Consequences of Global Change on Terrestrial Ecosystems: Integration and Scaling of Increased UV-B Radiation Effects on Ecological Processes from Molecular to Community Interactions

A Dissertation
Presented in Partial Fulfillment of the Requirements for the
Degree of Doctorate of Philosophy
with a
Major in Natural Resources
in the
College of Graduate Studies
University of Idaho
by

Vasile-Alexandru Suchar

Major Professor: Ronald Robberecht, Ph.D.
Committee Members: Stephen Bunting, Ph.D.; Brian Dennis, Ph.D.; Dennis Ferguson, Ph.D.
Department Administrator: Anthony Davis, Ph.D.

May 2015

Authorization to Submit Dissertation

This dissertation of Vasile-Alexandru Suchar, submitted for the degree of Doctorate of Philosophy with a Major in Natural Resources and titled "Consequences of Global Change on Terrestrial Ecosystems: Integration and Scaling of Increased UV-B Radiation Effects on Ecological Processes from Molecular to Community Interactions," has been reviewed in the final form. Permission, as indicated by the signatures and dates below, is now granted to submit final copies to the College of Graduate Studies for approval.

Major Professor: _____ Date: _____
Ronald Robberecht, Ph.D.

Committee Members: _____ Date: _____
Stephen Bunting, Ph.D.

_____ Date: _____
Brian Dennis, Ph.D.

_____ Date: _____
Dennis Ferguson, Ph.D.

Department Administrator: _____ Date: _____
Anthony Davis, Ph.D.

Abstract

This is the first mathematical model to integrate the effects of increased UV-B radiation through molecular level processes, whole plant growth and development, and community interactions. The model simulations showed that increased UV-B radiation-induced DNA damage significantly delayed cell division until the injury is repaired, resulting in significant reductions in leaf growth and development. Also, it significantly inhibited plant growth by delaying leaf expansion processes and increasing plant metabolic rates and/or reducing the photosynthesis rate. The costs of effective epidermal UV-B radiation absorptive compounds did not result in any significant changes in plant growth, but any associated metabolic costs can effectively reduce the potential plant biomass. There are significant interactions between UV-B radiation, temperature and any factor leading to inhibition of photosynthetic production or plant growth during the daytime, but the effects were not cumulative for all factors. Vegetative growth was significantly delayed in species that do not exhibit reproductive cycles during a growing season, but vegetative growth and reproductive yield in species completing their life cycle in one growing season did not appear to be delayed more than two to five days. The model showed significant differences between growth forms in the increased UV-B radiation effects of growth. In communities, the UV-B radiation sensitive species was constantly outcompeted by the resistant species. But small morphological and physiological changes can cancel the resistant species competitive advantage. A review of the relevant literature showed a wide range of values for the key parameters. Moreover, certain parameter values were inferred only from the calibration process. However our model allowed the testing of several to examine a variety of questions that were difficult to approach through experimental research.

Acknowledgments

I would like to thank my committee for all their help and support: Ronald Robberecht, Stephen Bunting, Brian Dennis, and Dennis Ferguson.

I would like to thank Stephan Flint for all his insightful comments and ideas.

I would like to acknowledge Andrew Ford contribution: he taught me the modeling technique that made this research possible.

This research was funded in part by the University of Idaho Student Grant Program No. UI07358.

Dedication

To my wife Livia – it is a privilege to share my life and love with you.

Table of Contents

List of Figures

List of Tables

Chapter 1

Integration and scaling of UV-B radiation effects on plants: from DNA to leaf

forthcoming in Ecology and Evolution

1.1. Abstract

A process-based model integrating the effects of UV-B radiation through epidermis, cellular DNA, and its consequences to the leaf expansion was developed from key parameters in the published literature. Enhanced UV-B radiation-induced DNA damage significantly delayed cell division, resulting in significant reductions in leaf growth and development. Ambient UV-B radiation-induced DNA damage significantly reduced leaf growth of species with high relative epidermal absorbance at longer wavelengths and average/low pyrimidine cyclobutane dimers (CPD) photorepair rates. Leaf expansion was highly dependent on the number of CPD present in the DNA, as a result of UV-B radiation dose, quantitative and qualitative absorptive properties of epidermal pigments, and repair mechanisms. Formation of pyrimidine-pyrimidone (6-4) photoproducts (6-4PP) has no effect on the leaf expansion. Repair mechanisms could not solely prevent the UV-B radiation interference with the cell division. Avoidance or effective shielding by increased or modified qualitative epidermal absorptance was required. Sustained increased UV-B radiation levels are more detrimental than short, high doses of UV-B radiation. The combination of low temperature and increased UV-B radiation was more significant in the level of UV-B radiation induced damage than UV-B radiation alone. Slow growing leaves were more affected by increased UV-B radiation than fast growing leaves.

1.2. Introduction

Ultraviolet (UV) radiation has been a natural environmental stress factor for organisms since the pre-Cambrian era (Cockell and Horneck, 2001; Lowry et al., 1980; Rettberg et al., 1998). Ultraviolet radiation induces injury to DNA, causes DNA mutations, inhibits photosynthetic processes, impairs membrane function, and can cause lethal cell damage (Britt, 1996; Rozema et al., 1999; Sancar and Sancar, 1988; Taylor et al., 1997; Weber, 2005). In addition to such direct UV-induced damage, DNA mutations may have been the catalysts for phylogenetic diversity through accelerated selection and evolution (Cockell, 2000; Sagan, 1973), and, as a result, be, at least partly, responsible for the success of terrestrial plant species (Lowry et al., 1980; Rozema et al., 1999; Stafford, 1991).

Current stratospheric ozone depletion and the potential associated UV-B radiation increase can significantly affect terrestrial plant species (Day and Neale, 2002; Searles et al., 2001), and these changes may be amplified across higher ecological scales and trophic levels (Caldwell et al., 1998a; van der Leun et al., 1998; Warren et al., 2002). Furthermore, stratospheric ozone depletion and global warming may be producing significant changes in both surface and stratospheric climate (Hartmann et al., 2000; United Nations Environment Programme, 2012). Thus, understanding how different levels of UV radiation environment of Earth affect terrestrial communities is important in predicting how the current stratospheric ozone depletion may affect life on Earth, and may interact with climate changes towards rapid global change. Also, it may provide insights in the UV radiation contribution as a selection agent throughout the evolutionary history of Earth.

Yet, experimental research on UV radiation effects on organisms has been mostly limited to individual and sub-individual plant levels. This is largely due to the technical difficulties in simulating an enhanced UV-B radiation regime at the scales required for higher ecological-level experiments (DeLucia et al., 2001). A modeling research approach, which integrates and scales the effects of enhanced UV-B radiation on terrestrial plant communities, was therefore used to understand plant response mechanisms to UV-B radiation and their broader consequences, identify the processes insufficiently addressed by past research, as well as to investigate hypotheses that were untestable by experimental research.

We modeled the pathway of UV-B radiation in leaf, its qualitative and quantitative attenuation in epidermis, its effects upon plant DNA, cellular responses to DNA injury, and

their potential consequences on leaf growth and development. Our primary hypothesis was that enhanced solar UV-B radiation-induced DNA damage significantly reduces leaf growth and development. Damage to DNA above ambient levels might delay cell division until the injury is repaired, or might delay cell expansion (de Lima-Bessa et al., 2008; Hectors et al., 2010; Lo et al., 2005; Srivastava, 2002). Delays in cell division and expansion during leaf expansion, and possible cell apoptosis might lead to modifications in leaf morphology, such as decreased leaf size, or even premature leaf senescence. These processes can significantly reduce the photosynthetic capacity of leaves, with consequences upon whole plant growth and development (Caldwell et al., 1998a; Milchunas et al., 2004; Rozema et al., 1997).

Although there is considerable research regarding the effects of UV-B radiation on concentration of flavonoids and related phenolics compounds, UV-B induced DNA injuries, and the effects of UV-B radiation on leaf morphology, our model integrated these processes and showed how changes in molecular and cellular processes can result in whole organ changes. We were able to examine a variety of questions that were difficult to approach through experimental research, including: (1) Are long, sustained increased UV-B radiation levels more detrimental than short, high doses of UV-B radiation? (2) Are fast growing leaves more adaptive than slow growing leaves? (3) Are different relative absorption spectra of flavonoids and related phenolics compounds responsible for the observed physiology of the leaf? (4) How important is DNA repair in leaf development? (5) There is an interaction between temperature and UV-B radiation induced effects?

1.3 Model framework

The model is comprised of four major components: UV-B radiation, leaf optical properties, DNA damage and repair mechanisms, and leaf cell division and expansion. The dose rate of UV-B radiation is a fundamental component of the model, since it influences the quantity of epidermal pigments, their absorption spectra, the quantity and quality of damaging radiation reaching the DNA.

Once incident on the leaf, the UV-B radiation pathway into the leaf is determined by the leaf optical properties (reflectance, absorptance, and transmittance). Most plant species exhibit low levels of UV-B leaf surface reflectance (5-6%), although some species can reflect up to 70% (Gausman et al., 1975; Robberecht and Caldwell, 1978; Robberecht et al., 1980).

Generally, 85 to 95% of the UV-B radiation is absorbed by the leaf, and the remaining UV-B radiation is transmitted (Bieza and Lois, 2001; Gausman et al., 1975; Robberecht and Caldwell, 1978; Robberecht et al., 1980). Pigments, primarily flavonoids, isoflavonoids, sinapate esters, flavons, and anthocyanins, are the most important leaf constituents that absorb UV-B radiation (Dixon and Paiva, 1995; Koes et al., 1994; Robberecht and Caldwell, 1978; Robberecht et al., 1980; Winkel-Shirley, 2002). Increases in UV-B radiation generally stimulate the production of secondary metabolites and results in changes in epidermal absorption (Dixon and Paiva, 1995; Koes et al., 1994; Li et al., 1993b; Schmelzer et al., 1988b; Winkel-Shirley, 2002). The relative changes in the quantity and quality of secondary metabolites vary with species (Chalker-Scott, 1999; Dixon and Paiva, 1995; Li et al., 1993b). Regardless of the compounds and amounts produced, their relative absorption spectra follow three general patterns (Day et al., 1994; Lavola et al., 1997; Qi et al., 2003; Schmelzer et al., 1988b; Sisson, 1981). Most evergreen species, deciduous trees, shrubs and vines show a maximum absorption at shorter wavelength (280 nm), and lower relative absorption at longer UV-B radiation wavelengths. Most grasses and herbaceous plants show minimum absorption at shorter wavelengths, and greater relative absorption at longer UV-B radiation wavelengths.

The major DNA lesions induced by UV-B radiation include pyrimidine cyclobutane dimers (CPD) and pyrimidine-pyrimidone (6-4) photoproducts (6-4PP) (Britt, 1996; Sancar and Sancar, 1988; Taylor et al., 1997; Weber, 2005). Low UV-B radiation doses induce CPD to 6-4PP ratio of approximately 9:1, while very high UV-B radiation doses result in 6:4 ratios (Sancar, 2003). Photoproducts are reversed through photorepair and nucleotide excision repair (NER) or dark repair. The CPD photolyase and 6-4PP photolyase bind to the DNA injury and reverse the damage using 350-450 nm light as energy source (Sancar, 2003; Weber, 2005). The 6-4PP photorepair is more efficient than CPD photorepair (Chen et al., 1994; Jiang et al., 1997). But, the CPD photolyase quantum yields are higher than those of 6-4 photolyase (Sancar, 2003). Nucleotide excision repair (NER) is an ATP-dependent, complex, repair pathway, involved in the removal of a variety of bulky DNA lesions including CPDs and 6-4PPs. NER repair of 6-4PPs is 9.5-10.7 faster than NER repair of CPDs (de Lima-Bessa et al., 2008; Lo et al., 2005; Sancar, 2003).

Induction and repair mechanisms rates are temperature dependent. Photoproducts induction rates at 0^{o}C are the lowest, increase with temperature, and stabilize or decline above

30^{o}C (Li et al., 2002; Takeuchi et al., 1996; Waterworth et al., 2002). The photoproducts repair rates are also temperature-dependent: negligible at 0^{o}C, increase with temperature, and remain steady or decline above 30^{o}C (Li et al., 2002; Takeuchi et al., 1996; Waterworth et al., 2002). But, the potential accumulation of photoproducts in plants growing in low-temperature environments as a result of low rate of UV-B radiation photoproducts induction and negligible repair rates might be mediated by a low-temperature stimulation of screening compounds production (Bilger et al., 2007).

Small unrepaired CPDs and 6-4PPs numbers arrest the cell cycle to allow effective repair, while major damage can induce apoptosis (Lo et al., 2005). Unrepaired 6-4PPs trigger apoptosis, whereas unrepaired CPDs rather induce cell cycle arrest (Lo et al., 2005) . In NER-deficient cells, both CPDs and 6-4PPs lead to apoptosis, while in NER-proficient cells, CPDs were solely responsible for apoptosis since 6-4PPs were rapidly repaired by NER (de Lima-Bessa et al., 2008). However, either DNA lesions, if unrepaired after 24 hours, lead to apoptosis at non-cumulative rates (de Lima-Bessa et al., 2008; Lo et al., 2005). Apoptosis triggering by UV-B radiation induced lesions is delayed minimum 8-16 hours, probably to allow time for damage removal (de Lima-Bessa et al., 2008; Lo et al., 2005). While these results were recorded for human cells, it is plausible that similar mechanisms may also regulate plant cells life cycles.

Enhanced UV-B radiation has been shown to induce smaller leaves in many plant species (González et al., 1998; Teramura et al., 1991), as a result of decreased leaf growth rates mainly during the day period(Hopkins et al., 2002). The leaf growth process is driven initially by active cell division, followed by cell expansion and differentiation, and leaf maturity (Beemster et al., 2005). Ultraviolet radiation may inhibit cell division (González et al., 1998; Rousseaux et al., 2004), cell expansion (Hectors et al., 2010; Wargent et al., 2009b), or both (Hofmann et al., 2003; Hopkins et al., 2002; Wargent et al., 2009b). While the connection between UV-B radiation, induction of DNA damage, and cell cycle arrest or apoptosis seems clear (Britt, 1996; de Lima-Bessa et al., 2008; Lo et al., 2005; Weber, 2005), the mechanisms of UV-B radiation induced reduced cell expansion rates are less understood (Hectors et al., 2010). While DNA is a key receptor of UV-B radiation, several plant stress signaling components (e.g., NADPH oxidase-derived reactive oxygen species, jasmonic acid, nitric oxide, mitogen-activated protein kinases) may be affected by enhanced UV-B radiation,

with possible inhibitory effects on leaf expansion (Ballare et al., 2011; Wargent et al., 2009a). Ultraviolet-B specific signaling proteins (e.g., as UVR8) have been shown to regulate gene activity responsible for secondary metabolites production and photorepair of DNA lesions (Ballare et al., 2011; Brown et al., 2005). For example, in *Arabidopsis thaliana* UVR8 is involved in leaf growth and photomorphogenesis by controlling leaf cell expansion, but it has no effect on cell division (Wargent et al., 2009a). Primary literature presenting these effects were discussed in previous papers (Ballare et al., 2011; Wargent et al., 2009a; Wargent et al., 2009b).

Our research modeled these processes for a hypothetical generalized leaf (a simple, planophyllic, glabrous, green plant leaf) and integrated the effects of UV-B radiation on DNA and the consequences on the leaf expansion over one growing season. This generalized leaf allowed us to model the influence of UV-B radiation under a variety of scenarios, including variations in leaf characteristics, UV-B irradiance, and repair mechanisms.

1.4 Model architecture

Our model simulated the leaf optical properties (reflectance, absorptance, and transmittance) under various levels of UV-B irradiation, the absorptance of epidermal secondary metabolites, the UV-B radiation targeting of signaling proteins and their photomorphogenic effects on cell expansion, the UV-B radiation induction of DNA injuries, their repair through UVA/PAR or ATP catalyzed repair mechanisms, their consequences on leaf cell cycle, and leaf expansion (Figure 1.1). A complete presentation of the mathematical model and parameters estimation is presented in Appendix A.

The model was created in Vensim modeling software (Systems, 2009). Data compilation, preparation, and analysis were done in various programs such as Microsoft Access, Excel, and R-language (Team, 2010).

The models were verified for consistency and units, for correctness of the mathematics and for accuracy of the conceptual logic (Rykiel, 1996), calibrated and validated (Gardner and Urban, 2003; Rykiel, 1996; Shugart, 1984). Prior to this, sensitivity analysis procedure were performed (Aber et al., 2003; Plentinger and Penning de Vries, 1996; Rykiel, 1996).

1.5. Model analysis

1.5.1 Sensitivity analysis

The ranges derived for the major model parameters were used for the allowable limits used in the model sensitivity analysis and calibration. The following parameters were tested: leaf optical properties (k_R, and k_T), CPD induction ($k_{A,DNA,CPD}$ and k_c), CPD/6-4PP photorepair and dark repair (a and r_{max}), CPD/6-4PP levels over which cell division is delayed (k_{cdd}), and duration of the cell division delay (t_{cdd}). The relative maximum number of CPDs and 6-4 PPs during the leaf growing period, and the relative mature leaf area were measured across the tested model parameters (Figure 1.7).

Our results show that the number of CPDs during the leaf growing period is sensitive to the amount of UV-B radiation reaching the DNA, the rate of CPD induction, and CPD photorepair and dark repair rate multipliers. The number of 6-4PPs during the leaf growing period is sensitive to the amount of UV-B radiation reaching the DNA, the rate of 6-4PPs induction, and 6-4PP photorepair and dark repair rate multipliers. The CPD and 6-4 PP in DNA do not reach the levels corresponding to the maximum CPDs and 6-4 PPs photorepair and dark repair rates, for either UV-B radiation dose. Leaf area is sensible only to changes in CPDs levels. The 6-4 PPs do not reach any levels that can influence leaf growth and expansion. Also, it is sensible to the CPD level at which cell division is delayed.

DNA lesions induction rate is the most influential factor, and it is responsible for the highest variation in CPDs and 6-4 PPs numbers, and relative leaf area. Repair of CPDs is less influential on the model, while the model is resistant to changes in 6-4PPs concentrations.

1.5.2 Calibration and validation

Because the diversity of experiments used to infer the parameter values prevented a species-specific calibration and validation, the model was calibrated by trial-and-error adjustment of the most sensitive parameters. Data collected for rice species were used for the evaluation of the CPD induction and repair rates (Hidema et al., 2001; Hidema et al., 2007; Iwamatsu et al., 2008; Kang et al., 1998; Quaite et al., 1994). Field-grown rice showed concentrations of 3 to 6 CPDs Mb^{-1} during the day, if grown under ambient UV-B conditions and higher values under UV-B supplementation (Hidema et al., 2001; Hidema et al., 2000). Moreover, rice cultivars seem to show decreases of about 50% decrease in dry weight under

increased UV-B radiation exposure (Hidema et al., 2001; Iwamatsu et al., 2008), depending on the effectiveness of their CPD repair mechanisms. For the calibration purposes, we considered these values to be equivalent to roughly 50% decrease in leaf area.

As validation, we considered that the model should show the general trends observed in previous experiments. First, leaves of many tree species do not have significantly smaller leaf size at higher UV-B radiation doses. Second, different combinations of repair rates should result in decreases about 20 to 90% dry weight (or approximate 20 to 90% leaf area) when plants are exposed to no UV-B and to 3.6 KJ m^{-2} h^{-1} (Iwamatsu et al., 2008).

The parameter estimates following the model calibration are presented in Table 1.1. For the CPD/6-4PP levels over which cell division is delayed, we choose, instead of a singular value, a range of 5 to 14 CPD/6-4PP. For CPD/6-4PP values smaller or equal than 5 CPD/6-4PP the cell division is not delayed, for values above 14 CPD/6-4PP, cell division is 100 percent delayed, and for values between 5 and 14 CPD/6-4PP cell division is proportionally delayed. The value of the supplemental UV-B radiation absorbed by epidermal pigments, when exposed to increased UV-B radiation ($k^*_{A,SM}$) was more difficult to estimate. The range of the values was inferred from a range of experimental designs (Bornman et al., 1997; Day and Demchik, 1996; de la Rosa et al., 2001; Kolb et al., 2001; Li et al., 1993b; Liu et al., 1995; Meijkamp et al., 1999; Olsson et al., 1998; Sheahan, 1996; Tegelberg et al., 2003; Tevini et al., 1981; Tevini et al., 1982, 1983; Vandestaaij et al., 1995). Epidermal pigment content was compared between plants grown with no UV-B radiation exposure, and under various UV-B radiation doses. Moreover, solar UV-B radiation might have a greater influence on the epidermal pigments content than the increased UV-B radiation (Ryan et al., 1998; Ryan et al., 2002). Since the range is too wide, study conditions were too diverse, and extrapolation of rates from one range of UV-B radiation doses to a different one is problematic, we considered for this model that the epidermal UV-B absorptance is constant (0.94) for any level of UV-B irradiance. We recognize that this value might lead to imprecise model predictions especially at increased UV-B radiation levels. Rather than addressing a particular species, our model examined the patterns common in most species.

The parameter values resulting in the best fit for the models are presented in Table 1.1. Supplemental model calibration, optimization and testing can be readily done as more comprehensive experimental data becomes available.

1.6 Results

In addition to model analysis simulations, the following scenarios were considered: increased UV-B radiation in combination with different epidermal absorption spectra and CPD repair rates; increased UV-B radiation dose concentrated spread over the leaf expansion period or concentrated in a one, two or three days; leaves growing indifferent periods of the growing season under increased UV-B radiation; leaves growing under three temperature regimes under increased UV-B radiation; slow, medium , and fast growing leaves under increased UV-B radiation regime.

The sensitivity analysis showed that the number of 6-4 PP induced by UV-B radiation (at either ambient or increased levels), are never high enough to interfere with the leaf growth and development. Also, photorepair of the DNA lesions is never saturated and the differences between the UV-B resistant and sensitive species seem to be in the rate of repair. Increased UV-B radiation does not induce sustained levels of DNA lesions to actually trigger apoptosis in leaf cells. The model was very sensitive to the number of CPD that actually induces cell division delays. In our model we simulated a range that satisfied the calibration and validation requirements. DNA lesions induction rate was the most influential factor, and it was responsible for the highest variation in CPDs and 6-4 PPs numbers, and relative leaf area. Repair of CPDs were less influential on the model, while the model was resistant to changes in 6-4PPs concentrations (Figure 1.2).

