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REVIEW ARTICLE

Disruption of the 'disease triangle' by chemical and physical environmental change

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ABSTRACT

The physical and chemical environment of the Earth has changed rapidly over the last 100 years and is predicted to continue to change into the foreseeable future. One of the main concerns with potential alterations in climate is the propensity for increases in the magnitude and frequency of extremes to occur. Even though precipitation is predicted to increase in some locations, in others precipitation is expected to decrease and evapotranspiration increase with air temperature, resulting in exacerbated drought in the future. Chemical [ozone (O_3) and other air contaminants] and subsequent physical alterations in the environment will have a profound effect on the 'disease triangle' (a favourable environment, a susceptible host and a virulent pathogen) and should be included in any analysis of biological response to climate change. The chemical and physical environment affects plant health and alters plant susceptibility to insect and pathogen attack through increased frequency, duration and severity of drought and reduction in host vigour. The potential effects of climate change and O_3 on tree diseases with emphasis on the western United States are discussed. We describe a generalised modelling approach to incorporate the complexities of the 'disease triangle' into dynamic vegetation models.

INTRODUCTION

The chemical and physical environment of the Earth has changed over the last century and is predicted to continue to change dramatically in the foreseeable future (Christensen *et al.* 2007; US Climate Change Science Program 2008; IPPC 2014). Increased greenhouse gas emissions and secondary air pollutants are linked concomitantly to climatic disturbances: airborne contaminants are the source of climate change. One of the main concerns with climate change is the likelihood of increased magnitude and frequency of meteorological extremes (Easterling *et al.* 2000), especially multi-year chronic and/or acute drought (Dale *et al.* 2001; Seager *et al.* 2009). For example, California experienced extreme drought in 2014, preceded by several years of chronic drought (Howitt *et al.* 2014). This occurrence was a one in 500 year event, that itself was preceded by a one in 250 year drought in 2002. Evidence is mounting that these shifts are likely the result of climate change (Mote *et al.* 2005; Mote 2006) .

In the western United States global circulation models (GCMs) predict greater water deficits due to air temperature-driven evaporative demand (Mote *et al.* 2005; Mote 2006). Most of the GCMs predict a negligible change (1–2% increase or decrease, depending on the model) in total annual precipitation and a shift in its timing in the western states, except for areas in southern Arizona and New Mexico. All 42 GCMs agree (IPCC 2014) that air temperature will increase 3.5 °C to 7.5 °C by mid-century. If there were no change in total annual precipitation, this temperature increase would likely result in a ~15% decrease in water available to biological systems (Ron Neilson, personal communication). Drought dramatically affects crop and timber production, with losses in the billions of dollars (NOAA National Climatic Center 2006; Howitt *et al.* 2014; Hartmann *et al.* 2015). Droughts are more prevalent in the summer months, but in recent years have occurred with increasing frequency in spring and autumn.

The world's population continues to multiply at an alarming rate (UNPD 2005). Urbanisation, agricultural production, industrial activities and increased fossil fuel consumption for transportation have dramatically increased greenhouse gas emissions and secondary pollutant production [NO_y, ozone (O_3)]. For example, central and southern California has experienced rapid population growth over the last six decades, resulting in increased precursors of air pollutants (Staszak *et al.* 2007). Despite more stringent air quality regulations, daily 24 h O_3 concentrations continue to increase, and many areas in the United States are not in compliance with current US EPA National Ambient Air Quality Standards (NAAQS) for tropospheric O_3, such as those downwind from large metropolitan areas, as well as combined, cumulative effects of smaller cities and industrial areas in the eastern USA (US EPA 2006).

Environmental factors (both chemical and physical atmospheric changes) alter plant health, their susceptibility to physiological drought stress (Grulke *et al.* 2009; Hartmann *et al.* 2015), as well as subsequent pathogen attack (Manning & von Tiedemann 1995). The interactions among environmental

conditions, air pollution, hosts, insect vectors and plant pathogens are highly complex. We have modified the classic 'disease triangle' (Fig. 1; Sandermann 1996): the relationships between favourable environmental conditions – host susceptibility – and a virulent pathogen, to incorporate the complexities of the chemical and physical environment, as well as the effects on plant pathogens, including insect vectors of these pathogens (Fig. 2). Considering the effects of temperature alone, we expect increases in minimum winter temperatures, earlier onset of spring, later onset of autumn and a longer duration of summer with higher maximum temperatures (Easterling *et al.* 2000; Intergovernmental Panel on Climate Change 2014). Because of warmer winter temperatures, more precipitation will occur as rain, and wetter autumns and springs are expected in lieu of lower precipitation during winter. The onset of summer water deficits will be earlier in summer-dry climates (Fig. 3).

