cardenolides, defensive compounds produced in milkweed (*Asclepias syriaca*) [27]. Rather than being regulated by resource/substrate availability, most PSM synthesis is tightly regulated by gene expression and, as such, selection from pathogens and herbivores can lead to great variation in the types of plant defenses [48,102].

e[CO_2] and plant defense hormones

Of the various greenhouse gases implicated in global climate change, CO_2 is by far the best studied in terms of its effects on plant signaling hormone responses (see review by Zavala et al. [28••]). Even so, most of our understanding of how CO_2 interacts with plant signaling hormone defenses against herbivores and plant pathogens is limited to studies involving two families: the Brassicaceae, including the model plant *A. thaliana*, and the Solanaceae, especially tobacco (*Nicotiana* spp.) and tomato (*Lycopersicon* spp.). Nevertheless, many of the mechanisms studied in these two plant families likely hold true in other plants. We summarize the salient mechanisms in Figure 1.

Elevated CO_2 suppresses the synthesis of JA and stimulates the synthesis of SA [28••,29,30••]. In general, e[CO_2] is expected to compromise a plant's ability to mount a successful induced defense against herbivores while increasing resistance to plant pathogens. Currently, the mechanism by which e[CO_2] induces the SA pathway is not known and we are just beginning to understand how CO_2 interferes with JA induction [28••]. e[CO_2] appears to interfere with the activity of mitogen-activated protein kinases, which are involved in the transduction of wound-damage and herbivore-damage signals to the chloroplast where the octadecanoid pathway is initiated [28••,31].

While the severity of damage incurred by plants due to pathogen attack is predicted to decline under conditions of e[CO_2], observed responses to e[CO_2] range from decreased severity [20,32] to increased severity [20,33,34]. The roles of JA and SA defense pathways were not examined in these studies and the observed differences among these systems may be explained by the effects of CO_2 on plant growth rates, canopy densities, stomatal characteristics, and wax deposition that affect overall humidity levels and susceptibility to pathogen

attack [20,28••]. However, to the extent that e[CO_2] may reduce plant disease, increased primary productivity and improved plant vigor may be expected to favor greater overall rates of herbivory and to sustain trophic interactions across multiple trophic levels. e[CO_2] is thought to exacerbate cross-talk between the SA and JA pathways (Figure 1), inducing SA accumulation and suppressing JA defense responses via antagonistic cross-talk between these two defense pathways [28••]. Correspondingly, e[CO_2] should result in reduced success rates for attacks by plant pathogens, but increased damage done by chewing herbivores. Studies of the parasitic root-knot nematode *Meloidogyne incognita* and the corn earworm *Helicoverpa armigera* on tomato (*Lycopersicon esculentum*) have shown that e[CO_2] induces the SA-signaling defense pathway of pathogenesis-related proteins (e.g. PAL) and reduces the JA-signaling defense pathway [35,36]. Similarly, soybeans (Fabaceae: *Glycine max*) grown under conditions of e[CO_2] produced increased levels of SA-regulated phenolics but decreased levels of JA-regulated cysteine proteinase (the major feeding deterrents to the western corn rootworm (*Diabrotica virgifera virgifera*) and the Japanese beetle (*Popillia japonica*) in soybean), resulting in increased levels of herbivory by these beetles [37,38]. Similar effects of e[CO_2] on pathway cross-talk has been shown in green peach aphids (*Myzus persicae*) feeding on *A. thaliana* [30••].