Combinations of UV-B radiation doses, epidermal absorptance spectra, and CPD repair rates simulations indicate that plants with relative high epidermal absorptance at short UV-B radiation wavelengths were mostly unaffected by UV-B radiation increases (Figure 1.3). Only plants with deficient photorepair rates exhibited relative leaf area losses at increased UV-B radiation. Plants with relatively high epidermal absorptance at long UV-B radiation wavelengths were the most sensible to increases in UV-B radiation (Figure 1.3), while plants with equal epidermal absorptance across wavelengths exhibited intermediary patterns (Figure 1.3).

When we compared the effect of sustained UV-B radiation increases with short-term increased UV-B bursts, we found that sustained increased UV-B radiation had a higher effect on the final leaf area (Figure 1.4). The one day single dose was the only one that induced a large enough number of CPDs to trigger apoptosis. Also leaves growing in mid-summer were

more affected by increased UV-B radiation than leaves growing in the beginning of the growing season (Figure 1.5).

Low temperature had an effect on leaf growth, especially when plants were exposed to increased UV-B radiation. Leaves grown at ambient and high temperatures reached similar relative leaf areas (Figure 1.6). Also, slow growing leaves exhibited the lowest relative leaf area when exposed to increased UV-B radiation (Figure 1.7).

1.7 Discussion

Our model simulations showed that UV-B radiation does not induce enough 6-4PPs to interfere directly with the leaf growth and development. This is due to a lower 6-4PP induction rate (Sancar, 2003) than for CPD, but also higher photorepair and dark repair (de Lima-Bessa et al., 2008; Lo et al., 2005; Sancar, 2003). Since 6-4PPs are more readily to trigger apoptosis than CPDs (Lo et al., 2005), a significant finding was that that 6-4PPs might not interfere directly with the leaf growth and development, although they may influence mutagenesis and premature cellular aging (Britt, 1996).

The amount of CPD in DNA appeared to be a significant factor for the leaf growth. The number of CPDs is controlled by the quality and quantity of UV-B radiation reaching the DNA (thus, by the absorptance properties on the epidermal secondary metabolites) and by the CPD photorepair and dark repair rates. Regardless of these rates, the model showed that the repair processes do not reach saturation, and given enough time could repair any amount of damage. While the sustained increased UV-B radiation may be successfully mediated, depending on the epidermal absorptance properties and the rates of repair, occasional extremely high UV-B radiation bursts can be mediated successfully by the repair mechanisms, regardless of their rates.

Moreover, the model showed that increased UV-B radiation did not result in immediate apoptosis of the leaf cells. This simulation does not imply that increased UV-B radiation is instantly lethal to the leaf, but that DNA repair processes were well equipped to handle a severe radiation stress. If the leaf cells are instantaneously apoptotic when exposed to severely high UV-B radiation doses, the mechanisms that induce their death do not seem to be related directly to amount of DNA lesions induced.

Model simulations of increased UV-B radiation in combination with different combinations of the qualitative epidermal absorptance and repair rates (Figure 1.3) explained why many tree species show little or no significant decreases in leaf size when grown with increased UV-B radiation (Figure 1.3.2). Only when coupled with low rates of photorepair, the effects of increased UV-B radiation were highly significant. However, species that exhibit relatively higher absorptance at the long UV-B radiation wavelengths (e.g., most grasses), seem to be more susceptible to UV-B radiation induced leaf reduction (Figure 1.3. 5 and 7). This confirms previous experimental results that show monocots exhibiting higher sensitivity to increased UV-B radiation than dicots (Barnes et al., 1990). Since we simulated equal quantitative epidermal absorptance for all scenarios (Figure 1.3), the modeled results suggested different plant strategies in dealing with increased UV-B radiation. For example, trees, with leaves present over the entire growing season, seem to have developed UV-B radiation resistance by qualitative changes in epidermal absorptance (i.e., reducing the effective UV-B radiation reaching the DNA). However, grasses seem more susceptible to increased UV-B radiation. Therefore, grass species may cope with increased UV-B radiation by increasing the epidermal pigments concentration, or by avoidance of elevated UV-B radiation seasons.

When we examined the effect of sustained increased UV-B radiation versus similar single doses (Figure 1.4), we observed that, at least from the DNA damage – repair perspective, sustained increased UV-B radiation doses were more detrimental to the leaf area than single extreme doses. A 30-fold UV-B radiation dose for a single day caused sufficient DNA lesions to induce partial leaf cell apoptosis, though the leaf seem to recover shortly. The same dose spread for two and three days had little to no effect. Again, the model does not account for other cellular damage that might trigger instantaneous apoptosis.

When we simulated leaves growing at different months of the growing season (Figure 1.5), we observed that ambient UV-B radiation did not have an effect on the final leaf area. It confirms that the timing of leaf growth is controlled by other mechanisms rather than UV-B radiation. But under elevated UV-B radiation, leaves growing in the beginning of the growing season have the least damage.

Simulations on the effect of temperature (Figure 1.6) showed that the plants were highly vulnerable to the combination of low temperature and increased UV-B radiation. Our

model results agree with previous results (Li et al., 1993b; Takeuchi et al., 1996; Waterworth et al., 2002). However, it is possible that supplemental epidermal pigments induced by low temperature environment (Bilger et al., 2007) can successfully complement the diminished repair capacity of cold climate plants.

The duration of the leaf growth appeared to be a factor in the final leaf size (Figure 1.7). Increased UV-B radiation has to have the least effect on fast growing plants, and the highest effect on slow growing plants. Therefore, our model predicts that the total UV-B radiation dose during the growing time is the most important factor in the final leaf area.

Improved model predictability can be achieved if some of the model parameters would be estimated for specific species. We recognize that some of the parameters were estimated from maybe dated research, research considering some unrealistic conditions, research performed on a limited number of plant species, or many times not duplicated. Acknowledging that these estimates might hinder the predictive power of the model, they were considered acceptable (for the purpose of the model), at least until better alternatives are available. Also the non-inclusion of the enhanced UV-B radiation photomorphogenic effects on plant growth and development may have affected the predictive power of the model. Improvements in the model can only be considered when the quantitative relationship between UV-B radiation dose and photomorphogenic responses is better understood. The inclusion of such responses in the model, together with species-specific quantification of UV-B radiation dependent epidermal absorptance, will allow us to separate and rank the relative importance of those mechanisms in the plant responses to increased UV-B radiation. While we reserve the right to re-visit the model in the future, we believe that it is essential to present the model at this stage, despite its shortcomings. First, while the magnitude of effects of UV-B radiation proposed by the results of the model might not be precise, we believe that the direction of the effects and their causes are essentially correct. Second, the model shows the strengths and weaknesses of our understanding of the effects of UV-B radiation in plants.

To name of few of those: first, although the epidermal UV-B radiation absorptance is a very important factor in the dynamics of the model, the range of values inferred from the literature was too wide and extrapolation of rates from one range of UV-B doses to a different one is problematic. Second, the conversion factor of the UV-B radiation reaching the DNA in the number of CPDs induced in DNA was estimated through the model calibration processes,

from a wide range produced by the literature. Third, while the literature presents a wide array of studies of the photorepair and dark CPD repair rates, most of those studies refer only to rice species, and did not offer enough information to detail the Michaelis-Menten enzyme-driven repair models parameters. These parameters are critical in estimating the dynamics of induction and repair of DNA photoproducts, and determinant to the associated cell division and leaf expansion processes. Finally, the quantification of the relationship between UV-B radiation dose and photomorphogenic responses is essential for a complete and predictive model.

1.8. Conclusions

This is the first (but probably not the last) mathematical model to integrate the effects of increased UV-B radiation through leaf epidermis, DNA, and leaf growth and development. We intend to re-visit the model as more data becomes available. Enhanced UV-B radiation-induced DNA damage significantly delayed cell division until the injury is repaired, resulting in significant reductions in leaf growth and development. A review of the relevant literature showed a wide range of values for the key parameters. Moreover, certain parameter values were inferred only from the calibration process. However, our model allowed the testing of a variety of questions that were difficult to approach through experimental research. Moreover the model predicts that the total UV-B radiation dose reaching the DNA during the growing time may be an important factor in the final leaf area.

1.9. Acknowledgments

We acknowledge the insightful comments of Stephan Flint, Brian Dennis, Dennis Ferguson, and Steve Bunting. This research was funded in part by the University of Idaho Student Grant Program No. UI07358.

1.10 References

Aber, J.D., Bernhardt, E.S., Dijkstra, F.A., Gardner, R.H., Macneale, K.H., Parton, W.J., S.T.A., P., Urban, D.L., Weathers, K.C., 2003. Standards of practice for review and publication of models: summary of discussion, in: Canham, C.D., Cole, J.J., Lauenroth, W.K. (eds.), Models in Ecosystem Science. Princeton University Press, Princeton, NJ, pp. 204-210.

Ballare, C.L., Caldwell, M.M., Flint, S.D., Robinson, S.A., Bornman, J.F., 2011. Effects of solar ultraviolet radiation on terrestrial ecosystems. Patterns, mechanisms, and interactions with climate change. Photochem Photobiol Sci 10, 226-241.

Barnes, P.W., Flint, S.D., Caldwell, M.M., 1990. Morphological Responses of Crop and Weed Species of Different Growth Forms to Ultraviolet-B Radiation. Am J Bot 77, 1354-1360.

Beemster, G.T., De Veylder, L., Vercruysse, S., West, G., Rombaut, D., Van Hummelen, P., Galichet, A., Gruissem, W., Inze, D., Vuylsteke, M., 2005. Genome-wide analysis of gene expression profiles associated with cell cycle transitions in growing organs of Arabidopsis. Plant Physiol 138, 734-743.

Bieza, K., Lois, R., 2001. An Arabidopsis Mutant Tolerant to Lethal Ultraviolet-B Levels Shows Constitutively Elevated Accumulation of Flavonoids and Other Phenolics. Plant Physiol 126, 1105-1115.

Bilger, W., Rolland, M., Nybakken, L., 2007. UV screening in higher plants induced by low temperature in the absence of UV-B radiation. Photochemical & Photobiological Sciences 6, 190-195.

Bornman, J.F., Reuber, S., Cen, Y.-O., Weissenbock, G., 1997. Ultraviolet radiation as a stress factor and the role of protective pigments, in: Lumsden, P.J. (ed.), Plants and UV-B:

responses to environmental change. Cambridge University Press, Cambridge, UK, pp. 157-168.

Britt, A.B., 1996. DNA damage and repair in plants. Annual Review of Plant Molecular Biology 47, 75-100.

Brown, B.A., Cloix, C., Jiang, G.H., Kaiserli, E., Herzyk, P., Kliebenstein, D.J., Jenkins, G.I., 2005. A UV-B-specific signaling component orchestrates plant UV protection. P Natl Acad Sci USA 102, 18225-18230.

Caldwell, M.M., Bjorn, L.O., Bornman, C.H., Flint, S.D., Kulandaivelu, G., Teramura, A.H., Tevini, M., 1998. Effects of increased solar ultraviolet radiation on terrestrial ecosystems. Journal of Photochemistry and Photobiology B: Biology 46, 40-52.

Chalker-Scott, L., 1999. Environmental Significance of Anthocyanins in Plant Stress Responses. Photochem Photobiol 70, 1-9.

Chen, J.J., Mitchell, D.L., Britt, A.B., 1994. Light-Dependent Pathway for the Elimination of Uv-Induced Pyrimidine-(6-4) Pyrimidinone Photoproducts in *Arabidopsis*. Plant Cell 6, 1311-1317.

Cockell, C.S., 2000. The ultraviolet history of the terrestrial planets - implications for biological evolution. Planetary and Space Science 48, 203-214.

Cockell, C.S., Horneck, G., 2001. The history of the UV radiation climate of the Earth - theoretical and space-based observations. Photochem Photobiol 73, 447-451.

Day, T.A., Demchik, S.M., 1996. Influence of enhanced UV-B radiation on biomass allocation and pigment concentrations in leaves and reproductive structures of greenhouse-grown *Brassica rapa*. Vegetatio 127, 109-116.

Day, T.A., Howells, B.W., Rice, W.J., 1994. Ultraviolet absorption and epidermal-transmittance spectra in foliage. Physiol Plantarum 92, 207-218.

Day, T.A., Neale, P.J., 2002. Effects of UV-B Radiation on Terrestrial and Aquatic Primary Producers. Annu Rev Ecol Syst 33, 371-396.

de la Rosa, T.M., Julkunen-Tiitto, R., Lehto, T., Aphalo, P.J., 2001. Secondary metabolites and nutrient concentrations in silver birch seedlings under five levels of daily UV-B exposure and two relative nutrient addition rates. New Phytol 150, 121-131.

de Lima-Bessa, K.M., Armelini, M.G., Chigancas, V., Jacysyn, J.F., Amarante-Mendes, G.P., Sarasin, A., Menck, C.F., 2008. CPDs and 6-4PPs play different roles in UV-induced cell death in normal and NER-deficient human cells. DNA Repair (Amst) 7, 303-312.

DeLucia, E.H., Coleman, J.S., Dawson, T.E., Jackson, R.B., 2001. Plant physiological ecology: linking the organism to scales above and below - Ecological Society of America Meeting Snowbird, UT, USA, August 2000. New Phytol 149, 12-16.

Dixon, R.A., Paiva, N.L., 1995. Stress-Induced Phenylpropanoid Metabolism. Plant Cell 7, 1085-1097.

Gardner, R.H., Urban, D.L., 2003. Model validation and testing: past lessons, present concerns, future prospects, in: Canham, C.D., Cole, J.J., Lauenroth, W.K. (eds.), Models in Ecosystem Science. Princeton University Press, Princeton, NJ, pp. 184-203.

Gausman, H.W., Rodriguez, R.R., Escobar, D.E., 1975. Ultraviolet Radiation Reflectance, Transmittance, and Absorptance by Plant Leaf Epidermises1. Agron. J. 67, 720-724.

González, R., Mepsted, R., Wellburn, A.R., Paul, N.D., 1998. Non-photosynthetic mechanisms of growth reduction in pea (*Pisum sativum* L.) exposed to UV-B radiation. Plant, Cell & Environment 21, 23-32.

Hartmann, D.L., Wallace, J.M., Limpasuvan, V., Thompson, D.W.J., Holton, J.R., 2000. Can ozone depletion and global warming interact to produce rapid climate change? Proceedings of the National Academy of Sciences 97, 1412-1417.

Hectors, K., Jacques, E., Prinsen, E., Guisez, Y., Verbelen, J.P., Jansen, M.A., Vissenberg, K., 2010. UV radiation reduces epidermal cell expansion in leaves of *Arabidopsis thaliana*. J Exp Bot 61, 4339-4349.

Hidema, J., I.-K., S., Sato, T., Kumagai, T., 2001. Relationship between ultraviolet-B sensitivity and cyclobutane pyrimidine dimer photorepair in rice. J Radiat Res 42, 295-303.

Hidema, J., Kumagai, T., Sutherland, B.M., 2000. UV radiation-sensitive Norin 1 rice contains defective cyclobutane pyrimidine dimer photolyase. Plant Cell 12, 1569-1578.

Hidema, J., Taguchi, T., Ono, T., Teranishi, M., Yamamoto, K., Kumagai, T., 2007. Increase in CPD photolyase activity functions effectively to prevent growth inhibition caused by UVB radiation. Plant J 50, 70-79.

Hofmann, R.W., Campbell, B.D., Bloor, S.J., Swinny, E.E., Markham, K.R., Ryan, K.G., Fountain, D.W., 2003. Responses to UV-B radiation in *Trifolium repens* L. - physiological links to plant productivity and water availability. Plant Cell Environ 26, 603-612.

Hopkins, L., Bond, M.A., Tobin, A.K., 2002. Ultraviolet-B radiation reduces the rates of cell division and elongation in the primary leaf of wheat (*Triticum aestivum* L. cv Maris Huntsman). Plant, Cell and Environment 25, 617-624.

Iwamatsu, Y., Aoki, C., Takahashi, M., Teranishi, M., Ding, Y., Sun, C., Kumagai, T., Hidema, J., 2008. UVB sensitivity and cyclobutane pyrimidine dimer (CPD) photolyase genotypes in cultivated and wild rice species. Photochem Photobiol Sci 7, 311-320.

Jiang, C.-Z., Yee, J., Mitchell, D.L., Britt, A.B., 1997. Photorepair mutants of Arabidopsis. Proceedings of the National Academy of Sciences 94, 7441-7445.

Kang, H.S., Hidema, J., Kumagai, T., 1998. Effects of light environment during culture on UV-induced cyclobutyl pyrimidine dimers and their photorepair in rice (*Oryza sativa* L.). Photochem Photobiol 68, 71-77.

Koes, R.E., Quattrocchio, F., Mol, J.N.M., 1994. The Flavonoid Biosynthetic-Pathway in Plants - Function and Evolution. Bioessays 16, 123-132.

Kolb, C.A., Kaser, M.A., Kopecky, J., Zotz, G., Riederer, M., Pfundel, E.E., 2001. Effects of natural intensities of visible and ultraviolet radiation on epidermal ultraviolet screening and photosynthesis in grape leaves. Plant Physiol 127, 863-875.

Lavola, A.N.U., Julkunen-Tiitto, R., Aphalo, P., De La Rosa, T., Lehto, T., 1997. The effect of u.v.-B radiation on u.v.-absorbing secondary metabolites in birch seedlings grown under simulated forest soil conditions. New Phytol 137, 617-621.

Li, J., Ou-Lee, T.M., Raba, R., Amundson, R.G., Last, R.L., 1993. Arabidopsis Flavonoid Mutants Are Hypersensitive to UV-B Irradiation. The Plant Cell Online 5, 171-179.

Li, S.S., Paulsson, M., Bjorn, L.O., 2002. Temperature-dependent formation and photorepair of DNA damage induced by UV-B radiation in suspension-cultured tobacco cells. J Photoch Photobio B 66, 67-72.

Liu, L., Gitz, D.C., Mcclure, J.W., 1995. Effects of Uv-B on Flavonoids, Ferulic Acid, Growth and Photosynthesis in Barley Primary Leaves. Physiol Plantarum 93, 725-733.

Lo, H.L., Nakajima, S., Ma, L., Walter, B., Yasui, A., Ethell, D.W., Owen, L.B., 2005. Differential biologic effects of CPD and 6-4PP UV-induced DNA damage on the induction of apoptosis and cell-cycle arrest. BMC Cancer 5, 135.

Lowry, B., Lee, D., Hebant, C., 1980. The origins of land plants: a new look at an old problem. Taxon 29, 183-197.

Meijkamp, B., Aerts, R., van der Staaij, J., Tosserams, M., Ernst, W., Rozema, J., 1999. Effects of UV-B on secondary metabolites on plants, in: Rozema, J. (ed.), Stratospheric Ozone Depletion: The Effects of Enhanced Uv-B Radiation on Terrestrial Ecosystems. Backhuys Publishers, Leiden, The Netherlands, pp. 71-100.

Milchunas, D.G., King, J.Y., Mosier, A.R., Moore, J.C., Morgan, J.A., Quirk, M.H., Slusser, J.R., 2004. UV radiation effects on plant growth and forage quality in a shortgrass steppe Ecosystem. Photochem Photobiol 79, 404-410.

Olsson, L.C., Veit, M., Weissenbock, G., Bornman, J.F., 1998. Differential flavonoid response to enhanced UV-B radiation in Brassica napus. Phytochemistry 49, 1021-1028.

Plentinger, M.C., Penning de Vries, F.W.T. (eds.) 1996. CAMASE register of agro-ecosystems models, http://library.wur.nl/way/bestanden/clc/1763788.pdf electronic ed 420 pp.

Qi, Y., Bai, S., Heisler, G.M., 2003. Changes in ultraviolet-B and visible optical properties and absorbing pigment concentrations in pecan leaves during a growing season. Agr Forest Meteorol 120, 229-240.

Quaite, F.E., Takayanagi, S., Ruffini, J., Sutherland, J.C., Sutherland, B.M., 1994. DNA damage levels determine cyclobutil pyrimidine dimer repair mechanisms in alfalfa seedlings. The Plant Cell 6, 1635-1641.

Rettberg, P., Horneck, G., Strauch, W., Facius, R., Seckmeyer, G., 1998. Simulation of planetary UV radiation climate on the example of the early Earth. Advances in Space Research 22, 335-339.

Robberecht, R., Caldwell, M.M., 1978. Leaf Epidermal Transmittance of Ultraviolet-Radiation and Its Implications for Plant Sensitivity to Ultraviolet-Radiation Induced Injury. Oecologia 32, 277-287.

Robberecht, R., Caldwell, M.M., Billings, W.D., 1980. Leaf ultraviolet optical properties along a latitudinal gradient in the arctic-alpine life zone. Ecology 61, 612-619.

Rousseaux, M.C., Flint, S.D., Searles, P.S., Caldwell, M.M., 2004. Plant responses to current solar ultraviolet-B radiation and to supplemented solar ultraviolet-B radiation simulating ozone depletion: an experimental comparison. Photochem Photobiol 80, 224-230.

Rozema, J., Van De Staaij, J., Bjorn, L.O., De Bakker, N., 1999. Depletion of stratospheric ozone and solar UV-B radiation: evolution of land plants, UV-screens and function of polyphenolics, in: Rozema, J. (ed.), Stratospheric ozone depletion: the effects of enhanced UV-B radiation on terrestrial ecosystems. Backhyn Publishers, Leiden, The Netherlands.

Rozema, J., van der Staaij, J.W.M., Tosserams, M., 1997. Effects of UV-B radiation on plants from agro- and natural ecosystems, in: Lumsden, P.J. (ed.), Plants and UV-B: responses to environmental change. Cambridge University Press, Cambridge, UK, pp. 213-232.