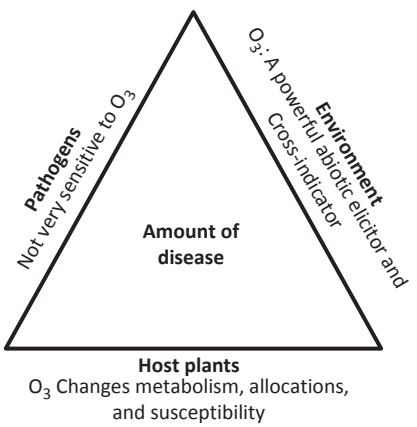

Fig. 1. The classic disease triangle, attributing the effect of the environment and including O_3 exposure on plant pathogens (negligible direct effects) and host plant (significant direct effects) on the amount of disease experienced by the host plant (text modified from Sandermann 1996).

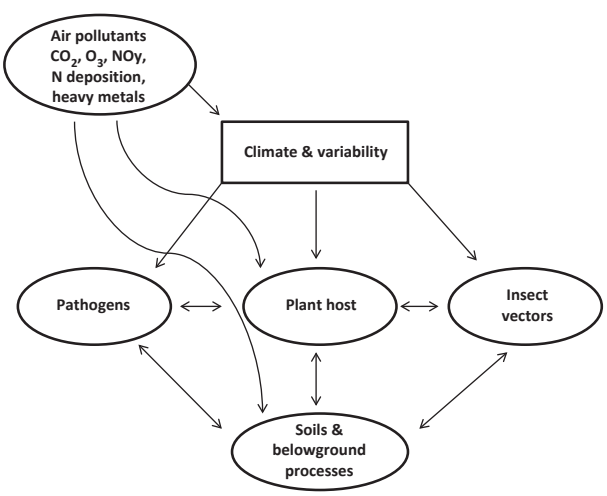

Fig. 2. Expansion of the disease triangle incorporating the influence of air chemistry and environmental variability on the health of the plant host and its susceptibility, the virulence of the pathogen, insect vectors (if part of the pathogen–plant system), and feedbacks between these components, soils and belowground processes critical for also understanding root pathogens (amended from Grulke 2011). NOy = ∑ of NOx+ compounds produced from oxidation of NOx.

These environmental effects combined with the interplay and outcome of fertilisation effects of both elevated CO_2 and N deposition and the deleterious effects of atmospheric pollutants (O_3, NO_y) on total plant biomass within-plant resource allocation and plant susceptibility to pathogens and insect vectors is complex. There will be a greater asynchrony with plant host, pathogen and insect vector phenologies and weather conditions (Manning & von Tiedemann 1995; Chakraborty 2005; Grulke 2011). Higher temperatures will promote growth and/or increase pathogen populations earlier in the year, but water deficits and altered nutritional balance and carbohydrate reserves in plant tissue will constrain growth. In addition, root pathogens may be constrained by lower soil moisture, alterations in plant carbon reserves and competition from an altered microbial and fungal community structure.

With emphasis on the western USA, we review the potential effects of the changing physical and chemical atmosphere on trees, the likely changes in tree susceptibility to diseases, and present a generalised modelling approach to incorporate these disruptions to the 'disease triangle' into dynamic vegetation models such as MC1 (Bachelet *et al.* 2001) and ED2 (Medvigy *et al.* 2009). Since O_3 is the most serious phytotoxic pollutant due to transport from urban to rural and forested regions (US EPA 2013; Cooper *et al.* 2014), it will be used as a case study, but the principles illustrated can be used in other plant pathogen–air pollution interaction studies.