e[CO_2], volatile organic compounds, and the third trophic level

Most studies of e[CO_2]-mediated effects of plant chemistry on parasitoids have focused on volatile organic compounds (VOCs), which form a direct link between plants and the third trophic level. Parasitoids (and, to a lesser degree, predators) of insect herbivores use plant VOCs released after damage by herbivores to locate potential hosts/prey items [39–41]. Plant synthesis of a wide variety of VOCs is known to be influenced by e[CO_2], which may alter the ability of parasitoids to locate suitable hosts. However, whether e[CO_2] induces or suppresses VOCs is both system-specific and compound-specific [42–44]. In cases where e[CO_2] leads to increased feeding rates by herbivores, increased VOC production in response to wounding is also likely [44–46]. In one study, e[CO_2] substantially increased production of 11 different terpenoids by non-*Bt* and *Bt* canola plants (*Brassica napus*); but despite a doubling (or more) of some compounds, attraction of the parasitoid *Cotesia vestalis* to plants damaged by the diamondback moth (*Plutella xylostella*) was unaffected [46]. In contrast, another study of diamondback moth herbivory (this time on cabbage, *B. oleracea*) found that monoterpene emissions were depressed under e[CO_2] [43]. Interestingly, the pentatomid predator *Podisus maculiventris* did not show a preference for damaged over undamaged plants under e[CO_2] and the parasitoid *C. vestalis* showed a decreased ability to discriminate between moth-damaged and undamaged cabbage plants when grown under e[CO_2] [43]. The effectiveness of VOC bouquets as signals is often dependent upon their proportional composition, sometimes quite independently of the concentrations of any single components or of the mixture in total [47,48]. Thus, idiosyncratic responses of individual compounds to e[CO_2] across biosynthetic pathways may diminish signal fidelity, whether overall synthesis is up-regulated or down-regulated.

e[CO_2], PSMs and herbivore quality, and the third trophic level

In addition to the direct effects of volatile attractants (above), plant chemistry can influence the third trophic level indirectly via effects on herbivore quality [49,50]. The few studies linking the effects of e[CO_2] on plant defensive chemistry with parasitoid fitness have shown either no relationship [51,52] or a negative relationship [53••,54]. Whether herbivore quality is affected by e[CO_2]-mediated plant defensive chemistry depends in part on the detoxification and/or sequestration capabilities of herbivores. Parasitoids that attack herbivores that avoid or effectively detoxify plant toxins upon feeding, may experience little direct effect of plant chemistry. On the other hand, parasitoids attacking herbivores that are unable to detoxify and eliminate plant toxins (or which sequester them as a defense strategy) may directly encounter plant toxins themselves in the host tissues on which they feed [55–58].

Plant defensive chemistry may also mediate the ability of an herbivore to mount a successful defense against parasitoids. Insect defense against parasitoids (and other foreign invaders) is frequently via encapsulation followed by melanisation [59,60]. Once the encapsulation response is triggered, hemocytes adhere to a foreign body to form an envelope followed by melanization, which effectively suffocates the invader [61–63]. To our knowledge, the influence of e[CO_2] or e[O_3] on herbivore immune responses has not been investigated, but insects encountering nutrient stress are typically less successful in mounting immune responses [64] and may therefore succumb more easily to parasitism. Conversely, increased concentrations of some PSMs, including antioxidants and alkaloids, have been found to promote encapsulation and improved immune responses in some insect herbivores [65,66]. The longer larval development times typically associated with reduced leaf quality may also increase the duration of vulnerable exposure to parasitoids. Given the abundance of studies illustrating the effects of e[CO_2] and e[O_3] on PSMs, we suggest that changes in insect immune responses may be an important factor underpinning such herbivore–parasitoid interactions.

How e[CO_2] influences the interactions between herbivorous insects and their natural enemies depends not only