Ryan, K.G., Markham, K.R., Bloor, S.J., Bradley, J.M., Mitchell, K.A., Jordan, B.R., 1998. UVB radiation induced increase in quercetin: Kaempferol ratio in wild-type and transgenic lines of Petunia. Photochem Photobiol 68, 323-330.

Ryan, K.G., Swinny, E.E., Markham, K.R., Winefield, C., 2002. Flavonoid gene expression and UV photoprotection in transgenic and mutant Petunia leaves. Phytochemistry 59, 23-32.

Rykiel, J.E.J., 1996. Testing ecological models: the meaning of validation. Ecol Model 90, 229.

Sagan, C., 1973. Ultraviolet Selection Pressure on Earliest Organisms. J Theor Biol 39, 195-200.

Sancar, A., 2003. Structure and Function of DNA Photolyase and Cryptochrome Blue-Light Photoreceptors. Chemical Reviews 103, 2203-2238.

Sancar, A., Sancar, G.B., 1988. DNA-Repair Enzymes. Annu Rev Biochem 57, 29-67.

Schmelzer, E., Jahnen, W., Hahlbrock, K., 1988. In situ localization of light-induced chalcone synthase mRNA, chalcone synthase, and flavonoid end products in epidermal cells of parsley leaves. Proceedings of the National Academy of Sciences 85, 2989-2993.

Searles, P.S., Flint, S.D., Caldwell, M.M., 2001. A meta analysis of plant field studies simulating stratospheric ozone depletion. Oecologia 127, 1-10.

Sheahan, J.J., 1996. Sinapate esters provide greater UV-B attenuation than flavonoids in *Arabidopsis thaliana* (Brassicaceae). Am J Bot 83, 679-686.

Shugart, H.H., 1984. A Theory on Forest Dynamics. The Ecological Implications of Forest Succesion Models. Springer-Verlag, New York, NY 278 pp.

Sisson, W.B., 1981. Photosynthesis, Growth, and Ultraviolet Irradiance Absorbance of *Cucurbita pepo* L. Leaves Exposed to Ultraviolet-B Radiation (280-315 nm). Plant Physiol 67, 120-124.

Srivastava, L.M., 2002. Plant growth and development: hormones and environment. Academic Press, San Diego, CA.

Stafford, H.A., 1991. Flavonoid Evolution - an Enzymatic Approach. Plant Physiol 96, 680-685.

Ventana Systems, 2009. Vensim: Ventana Simulation Environment, 5.6 ed, http://www.vensim.com.

Takeuchi, Y., Murakami, M., Nakajima, S., Kondo, S., Nikaido, O., 1996. Induction and repair of damage to DNA in cucumber cotyledons irradiated with UV-B. Plant Cell Physiology 37, 181-187.

Taylor, R.M., Tobin, A.K., Bray, C.M., 1997. DNA damage and repair in plants, in: Lumsden, P.J. (ed.), Plants and UV-B Responses to Environmental Change. Cambridge University Press, Cambridge, UK, pp. 53-76.

Team, R.D.C., 2010. R: A language and environment for statistical computing. R Foundation for Statistical Computing, Vienna, Austria http://R-project.org.

Tegelberg, R., Veteli, T., Aphalo, P.J., Julkunen-Tiitto, N., 2003. Clonal differences in growth and phenolics of willows exposed to elevated ultraviolet-B radiation. Basic Appl Ecol 4, 219-228.

Teramura, A.H., Ziska, L.H., Sztein, A.E., 1991. Changes in growth and photosynthetic capacity of rice with increased UV-B radiation. Physiol Plantarum 83, 373-380.

Tevini, M., Iwanzik, W., Thoma, U., 1981. Some Effects of Enhanced Uv-B Irradiation on the Growth and Composition of Plants. Planta 153, 388-394.

Tevini, M., Thoma, U., Iwanzik, W., 1982. Effect of enhanced UV-B radiation on development and composition of plants, in: Bauer, H., Caldwell, M.M., Tevini, M., Worrest, R.C. (eds.), Biological Effects of UV-B Radiation: Workshop : Papers. Gesellschaft fur Strahlen- und Umweltforschung, Munich, Germany, pp. 71-82.

Tevini, M., Thoma, U., Iwanzik, W., 1983. Effects of Enhanced Uv-B Radiation on Germination, Seedling Growth, Leaf Anatomy and Pigments of Some Crop Plants. Z Pflanzenphysiol 109, 435-448.

United Nations Environment Programme, E.E.A.P., 2012. Environmental effects of ozone depletion and its interactions with climate change: progress report, 2011. Photochemical & Photobiological Sciences 11, 13-27.

Authro, 1998. Environmental Effects of Ozone Depletion: 1998 Update.

Vandestaaij, J.W.M., Ernst, W.H.O., Hakvoort, H.W.J., Rozema, J., 1995. Ultraviolet-B (280-320 Nm) Absorbing Pigments in the Leaves of *Silene Vulgaris* - Their Role in Uv-B Tolerance. J Plant Physiol 147, 75-80.

Wargent, J.J., Gegas, V.C., Jenkins, G.I., Doonan, J.H., Paul, N.D., 2009a. UVR8 in *Arabidopsis thaliana* regulates multiple aspects of cellular differentiation during leaf development in response to ultraviolet B radiation. New Phytol 183, 315-326.

Wargent, J.J., Moore, J.P., Roland Ennos, A., Paul, N.D., 2009b. Ultraviolet Radiation as a Limiting Factor in Leaf Expansion and Development. Photochem Photobiol 85, 279-286.

Warren, J.M., Bassman, J.H., Eigenbrode, S., 2002. Leaf chemical changes induced in *Populus trichocarpa* by enhanced UV-B radiation and concomitant effects on herbivory by *Chrysomela scripta* (Coleoptera: Chrysomidae). Tree Physiol 22, 1137-1146.

Waterworth, W.M., Jiang, O., West, C.E., Nikaido, M., Bray, C.M., 2002. Characterization of *Arabidopsis* photolyase enzymes and analysis of their role in protection from ultraviolet-B radiation. J Exp Bot 53, 1005-1015.

Weber, S., 2005. Light-driven enzymatic catalysis of DNA repair: a review of recent biophysical studies on photolyase. Bba-Bioenergetics 1707, 1-23.

Winkel-Shirley, B., 2002. Biosynthesis of flavonoids and effects of stress. Curr Opin Plant Biol 5, 218-223.

Table 1.1: Summary of the model parameters estimators

Parameter	Definition	Unit	Range	Assigned values*
Leaf optical properties				
1 k_R	total solar UV-B radiation incident on the leaf reflected multiplier	%	0.05-0.7	0.05
2 k_T	total solar UV-B radiation incident on the leaf transmitted multiplier	%	0.01-0.1	0.05
3 $k_{A,SM}$	UV-B radiation absorbed by pigments multiplier	%	0.94	0.94
4 $k^*_{A,SM}$	supplemental increased UV-B radiation absorbed by pigments multiplier	% kJ m^{-2} d^{-1}	-0.2-1	0.94
CPD/6-4PP induction				
5 $k_{A,DNA,CPD}$	UV-B radiation reaching the DNA - CPD frequency conversion factor	CPD Mb^{-1} kJ^{-1} m^2 h	5-74	15
6 $k_{A,DNA,6-4PP}$	UV-B radiation reaching the DNA – 6-4PP frequency conversion factor	6-4PP Mb^{-1} kJ^{-1} m^2 h	$0.11 - 0.67\ CPD$	
7 k_c	correction factor multiplier due to differences in epidermal absorption spectra	%	0.3-1.7	0.65-1.35
CPD photorepair[a]				
8 a	CPD photorepair rate multiplier	CPD Mb^{-1} h^{-1}	0.3-0.7	
9 r_{max}	maximum rate of CPD photorepair	CPD Mb^{-1} h^{-1}	70-150	
10 k_s	enzyme saturation point	CPD Mb^{-1}	300	
11 k_a	the level of DNA damage that causes instant cellular apoptosis	CPD Mb^{-1}	500	
CPD dark repair[a]				
12 a	CPD dark repair rate multiplier	CPD Mb^{-1} h^{-1}	0.1-0.3	
13 r_{max}	maximum rate of CPD dark repair	CPD Mb^{-1} h^{-1}	5-7.5	
6-4PP photorepair[a]				
14 a	6-4PP photorepair rate multiplier	6-4PP Mb^{-1} h^{-1}	0.5-0.9	
15 r_{max}	maximum rate of 6-4PP photorepair	6-4PP Mb^{-1} h^{-1}	70-150	
6-4PP dark repair[a]				
16 a	6-4PP dark repair rate multiplier	6-4PP Mb^{-1} h^{-1}	0.9	
17 r_{max}	maximum rate of 6-4PP dark repair	6-4PP Mb^{-1} h^{-1}	8-14	
Temperature dependence of CPD induction|6-4PP induction| CPD repair|6-4PP repair mechanisms				
18 $b_{°C,0}$	regression equation coefficient	%	0.58|0.57|0.12|0.12	
19 $b_{°C,1}$	regression equation coefficient	% $°C^{-1}$	0.023|0.021|0.066|0.044	
20 $b_{°C,2}$	regression equation coefficient	% $°C^{-2}$	-0.0004|-0.0002|-0.0014|-0.0006	
Leaf growth (fast/medium/slow)[b]				
21 $t_{b,g}$	time of the beginning of growth	h	anytime in growing season	
22 $t_{m,g}$	time of inflection	h	$(t_{e,g} - t_{b,g)/2}$	
23 $t_{e,g}$	time of cessation of growth	h	$t_{b,g}$+ 168|360|720	
Percent of apoptotic cells dependence on the quantity of DNA lesions				
24 $b_{a,1}$	regression equation coefficient	% CPD^{-1}	0.13	
25 $b_{a,2}$	regression equation coefficient	% $6\text{-}4PP^{-1}$	1.09	
26 k_{cdd}	CPD/6-4PP levels over which cell division is delayed	CPD/6-4PP	6-12	5-14
27 t_{cdd}	duration of the cell division delay	h	8-16	16

*where appropriate
[a]k_s and k_a have identical values for all repair processes
[b]leaf senescence coefficients were chosen to model identical trends as leaf growth processes, and timed for the ending of the growing season considered

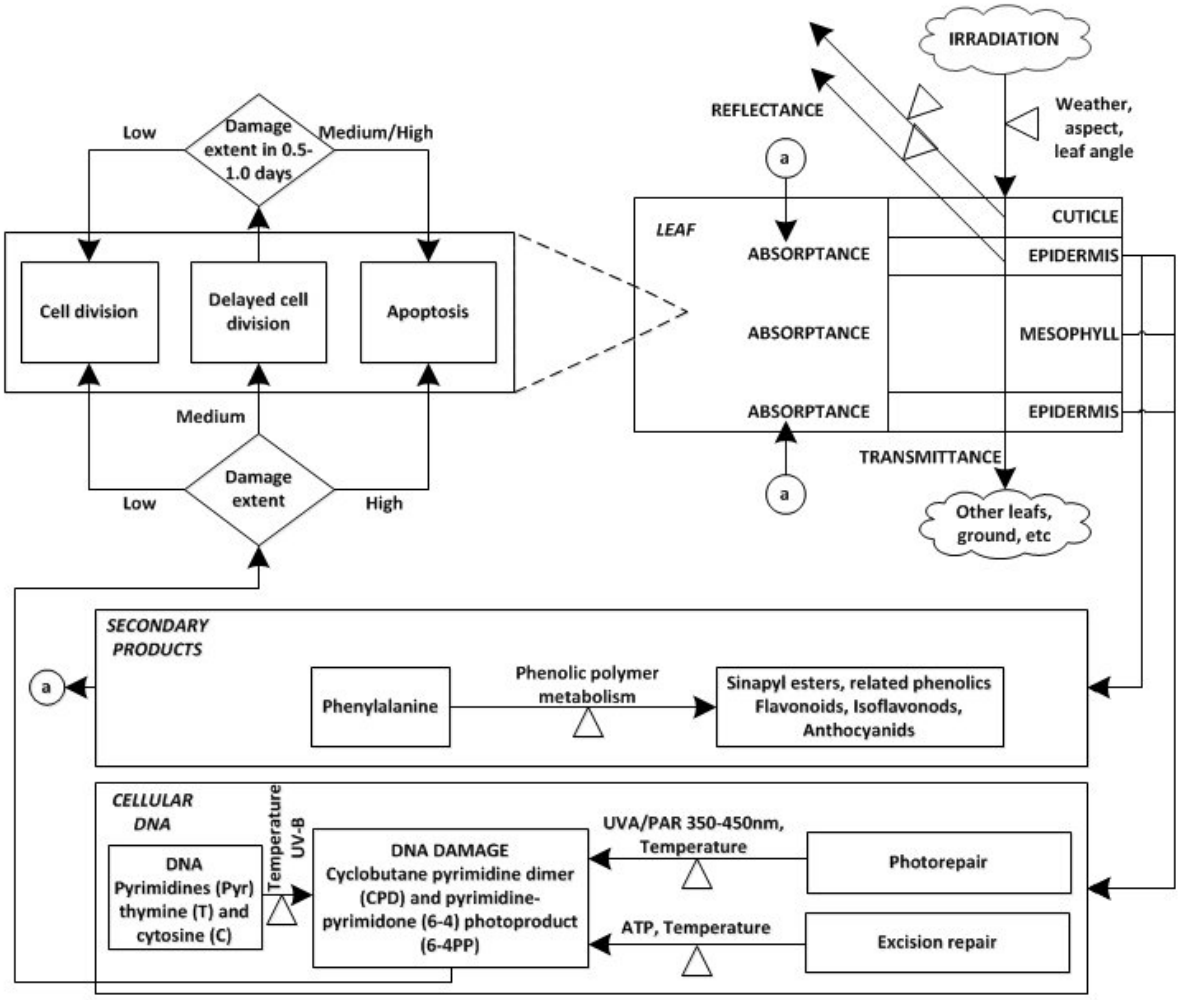

Figure 1.1: UV-B radiation is reflected, absorbed, or transmitted through the leaf. Most of the absorbed UV-B radiation is retained by epidermal secondary metabolites. Production of secondary metabolites is stimulated by increased UV-B radiation. Remaining absorbed UV-B radiation induces DNA injuries, which are repaired through UVA/PAR or ATP catalyzed repair mechanisms, or targets signaling proteins with photomorphogenic effects on cell expansion. Medium to high DNA injuries levels can arrest cell cycle or trigger apoptosis. Acronyms used in the figure: Pyrimidines (Pyr), Thymine (T), Cytosine (C), Cyclobutane pyrimidine dimmers (CPDs), Pyrimidine-pyrimidone (6-4) photoproducts (6-4PPs), Ultraviolet-A radiation (UVA), Photosynthetically Active Region (PAR), Adenosine Triphosphate (ATP) .

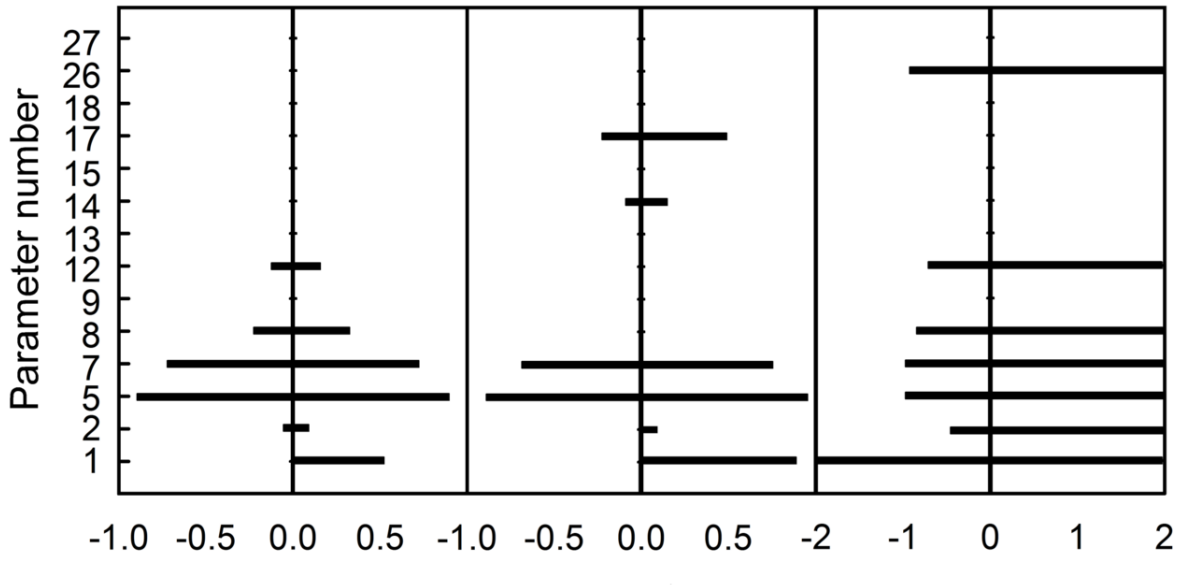

Figure 1.2: Sensitivity analysis: The relative maximum number of CPDs and 6-4 PPs during the leaf growing period, and the relative mature leaf area were measured across the leaf optical properties (k_R, and k_T), CPD induction ($k_{A,DNA,CPD}$ and k_c), CPD/6-4PP photorepair and dark repair (a and r_{max}), CPD/6-4PP levels over which cell division is delayed (k_{cdd}), and duration of the cell division delay (t_{cdd}) .

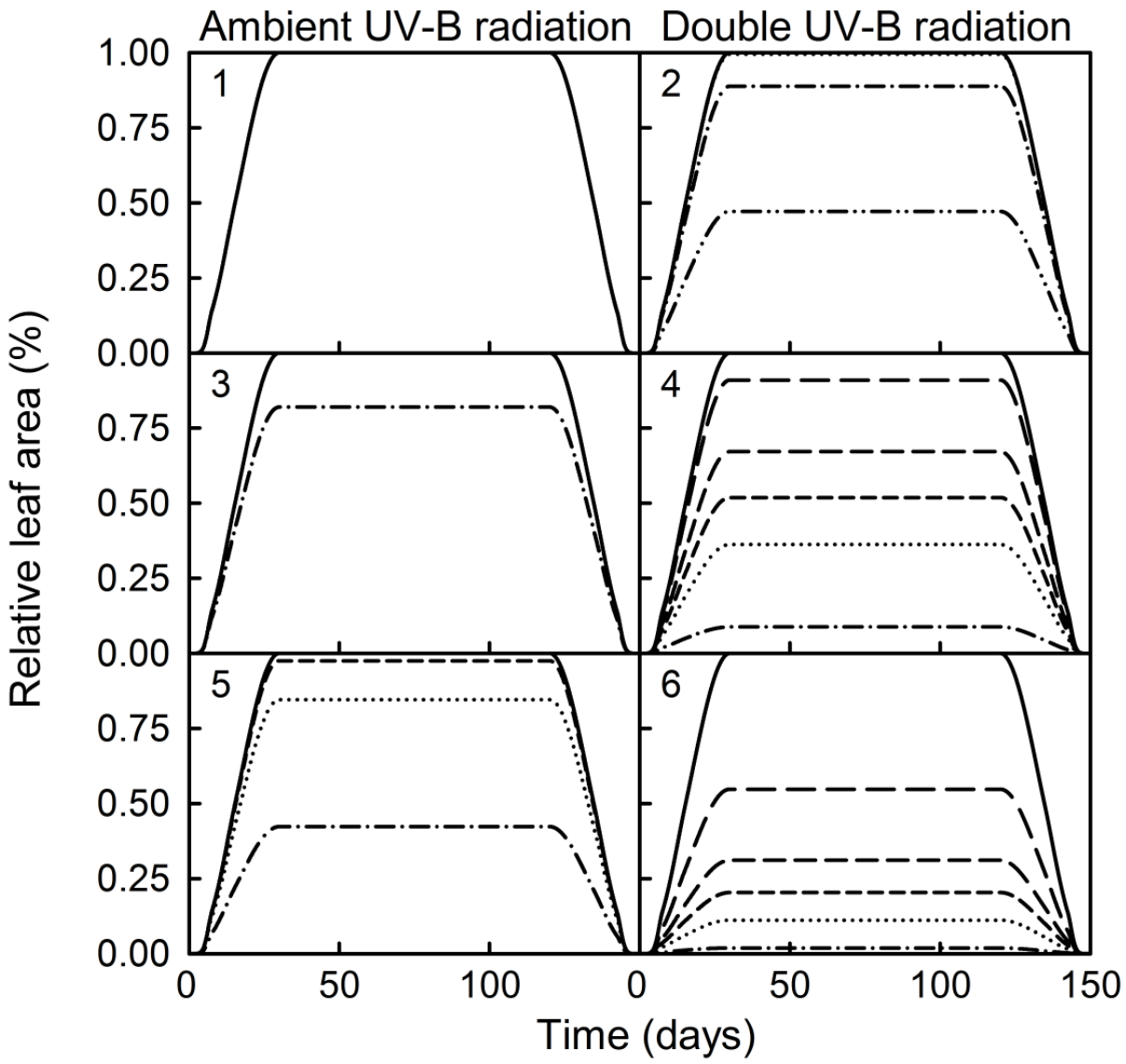

Figure 1.3: Effect of increased UV-B radiation (ambient and double the UV-B radiation), in combination with relative epidermal absorptance (relative high absorptance at low UV-B radiation wavelengths (1 and 2), equal absorptance at all UV-B radiation wavelengths (3 and 4), and relative high absorptance at low UV-B radiation wavelengths (5 and 6), and CPD repair rates combinations (no CPD inhibition of leaf growth (solid line), high photorepair and high dark repair rates (long dash), high photorepair rate – low dark repair rate (medium dash), average photorepair and dark repair rates (short dash), low photorepair rate – high dark repair rate(dotted line), and low photorepair and dark repair rates (dash-dotted line) on relative leaf area.