CONSIDERATIONS FOR CHEMICAL AND PHYSICAL ENVIRONMENTAL CHANGE EFFECTS TO THE DISEASE TRIANGLE

In the western USA, climate change has already affected host physiology (Matyssek *et al.* 2014) and insect vector (Bentz *et al.* 2010) and pathogen life cycles (Sturrock *et al.* 2011). Increased air temperature has prolonged the growing season, and resulted in changes in the form, amount and timing of precipitation (Mote 2006). Higher winter temperatures increase plant host respiratory costs, and improve over-winter survival of both plant pathogens and their insect vectors (Fig. 3). Higher air temperatures accelerate earlier bud break (as light induction precedes temperature induction by months) and plant growth, but will also stimulate earlier pathogen and insect vector life stages. The timing of all three phenologies – host, insect vector and pathogen – may or may not be synchronous. If an insect promotes tree inoculation with a pathogen, but the insect life cycle timing is significantly offset from that of the pathogen, pathogen infestation may decrease. With longer growing seasons, both pathogens and insect vectors may be able to complete multiple life cycles, thus increasing the potential for a large-scale plant disease epidemic to occur (Manning & von Tiedemann 1995; Bentz *et al.* 2010; Sturrock *et al.* 2011).

Throughout much of the western USA, an increase in frequency, duration and severity of drought has been observed (Cook *et al.* 2004; Pederson *et al.* 2011). Many GCMs also predict reduced plant-available moisture, either directly through reduced total annual precipitation or *via* increased temperature-mediated evapotranspiration for the Pacific and intermountain region (Intergovernmental Panel on Climate

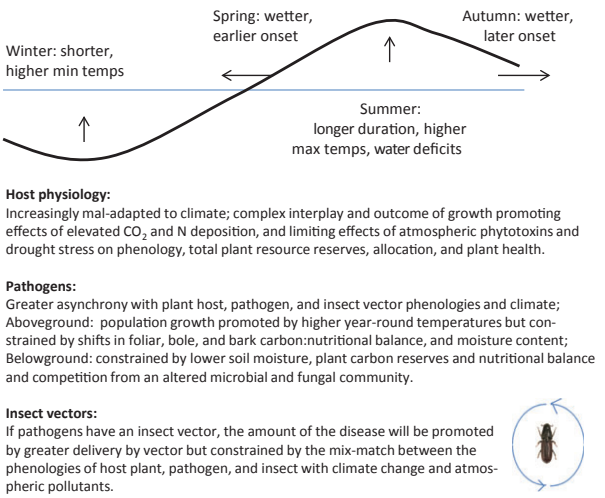

Fig. 3. Inclusion of air pollutants and other environmental change effects on components of the disease triangle with emphasis on timing and duration of climate events.

Change 2014). The environmental effects of climate change, as well as anthropogenic disturbance to soils, both decrease and alter mycorrhizal community diversity and composition, potentially leading to greater mismatch between belowground symbiont and/or pathogen and host tree (Allen *et al.* 2014; Wang *et al.* 2015). Consequently, lower soil moisture and less water transfer from mycorrhizae to the tree will increase plant physiological drought stress, increasing the likelihood of aboveground plant pathogens. However, a decrease in soil moisture may reduce such pathogens as root rots (Sturrock *et al.* 2011).

OZONE FORMATION

Combustion of any carbon-rich compound increases tropospheric concentrations of CO_2, CO, NOy (sum of NOx + compounds produced from oxidation of NOx) and nitrogen-rich particulates, as well as O_3, which is the result of secondary chemical reactions (photolytic cycle). In quantity, these alterations in atmospheric chemistry subsequently influence global temperature and precipitation patterns. During the 20th century tropospheric O_3 increased globally (Vingarzan 2004; Cooper *et al.* 2014). Ozone concentrations doubled in Europe between 1950–2000 (Cooper *et al.* 2014). From 1990–2010 in the eastern USA O_3 concentrations have decreased in the summer months and increased in the winter, while in the western USA O_3 concentrations have shown the largest increases in the spring months. Tropospheric O_3 is a significant greenhouse gas associated with climate change (Cooper *et al.* 2014; IPCC 2014).