on the plant's ability to produce defensive compounds but also the ability of herbivores to detoxify such plant toxins. Almost nothing is known about how, or even whether, e[CO_2] influences the detoxification capabilities of insect herbivores (either directly or indirectly), although extremely elevated temperatures are likely to increase the rates of most metabolic processes in ectotherms, including detoxification. If CO_2 does affect the ability of herbivores to detoxify plant defensive chemicals, we expect that this would indirectly affect the parasitism success of parasitoids developing within such herbivores. Gypsy moths (*Lymantria dispar*) fed aspen leaves (*Populus tremuloides*) grown at e[CO_2] exhibited increased activity of a suite of detoxification enzymes (cytochrome P450 monooxygenase, esterase, and carbonyl reductase) [67]. Interestingly, these effects were absent when larvae fed on sugar maple (*Acer saccharum*) grown under e[CO_2], indicating that the host-plant chemistry-mediated effects of e[CO_2] on herbivore detoxification abilities are system-specific. On the other hand, the brown plant hopper (*Nilaparvata lugens*) exhibited decreased activity of the detoxification enzyme glutathione S-transferase when reared under e[CO_2] [68•]. Whether e[CO_2] directly or indirectly (via changes in plant chemistry) influenced the activity of *N. lugens* detoxification enzymes were not determined in this study. Highly system-specific trophic interactions involving host defense chemistry have been observed in other systems [69], suggesting that predicting the multi-trophic effects of climate change will be difficult and will need far more study than currently has been invested.

Effects of e[O_3]

In addition to e[CO_2], the last few decades have seen an increase in tropospheric concentrations of trace gases such as O_3 [1]. Plants play an important role in absorbing O_3 from the atmosphere, however this process is inhibited by stomatal closure, meaning that e[CO_2], drought, and heat waves may all act to reduce plant exposure to O_3. At the same time, these factors significantly increase ground-level O_3 concentrations over short timescales, to the direct detriment of higher trophic levels especially in parts of the world with locally intensive NO_2 emissions [70,71].

e[O_3] and plant defense hormones

Ozone in the lower atmosphere is a potent source of oxidative stress for plants [72]. Photosynthetic rates, water use efficiency, free amino acid content, and biomass are often suppressed under conditions of e[O_3] [73,74]. Elevated O_3 also influences plant quality for herbivores by modulating the expression of both JA-induced and SA-induced defenses (see Figure 1). In general, plants respond similarly to e[O_3] as they do to pathogens and piercing-sucking insects (e.g. aphids and whiteflies). Elevated [O_3] induces the production of reactive oxygen species (ROS) and increases SA biosynthesis [75,76]. Elevated O_3 induces greater production of pathogenesis-related protein (PR1) and lowers phloem concentrations of soluble sugars and amino acids, negatively affecting piercing-sucking insects such as aphids and whiteflies [77,78,79••]. Ozone is also known to upregulate antioxidant defense systems associated with the shikimic acid pathway [45], resulting in the production of several nitrogen-containing compounds (e.g. alkaloids, cyanogenic glycosides, and glucosinolates) as well as phenolic compounds such as phenylpropanoids, flavonoids, coumarins, lignins, and tannins [80]. Other key molecules involved in plant defense against ROS caused by ozone are ascorbic acid and glutathione, the synthesis and concentrations of which have been variously observed to increase and decrease in different plant genotypes and species [81,82]. If increased, dietary ascorbate and glutathione concentrations are likely also to benefit chewing herbivores challenged by pro-oxidant tannins and other allelochemicals [83]. Evidently, the fitness effects of e[O_3] on both phloem-feeding and chewing insect herbivores as well as pathogens are highly variable across species [84–86,20,78]. Species-specific effects of O_3 on plant nutritive quality may explain some of the variation in fitness responses. Also likely is a role of O_3 in modifying cross-talk between the JA-defense and SA-defense signaling pathways. O_3 increases the release of linolenic acid from cell membranes, which triggers the production of JA and moderates the influence of O_3 on the SA pathway [87]. Furthermore, O_3 increases the production of ethylene (ET), which in addition to interacting strongly with many components of the JA pathway, in turn induces the synthesis of abscisic acid (ABA) that regulates stomatal conductance and drought tolerance in plants [88]. Clearly much remains to be discovered regarding the influence of O_3 on plant–herbivore and plant–pathogen interactions, let alone their interactions with other trophic levels.