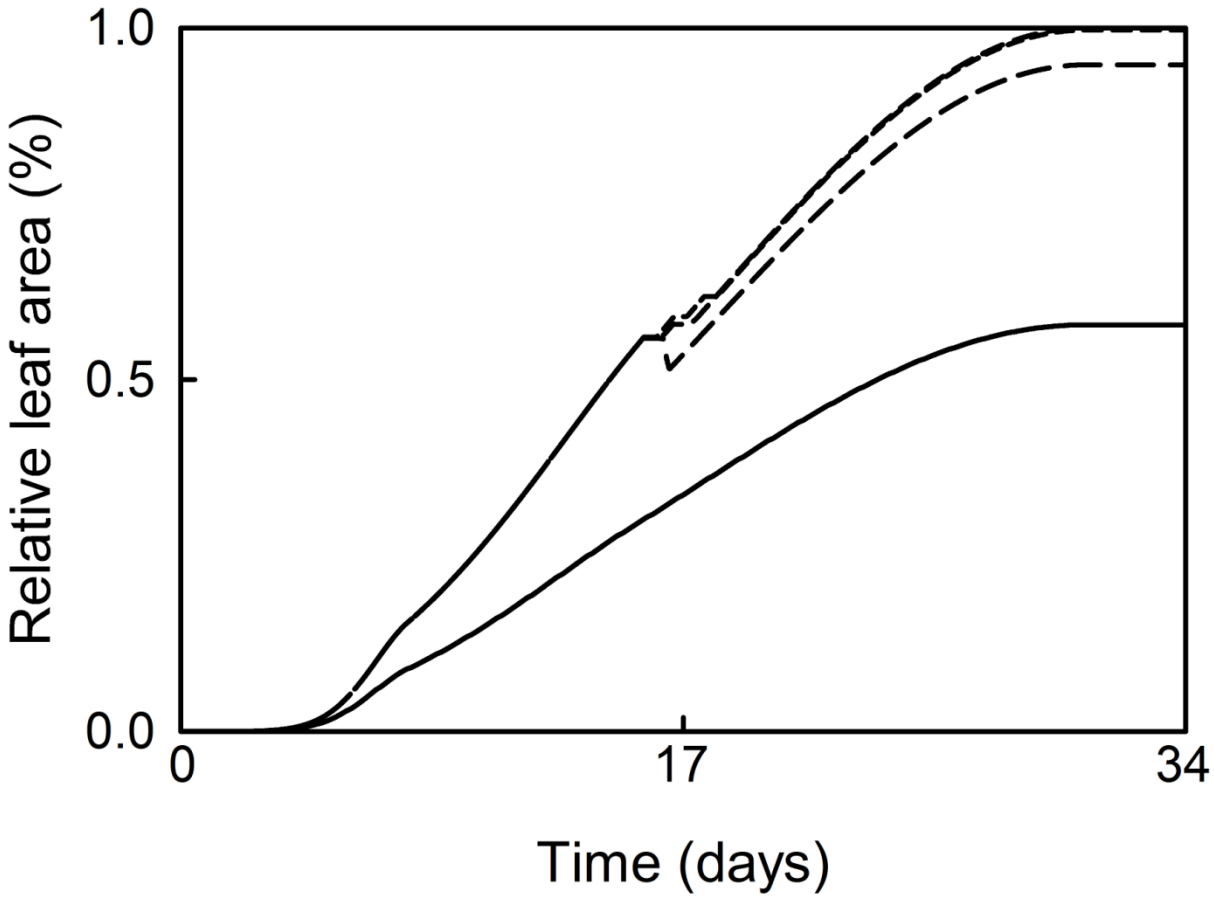

Figure 1.4: The effect of 100% increased UV-B radiation: spread along the leaf growth period (solid line), in one day dose (long dash), in two days dose (medium dash), and in three days dose (short dash). The simulations were done for plants with average rates of CPD repairs and equal epidermal absorptance across UV-B radiation wavelengths. Note that the data for one, two, and three days UV-B radiation doses plots are overlapping until the application of the treatment (in day 18) and became a solid line on the graph.

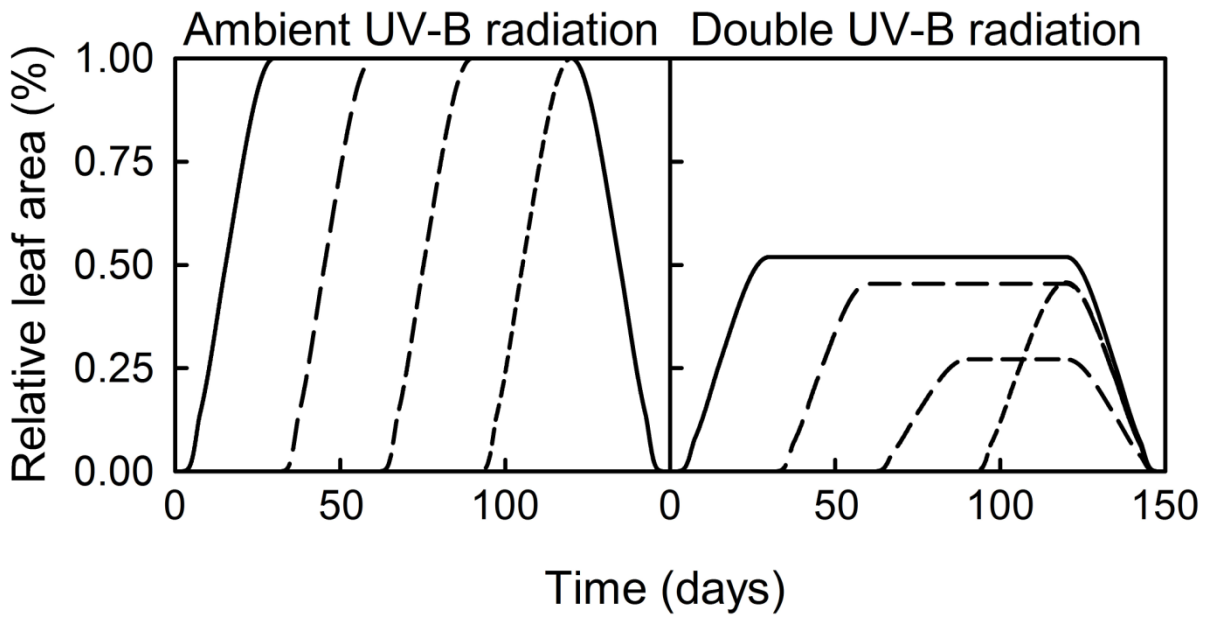

Figure 1.5: The effect of timing on leaf growth: relative leaf area for leaves growing in May (solid line), June (long dash), July (medium dash), and August dose (short dash), under ambient UV-B and double UV-B. The simulations were done for plants with average rates of CPD repairs and equal epidermal absorptance across UV-B radiation wavelengths.

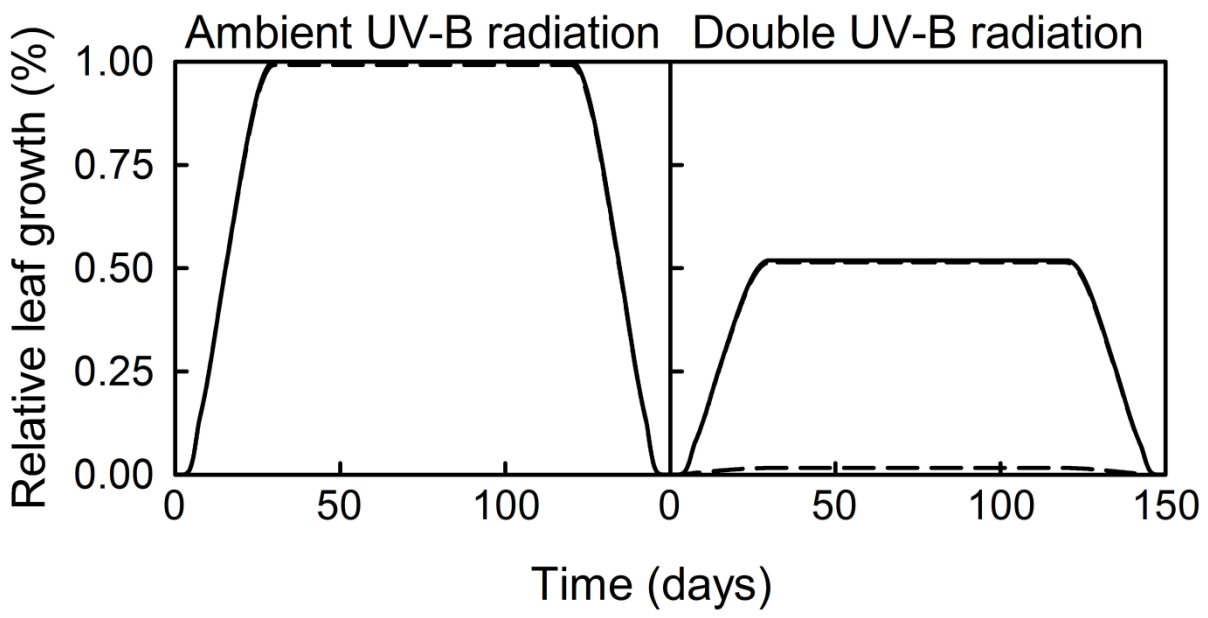

Figure 1.6: The effect of temperature on leaf growth: relative leaf area for leaves under ambient temperatures (solid line), low temperatures: ambient temperatures $-10℃$ (long dash), and high temperatures: ambient temperatures $+10℃$ (medium dash), under ambient UV-B and double UV-B. The simulations were done for plants growing in May, with average rates of CPD repairs and equal epidermal absorptance across UV-B radiation wavelengths.

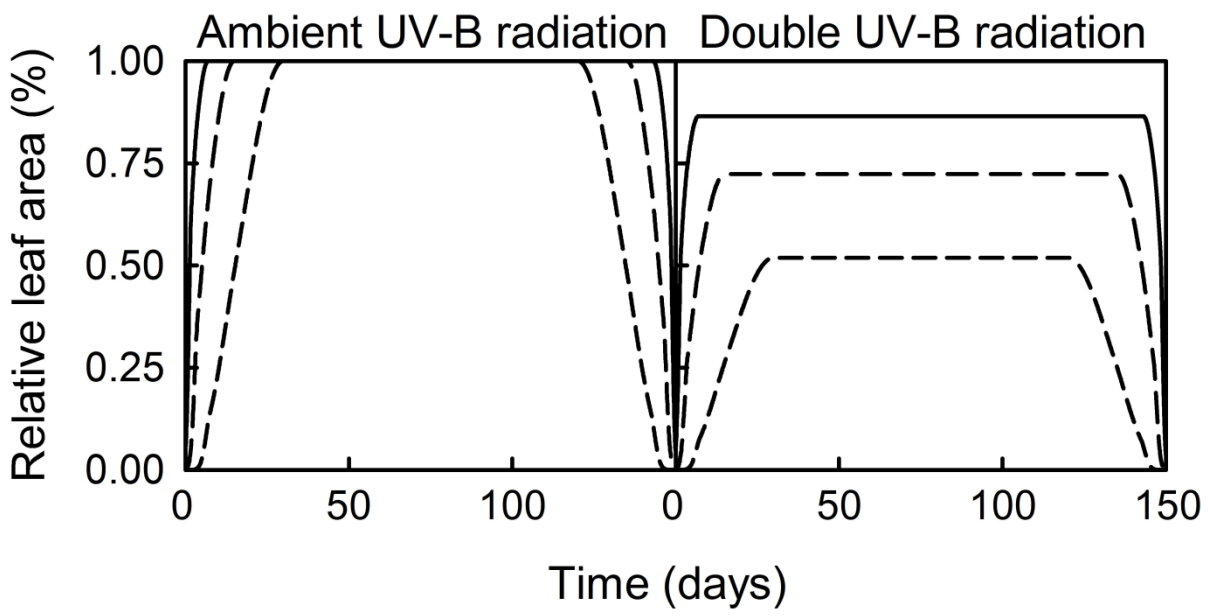

Figure 1.7: The effect of the duration of leaf growth: relative leaf area for fast growing leaves: 7 days (solid line), medium growing leaves: 15 days (long dash), and slow growing leaves: 30 days (medium dash), under ambient UV-B and double UV-B. The simulations were done for plants growing in May, with average rates of CPD repairs and equal epidermal absorptance across UV-B radiation wavelengths.

Chapter 2

Integration and scaling of UV-B radiation effects on plants: from molecular interactions to whole plant responses

submitted to Ecological Modelling

2.1 Abstract

A process based model integrating the effects of UV-B radiation to molecular level processes and their consequences to whole plant growth and development was developed from key parameters in the published literature. Enhanced UV-B radiation significantly inhibited plant growth by delaying leaf expansion processes and increasing plant metabolic rates and/or reducing the photosynthesis rate. The costs of effective epidermal UV-B radiation absorptive compounds did not result in any significant changes in plant growth, but any associated metabolic costs effectively reduced the potential plant biomass. The model showed significant interactions between UV-B radiation effects and temperature and any factor leading to inhibition of photosynthetic production or plant growth during the midday, but the effects were not cumulative for all factors. Vegetative growth was significantly delayed in species that do not exhibit reproductive cycles during a growing season, but vegetative growth and reproductive yield in species completing their life cycle in one growing season did not appear to be delayed more than two-five days, probably within the natural variability of the life cycles for many species. This is the first model to integrate the effects of increased UV-B radiation through molecular level processes and their consequences to whole plant growth and development.

2.2 Introduction

Integration among various ecological processes and scaling among various levels of organization are inherent in ecology and pose major challenges in understanding the consequences of global environmental problems (Levin, 1992). Although research on integrating ecological levels has been done (Clark, 1990), many ecological studies are still short-term and small-scale experiments. Such experiments have limited ecological relevance as more factors are added and the scale is increased (Carpenter, 1996; Schindler, 1998), and fail in testing the major theories about the natural world (Weiner, 1995). Our approach

modeled published molecular interactions and the relevant mechanisms responsible for the whole plant responses to ambient and enhanced UV-B radiation (280 – 320 nm). Enhanced UV-B radiation is the increase in irradiance and a shift to shorter wavelength as a consequence of stratospheric ozone depletion.

Ultraviolet (UV) radiation has been a natural environmental stress factor for organisms since the pre-Cambrian era (Cockell and Horneck, 2001; Lowry et al., 1980; Rettberg et al., 1998; Sagan, 1973). Before the early formation of the stratospheric ozone layer in the Cambrian period (approximately 520 million years ago), (Caldwell, 1979; Lowry et al., 1980; Margulis et al., 1976; Rozema, 1999), UV-C (100 – 280 nm) irradiance at the ground limited the early organisms to the aquatic environments (Castenholz, 2004; Cockell, 1998; Dillon and Castenholz, 1999). The development of a stratospheric ozone layer and the evolution of new biochemical processes contributed partly to the successful development of terrestrial plant species (Lowry et al., 1980; Rozema, 1999; Stafford, 1991), which led to feedback effects on atmospheric, terrestrial and aquatic systems (Kenrick and Crane, 1997). While the discovery that chlorofluorocarbons (CFCs) may catalyze the breakdown of ozone in the stratosphere (Molina and Rowland, 1974), and the discovery of the Antarctic ozone "hole" (Farman et al., 1985) stimulated research on the potential effects of enhanced UV-B radiation on terrestrial systems, technical difficulties limited experimental research mostly to individual and sub-individual plant levels. (DeLucia et al., 2001). Moreover, most of these studies focused on either molecular interactions or whole plant effects of enhanced UV-B radiation, and did not propose quantitative mechanisms linking various levels of ecological organization. However, three decades of research showed that a primary mechanism of UV-B radiation induced damage results from molecular level injuries in many plant species (Britt, 1996; Rozema, 1999; Sancar and Sancar, 1988; Taylor et al., 1997; Weber, 2005) and targeting of UV-B radiation specific and non-specific plant stress proteins (Ballare et al., 2011; Brown et al., 2005; Wargent et al., 2009a; Wargent et al., 2009b), which may result in direct effects on the whole plant growth, resource allocation, and reproduction, and indirect effects on the community structure and function (Caldwell et al., 1998b; Caldwell et al., 2007; Warren et al., 2002). Moreover, the potential UV-B induced changes may be amplified across higher ecological scales and trophic levels (Caldwell et al., 1998b; van der Leun et al., 1998; Warren et al., 2002). Furthermore, stratospheric ozone depletion and global warming may be

producing significant changes in both surface and stratospheric climate (Hartman et al., 2000; United Nations Environment Programme, 2012).

Thus, understanding how different levels of the UV-B radiation environment of Earth affect terrestrial communities is essential in predicting how the current stratospheric ozone depletion may affect life on Earth, and may interact with climate changes towards rapid global change. Also, understanding how UV-B radiation affects terrestrial communities may provide insights in the UV-B radiation contribution as a selection agent throughout the evolutionary history of Earth.

We modeled the function of an individual plant by integrating photosynthetic production, respiration, and resource allocation. To scale the effects of UV-B radiation in a plant, we included the molecular effects of increased UV-B radiation, the cellular responses to the molecular effects, and their potential consequences on leaf growth and development. Our primary hypothesis was that enhanced solar UV-B radiation-induced molecular changes significantly reduce plant growth and development. Damage to DNA can cause delays in cell division and expansion during leaf expansion, and possible cell apoptosis (de Lima-Bessa et al., 2008; Lo et al., 2005), potentially leading to modifications in leaf morphology and significantly reducing the photosynthetic capacity of leaves (Suchar and Robberecht, 2015b). Possible UV-B radiation-induced photosynthetic inhibition and increased metabolism, together with reduced photosynthetic capacity of leaves, may have significant consequences upon whole plant growth and development (Caldwell et al., 1998b; Milchunas et al., 2004; Rozema et al., 1997).

Although there is considerable research regarding the effects of UV-B radiation on sub-individual and individual plant levels, we used a modeling approach to integrate these processes and to examine how changes in molecular and cellular processes are scaled to effects at the whole plant level. We examined a variety of questions that would be difficult to approach through experimental research, including: (1) What are the most advantageous strategies for the plant to optimize its growth and potential fitness? (2) Does UV-B radiation interact with other environmental factors? (3) What is the effect of midday photosynthetic depression?

2.3 Model framework

The plant can be viewed as a system that dynamically balances the resource uptake and use. Plants optimize the resource allocation by investing resources in such a way that maximizes the returns, i.e., the growth of organs involved in the acquisition of the limiting resources is promoted (Bazzaz, 1997; Bloom et al., 1985; Cockell, 1998; Heilmeier et al., 1997; Wayne and Bazzaz, 1993). In general, environmental conditions lead to changes in resource allocation and storage, with species growing in variable environments being more plastic in their resource allocation than plants from more stable environments (Bazzaz, 1997; Chiariello and Gulmon, 1991; Miao et al., 1991; Weiner, 2004). This pattern may also apply to a comparison of species with an annual (more plastic) versus a perennial (less plastic) life span. Therefore, the whole plant is the consequence of its life history (Aphalo, 2010).

Photosynthetic fixed carbon is synthesized in carbohydrate, then exported to the other plant organs or converted in starch for storage for short- or long- term carbohydrate plant needs (Smith, 2005). The sink strength of various plant organs regulates the production and allocation of carbohydrates in plants (Cournede et al., 2006; de Reffye et al., 2008; Mathieu et al., 2009). Growth is in part controlled by nitrogen (N) uptake. When nitrogen is not limiting, growth is proportional to the photosynthesis rate. When N becomes limiting, growth rate slows and carbohydrates are accumulated as starch (Fichner et al., 1995; Schulze and Schulze, 1995). While whole plant carbon fixation and nutrient uptake rate are influenced by environmental conditions, carbon fixation rates vary in different leaves on the plant, as well as nutrient uptake rates of different root segments (Bazzaz, 1997). The correlation between growth and carbon fixation is generally weak, in part because of the variability in the cost in growth due to other resources availability (i.e., type of N source in soil), and in part as a result of variations in resource allocation patterns (Bazzaz, 1997; Crabtree and Bazzaz, 1993; Korner et al., 1979).

Resource allocation ratios within plant parts changes with ontogeny (Bazzaz, 1997; Gedroc et al., 1996; Weiner, 2004), but the annual growth rates of leaves, stems, and roots appear to follow similar isometric scale across many seed plant species (Enquist and Niklas, 2002; Niklas and Enquist, 2002b). These allometric models consider leaves as the only photosynthetic organs, and assumed that biomass allocated to reproductive plants was either negligible or equally drawn from the pools of leaves, stems and roots (Enquist and Niklas,

2002; Niklas and Enquist, 2002b). From the plant architecture perspective, plants are composed of repeating structural elements, with identical or similar combination of organs, specific to individual species (Barthelemy and Caraglio, 2007; de Reffye et al., 2008; Nygren and Pallardy, 2008). These confirm the similar isometric scaling among plant species, at least for aboveground vegetative organs.

In many species, resource allocation towards reproductive parts occurs only after the plant reaches a certain mass, size, or age (Bazzaz and Catovsky, 2001). The importance of mass, size, or age as the trigger of the reproductive parts growth, depends on the species. Also, the required size varies with plant age within same plant species (Bazzaz, 1997; Schmid et al., 1995). Regardless of the trigger mechanism, re-allocation of resources towards reproduction can be complete, gradual, or resource-availably based (Bazzaz, 1997; King and Roughgarden, 1982a, b; Reekie and Bazzaz, 1987). Moreover, the allocation to reproductive organs can exceed the maturation capacity of plants, and result in abortion of some of the reproductive organs (Bazzaz, 1997; Lee and Bazzaz, 1982, 1986; Marshall and Ellstrand, 1988). Allocation towards secondary metabolites results in resources re-allocated from immediate plant growth, but can result in greater benefits in the long run (Gayler et al., 2008). For example, secondary metabolites are the most important leaf constituents that absorb UV-B radiation and can prevent the bulk of the incident radiation from reaching the cellular DNA, photosystems and membranes (Dixon and Paiva, 1995; Koes et al., 1994; Robberecht et al., 1980; Winkel-Shirley, 2002).