OZONE EFFECTS ON PLANTS

Since Middleton (1956) first recognised O_3 as phytotoxic, evidence has mounted that it is the most significant regional air pollutant deleteriously affecting vegetation in the USA (US EPA 1996, 2006, 2013). O_3 concentrations during pre-industrial times averaged <0.02 ppm ($\mu l \cdot l^{-1}$), but presently ranges between 0.02–0.045 ppm in non-source areas of the world (NARSTO 2000). Initially believed to be an urban problem, O_3 is transported downwind, such that many of the world's most productive agricultural and forested regions are currently affected (Chameides *et al.* 1994; Ashmore 2005; Ren *et al.* 2007, 2011).

Ozone affects nearly every aspect of plant physiology and structure, either primarily or secondarily. The effects have been reviewed from several different perspectives (Chappelka & Chevone 1992; Matyssek *et al.* 1995; Heath & Taylor 1997; Pell *et al.* 1997; Schraudner *et al.* 1997; Chappelka & Samuelson 1998; De Kok & Tausz 2001; Grulke 2003).

The deleterious effects of O_3 originate with subcellular and cellular injury, which causes metabolic changes and eventually, alterations in growth if the exposure to O_3 is sufficient and plant protective or repair mechanisms are overcome. In general, strong oxidant (O_3, NO_y, SO_2) exposure lowers photosynthetic capacity, increases respiration, results in stomatal dysfunction, reduces nutritional content and results in elemental imbalances. Strong oxidant exposure alters within-plant priorities for resources: more reparation activities result in less C allocation to roots, less foliar biomass retention, higher nutrient content of excised tissues and shifts in quantity and location of stored carbohydrates (Grulke 2003; Paoletti & Grulke 2010).

Trees can exhibit visible symptoms due to O_3 exposures. These symptoms can vary from stippling on deciduous species to chlorotic mottling and decreases in foliar retention in the various needle age classes in conifers (Chappelka & Chevone 1992; Chappelka & Samuelson 1998). In the western US, three methodologies have been developed to assess visible O_3 injury in pines (Pronos *et al.* 1978; Miller *et al.* 1996; Grulke & Lee 1997). In sensitive species and individuals, greater visible injury is often correlated to overall reduced health (lower photosynthetic capacity, biomass, foliar nutrient content). Some species (such as white fir, *Abies concolor* Gord. & Glend.) are asymptomatic but have compromised carbon acquisition (Retzlaff *et al.* 2000).

Ozone uptake at moderately high levels and above is likely to negate the stimulation of plant carbon sequestration by elevated CO_2 concentrations. For example, whereas elevated CO_2 increases plant carbon allocation to roots (Matyssek *et al.* 2014), elevated O_3 exposure decreases allocation to roots, predisposing trees to drought stress (Grulke *et al.* 2009). Both gases and elevated temperature increase plant respiration, reducing carbon sequestration in storage organs. Both O_3 (Grulke *et al.* 2009) and CO_2 (Matyssek *et al.* 2014) also increase leaf turnover, reduce nutrient content of excised leaves and roots, and will alter nutritional balance of both retained and excised tissue and, in addition at a longer time scale, alter elemental ratios in soil.

OZONE AND PLANT DISEASE

Plant response to various disease-causing pathogens can be changed through introducing O_3 into the system (Heagle 1973; Chappelka & Chevone 1992). However, these effects are variable, depending on timing and exposure of both the pathogen and O_3, and other environmental factors (Fuhrer 2003). Ozone can alter the tree vigour and reduce defensive compounds, which allows predisposition of the plants to infection and colonisation by the pathogen (San-

dermann 1996; Matyssek *et al.* 2012). However, both O_3 exposure and fungal pathogens induce antioxidant defences in plants (Sandermann 1996). The effects on individual tree species and ecosystems are variable, depending on underlying genetics and environmental conditions, O_3 concentration, timing and exposure (and uptake), underlying capacity for resource acquisition and antioxidant defence and all combined effects on both the tree and the pathogen (Fig. 2).

Regarding O_3–plant pathogen interactions there are four classes of critical features to be considered. These are physical and chemical characteristics of the environment, defence features of both host and pathogen, population-level features and interspecific interactions (Table 1).