e[O_3] and VOCs

Ozone has important direct effects on insect herbivores and their natural enemies, primarily by interfering with VOCs used by herbivores to locate suitable host plants and by natural enemies to locate suitable hosts. e[O_3] can interact with volatiles attractive to parasitoids in complex ways. On the one hand, exposure to e[O_3] has been shown to increase emissions of MeSA as well as various terpenoids implicated in attracting parasitoids of insect herbivores [46,89]. As discussed above, O_3 strongly induces intracellular reactive oxygen species (ROS), which promotes the formation of free fatty acids such as linolenic acid, ultimately producing VOCs via the octadecanoid pathway [87,90]. e[O_3] induces the emission of several terpenoids by lima beans, *Phaseolus lunatus*, that are attractive to predatory mites, *Phytoseiulus persimilis*, which attack the herbivorous two-spotted spider mite, *Tetranychus urticae* [91]. However, higher O_3 levels can also react chemically with many terpenoids, degrading their biological activity [92] and potentially disrupting

plant-signaling interactions with both herbivores and higher trophic levels (e.g. parasitoids and predators) [93,94]. Nevertheless, moderately increased O_3 levels did not hinder the ability of the parasitoid *C. vestalis* to locate and successfully attack its host, diamondback moth *Plutella xylostella*, in the field [95] suggesting that volatiles other than, or in addition to, terpenoids are responsible for host location and acceptance by *C. vestalis* (e.g. nitriles, MeSA, MeJA, O_3-tolerant terpenoids such as 1,8-cineole [44,46,96,97]).

Conclusions, ideas, and future directions

As pointed out elsewhere (e.g. [98•]), we know very little about how atmospheric and climate change influence the chemical ecology of most trophic interactions, especially those involving herbivores and natural enemies [3••]. We are, however, making steady progress in the field and key references in this paper indicate there are some emerging patterns, especially in terms of how e[CO_2] affects signaling in plants, which will ultimately determine the expression of PSMs that affect herbivores, pathogens and other trophic groups [28••]. However, the response of plants, herbivores, and natural enemies to climate change is highly variable, and can be very system-specific, so it seems germane that scientists should include a wider diversity of study systems beyond Brassicaceae and Solanaceae, important though these are.

As discussed by Facey *et al.* [2] elsewhere in this volume, there is a clear need to incorporate multiple atmospheric and climatic variables when investigating these interactions. Logistic constraints often prevent adequate replication in such factorial experiments. The fact that most studies that investigate more than one variable report some sort of interaction (sometimes canceling one another out) [3••], however, suggests this is very important for getting a realistic insight into how future climates will shape multi-trophic interactions.

Some herbivore feeding guilds limit or avoid triggering defensive responses (especially the JA-pathway) in plants. These include piercing-sucking insects (e.g. aphids), herbivores that cut plant tissue cleanly with razor-like mandibles or those that trench or sever plant veins to deactivate delivery of PSMs (e.g. caterpillars feeding on milkweed). Given that e[CO_2] usually increases leaf toughness and thickness, this raises the intriguing possibility that some herbivores may be less able to make 'clean cuts' into plant tissue, causing more cellular damage (particularly as mandibles incur greater wear) and thereby may induce stronger defensive responses in the plant. We are unaware of any demonstrations of this, but it seems at least a plausible mechanism by which PSMs could be additionally triggered by herbivores, with consequences for their natural enemies.

In writing this article, we aimed to stimulate further interest in investigating how global change might affect PSMs, and how these changes might have far reaching consequences for pathogens as well as herbivores and their natural enemies. In addition to reports of e[CO_2] and e[O_3] increasing PSMs in plants, usually with deleterious effects on herbivores and (albeit less reported) their natural enemies, we identify other mechanisms by which altered PSMs could affect herbivore immunology and feeding behavior. From the limited information available, it does seem that with anthropogenic atmospheric change we are indeed reshaping the building blocks of multitrophic interactions.

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