Ultraviolet-B radiation can interfere with the plant growth and development in several ways. Ultraviolet-B radiation-induced DNA lesions (Britt, 1995, 1996; Sancar, 1994; Taylor et al., 1997), inhibited cell division (Gonzalez et al., 1998; Rousseaux et al., 2004), reduced cell expansion (Hectors et al., 2010; Wargent et al., 2009b), or both (Hoffman et al., 2003; Hopkins et al., 2002). These delays in cell division and expansion may result in significant reduction in leaf area (Suchar and Robberecht, 2015b). Although photosynthetic rates are not well correlated to total leaf area (Bazzaz, 1997), a reduction in leaf area may result in reduction in the carbohydrate production of the plant. Moreover, plant protection against increased UV-B radiation requires investment of resources in metabolic processes. For example, increases in UV-B radiation generally stimulate the species-specific production of secondary metabolites and results in changes in the quantity and quality of epidermal

absorption (Dixon and Paiva, 1995; Li et al., 1993a; Schmelzer et al., 1988a; Winkel-Shirley, 2002). Also, photoproducts are reversed through enzyme-driven repair mechanisms (Sancar, 1994), that might also affect the plant metabolic costs.

The UV-B radiation interference with plant photosynthesis is more complex. Many studies conducted under glasshouse and environmental chamber conditions show that enhanced UV-B radiation can impair the photosynthesis by affecting the photosystems and phosphorylation reactions, chloroplast structure, and enzyme activity (Allen et al., 1998; Sullivan and Rozema, 1999; Zhou et al., 2007). Field studies using modulated field radiation systems that supplement UV-B radiation proportionally to the ambient UV-B regiment show that enhanced UV-B radiation has no significant effects on the photosynthesis (Bassman et al., 2002; Bassman and Robberecht, 2006; Caldwell et al., 2007; Searles et al., 2001).

Although those field studies demonstrated that photosynthesis is seldom affected by enhanced UV-B radiation, morphological changes such as reduced leaf area, shoot mass and plant height are more frequently present (Caldwell et al., 2003; Caldwell et al., 2007; Searles et al., 2001). Changes in resource allocation and timing of reproduction has been observed (Demchik and Day, 1996; Koti et al., 2007; Koti et al., 2005), but it is not definitive that such changes are direct consequences of increased UV-B radiation or indirect effects caused by diminished carbohydrates production, or changes in nutrient uptake.

Our research modeled these processes for a hypothetical generalized flowering plant with simple, planophyllic, glabrous, green leaves, and integrated the effects of UV-B radiation on DNA and the consequences on the plant growth, development and reproduction over one growing season. This generalized flowering plant allowed us to model the influence of UV-B radiation under a variety of scenarios, including variations in growth characteristics and UV-B irradiance.

2.4 Conceptual Model

We chose a process based model to illustrate the effect of UV-B radiation on the whole plant (Figure 2.1). To emphasize the molecular-to-whole plant integration under various levels of UV-B radiation, our model focused on the whole plant function, instead of the plant architecture. Leaf angle can greatly influence the daily effective UV-B radiation

dose intercepted by individual leaves. For example, vertical leaves may receive about 5-41% less daily UV-B radiation, depending on the latitude and elevation (Caldwell et al., 1980). But it can be also true that some leaves angles will increase the UV-B radiation interception. Also, since our UV-B radiation – leaf area model (Suchar and Robberecht, 2015b) applies to new growth only, it can be assumed that self-shading is negligible. Total leaf area determines the gross primary production. A fraction of the photosynthetic production is used for respiration, while the remaining production is used towards the growth (Haefner, 2005). The remaining photosynthetic production is differentially allocated towards plant organs, following the same proposed isometric rates across the growing season (Enquist and Niklas, 2002; Niklas and Enquist, 2002a, b). Leaf biomass is correlated to leaf area, leaf area ratio (leaf area per leaf weight) is species specific, and respiration rates vary with the total biomass of the plant. Also light interception is proportional with leaf area, and carbon and nitrogen sources and sinks do not interact significantly (i.e., plant growth is not limited by nitrogen uptake). The UV-B radiation affects whole-plant growth and development by interfering with leaf expansion, with photosynthesis processes, and respiration (Figure 2.1). We considered a generic plant growing over a local growing season. Light interception is proportional with the LAI and plant leaf architecture effects were considered negligible.

Ultraviolet-B radiation data were obtained from the UV-B Monitoring and Research Program (UVMRP) for ten years 2000-2009, Pullman, Washington, which is a location that is representative of UV-B radiation for the northern temperate zone. We used UV-B Langley calibrated data, considered more appropriate than lamp calibrated data for sunny and dry locations (UVMRP 2010). Ultraviolet-B radiation data were averaged for the 10-year period, and for each month of the local growing season (May-September). Hourly temperature data were obtained for Spokane, Washington from National Oceanic and Atmospheric Administration - National Climatic Data Center (NOAA 2011).

2.5 Mathematical Model

For the model, the plant was considered to have the following organs: roots (R), aboveground structural organs (S), such as stems, or sheaths and stolons, leaves (L), reproductive organs (Ro), and seeds (Sd).

Since the model considers only the plant function, only the carbon content and its use by different plant pools was considered (Haefner, 2005; Kerkhoff et al., 2005). As the plant architecture was not considered, and the modularity of plant structure was not an issue, we modeled the plant growth (i.e., organ appearance) as continuous (Mathieu et al., 2009) and resulting from the source-sink relationships presented subsequently.

2.5.1 Total plant production

Under the assumption that leaves are the only photosynthetic organs, total production (P, g time^{-1}) is direct proportional with the total leaf mass (Enquist and Niklas, 2002; Niklas and Enquist, 2002a, b):

$$P = k_1 M_L \quad (1)$$

Where, M_L is the leaf mass of the plant (g), k_1 is the plant mass photosynthetic production rate multiplier (time^{-1}).

Since, generally, the photosynthetic capacity of leaves exhibit a decline after their expansion (Ackerly and Bazzaz, 1995; Kitajima et al., 2002), a linear adjustment factor of the decrease of the photosynthetic capacity with time was considered (Kikuzawa, 1991; Kitajima et al., 2002). Under the assumption that all leaves in a plant have identical thickness, equation 1 becomes:

$$P = k_1 k_2 A_L \left(1 - b_{pd} t\right) \quad (2)$$

Where, A_L is the total leaf area of the plant (m^2), k_2 is the total leaf area – total plant mass multiplier (g m^{-2}), b_{pd} is the slope of the linear photosynthetic capacity decline (time^{-1}), and t is time.

2.5.2 Total plant growth

The total photosynthetic production available for growth (G, g time^{-1}) is a function of the total production (P, g time^{-1}), the maintenance respiration (R, g time^{-1}) and the production allocated to/from storage (S, g time^{-1}).

$$G = P - R \pm S \quad (3)$$

2.5.3 Respiration

It was considered that maintenance respiration (R, g time-1) is a function of total plant mass (M_T, g).

$$R = k_3 M_T \tag{4}$$

Where k_3 is the plant mass respiration rate multiplier (time^{-1}).

2.5.4 Plant organ growth

In the basic model, we assumed that all production is allocated to new organ growth from to the common pool of resources.

Under these assumption, the new growth for a new plant organs (G_O, g time^{-1}) becomes

$$G_O = k_{4,O} k_{5,O} G \tag{5}$$

Where, "O" denotes the organ considered (i.e., roots (R), above ground structural organs (S), leaves (L), reproductive organs (Ro), and seeds (Sd)), $k_{4,O}$ (unitless) is the conversion efficiency in biomass of photosynthetic production, and $k_{5,O}$ (unitless) is the percent of total photosynthetic production allocated to the growth of plant organs (Bazzaz, 1997; Enquist and Niklas, 2002; Kerkhoff et al., 2005; Niklas and Enquist, 2002a, b).

For most species, the plant reproduction is associated with a critical plant mass (Geber et al., 1997). However, photoperiod and environmental stress can also initiate flowering in some species (Putterill et al., 2004). Regardless, the minimum mass associated with reproduction can vary with plant age and resource availability (Bazzaz, 1997). Since we considered a generalized plant over one growing season, we considered that the plant reproduction is triggered sometime during the growing season, and we simulated different times of beginning of reproduction effect on plant fitness.

For the resource allocation to reproductive parts, we considered a gradual allocation of resources instead complete allocation of resources towards reproductive parts. In this case, the percent of total photosynthetic production allocated towards reproductive parts becomes:

$$k_{5,RO} = \begin{cases} 0 & if\ t < t_{br} \\ a_t t & if\ t_{br} \le t < \frac{1}{a_t} \\ 1 & if\ t \ge \frac{1}{a_t} \end{cases} \tag{6}$$

Where, a_t (time^{-1}) is the linear increase in photosynthetic production allocation to reproductive parts.

Thus, the proportion of total photosynthetic production allocated towards fruits and seeds follow the same scenario portrayed in equation (6), and it is limited by the resources available for allocation. The processed is considered to be delayed by Δt_r (time), the interval necessary for reproduction (i.e., going from flowers to seeds).

2.5.5 UV-B radiation effects on whole plant growth and development

The plant model equations (1, 2, 4, 5) are adjusted for the effects of UV-B radiation as follows:

$$P = k_{1,UVB} k_1 M_L \tag{1.1}$$

$$P = k_{1,UVB} k_1 k_{2,UVB} k_2 A_L \left(1 - b_{pd} t\right) \tag{2.1}$$

$$R = k_{3,UVB} k_3 M_T \tag{4.1}$$

$$G_L = k_{6,UVB} k_{4,L,UVB} k_{5,L} G \tag{5.1}$$

Where, $k_{1,UVB}$ is an adjustment factor due to the effects of UV-B radiation on photosynthesis, $k_{2,UVB}$ is an adjustment factor due to effects of UV-B radiation on leaf thickness, $k_{3,UVB}$ is an adjustment factor due to the effects of UV-B radiation on metabolic processes, $k_{4,L,UVB}$ is the conversion efficiency in leaf biomass of photosynthetic production when plant is exposed to increased UV-B radiation, and $k_{6,UVB}$ is an leaf growth adjustment factor due to the effects of UV-B radiation on leaf expansion.

To simulate the UV-B radiation effects on the leaf area, we used the Suchar and Robberecht (2015) model that simulates relative leaf area for various UV-B radiation-induced DNA lesions and rates of photorepair and dark repair.

The variables of interest in our model were UV-B radiation-induced relative changes in organ biomass:$M_{O,UVB}/M_O$ ("O" denotes the organ considered (i.e., roots (R), stems (S), leaves (L), reproductive organs (Ro), and seeds (Sd)) for the scenarios considered.

2.6 Parameter estimation

Since we modeled a hypothetical generalized plant, the parameter estimators considered were means calculated for large arrays of species. Therefore, many of these values were obtained from comprehensive plant traits papers (Kattge et al., 2011; Poorter et al.,

2009; Poorter and Remkes, 1990; Searles et al., 2001; Wright et al., 2004), but not limited to their results.

2.6.1 Total plant production

Under the assumption that leave are the only photosynthetic organs, total production (P, g time^{-1}) is direct proportional with the total leaf mass (Enquist and Niklas, 2002; Niklas and Enquist, 2002a, b):

$$P = k_1 M_L \quad (1)$$

Where, M_L is the leaf mass of the plant (g), k_1 is the plant mass photosynthetic production rate multiplier (time^{-1}).

Under field radiation conditions photosynthesis follows two general patterns: first pattern exhibit an increase in photosynthesis in the morning until it reaches saturation, followed by a decrease in the afternoon; second pattern exhibit a gradual increase in photosynthesis in the morning, followed by a midday depression in photosynthesis rates, and another peak in photosynthesis during the afternoon (Larcher, 2003; Xu and Shen, 2005). The proposed causes for the midday photosynthetic depression include air and leaf temperature, CO_2 concentration, air and soil moisture content, decrease in leaf water potential, stomatal closure, increases in respiration, photorespiration and mesophyll resistance, developmental stage, circadian rhythm, photosynthate accumulation, decrease in Rubisco activity and photochemical efficiency, and enhanced abscisic acid production (Larcher, 2003; Mc Donald, 2003; Tay et al., 2007; Xu and Shen, 2005). The second peak in net photosynthesis is usually not as pronounced as the first peak (Xu and Shen 2005). Midday depression might be responsible for decreases in productivity of 30-50% or more (Xu and Shen 2005). We simulated two theoretical scenarios: one peak in net photosynthesis, and two-peak photosynthesis. The maximum net photosynthesis values ranges from 0.008 to 0.14 h^{-1} for herbaceous plant species, and from 0.003 to 0.03 h^{-1} for woody species (Larcher, 2003). For our model we considered a mid-value from the interval of maximum net photosynthesis range which led to a maximum value for $k_1 = 0.1$ plus the maintenance respiration (Larcher 2003). To account for daily changes in photosynthesis, we considered a generic trend, as follow:

$$k_1 = aTime + bTime^2 \quad (7)$$

Where time denotes the time step, and ranges from sunrise until sunset (adjusted for time of the year), and a and b coefficients were calculated for the maximum value for k_1 considered, and the time range. Equation coefficients were adjusted for each month of the growing season considered.

For the second trend, plant species exhibiting midday depression, we considered a reduction in photosynthesis around the midday resulting in an average daily reduction in photosynthesis of 40% (Xu and Shen 2005). More specific relationships can be readily substituted for the species of interest.

For 45,733 entries, the average specific leaf area was calculated to be 16.6 mm^2 mg^{-1} (Kattge et al., 2011), which leads to a value for the total leaf area – total plant mass multiplier k_2 of 60.24 g m^{-2}.

The leaf photosynthetic capacity decline rate seems to be positively correlated with leaf lifespan (Ackerly and Bazzaz, 1995; Kitajima et al., 2002). For leaves with longer lifespan (>170 days) as those considered in our model, we considered a loss in photosynthetic capacity of approximately $b_{pd} = 0.1\%\ day^{-1}$ or $b_{pd} = 0.004\%\ h^{-1}$ (Kitajima et al., 2002).

2.6.2 Respiration

Respiration, the fraction of daily production used in the same time, is sensitive to a series of factors including nutrient content, growth and photosynthesis rates, temperature, and plant organs (Atkin et al., 2005; Atkin and Tjoelker, 2003; Lambers et al., 2005; Loveys et al., 2003; Poorter et al., 1991). Although it has been shown that different plant organs exhibit different respiration rates (Reich et al., 2008), since we modeled a generalized plant, we assumed that the respiration rates are identical in all plant organs. These rates can be easily adjusted in case of modeling specific species. For the temperature-dependence of respiration, we considered a general Q10 value of 2.0 (i.e. respiration doubles per 10℃ rise in temperature). While the Q10 respiration value is not constant and it is dependent on the temperature range used in its calculations and the temperature-response curve used (Atkin et al., 2005; Atkin and Tjoelker, 2003), it was considered a reasonable approximation since all the other parameter estimators in the model are generalized values, averaged over a wide range of species.

Thus, the plant reaches a maximum relative respiration rate at about 50℃, half of the maximum relative respiration rate at 40℃, and negligible respiration at 0℃. At 20℃, the maintenance respiration rates at the beginning of the night range from 0.001 to 0.008 g g^{-1} DM h^{-1} in deciduous species (Larcher 2003). Also, during the night respiration rates continuously decrease by 40-50% until the sunrise (Larcher 2003). A mid-value was considered. Thus, k_3 equals 0.0045 h^{-1} during the day and the beginning of the night, and reaches 0.0025 h^{-1} at daylight, with the linear night decline in maintenance respiration.

2.6.3 Plant organ growth

The allometric relationships proposed for a broad range of plant species (Enquist and Niklas, 2002; Niklas and Enquist, 2002a, b) suggest biomass allocation ratio for Leaves (L) : Roots (R) : Stems (S) of approximately 0.3:0.13:0.57. By combining these values with the values for the conversion efficiency $k_{4,O}$ of 0.67:0.75:0.69 (L:R:S) (Poorter and Villar, 1997), the estimates for the percent of total photosynthetic production allocated to the growth of plant organs $k_{5,O} = 0.31: 0.12: 0.57$ (L:R:S). For a wide range of species, the conversion efficiencies $k_{4,O}$ to reproductive organs and seeds are 0.71 and 0.65 (Poorter and Villar, 1997).

We considered that the duration of flowering is about one to two weeks, and the fruit growth and seed maturation is about one month. As a result for a May to September growing season considered, the time of beginning of reproduction t_{br} should be at the latest the end of July. These result in values for $t_{br} = 2160\ h^{-1}$ and for $\Delta t_r = 168 - 336\ h^{-1}$.

If we consider a gradual allocation of resources towards reproductive parts of about two to four weeks, the value for the linear increase in photosynthetic production allocation to reproductive parts become approximately$a_t = 0.0004\ h^{-1}$.

2.6.4 UV-B radiation effects on whole plant growth and development

Studies conducted in glasshouse and growth chamber conditions indicated that enhanced UV-B radiation can impair the photosynthesis (Allen et al., 1998; Sullivan and Rozema, 1999; Zhou et al., 2007), while field studies with modulated field UV-B radiation systems indicated that enhanced UV-B radiation has no significant effects on the photosynthesis (Bassman et al., 2002; Bassman and Robberecht, 2006; Caldwell et al., 2007;

Searles et al., 2001). For our models we considered that enhanced UV-B radiation has no significant effects on photosynthetic rate, but we considered simulations with decreases in net production in the calibration and validation.

Increased UV-B radiation can induce increases in leaf thickness (Yamasaki et al., 2007), decreases in leaf thickness (Kakani et al., 2003), or non-significant changes in leaf thickness (Kotilainen et al., 2009). While many of the initial studies of the effects of UV-B radiation on plants reported increases in leaf thickness (Bornman and Vogelmann, 1991), analysis of field studies failed to reveal any significant UV-B radiation induced changes in leaf thickness (Ballare et al., 2011; Searles et al., 2001). Therefore we considered for our model a generic range for the adjustment factor due to effects of UV-B radiation on leaf thickness, $k_{2,UVB}$between 0.75 and 1.25, to be further investigated in the model calibration and validation.

While increases in leaf respiration were observed, when plants were subject to increased UV-B radiation (Ziska et al., 1992), there are very few studies investigating this aspect (Bassman et al., 2003; Gwynn-Jones, 2001). The studies of the effects of increased UV-B radiation showed increases in respiration rates from 0 to 280% (Bassman et al., 2003; Gwynn-Jones, 2001). The increases in maintenance respiration might be due to increases in resource demands by the plant tissues for protection and repair in both emerging and mature leaves (Gwynn-Jones, 2001). Since the respiration costs are comparable to the growth costs over a growing season in herbaceous plants, variations in those costs can significantly alter the overall plant growth and productivity (Amthor, 1984). Levels of UV-B radiation corresponding to a 25% decrease in stratospheric ozone induced no significant changes in dry weight, leaf area, and resource allocation of a native grass *Calamagrostis purpurea.* However, respiration rates in both young and mature leaves increased up to approximately 280% (Gwynn-Jones, 2001). Studies of the effects of increased UV-B radiation on perennial species showed no significant increases in respiration rates (Bassman et al., 2003). Therefore we considered $k_{3,UVB}$ a range of 1 to 4 as the adjustment factor due to the effects of UV-B radiation on metabolic processes. A final value was inferred from the model calibration procedures.

In general, plants exposed to increased UV-B radiation exhibit elevated levels of secondary metabolites. The construction costs of flavonoids and related phenolic compounds

are generally higher than the average for the leaf mass (Poorter and Villar, 1997), and therefore, lowers the conversion efficiency of photosynthetic production in leaf biomass down. For example, an increase in secondary metabolites production by 100% will lower the conversion efficiency from $k_{4,L} = 0.67$ to about $k_{4,L,UVB} = 0.66$ (Poorter and Villar 1997).

For the model of UV-B radiation effects on the leaf area, we used a Suchar and Robberecht (2015) model that simulates relative leaf area for various UV-B radiation-induced DNA lesions and rates of photorepair and dark repair.

2.7 Modeling methodology

The model was created in Vensim (Systems, 2009). Data compilation, preparation, and analysis were done in various programs such as Microsoft Access, Excel, and R-language.

The models were verified for consistency and units, for correctness of the mathematics and for accuracy of the conceptual logic (Rykiel, 1996), calibrated and validated (Gardner and Urban, 2003; Rykiel, 1996; Shugart, 1984). Prior to this, sensitivity analysis procedures were performed (Aber et al., 2003; Plentinger and Penning de Vries, 1996; Rykiel, 1996).

The variables of interest in our model were UV-B radiation-induced relative changes in organ biomass:$M_{O,UVB}/M_O$ ("O" denotes the organ considered (i.e., roots (R), stems (S), leaves (L), reproductive organs (Ro), and seeds (Sd)) for the scenarios considered.

2.8 Model analysis

2.8.1 Sensitivity analysis

The parameter values ±25% for the major plant growth model were used in the model sensitivity analysis. For the UV-B radiation effects on the plant growth and development, the ranges derived for the major model parameters were used for the allowable limits in the model sensitivity analysis. The relative biomass of roots, structural organs, leaves, and mature seeds were measured across the tested model parameters (Figure 2.2).

The sensitivity analysis of the model showed that all model output variables considered were highly sensitive to the net production available to growth (production per leaf mass, and respiration per plant mass), and the proportion of net production allocated to structural organs and leaves biomass. The measured variables were moderately sensitive to

the decline in leaves photosynthetic capacity in time, the proportion of net production allocated to roots, and the speed of reallocation of resources from vegetative biomass towards the reproductive biomass. The relative biomass of roots, structural organs, leaves, and mature seeds were somewhat or not influenced by changes in conversion efficiency of net production for any plant component. Only seed biomass was influenced by changes in the time required for reproduction. Seed biomass was relatively more sensitive than root, shoot, and leaf biomass to changes in decline in leaves photosynthetic capacity with age, and allocation ratio towards roots and reproductive organs.

The relative biomass of roots, structural organs, leaves, and mature seeds were highly sensitive to UV-B radiation induced changes in photosynthetic production and metabolism, but not very sensitive to increases in conversion efficiency to leaf biomass due to supplemental metabolic investment in secondary metabolites. The effects of UV-B radiation on leaf expansion were previously analyzed in Suchar and Robberecht (2015).