Research on the interactions of O_3 and forest tree pathogens is limited (Chappelka & Chevone 1992; Manning & von Tiedemann 1995). The earliest known work on O_3–disease interactions with forest trees was in southern California with ponderosa (*Pinus ponderosa* Laws) and Jeffrey (*Pinus Jeffreyi* Grev. & Balf.) pines, and their relationship with the root rot fungus, *Heterobasidion annosum* (James *et al.* 1980). These authors found that pines with visible oxidant injury (*e.g.* a symptom correlated with a compromised carbon acquisition system) had increased susceptibility to infection and colonisation by root rot in both the field and in fumigation chambers. In chambers with elevated O_3, more pine seedlings were infected with the fungus than those in O_3-free chambers. Fenn *et al.* (1990) investigated the effects of O_3 exposure on black stain root disease (*Leptographium wageneri var. ponderosum*) of ponderosa pine. In inoculated pine seedlings, there was more O_3 foliar injury and decreased stem growth; with increasing O_3 concentrations, the disease lesion length also increased. In a recent study, Pollastrini *et al.* (2015) working with *H. annosum* and *H. irregulare* found O_3 caused a decrease in terpene production in seedlings infected with the pathogens compared to the controls. This may possibly predispose the trees to increased colonisation. The above research (James *et al.* 1980; Fenn *et al.* 1990; Pollastrini *et al.* 2015) suggests intriguing interactions between air quality, pathogen virility and seedling growth of a pine species sensitive to O_3.

Lackner & Alexander (1983), in a field study, excavated roots from air pollution-sensitive and -tolerant trees in the Blue Ridge Parkway in Virginia. They recovered *Leptographium procerum*, *H. annosum*, *L. serpens* and *Graphium* sp. (24, 8, 8 and 4%, respectively) from the roots of the sensitive trees. No fungi were recovered from the tolerant trees. In addition, two weevil species were isolated from the sensitive trees.

Luedemann *et al.* (2005) in a phytotron study investigated the response to O_3 of two tree species (*Fagus sylvatica* and *Picea abies*) growing together and infected with the root pathogen *Phytophthora citricola*. They found that when grown together the susceptibility to the root pathogen was increased with O_3 for spruce compared to beech, and related this to shifts in resource allocation, thus increasing the defence capacity in beech.

Working with loblolly pine, Carey & Kelley (1994) reported that O_3 caused a predisposition to colonisation by the pitch canker fungus, *Fusarium subglutinans*. Cankers were significantly smaller for the pitch canker-resistant loblolly pine families compared with susceptible ones; however, elevated O_3 resulted in larger cankers regardless of sensitivity to the pathogen. These stresses in combination resulted in larger decreases in growth and productivity of the seedlings than when alone.

Paoletti *et al.* (2007) in a study with cork oak (*Quercus suber*) and turkey oak (*Q. cerris*) found that exposure to O_3 predisposed the leaves to infection and colonisation by the leaf pathogens, *Diplodia corticola* and *Biscogniauxia mediterranea*. The primary cause of the increased leaf injury appeared to be higher germination of spores in the O_3-exposed seedlings compared to control plants.

There is very little information available regarding interactions among O_3, or climate change in general, and other microbial organisms, such as bacteria and viruses (Manning & von Tiedemann 1995). Diseases caused by necrotrophic fungi are generally enhanced by O_3 exposure (Manning & von Tiedemann 1995), but O_3 generally inhibits biotrophic fungi (Manning & von Tiedemann 1995). Mycorrhizae can buffer the effects of O_3 (and drought) on plants (Bonello *et al.* 1993), but changes in metabolism can feed back to shifts in mycorrhizae morphotypes (Qiu *et al.* 1993) and community diversity and composition (Allen *et al.* 2014), likely altering plant nutrient and water uptake.

Although much has been written on tree response to the combined response of elevated CO_2, and elevated O_3, little research has been conducted on their concurrent effects regarding plant pathogens. There has been significantly more literature on O_3 in combination with CO_2 and insect herbivory

Table 1. Four classes of critical features to be considered in O_3–plant pathogen interactions[a].