2.8.2 Calibration and validation

Results from meta-analysis studies of the effects of UV-B radiation on plant characteristics were used in the calibration and validation process (Li et al., 2010; Searles et al., 2001). For field studies simulating 10-20% and >20% ozone depletion, the average reduction in aboveground vegetative biomass ranged from 6% to 9-15%, the average reduction in shoot biomass ranged from 6% to 16%, the average reduction in leaf area ranged from 1.4% to 16.8% (Searles et al., 2001). Non-significant changes were recorded for leaf mass per area and reproductive yield (Searles et al., 2001). Similar meta-analysis recorded for 10-20% and >20% ozone depletion, average reduction in total biomass ranging from 7% to 11.7% for herbaceous plant species, and from non-significant to 13.6% in woody plant species, average reduction in leaf area ranging from non-significant to 16.1% and 16.8% in herbaceous and wood plant species, respectively (Li et al., 2010). The changes in root: shoot ratios were non-significant for both ozone depletion categories (Li et al., 2010). The final values considered for our calibration and validation of our model, for conditions simulating 10-20% ozone reduction and >20% ozone depletion, were the following: for average decreases in aboveground biomass 6% to 12.5%; for average decreases in leaf area 1.4% to 16.5%; for shoot biomass 6% to 16%; for reproductive yield 0% for both ozone depletion

regimes. Since these averages had 95% confidence intervals of up to ±100%, we considered that if our generic model yields values within the same order of magnitude with those considered for calibration, the model is satisfactory. If it yields values outside these constrains, the model requires further refinement.

The model was calibrated by an iterative process to adjust the most sensitive parameters. The calibration process suggests that enhanced UV-B radiation may cause increased in the plant metabolic rates, but maybe not as high as suggested in literature (Gwynn-Jones, 2001). Our simulations suggest a 0.5% increase for UV-B radiation levels corresponding to about 10% ozone depletion, and a 1% increase for UV-B radiation levels corresponding to about 20% ozone depletion. Our model uses parameter estimators that were averaged over large numbers of species and experimental conditions, and it was expected to not be able to capture with a high degree of precision the study on *Calamagrostis purpurea* that showed increases in respiration rates of up to approximately 280% (Gwynn-Jones, 2001).

The average decrease in aboveground and structural organs biomass in our simulations for conditions simulating about 10% and 20% ozone depletion were 4% and 11%, below the values suggested by the literature of 6 and 12.5-16% (Li et al., 2010; Searles et al., 2001), but within the confidence limits pre-established. The underestimation may be due to simulation of single values for about 10% and 20% stratospheric ozone depletion, while the studies considered in the meta-analysis (Li et al., 2010; Searles et al., 2001) covered ranges of ozone depletion. The leaf area predicted by our model, overestimated the value suggested by the literature (average decrease of about 4%) for conditions simulating about 10% ozone depletion, but underestimated the value suggested by the literature for conditions simulating about 20% ozone depletion. This suggests that some of the linear relationships used in the model are non-linear, although it is not possible to identify which relationship has to be re-evaluated at this time, since our model used averaged values.

The meta-analysis of published studies suggest that these levels of stratospheric ozone depletion lead to non-significant changes in the reproductive yield of the species investigated (Searles et al., 2001). In contrast, our model simulations showed average decreases in the number of mature seeds of 5% to 12%, which may result from the fixed reproduction cycle interval used. If plants optimize the resource allocation by investing resources in such a way that maximizes the return (Bazzaz, 1997; Bloom et al., 1985; Cockell, 1998; Heilmeier et al.,

1997; Wayne and Bazzaz, 1993), it is likely that the reproduction will not begin at a fixed time in under environmental conditions. A second source of possible uncertainty in the yield of mature seed is related to the relationship between net production demand posed by fertilized flowers ready to "convert" to seeds and the net production available for growth. Since our model considered biomass as the measurable unit, it is not possible to evaluate the amount of net biomass necessary to convert a particular mass of flowers in a particular mass of seeds. Also, the model quantifies reproductive of seeds as a mass of seeds, and does not account for the variation in number of seeds: mass of seeds ratio.

Even though the source data for our model was relatively heterogeneous, our model was capable of addressing the objectives and major questions of our study. The parameter values resulting in the best fit for the models are given in Table 2.1. Improved model calibration, optimization and testing can be readily done in Vensim (2009) when most of these parameters are estimated for specific species, or more complete experimental data becomes available.

2.9 Results

In addition to the simulations used to analyze the model, we considered the following scenarios: (1) increased UV-B radiation in different periods of the growing season, (2) increased UV-B radiation in combination with different epidermal absorption spectra and UV-B radiation induced DNA lesions repair rates, (3) plants growing under three temperature regimes under increased UV-B radiation, (4) effects of expedited/delayed reproduction on plant growth and reproduction under increased UV-B radiation, and (5) effects of midday photosynthetic depression in plant growth under increased UV-B radiation. To investigate these scenarios, the relative changes in maximum roots, structural organs, leaves, and mature seeds biomass under ambient, 1.5X and 2X ambient UV-B radiation regime were recorded.

The sensitivity analysis indicated that increased UV-B radiation may decrease net production, resulting from either increased metabolic rates or reduced photosynthetic rates. Decreases in the conversion efficiency in leaf biomass, due to increased production of secondary metabolites, had no substantial influence on the vegetative parts and mature seeds biomass. Also, our model showed that increased UV-B radiation decreased the biomass of

mature seeds, which suggested the probability of reproductive timing shifts in plants as a response mechanism.

Increased UV-B radiation in different periods of the growing season simulations showed that plants are more vulnerable to radiation stress in the first part of the growing season, and less sensitive to increase UV-B radiation in the second part of the growing season (Figure 2.3). With fixed timing of reproduction, the biomass of mature seeds was more sensitive than vegetative biomass, and it was disproportionally more affected by increased UV-B radiation towards the end of the growing season.

Simulations of increased UV-B radiation in combination with different epidermal absorption spectra and CPD repair rates showed that increased metabolism was responsible for substantial decreases in vegetative biomass and the biomass of mature seeds. The latter was slightly more affected by exposure to UV-B radiation (Figure 2.4). Species with low CPD photorepair and dark repair rates were the most vulnerable. Species with high epidermal UV-B radiation absorption at short wavelengths exhibited the least growth inhibition even in combination with deficient CPD repair rates, while species with high epidermal UV-B radiation absorption at long wavelengths were sensitive even when they had high CPD repair rates. Mature seeds biomass showed slightly stronger declines than the whole plant biomass. Simulations of the combined effects of temperature and increased UV-B radiation, showed that the effect of increased UV-B radiation effect is confounded with the effects of low temperatures within the range of temperatures considered (Figure 2.5). The decrease in vegetative biomass and biomass of mature seeds, for the modeled low temperature range, was similar for the three levels of UV-B radiation (ambient, 1.5X and 2X ambient). At the higher temperature considered, there was an interaction between temperature and UV-B radiation. Relative to their growth at ambient temperatures, plants exposed to increased UV-B radiation exhibited less growth inhibition than plants exposed to ambient UV-B radiation (Figure 2.5 top). Relative to the growth exhibited by plants grown at ambient temperature and UV-B radiation, plants exposed to increased UV-B radiation exhibited reduced growth at ambient temperature, but still higher growth at the higher temperatures (Figure 2.5 bottom). Mature seeds exhibited similar trends.

Simulations on reproductive timing under increased UV-B radiation showed maximum delay in the vegetative biomass of about 5 and 15 days for plants exposed to 150%

and 200% UV-B radiation, respectively (Figure 2.6). These delays corresponded with a lack of mature seed production. The delay in vegetative biomass production corresponding to the maximum biomass production for mature seeds ranged from two to five days. Simulations of the effects of midday photosynthetic depression in plant growth under increased UV-B radiation showed that species exhibiting midday depression were less sensitive to the relatively high doses of UV-B radiation (Figure 2.7). At ambient and 1.5X ambient UV-B irradiance, species with midday photosynthesis depression exhibited similar growth inhibition, and at 2X ambient UV-B radiation levels, they exhibited less growth inhibition.

2.10 Discussion

Our simulations suggested that supplemental production of secondary metabolites leads to minor changes in plant biomass. It was suggested that supplemental investment in secondary metabolites might be a significant drain on the plant resources, and inevitably will affect growth (Feldheim and Conner, 1996; Johanson et al., 1995). Our model accounted only for the plant cost in net plant productivity in procuring this extra protection, without considering the potential higher metabolic costs to produce it. It is possible that these additional costs may lead to significant changes in plant biomass due to the production of secondary metabolites. Regardless, the availability of carbohydrates is important in the tradeoff between growth and plant chemical defenses (Gwynn-Jones, 2001), and it has been shown to induce qualitative changes in UV-B radiation-induced plant secondary metabolites (Lavola et al., 2003). But, our model clearly showed that even small changes in metabolic and/or photosynthetic rates can lead to significant changes in the final plant biomass (Figure 2.4). Moreover, species with more efficient and/or higher epidermal absorptance are less susceptible to increased UV-B radiation (Figure 2.4). This confirms previous experimental results that show monocots exhibiting higher sensitivity to increased UV-B radiation than dicots (Barnes et al., 1990). No level of DNA lesions repair rates can compensate for inefficient UV-B epidermal absorptance. Since the net production cost of secondary metabolites does not lead to significant decreases in plant biomass, it is plausible to assume that investment in protection to be the most efficient plant response to increased UV-B radiation. We were unable to identify the potential metabolic costs associated with secondary

metabolites production or with other cellular processes, but these aspects may be valuable components of future models.

The inhibition of seeds biomass due to increased UV-B radiation observed in our simulations contradicts the results of meta-analysis studies (Li et al., 2010; Searles et al., 2001) that showed non-significant changes in the reproductive yield. This inhibition of seed biomass may be an artifact of fixed reproductive timing in our model simulation comparisons. As shown in Figure 2.5, the delays in achieving the potential biomass under increased UV-B radiation are of maximum 5-15 days, and only when seed reproduction is not achieved. If we consider that the plant reproduction may be associated with some critical plant mass (Geber et al., 1997), and that the delays suggested by our model for species that aim to maximize seed production are much smaller (two to five days) for the growing season considered (probably within the natural variability exhibited within-species), it is possible that plants response to UV-B radiation stress may be to delay their reproductive timing, and, thus, maximize their fitness.

The timing of the increased UV-B irradiance in the environment event seems to be a significant factor (Figure 2.3). Plants exposed to increased UV-B radiation in the beginning of the growing season never recovered to their full biomass potential; while late growing season increased UV-B events had proportionally smaller effect. Plant species are more vulnerable to environmental stress during their establishment and initial growth period (Niinemets, 2010), and it appears that the effects of UV-B radiation are also significant during early plant growth and development. Similar results were observed for (*Pisum sativum*) matched pair experiments with combinations of low and high UV-B radiation levels (González et al., 1998). Regardless of the timing of exposure, plants receiving increased UV-B radiation exhibited reduced vegetative and/or seed biomass. While other environmental stress factors may require morphological and physiological responses to stress conditions (e.g., changes in root: shoot ratio, and/or quantitative/qualitative changes in solute content and concentrations) at the expenses of the vegetative growth, our model suggest that the observed growth inhibitions may be a result of the delay in growth and the timing of the delay, rather than resource availability (note that supplemental production of secondary metabolites do not seem to lead to significant reductions in plant growth and development).

Our model suggests a clear interaction between temperature and UV-B effect (Figure 2.5). For the temperature range considered, plants exhibited similar relative decreases in biomass at lower temperatures for all three levels of UV-B radiation. Plants exposed to higher temperatures exhibited less relative growth inhibition than plants exposed to ambient UV-B radiation (Figure 2.5 top), and showed higher relative growth at higher temperature than plants exposed to ambient temperature and UV-B (Figure 2.5 bottom). These results suggest that potential increased temperatures due to global change processes might effectively disguise the effects of potential increased UV-B radiation. The disparity between the effect of UV-B radiation at high temperatures and low temperatures may be an artifact of the particular low ambient temperatures considered in our model, and characteristic for our region. Simulated low temperatures reduced the photosynthetic production to very little. Therefore UV-B radiation – induced growth inhibitions were very small proportional with the potential growth. The high temperatures simulated actually increased the photosynthetic production, and the UV-B induced inhibitions were proportionally higher. This suggests that increases in UV-B radiation effects may be more visible in highly productive systems, while in low productive plant associations, these effects may be more subtle.

Similar results are suggested by the smaller UV-B radiation growth inhibition exhibited by species with midday photosynthetic depression (Figure 2.7). These results suggest that, generally, any environmental conditions that inhibit photosynthetic production or growth during the midday in particular, and growth in general, will lead to less pronounced UV-B radiation induced effects. This confirms the results of many studies showing that UV-B radiation and drought may have confounded effects (Alexieva et al., 2001; Nogués and Baker, 2000). Research also indicated that UV-B radiation and water stress may have synergistic effects (Bjorn et al., 1997), and the addition of UV-B radiation treatments to drought conditions may have beneficial effects (Balakumar et al., 1993). While we can see how the synergistic effects can emerge from our model under certain combinations of UV-B and drought simulations, the conditions that might lead to beneficial effects are not fully understood quantitatively and were not included in the model structure. The reason for this merged effect may be due to the nature of UV-B radiation induced plant growth inhibition. The accumulation of high enough UV-B radiation-induced DNA lesions that inhibit plant growth occurs during the midday and early afternoon. If other environmental conditions

prevent growth during the same period of the day, the effect of UV-B radiation cannot be separated. The effects of UV-B radiation on leaf model used (Suchar and Robberecht, 2015b) does not includes the photomorphogenic responses to UV-B radiation, which may regulate the gene activity responsible for secondary metabolites production and photorepair of DNA lesions, and may inhibit leaf cell expansion. If these photomorphogenic effects are highly sensitive to the UV-B radiation dose, and respond readily to changes in the radiation regime, it is possible that the observe effects of daytime environmental driven growth inhibitions and the effects of the UV-B radiation are confounded. If photomorphogenic responses are less plastic, it is possible that the interaction between the daytime environmental driven growth inhibitions and the effects of the UV-B radiation are less significant.

Overall, our model suggests that the effects of UV-B radiation in natural conditions might be less evident as previously thought and may be more in accordance with the results of the latest review studies. Many conditions, such as temperature and humidity can effectively mask the effects of UV-B radiation. Moreover, while some environmental factors effects can be cumulative with UV-B radiation effects, other factors might actually prevent the UV-B radiation to have observable effects to the plant growth (e.g., midday photosynthetic depression and moisture).

2.11 Conclusions

Our model is the first to integrate the effects of increased UV-B radiation through molecular level processes and their consequences to whole plant growth and development. We modeled the effects of UV-B radiation at molecular level, and proposed the possible mechanisms that lead to the observed whole plant dynamics. Enhanced UV-B radiation significantly inhibited plant growth by delaying leaf expansion processes and increasing plant metabolic rates and/or reducing the photosynthesis rate. The costs of effective epidermal UV-B radiation absorptive compounds did not result in any significant changes in plant growth, but any associated metabolic costs can effectively reduce the potential plant biomass. The model showed significant interactions between UV-B radiation effects and temperature and any factor leading to inhibition of photosynthetic production or plant growth during the midday, but the effects were not cumulative for all factors. Vegetative growth was significantly delayed in species that do not exhibit reproductive cycles during a growing

season, but vegetative growth and reproductive yield in species completing their life cycle in one growing season did not appear to be delayed more than two to five days, which is probably within the natural variability of the life cycles for many species. A review of the relevant literature showed a wide range of values for the key parameters. Moreover, certain parameter values were inferred only from the calibration process. However our model allowed the testing of several to examine a variety of questions that were difficult to approach through experimental research.

2.12 Acknowledgments

We acknowledge the insightful comments of our reviewers. This research was funded in part by the University of Idaho Student Grant Program No. UI07358.

2.13 References

Aber, J.D., Bernhardt, E.S., Dijkstra, F.A., Gardner, R.H., Macneale, K.H., Parton, W.J., S.T.A., P., Urban, D.L., Weathers, K.C., 2003. Standards of practice for review and publication of models: summary of discussion, in: Canham, C.D., Cole, J.J., Lauenroth, W.K. (eds.), Models in Ecosystem Science. Princeton University Press, Princeton, NJ, pp. 204-210.

Ackerly, D.D., Bazzaz, F.A., 1995. Leaf Dynamics, Self-Shading and Carbon Gain in Seedlings of a Tropical Pioneer Tree. Oecologia 101, 289-298.

Alexieva, V., Sergiev, I., Mapelli, S., Karanov, E., 2001. The effect of drought and ultraviolet radiation on growth and stress markers in pea and wheat. Plant, Cell & Environment 24, 1337-1344.

Allen, D.J., Nogues, S., Baker, N.R., 1998. Ozone depletion and increased UV-B radiation: is there a real threat to photosynthesis? J Exp Bot 49, 1775-1788.

Amthor, J.S., 1984. The Role of Maintenance Respiration in Plant-Growth. Plant Cell Environ 7, 561-569.

Aphalo, P.J., 2010. On how to disentangle the contribution of different organs and processes to the growth of whole plants. J Exp Bot 61, 626-628.

Atkin, O.K., Bruhn, D., Tjoelker, M.G., 2005. Response of plant respiration to changes in temperature: mechanisms and consequences of variations in Q_{10} values and acclimation, in: Lambers, H., Ribas-Carbo, M. (eds.), Plant Respiration: From Cell to Ecosystem. Springer, The Netherlands, pp. 95-135.

Atkin, O.K., Tjoelker, M.G., 2003. Thermal acclimation and the dynamic response of plant respiration to temperature. Trends Plant Sci 8, 343-351.

Balakumar, T., Vincent, V.H.B., Paliwal, K., 1993. On the interaction of UV-B radiation (280–315 nm) with water stress in crop plants. Physiol Plantarum 87, 217-222.

Ballare, C.L., Caldwell, M.M., Flint, S.D., Robinson, S.A., Bornman, J.F., 2011. Effects of solar ultraviolet radiation on terrestrial ecosystems. Patterns, mechanisms, and interactions with climate change. Photochem Photobiol Sci 10, 226-241.

Barnes, P.W., Flint, S.D., Caldwell, M.M., 1990. Morphological Responses of Crop and Weed Species of Different Growth Forms to Ultraviolet-B Radiation. Am J Bot 77, 1354-1360.

Barthelemy, D., Caraglio, Y., 2007. Plant architecture: A dynamic, multilevel and comprehensive approach to plant form, structure and ontogeny. Annals of Botany 99, 375-407.

Bassman, J.H., Edwards, G.E., Robberecht, R., 2002. Long-term exposure to enhanced UV-B radiation is not detrimental to growth and photosynthesis in Douglas-fir. New Phytol 154, 107-120.

Bassman, J.H., Edwards, G.E., Robberecht, R., 2003. Photosynthesis and growth in seedlings of five forest tree species with contrasting leaf anatomy subjected to supplemental UV-B radiation. Forest Science 49, 176-187.

Bassman, J.H., Robberecht, R., 2006. Growth and gas exchange in field-grown and greenhouse-grown *Quercus rubra* following three years of exposure to enhanced UV-B radiation. Tree Physiol 26, 1153-1163.

Bazzaz, F.A., 1997. Allocation of resources in plants: state of the science and critical questions, in: Bazzaz, F.A., Grace, J. (eds.), Plant Resource Allocation. Academic Press, San Diego, CA, pp. 1-37.

Bazzaz, F.A., Catovsky, S., 2001. Resource Partitioning, in: Editor-in-Chief: Simon, A.L. (ed.), Encyclopedia of Biodiversity. Elsevier, New York, pp. 173-184.

Bjorn, L.O., Callaghan, T.V., Johnsen, I., Lee, J.A., Manetas, Y., Paul, N.D., Sonesson, M., Wellburn, A.R., Coops, D., HeideJorgensen, H.S., Gehrke, C., GwynnJones, D., Johanson,

U., Kyparissis, A., Levizou, E., Nikolopoulos, D., Petropoulou, Y., Stephanou, M., 1997. The effects of UV-B radiation on European heathland species. Plant Ecol 128, 252-264.

Bloom, A.J., Chapin III, F.S., Mooney, H.A., 1985. Resource limitation in plants - An economic analogy. Annu Rev Ecol Syst 16, 363-392.

Bornman, J.F., Vogelmann, T.C., 1991. Effect of Uv-B Radiation on Leaf Optical-Properties Measured with Fiber Optics. J Exp Bot 42, 547-554.

Britt, A.B., 1995. Repair of DNA damage induced by ultraviolet radiation. Plant Physiol 108, 891-896.

Britt, A.B., 1996. DNA damage and repair in plants. Annual Review of Plant Molecular Biology 47, 75-100.

Brown, B.A., Cloix, C., Jiang, G.H., Kaiserli, E., Herzyk, P., Kliebenstein, D.J., Jenkins, G.I., 2005. A UV-B-specific signaling component orchestrates plant UV protection. P Natl Acad Sci USA 102, 18225-18230.

Caldwell, M.M., 1979. Plant Life and Ultraviolet-Radiation - Some Perspective in the History of the Earths Uv Climate. Bioscience 29, 520-525.

Caldwell, M.M., Ballare, C.L., Bornman, J.F., Flint, S.D., Bjorn, L.O., Teramura, A.H., Kulandaivelu, G., Tevini, M., 2003. Terrestrial ecosystems increased solar ultraviolet radiation and interactions with other climatic change factors. Photochemical & Photobiological Sciences 2, 29-38.

Caldwell, M.M., Bjorn, L.O., Bornman, J.F., Flint, S.D., Kulandaivelu, G., Teramura, A.H., Tevini, M., 1998. Effects of increased solar ultraviolet radiation on terrestrial ecosystems. Journal of Photochemistry and Photobiology B: Biology 46, 40-52.

Caldwell, M.M., Bornman, J.F., Ballare, C.L., Flint, S.D., Kulandaivelu, G., 2007. Terrestrial ecosystems, increased solar ultraviolet radiation, and interactions with bother climate change factors. Photochemical & Photobiological Sciences 6, 252-266.

Caldwell, M.M., Robberecht, R., Billings, W.D., 1980. A steep latitudinal gradient of solar ultraviolet-B radiation in the arctic-alpine life zone. Ecology 61, 600-611.

Carpenter, S.R., 1996. Microcosm experiments have limited relevance for community and ecosystem ecology. Ecology 77, 677-680.