A. Physical and chemical characteristics of the environment
Climate (temperature, season length, and frequency, severity and duration of drought)
Site (location, microsite, aspect, nutrition)
Light penetration for lower canopy and understorey
High light (oxidative stress) in mid- and upper canopy
Mix of pollutants
Multiple stressors
B. Biological and chemical (nutrition, defences) features of the host and pathogen
Nutritional suitability of the host plant
Biochemical and physical defences of the host plant
Vulnerability of the host
Functional type of pathogen
Necrotropic
Biotrophic
C. Population-level features
Host (age structure, distribution within microsites and the landscape, proximity to another host, proportion of population poor, average and above average health)
Pathogen (density, distribution pattern [disease propagation and progression], simple interest (single generation), compound interest (multiple generation)
D. Interspecific interactions
Pathogens, symbionts
Insect vectors
Competition between trees, nearest neighbour (NN) and species
Community succession

[a]Adapted from Bedford (1987) and Chappelka & Chevone (1992).

(Valkama *et al.* 2007). In an observational study at the Aspen FACE site (Rhinelander, WI, USA) Karnosky *et al.* (2002) investigated the interaction of elevated CO_2 and/or O_3 on the occurrence and severity of aspen leaf rust (*Melampsora medusae Thuem. f. sp. tremuloidae*) on trembling aspen (*Populus tremuloides* Michx.). Depending on the clone investigated, the rust fungus increased three- to four-fold with O_3 alone or CO_2 + O_3 after 2 years of fumigation. The authors concluded that O_3 altered leaf surfaces (reduction in waxes, etc.) and predisposed plants to increased infection by aspen leaf rust.

Enhanced fungal activity under aspen (*Populus tremuloides* Michx.) grown in elevated CO_2 was not maintained when aspen was also exposed to elevated O_3 (Phillips *et al.* 2002). Kasurinen *et al.* (1999) found that Scots pine (*Pinus sylvestris* L.) exposed to two times ambient CO_2 did not increase carbon allocation to roots or mycorrhizae, and elevated O_3 did not cause root growth limitation in seedlings after 3 years. In a prior study with Scots pine, Perez-Soba *et al.* (1995) found that elevated CO_2 did not alleviate the deleterious effects of O_3 on mycorrhizae. Perhaps these results can be applied to pathogenic biotrophic fungi. Wang *et al.* (2015) reported the colonisation of ectomycorrhizal fungi and species abundance of a hybrid larch (*Larix gmelinii* var. *japonica* × *Larix kaempferi*) under elevated CO_2 and O_3. After two growing seasons, ectomycorrhizal colonisation and root biomass increased under elevated CO_2, but O_3 fumigation resulted in decreased ectomycorrhizal colonisation and species richness and a decrease in stem biomass. No effects of combined fumigation (CO_2 + O_3) were observed on ectomycorrhizal colonisation.

Successful pathogen infection and colonisation may or may not occur for several reasons. These include abiotic factors such as environmental conditions that are favourable for plant susceptibility to infection (*e.g.* drought stress or waterlogging) or pathogen survival and/or growth (*e.g.* extremes in temperature or moisture that keep growth in check or are lethal, or favourable conditions for growth, propagation and transport) as shown in Figs 2 and 3. Biotic factors that influence the host–pathogen relationship include physical attributes of the host that prevent or promote infection (*e.g.* thick bark or a vigorous host callus response that blocks further infection or, conversely, a mechanical wound); host chemical attributes (*e.g.* host is the wrong (or right) species; insufficient *versus* sufficient tree nutritional composition or host defence response (Sandermann 1996); presence or absence of biotic vectors; multiple challenges to the host). Whether an outbreak occurs at the stand level is dependent on enuvironmental factors, forest stand attributes such as the proximity of nearest neighbors that are also susceptible, pathogen virulence and functional type (necrotropic, biotrophic) and whether pathogens have multiple generations within a year (Bedford 1987; Chappelka & Chevone 1992).

GENERALISED MODELLING APPROACH FOR HOST–PATHOGEN INTERACTIONS

As climate continues to change, the effects on forest tree diseases and subsequent effects to forest ecosystem health will change (Sturrock *et al.* 2011). One way to examine these effects within a landscape is through modelling (Scherm 2004; Chakraborty 2005). The current approach to incorporating pathogens into dynamic vegetation models is pathogen- and species-specific (*e.g.* Swiss needle cast, *Phaeocryptopus gäumannii,* and Douglas-fir, *Pseudotsuga menziesii*; Manter *et al.* 2005). There is a great need to incorporate such information into dynamic vegetation models for the purpose of predicting climate change-driven forest dynamics, but it is impractical to continue to develop single-species empirical models, even for economically important species.