Castenholz, R.W., 2004. Phototrophic bacteria under UV stress, in: Seckbach, J. (ed.), Origins: Genesis, Evolution and Diversity of Life. Kluwer Academic Publishers, Dordrecht, The Netherlands, pp. 447-461.

Chiariello, N.R., Gulmon, S.L., 1991. Stress effects on plant reproduction, in: Mooney, H.A., Winter, W.E., Pell, E.J. (eds.), Response of plants to multiple stresses. Academic Press, San Diego, pp. 161-188.

Clark, J.S., 1990. Integration of Ecological Levels - Individual Plant-Growth, Population Mortality and Ecosystem Processes. The Journal of Ecology 78, 275-299.

Cockell, C.S., 1998. Biological effects of high ultraviolet radiation on early Earth - a theorethical evaluation. J Theor Biol 193, 717-729.

Cockell, C.S., Horneck, G., 2001. The history of the UV radiation climate of the Earth - theoretical and space-based observations. Photochem Photobiol 73, 447-451.

Cournede, P.H., Kang, M.Z., Mathieu, A., Barczi, J.F., Yan, H.P., Hu, B.G., de Reffye, P., 2006. Structural Factorization of Plants to Compute Their Functional and Architectural Growth. Simulation 82, 427-438.

Crabtree, R.C., Bazzaz, F.A., 1993. Seedling Response of 4 Birch Species to Simulated Nitrogen Deposition - Ammonium Vs Nitrate. Ecol Appl 3, 315-321.

de Lima-Bessa, K.M., Armelini, M.G., Chigancas, V., Jacysyn, J.F., Amarante-Mendes, G.P., Sarasin, A., Menck, C.F., 2008. CPDs and 6-4PPs play different roles in UV-induced cell death in normal and NER-deficient human cells. DNA Repair (Amst) 7, 303-312.

de Reffye, P., Heuvelink, E., Barthélémy, D., Cournède, P.H., 2008. Plant Growth Models, in: Editors-in-Chief: Sven Erik, J., Brian, F. (eds.), Encyclopedia of Ecology. Academic Press, Oxford, pp. 2824-2837.

DeLucia, E.H., Coleman, J.S., Dawson, T.E., Jackson, R.B., 2001. Plant physiological ecology: linking the organism to scales above and below - Ecological Society of America Meeting Snowbird, UT, USA, August 2000. New Phytol 149, 12-16.

Demchik, S.M., Day, T.A., 1996. Effect of enhanced UV-B radiation on pollen quantity, quality, and seed yield in *Brassica rapa* (Brassicaceae). Am J Bot 83, 573-579.

Dillon, J.G., Castenholz, R.W., 1999. Scytonemin, a cyanobacterial sheath pigment, protects against UVC radiation: implications for early photosynthetic life. Journal of Phycology 35, 673-681.

Dixon, R.A., Paiva, N.L., 1995. Stress-Induced Phenylpropanoid Metabolism. Plant Cell 7, 1085-1097.

Enquist, B.J., Niklas, K.J., 2002. Global allocation rules for patterns of biomass partitioning in seed plants. Science 295, 1517-1520.

Farman, J.C., Gardiner, B.G., Shanklin, J.D., 1985. Large losses of total ozone in Antarctica reveal seasonal ClO_x-NO_x interaction. Nature, 207-210.

Feldheim, K., Conner, J.K., 1996. The effects of increased UV-B radiation on growth, pollination success, and lifetime female fitness in two *Brassica* species. Oecologia 106, 284-297.

Fichner, K., Koch, G.W., Mooney, H.A., 1995. The photosynthesis-nitrogen relationship in wild plants, in: Schulze, E.-D., Caldwell, M.M. (eds.), Ecophophysiology of Photosynthesis. Springer-Verlag, Berlin, pp. 133-144.

Gardner, R.H., Urban, D.L., 2003. Model validation and testing: past lessons, present concerns, future prospects, in: Canham, C.D., Cole, J.J., Lauenroth, W.K. (eds.), Models in Ecosystem Science. Princeton University Press, Princeton, NJ, pp. 184-203.

Gayler, S., Grams, T.E.E., Heller, W., Treutter, D., Priesack, E., 2008. A dynamical model of environmental effects on allocation to carbon-based secondary compounds in juvenile trees. Ann Bot-London 101, 1089-1098.

Geber, M.A., Watson, M.A., de Kroon, H., 1997. Organ preformation, development, and resource allocation in perennials, in: Bazzaz, F.A., Grace, J. (eds.), Plant Resource Allocation. Academic Press, San Diego, CA, pp. 113-141.

Gedroc, J.J., McConnaughay, K.D.M., Coleman, J.S., 1996. Plasticity in root shoot partitioning: Optimal, ontogenetic, or both? Funct Ecol 10, 44-50.

Gonzalez, R., Mepsted, R., Wellburn, A.R., Paul, N.D., 1998. Non-photosynthetic mechanisms of growth reduction in pea (*Pisum sativum* L.) exposed to UV-B radiation. Plant Cell Environ 21, 23-32.

González, R., Mepsted, R., Wellburn, A.R., Paul, N.D., 1998. Non-photosynthetic mechanisms of growth reduction in pea (*Pisum sativum* L.) exposed to UV-B radiation. Plant, Cell & Environment 21, 23-32.

Gwynn-Jones, D., 2001. Short-term impacts of enhanced UV-B radiation on photo-assimilate allocation and metabolism: a possible interpretation for time-dependent inhibition of growth. Plant Ecol 154, 65-73.

Haefner, J.W., 2005. Modeling biological systems: principles and applications, 2 ed. Springer Science+Business media, New York, NY.

Hartman, D.L., Wallace, J.M., Limpasuvan, V., Thompson, D.W.J., Holton, J.R., 2000. Can ozone depletion and global warming interact to produce rapid climate change? Proceedings of the National Academy of Sciences 97, 1412-1417.

Hectors, K., Jacques, E., Prinsen, E., Guisez, Y., Verbelen, J.P., Jansen, M.A., Vissenberg, K., 2010. UV radiation reduces epidermal cell expansion in leaves of *Arabidopsis thaliana*. J Exp Bot 61, 4339-4349.

Heilmeier, H., Erhard, M., Schulze, E.-D., 1997. Biomass allocation and water use under arid conditions, in: Bazzaz, F.A., Grace, J. (eds.), Plant Resource Allocation. Academic Press, San Diego, CA, pp. 93-111.

Hoffman, R.W., Campbell, B.D., Bloor, S.J., Swinny, E.E., Markham, K.R., Ryan, K.G., Fountain, D.W., 2003. Responses to UV-B radiation in *Trifolium repens* l. - physiological links to plant productivity and water availability. Plant, Cell and Environment 26, 603-612.

Hopkins, L., Bond, M.A., Tobin, A.K., 2002. Ultraviolet-B radiation reduces the rates of cell division and elongation in the primary leaf of wheat (*Triticum aestivum* L. cv Maris Huntsman). Plant, Cell and Environment 25, 617-624.

Johanson, U., Gehrke, C., Bjorn, L.O., Callaghan, T.V., 1995. The Effects of Enhanced Uv-B Radiation on the Growth of Dwarf Shrubs in a Sub-Arctic Heathland. Funct Ecol 9, 713-719.

Kakani, V.G., Reddy, K.R., Zhao, D., Mohammed, A.R., 2003. Effects of ultraviolet-B radiation on cotton (*Gossypium hirsutum* L.) morphology and anatomy. Ann Bot-London 91, 817-826.

Kattge, J., Diaz, S., Lavorel, S., Prentice, C., Leadley, P., Bonisch, G., Garnier, E., Westoby, M., Reich, P.B., Wright, I.J., Cornelissen, J.H.C., Violle, C., Harrison, S.P., van Bodegom, P.M., Reichstein, M., Enquist, B.J., Soudzilovskaia, N.A., Ackerly, D.D., Anand, M., Atkin, O., Bahn, M., Baker, T.R., Baldocchi, D., Bekker, R., Blanco, C.C., Blonder, B., Bond, W.J., Bradstock, R., Bunker, D.E., Casanoves, F., Cavender-Bares, J., Chambers, J.Q., Chapin, F.S., Chave, J., Coomes, D., Cornwell, W.K., Craine, J.M., Dobrin, B.H., Duarte, L., Durka, W., Elser, J., Esser, G., Estiarte, M., Fagan, W.F., Fang, J., Fernandez-Mendez, F., Fidelis, A., Finegan, B., Flores, O., Ford, H., Frank, D., Freschet, G.T., Fyllas, N.M., Gallagher, R.V., Green, W.A., Gutierrez, A.G., Hickler, T., Higgins, S.I., Hodgson, J.G., Jalili, A., Jansen, S., Joly, C.A., Kerkhoff, A.J., Kirkup, D., Kitajima, K., Kleyer, M., Klotz, S., Knops, J.M.H., Kramer, K., Kuhn, I., Kurokawa, H., Laughlin, D., Lee, T.D., Leishman, M., Lens, F., Lenz, T., Lewis, S.L., Lloyd, J., Llusia, J., Louault, F., Ma, S., Mahecha, M.D., Manning, P., Massad, T., Medlyn, B.E., Messier, J., Moles, A.T., Muller, S.C., Nadrowski, K., Naeem, S., Niinemets, U., Nollert, S., Nuske, A., Ogaya, R., Oleksyn, J., Onipchenko, V.G., Onoda, Y.,

Ordonez, J., Overbeck, G., Ozinga, W.A., Patino, S., Paula, S., Pausas, J.G., Penuelas, J., Phillips, O.L., Pillar, V., Poorter, H., Poorter, L., Poschlod, P., Prinzing, A., Proulx, R., Rammig, A., Reinsch, S., Reu, B., Sack, L., Salgado-Negre, B., Sardans, J., Shiodera, S., Shipley, B., Siefert, A., Sosinski, E., Soussana, J.F., Swaine, E., Swenson, N., Thompson, K., Thornton, P., Waldram, M., Weiher, E., White, M., White, S., Wright, S.J., Yguel, B., Zaehle, S., Zanne, A.E., Wirth, C., 2011. TRY - a global database of plant traits. Global Change Biol 17, 2905-2935.

Kenrick, P., Crane, P.R., 1997. The origin and early evolution of plants on land. Nature 389, 33-39.

Kerkhoff, A.J., Enquist, B.J., Elser, J.J., Fagan, W.F., 2005. Plant allometry, stoichiometry and the temperature-dependence of primary productivity. Global Ecology and Biogeography 14, 585-598.

Kikuzawa, K., 1991. A Cost-Benefit-Analysis of Leaf Habit and Leaf Longevity of Trees and Their Geographical Pattern. Am Nat 138, 1250-1263.

King, D., Roughgarden, J., 1982a. Graded Allocation between Vegetative and Reproductive Growth for Annual Plants in Growing Seasons of Random Length. Theor Popul Biol 22, 1-16.

King, D., Roughgarden, J., 1982b. Multiple Switches between Vegetative and Reproductive Growth in Annual Plants. Theor Popul Biol 21, 194-204.

Kitajima, K., Mulkey, S.S., Samaniego, M., Joseph Wright, S., 2002. Decline of photosynthetic capacity with leaf age and position in two tropical pioneer tree species. Am J Bot 89, 1925-1932.

Koes, R.E., Quattrocchio, F., Mol, J.N.M., 1994. The Flavonoid Biosynthetic-Pathway in Plants - Function and Evolution. Bioessays 16, 123-132.

Korner, C., Scheel, J.A., Bauer, H., 1979. Maximum Leaf Diffusive Conductance in Vascular Plants. Photosynthetica 13, 45-82.

Koti, S., Reddy, K.R., Kakani, V.G., Zhao, D., Gao, W., 2007. Effects of carbon dioxide, temperature and ultraviolet-B radiation and their interactions on soybean (*Glycine max* L.) growth and development. Environ Exp Bot 60, 1-10.

Koti, S., Reddy, K.R., Reddy, V.R., Kakani, V.G., Zhao, D., 2005. Interactive effects of carbon dioxide, temperature, and ultraviolet-B radiation on soybean (*Glycine ma*x L.) flower and pollen morphology, pollen production, germination, and tube lengths. J Exp Bot 56, 725-736.

Kotilainen, T., Venäläinen, T., Tegelberg, R., Lindfors, A., Julkunen-Tiitto, R., Sutinen, S., O'Hara, R.B., Aphalo, P.J., 2009. Assessment of UV Biological Spectral Weighting Functions for Phenolic Metabolites and Growth Responses in Silver Birch Seedlings. Photochem Photobiol 85, 1346-1355.

Lambers, H., Robinson, S.A., Ribas-Carbo, M., 2005. Regulation of respiration in vivo, in: Lambers, H., Ribas-Carbo, M. (eds.), Plant Respiration: From Cell to Ecosystem. Springer, The Netherlands, pp. 1-15.

Larcher, W., 2003. Physiological plant ecology, 4 ed. Springer-Verlag, Berlin 513 pp.

Lavola, A., Aphalo, P.J., Lahti, M., Julkunen-Tiitto, R., 2003. Nutrient availability and the effect of increasing UV-B radiation on secondary plant compounds in Scots pine. Environ Exp Bot 49, 49-60.

Lee, T.D., Bazzaz, F.A., 1982. Regulation of Fruit Maturation Pattern in an Annual Legume, Cassia-Fasciculata. Ecology 63, 1374-1388.

Lee, T.D., Bazzaz, F.A., 1986. Maternal Regulation of Fecundity - Nonrandom Ovule Abortion in Cassia-Fasciculata Michx. Oecologia 68, 459-465.

Levin, S.A., 1992. The problem of pattern and scale in ecology. Ecology 73, 1943-1967.

Li, F.-R., Peng, S.-L., Chen, B.-M., Hou, Y.-P., 2010. A meta-analysis of the responses of woody and herbaceous plants to elevated ultraviolet-B radiation. Acta Oecologica 36, 1-9.

Li, J., Ou-Lee, T., Raba, R., Admundson, R.G., Last, R.L., 1993. Arabidopsis flavonoids mutants are hypersensitive to UV-B irradiation. The Plant Cell 5, 171-179.

Lo, H.L., Nakajima, S., Ma, L., Walter, B., Yasui, A., Ethell, D.W., Owen, L.B., 2005. Differential biologic effects of CPD and 6-4PP UV-induced DNA damage on the induction of apoptosis and cell-cycle arrest. BMC Cancer 5, 135.

Loveys, B.R., Atkinson, L.J., Sherlock, D.J., Roberts, R.L., Fitter, A.H., Atkin, O.K., 2003. Thermal acclimation of leaf and root respiration: an investigation comparing inherently fast- and slow-growing plant species. Global Change Biol 9, 895-910.

Lowry, B., Lee, D., Hebant, C., 1980. The origins of land plants: a new look at an old problem. Taxon 29, 183-197.

Margulis, L., Walker, J.C.G., Rambler, M., 1976. Reassessment of Roles of Oxygen and Ultraviolet-Light in Precambrian Evolution. Nature 264, 620-624.

Marshall, D.L., Ellstrand, N.C., 1988. Effective Mate Choice in Wild Radish - Evidence for Selective Seed Abortion and Its Mechanism. Am Nat 131, 739-756.

Mathieu, A., Cournede, P.H., Letort, V., Barthelemy, D., de Reffye, P., 2009. A dynamic model of plant growth with interactions between development and functional mechanisms to study plant structural plasticity related to trophic competition. Ann Bot 103, 1173-1186.

Mc Donald, M.S., 2003. Photobiology of higher plants. John Wiley & Sons, England 354 pp.

Miao, S.L., Bazzaz, F.A., Primack, R.B., 1991. Effects of Maternal Nutrient Pulse on Reproduction of Two Colonizing Plantago Species. Ecology 72, 586-596.

Milchunas, D.G., King, J.Y., Mosier, A.R., Moore, J.C., Morgan, J.A., Quirk, M.H., Slusser, J.R., 2004. UV radiation effects on plant growth and forage quality in a shortgrass steppe Ecosystem. Photochem Photobiol 79, 404-410.

Molina, M.J., Rowland, F.S., 1974. Startospheric sink for chlorofluoromethanes: chlorine atomc-atalyzed destruction of ozone. Nature 249, 810-812.

Niinemets, U., 2010. Responses of forest trees to single and multiple environmental stresses from seedlings to mature plants: Past stress history, stress interactions, tolerance and acclimation. Forest Ecol Manag 260, 1623-1639.

Niklas, K.J., Enquist, B.J., 2002a. Canonical rules for plant organ biomass partitioning and annual allocation. Am J Bot 89, 812-819.

Niklas, K.J., Enquist, B.J., 2002b. On the vegetative biomass partitioning of seed plant leaves, stems, and roots. Am Nat 159, 482-497.

Nogués, S., Baker, N.R., 2000. Effects of drought on photosynthesis in Mediterranean plants grown under enhanced UV-B radiation. J Exp Bot 51, 1309-1317.

Nygren, P., Pallardy, S.G., 2008. Applying a universal scaling model to vascular allometry in a single-Stemmed, monopodially branching deciduous tree (Attim's model). Tree Physiology 28, 1-10.

Plentinger, M.C., Penning de Vries, F.W.T. (eds.) 1996. CAMASE register of agro-ecosystems models, http://library.wur.nl/way/bestanden/clc/1763788.pdf electronic ed 420 pp.

Poorter, H., Niinemets, U., Poorter, L., Wright, I.J., Villar, R., 2009. Causes and consequences of variation in leaf mass per area (LMA): a meta-analysis. New Phytol 182, 565-588.

Poorter, H., Remkes, C., 1990. Leaf-Area Ratio and Net Assimilation Rate of 24 Wild-Species Differing in Relative Growth-Rate. Oecologia 83, 553-559.

Poorter, H., Vanderwerf, A., Atkin, O.K., Lambers, H., 1991. Respiratory Energy-Requirements of Roots Vary with the Potential Growth-Rate of a Plant-Species. Physiol Plantarum 83, 469-475.

Poorter, H., Villar, R., 1997. Fate of acquired carbon in plants: chemical composition and construction costs, in: Bazzaz, F.A., Grace, J. (eds.), Plant Resource Allocation. Academic Press, San Diego, CA, pp. 39-72.

Putterill, J., Laurie, R., Macknight, R., 2004. It's time to flower: the genetic control of flowering time. Bioessays 26, 363-373.

Reekie, E.G., Bazzaz, F.A., 1987. Reproductive Effort in Plants .1. Carbon Allocation to Reproduction. Am Nat 129, 876-896.

Reich, P.B., Tjoelker, M.G., Pregitzer, K.S., Wright, I.J., Oleksyn, J., Machado, J.L., 2008. Scaling of respiration to nitrogen in leaves, stems and roots of higher land plants. Ecol Lett 11, 793-801.

Rettberg, P., Horneck, G., Strauch, W., Facius, R., Seckmeyer, G., 1998. Simulation of planetary UV radiation climate on the example of the early Earth. Advances in Space Research 22, 335-339.

Robberecht, R., Caldwell, M.M., Billings, W.D., 1980. Leaf ultraviolet optical properties along a latitudinal gradient in the arctic-alpine life zone. Ecology 61, 612-619.

Rousseaux, M.C., Flint, S.D., Searles, P.S., Caldwell, M.M., 2004. Plant responses to current solar ultraviolet-B radiation and to supplemented solar ultraviolet-B radiation simulating ozone depletion: an experimental comparison. Photochem Photobiol 80, 224-230.

Rozema, J., 1999. UV-B radiation and terrestrial ecosystems: processes, structure and feedback loops, in: Rozema, J. (ed.), Stratospheric Ozone Depletion: The Effects of Enhanced UV-B Radiation on Terrestrial Ecosystems. Backhuys Publishers, Leiden, The Netherlands, pp. 101-116.

Rozema, J., van der Staaij, J.W.M., Tosserams, M., 1997. Effects of UV-B radiation on plants from agro- and natural ecosystems, in: Lumsden, P.J. (ed.), Plants and UV-B: responses to environmental change. Cambridge University Press, Cambridge, UK, pp. 213-232.

Rykiel, J.E.J., 1996. Testing ecological models: the meaning of validation. Ecol Model 90, 229.

Sagan, C., 1973. Ultraviolet Selection Pressure on Earliest Organisms. J Theor Biol 39, 195-200.

Sancar, A., 1994. Structure and function of DNA photolyase. Biochemistry-Us 33, 2-9.

Sancar, A., Sancar, G.B., 1988. DNA-Repair Enzymes. Annu Rev Biochem 57, 29-67.

Schindler, D.W., 1998. Whole-Ecosystem Experiments: Replication Versus Realism: The Need for Ecosystem-Scale Experiments. Ecosystems 1, 323-334.

Schmelzer, E., Jahnen, W., Hahlbrock, K., 1988. In situ localization of light-induced chalcone synthase mRNA, chalcone synthase, and flavonoid end products in epidermal cells of parsley leaves. P Natl Acad Sci USA 85, 2989-2993.

Schmid, B., Bazzaz, F.A., Weiner, J., 1995. Size Dependency of Sexual Reproduction and of Clonal Growth in 2 Perennial Plants. Can J Bot 73, 1831-1837.

Schulze, W., Schulze, E.-D., 1995. The significance of assimilatory starch for growth in *Arabidopsis thaliana* wild-type and starchless mutants, in: Schulze, E.-D., Caldwell, M.M. (eds.), Ecophophysiology of Photosynthesis. Springer-Verlag, Berlin, pp. 123-131.

Searles, P.S., Flint, S.D., Caldwell, M.M., 2001. A meta analysis of plant field studies simulating stratospheric ozone depletion. Oecologia 127, 1-10.

Shugart, H.H., 1984. A Theory on Forest Dynamics. The Ecological Implications of Forest Succesion Models. Springer-Verlag, New York, NY 278 pp.

Smith, B.N., 2005. Photosynthesis, respiration, and growth, in: Pessarakli, M. (ed.), Handbook of Photosynthesis. 2 ed. Taylor & Francis Group, Boca Raton, FL, pp. 671-677.

Stafford, H.A., 1991. Flavonoid Evolution - an Enzymatic Approach. Plant Physiol 96, 680-685.