A generic base model that relies on environmental databases for incorporating pathogen responses to environmental conditions into dynamic vegetation models could be managed using a matrix table of varying functions to call up appropriate environmental attributes to estimate biological responses for the different functional types of pathogens. We present a two-dimensional model for simplicity; however, the model should be multi-dimensional. This model is schematised in Fig. 4 and could be implemented, *via* a two-dimensional array of function pointers, in C++ or any other language with function/subroutine pointers. Similarly, without the analogy of a matrix, it could also be implemented *via* a series of switch-case statements in C/C++, Matlab, Python, etc.

In this paper, we proposed modifications to the original disease triangle to capture the complexities of environmental change effects on the plant host and pathogen (Fig. 2; see also Grulke 2011) to support development of this generalised modelling approach. We considered physical and chemical atmosphere attributes, their effects on soil, plant host (s), insect vector (if present) and pathogen dynamics. Attributes such as host density, heterogeneity, as well as available host size classes should also be incorporated into the model.

Dynamic vegetation models already reference down-scaled climatic data ('environmental data stack'; Fig. 4), have well-developed physical, chemical and hydrological soil attributes, ecosystem processes and current and future assemblages of plant functional types (plant host 1, 2; Fig. 4) to govern level of host availability, susceptibility, virility of pathogen and strength of pathogen vectoring. Our purpose in proposing this generalised model is to incorporate pathogen dynamics into dynamic global vegetation models (*e.g.* MC1, ED2; Bachelet *et al.* 2001; Medvigy *et al.* 2009).

We suggest that pathogen success (equation 1 below; Fig. 4) in the current year (t_0), is a function of:

$$PathogenSuccess = f_{(t0)}(\mathrm{PRESENCE}_{(t-1)}, \mathrm{ENV}, \mathrm{PATH\ VIRIL}, \mathrm{HOST\ SUSC}(1,2), \mathrm{INSECT}, \mathrm{INSECT\ PRED}) \quad (1)$$

where PRESENCE is prior year presence ($t-1$) in the cell (pixel); ENV is favourable or unfavourable environmental conditions, defined by a threshold value, or a function of multiple environmental conditions, such as minimum winter temperature, maximum summer temperature, early summer relative humidity (Stone *et al.* 2008); PATH VIRIL is pathogen virility, such as Dwarf Mistletoe Ranking or Colonisation Index for Swiss Needle Cast (Stone *et al.* 2008); HOST SUSC is host susceptibility, which depends on physiological status in its known entirety (already parameterised in MC2); INSECT is a pathogen that is carried by an insect vector (otherwise set to null);

and INSECT PRED is an insect predator that constrains the vector.

Pathogen virility (PATH VIRIL) could be defined as (equation 2):

$$\text{Pathogen Virility} = f_{t0}(\text{MIN T}, \text{MAX T}, \text{RH}, \text{WS}, \text{DD}, \text{CD}) \quad (2)$$

where MIN T is a function of current year minimum winter temperature, with values below a threshold returning a null value (*e.g.* lethal) or minima above a threshold increasing population size (insert a growth function as defined in literature). MAX T is a function of maximum summer temperature, with values below a threshold not affecting virility but values above a threshold returning a null value (lethal). RH is the relative humidity at the time of dispersal. WS is the wind speed at the time of dispersal. DD is cumulative growing season degree-day after host bud break, the length of the pathogen growing season promoted by air temperature and high relative humidity conducive to growth, constrained by low relative humidity restrictive to growth, and lethal air temperatures in the future. CD is community diversity, a measure of microbial and fungal biodiversity relevant for pathogen–pathogen competition.

Host susceptibility could be defined as (equation 3):

$$\text{Host Susceptibility}(1,2) = f_{t(0..-5)}(\text{PPT}, \text{DD}, \text{MAX T}, \text{C:N}) \quad (3)$$

as a function of the level of water, nutritional or carbon deficit-induced plant stress of the primary host, or both the host–alternate host system. Dynamic vegetation models are already well described and parameterised for evaluating these types and levels of tree physiological stress in physiognomic plant classes (contain physiological parameterisation for multiple but species with similar constraints). The proposed equation (equation 3) reflects current and prior years (through $t-5$) total annual precipitation (PPT), cumulative degree-day (DD), summer maximum air temperature (MAX T) and a measure of aridity such as the Palmer Drought Index or the difference between potential and actual evapotranspiration. For some pathogen–host relationships, host susceptibility may not be attributable to any known environmental stressor, in which case this attribute is input as null. If the pathogen does not have an alternative host (HOST 2), it is not used.

The insect vector could be defined by the following (equation 4):

$$\text{Insect Vector} = f_{t0}(f_{(t-1)}(p/a), \text{MIN T}, \text{MAX T}, \text{DD}, \text{WS}) \quad (4)$$

where the function for prior year carrier insect presence in the cell or shape file or adjacent cell as for the pathogen, and all other variants have been described previously except for insects, could have different thresholds or driving environmental factors. Since the environmental data is in a database, the functions can call up whatever is appropriate to the organism, or guild. Similar to that for pathogens, MIN T will increase over-winter mortality, MAX T will increase summer mortality due to potentially (future) lethal temperatures, DD will govern the number of generations of insect vectors within a growing season, and WS enables distribution of the insect vector.

Insect predation will reduce the effectiveness of the insect vector of pathogen (equation 5):

$$\text{INSECT PRED} = f_{(t0)}(p/a) \quad (5)$$

where INSECT PRED is the presence of the predator in the cell in the current year.

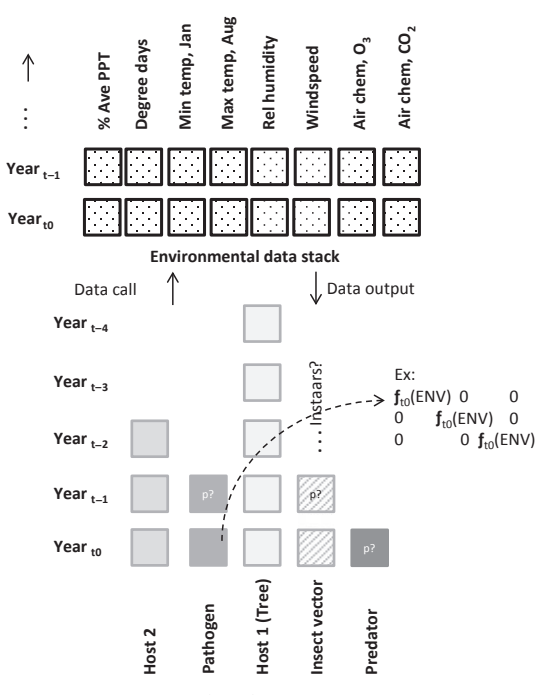

Fig. 4. A generalized modelling approach to incorporate plant pathogen and insect vectors into dynamic vegetation models using existing monthly environmental data stacks (dotted squares above), already parameterised plant processes (using physiognomic plant classes, here 'host 1, 2') determined as susceptible or resistant based on functions called up and applied using matrix algebra (represented by white to grey squares below). Prior year ($t-1$) presence (p?) of pathogen and insect vector will promote probability for an outbreak and current year (t) presence of insect predator will constrain insect population.

SUMMARY

Changes in air chemistry and subsequent physical changes in the environment have a profound effect on the disease triangle and should be incorporated into research designs, their analyses and in empirical modelling of pathogen effects on forest ecosystems. Trees in the western USA, especially pine, may be predisposed to pathogen attack by increasing O_3 exposure. Combined elevated O_3 and CO_2 effects on plant pathogens within controlled or natural setting are not well quantified, and new research is needed in this arena. Of the studies examined with concurrent exposure to both gases, none demonstrated that elevated CO_2 would mitigate the deleterious effects of elevated O_3 on mycorrhizae colonisation frequency, growth or longevity. Perhaps these results for symbiotic fungi can be used to guide expectations for pathogenic fungi in a current and future CO_2- and O_3-enriched troposphere. We suggest using

our generalised modelling approach to incorporate the complexities of environment–plant host–pathogen relationships into dynamic vegetation models.

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