Suchar, V.A., Robberecht, R., 2015. Integration and scaling of UV-B radiation effects on plants: from DNA to leaf. Ecology and Evolution, (in press).

Sullivan, J., Rozema, J., 1999. UV-B effects on terrestrial plant growth and photosynthesis, in: Rozema, J. (ed.), Stratospheric ozone depletion: the effects of enhanced UV-B radiation on terrestrial ecosystems. Backhuys Publishers Leiden, The Netherlands, pp. 39-58.

Systems, V., 2009. Vensim: Ventana Simulation Environment, 5.6 ed, http://www.vensim.com.

Tay, A., Abdullah, A., Awang, M., Furukawa, A., 2007. Midday depression of photosynthesis in *Enkleia malaccensis* , a woody climber in a tropical rainforest. Photosynthetica 45, 189-193.

Taylor, R.M., Tobin, A.K., Bray, C.M., 1997. DNA damage and repair in plants, in: Lumsden, P.J. (ed.), Plants and UV-B Responses to Environmental Change. Cambridge University Press, Cambridge, UK, pp. 53-76.

United Nations Environment Programme, E.E.A.P., 2012. Environmental effects of ozone depletion and its interactions with climate change: progress report, 2011. Photochemical & Photobiological Sciences 11, 13-27.

United Nations Environment Programme, 1998. Environmental Effects of Ozone Depletion: 1998 Update.

Wargent, J.J., Gegas, V.C., Jenkins, G.I., Doonan, J.H., Paul, N.D., 2009a. UVR8 in *Arabidopsis thaliana* regulates multiple aspects of cellular differentiation during leaf development in response to ultraviolet B radiation. New Phytol 183, 315-326.

Wargent, J.J., Moore, J.P., Roland Ennos, A., Paul, N.D., 2009b. Ultraviolet Radiation as a Limiting Factor in Leaf Expansion and Development. Photochem Photobiol 85, 279-286.

Warren, J.M., Bassman, J.H., Eigenbrode, S., 2002. Leaf chemical changes induced in *Populus trichocarpa* by enhanced UV-B radiation and concomitant effects on herbivory by *Chrysomela scripta* (Coleoptera: Chrysomidae). Tree Physiol 22, 1137-1146.

Wayne, P.M., Bazzaz, F.A., 1993. Morning vs afternoon sun patches in experimental forest gaps: consequences of temporal incongruency of resources to birch regeneration. Oecologia 94, 235-243.

Weber, S., 2005. Light-driven enzymatic catalysis of DNA repair: a review of recent biophysical studies on photolyase. Bba-Bioenergetics 1707, 1-23.

Weiner, J., 1995. On the Practice of Ecology. J Ecol 83, 153-158.

Weiner, J., 2004. Allocation, plasticity and allometry in plants. Perspectives in Plant Ecology, Evolution and Systematics 6, 207-215.

Winkel-Shirley, B., 2002. Biosynthesis of flavonoids and effects of stress. Curr Opin Plant Biol 5, 218-223.

Wright, I.J., Reich, P.B., Westoby, M., Ackerly, D.D., Baruch, Z., Bongers, F., Cavender-Bares, J., Chapin, T., Cornelissen, J.H.C., Diemer, M., Flexas, J., Garnier, E., Groom, P.K., Gulias, J., Hikosaka, K., Lamont, B.B., Lee, T., Lee, W., Lusk, C., Midgley, J.J., Navas, M.L., Niinemets, U., Oleksyn, J., Osada, N., Poorter, H., Poot, P., Prior, L., Pyankov, V.I., Roumet, C., Thomas, S.C., Tjoelker, M.G., Veneklaas, E.J., Villar, R., 2004. The worldwide leaf economics spectrum. Nature 428, 821-827.

Xu, D.-Q., Shen, Y.-K., 2005. External and internal factors responsible for midday depression of photosynthesis, in: Pessarakli, M. (ed.), Handbook of Photosynthesis. 2 ed. Taylor & Francis Group, Boca Raton, FL, pp. 287-297.

Yamasaki, S., Noguchi, N., Mimaki, K., 2007. Continuous UV-B irradiation induces morphological changes and the accumulation of polyphenolic compounds on the surface of cucumber cotyledons. J Radiat Res 48, 443-454.

Zhou, Y.H., Lam, H.M., Zhang, J.H., 2007. Inhibition of photosynthesis and energy dissipation induced by water and high light stresses in rice. J Exp Bot 58, 1207-1217.

Ziska, L.H., Teramura, A.H., Sullivan, J.H., 1992. Physiological Sensitivity of Plants Along an Elevational Gradient to Uv-B Radiation. Am J Bot 79, 863-871.

Table 2.1: Summary of the model parameters estimators

Parameter	Definition	Unit	Range	Assigned values*
Total mass production				
1 k_1	plant mass photosynthetic production rate multiplier	hour^{-1}	See eq. 7	
2 k_2	total leaf area – total plant mass multiplier	g m^{-2}	60.24	
3 b_{pd}	slope of the linear photosynthetic capacity decline	% hour^{-1}	0.004	
Respiration				
4 k_3	plant mass respiration rate multiplier	hour^{-1}	0.0025 – 0.0045	
Plant organs growth				
5 $k_{4,R}$	conversion efficiency in root biomass of photosynthetic production	unitless	0.75	
6 $k_{5,R}$	percent of total photosynthetic production allocated to roots growth	unitless	0.12	
7 $k_{4,S}$	conversion efficiency in structural organs biomass of photosynthetic production	unitless	0.69	
8 $k_{5,S}$	percent of total photosynthetic production allocated to structural organs growth	unitless	0.57	
9 $k_{4,L}$	conversion efficiency in leaf biomass of photosynthetic production	unitless	0.67	
10 $k_{5,L}$	percent of total photosynthetic production allocated to leaf growth	unitless	0.31	
11 $k_{4,RO}$	conversion efficiency in reproductive organs biomass of photosynthetic production	unitless	0.71	
12 $k_{5,RO}$	percent of total photosynthetic production allocated to reproductive organs growth	unitless	0-1	
13 t_{br}	time triggering reproduction	hour	2160	
14 a_t	linear increase in photosynthetic production allocation to reproductive parts	hour^{-1}	0.0004	
15 $k_{4,Sd}$	conversion efficiency in seed biomass of photosynthetic production	unitless	0.65	
16 $k_{5,Sd}$	percent of total photosynthetic production allocated to seed growth	unitless	0-1	
17 Δt_r	interval necessary for reproduction	hour	168-336	
UV-B radiation effects on whole plant growth and development				
18 $k_{1,UVB}$	adjustment factor due to the effects of UV-B radiation on photosynthesis	unitless	0.75-1	1
19 $k_{2,UVB}$	adjustment factor due to effects of UV-B radiation on leaf thickness	unitless	0.75-1.25	1
20 $k_{3,UVB}$	adjustment factor due to the effects of UV-B radiation on metabolic processes	unitless	1-4	1.0125|1.025
21 $k_{4,L,UVB}$	conversion efficiency in leaf biomass of photosynthetic production under increased UV-B radiation	unitless	0.66	
22 $k_{6,UVB}$	adjustment factor due to the effects of UV-B radiation on leaf expansion	Suchar and Robberecht 2015		

*where appropriate

[a]leaf senescence coefficients were chosen to model identical trends as leaf growth processes, and timed for the ending of the growing season considered

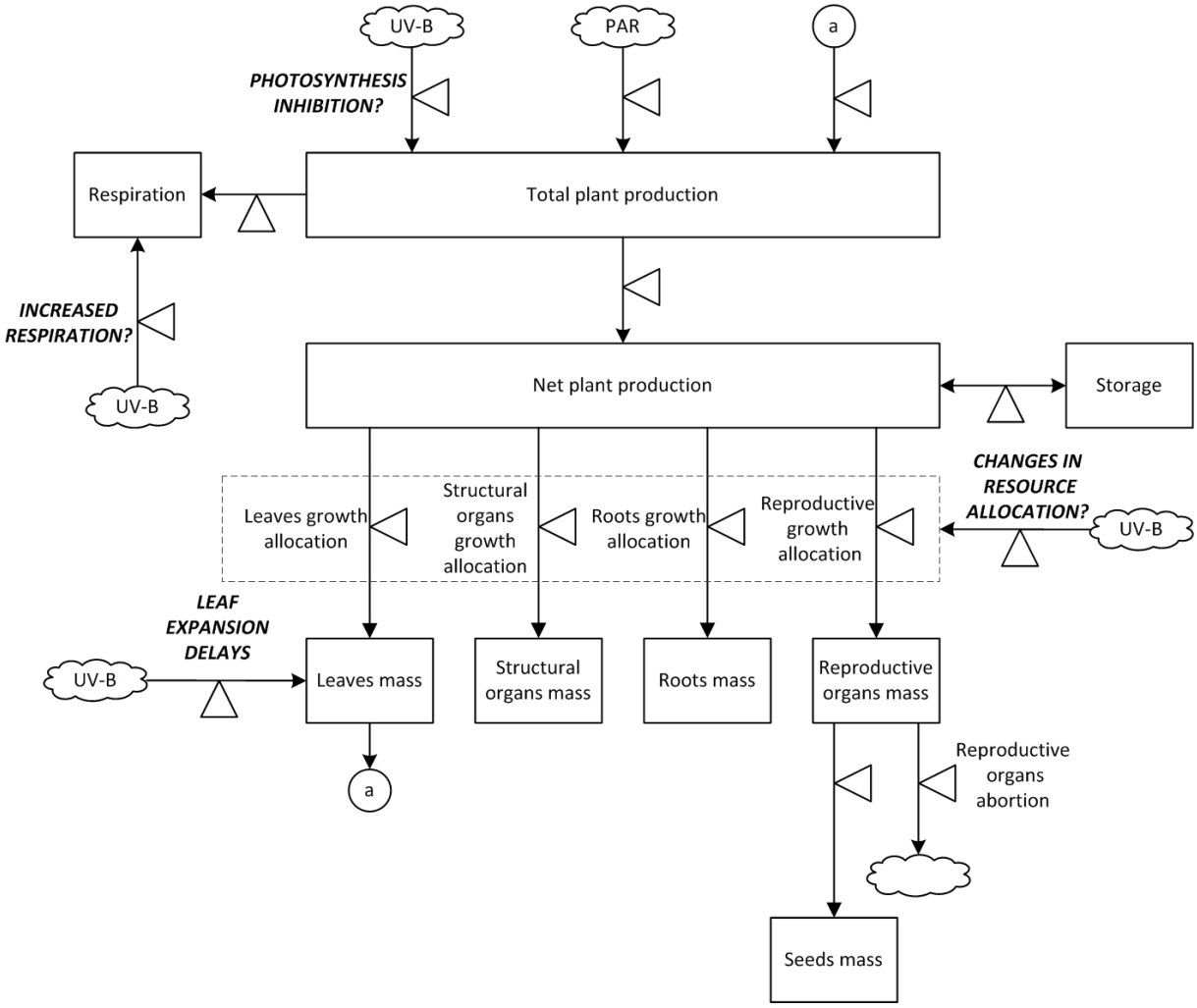

Figure 2.1: Conceptual model of UV-B radiation effects on the whole plant.

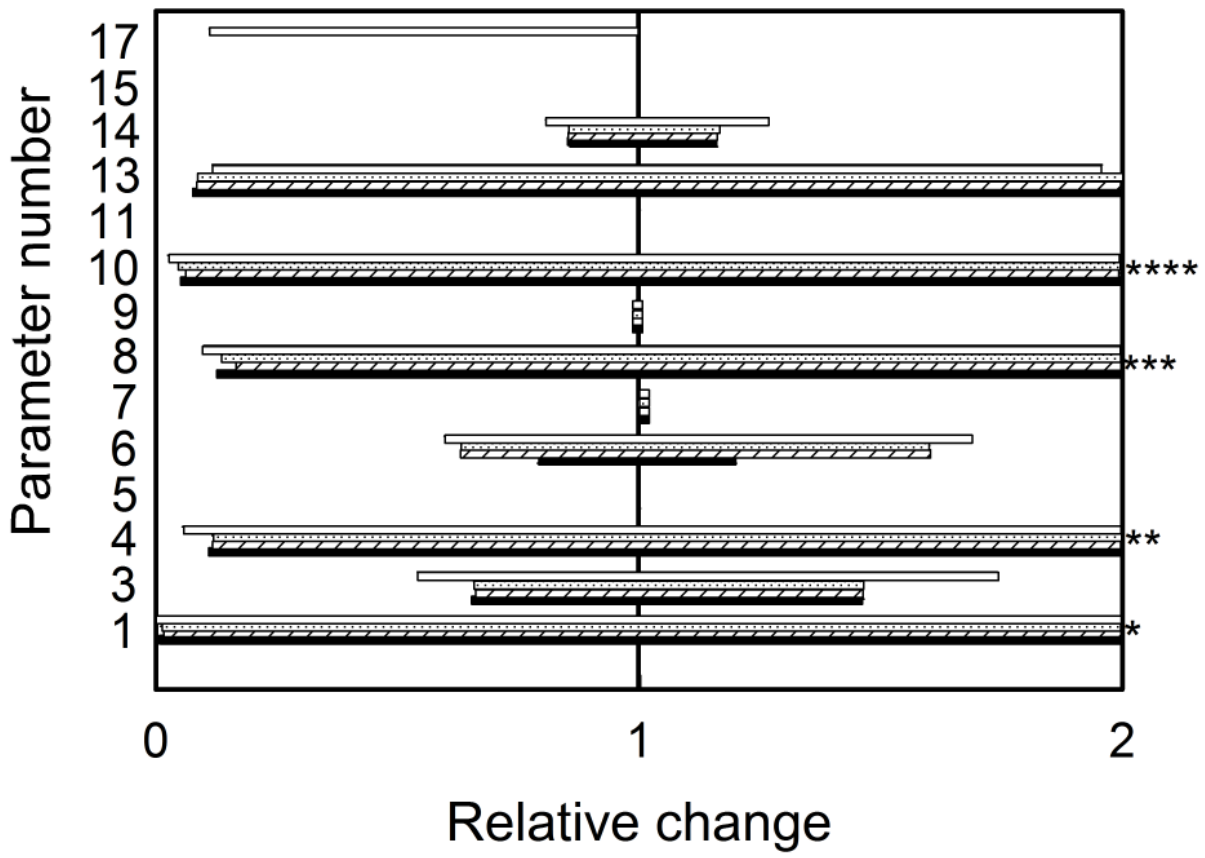

Figure 2.2: Sensitivity analysis: relative change in roots (solid bar), structural organs (right dash bar), leaves (dotted bar), and mature seeds biomass (no fill bar).

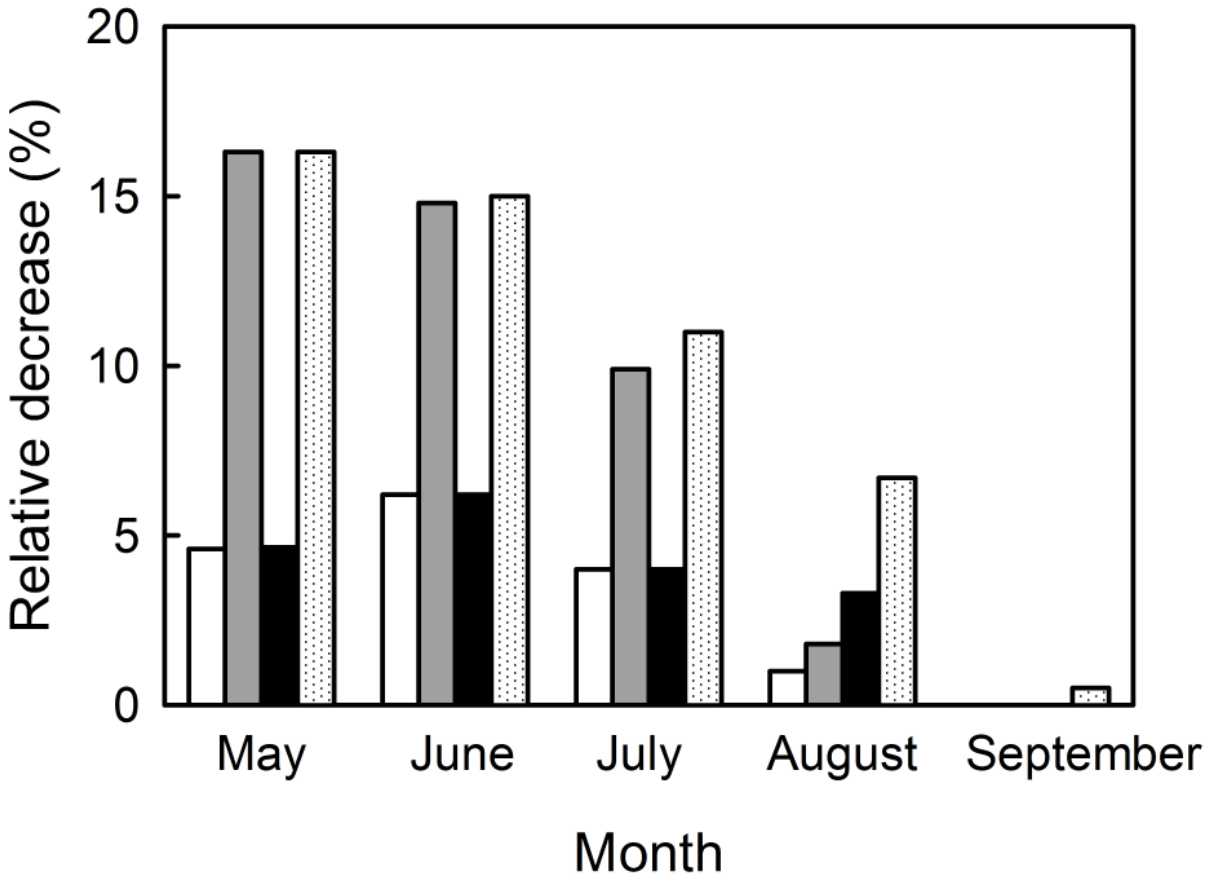

Figure 2.3: The effect of timing of the increased UV-B radiation event: relative vegetative and mature seeds biomass decrease for plants exposed to increased UV-B radiation in May, June, July, August, and September (vegetative biomass-150% UV-B (no fill bar), vegetative biomass-200% UV-B (gray bar), mature seeds biomass-150% UV-B (black bar), mature seeds biomass-200% UV-B (dotted bar)).

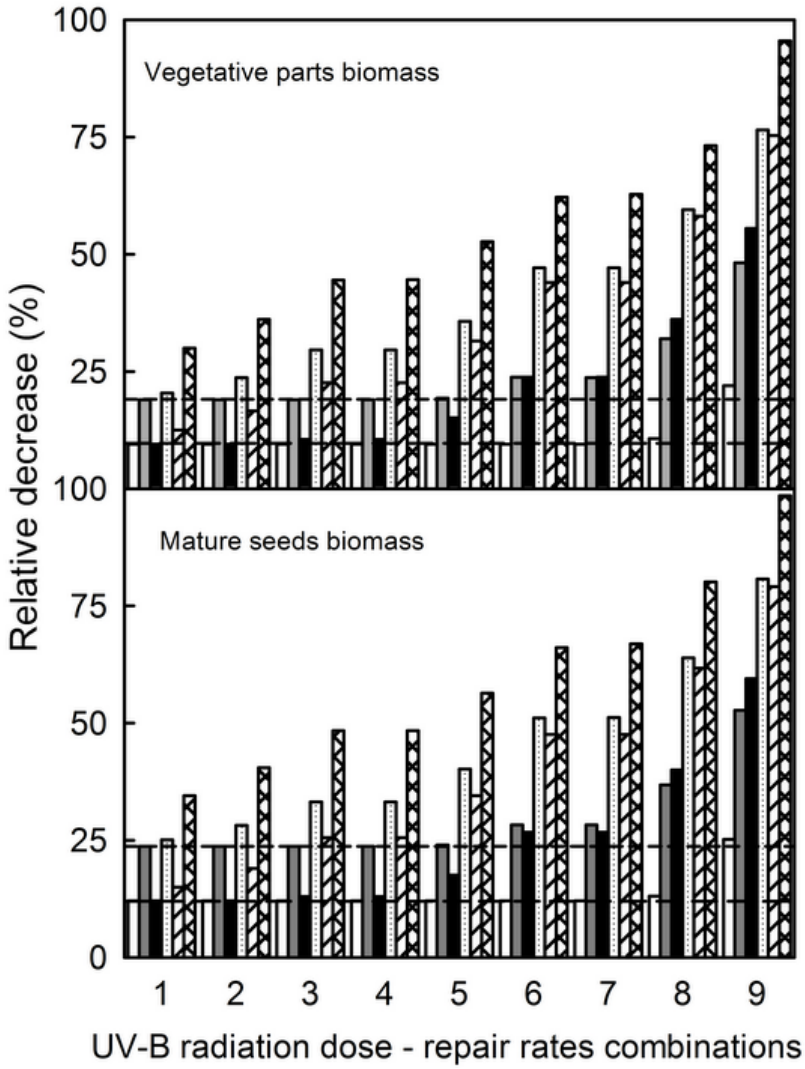

Figure 2.4: Effect of increased UV-B radiation - repair rates combinations (1 = high CPD photorepair rate (HP) – high CPD dark repair rate (HD), 2 = HP – average CPD dark repair rate (AD), 3 = HP – low CPD dark repair rate (LD), 4 = average CPD photorepair rate (AP) – HD, 5 = AP – AD, 6 = AP - LD, 7 = low CPD photorepair rate (LP) – HD, 8 = LP – AD, and 9 = LP – LD), relative epidermal absorptance (relative high absorptance at short UV-B radiation wavelengths: 1.5X UV-B (no fill bar) and 2X UV-B (gray bar), equal absorptance at all UV-B radiation wavelengths: 1.5X UV-B (black bar) and 2X UV-B (dotted bar), and relative high absorptance at long UV-B radiation wavelengths: 1.5X UV-B (left dash bar) and 200% UV-B (crisscross bar). Horizontal lines: the relative decrease in plant growth due to increased metabolism at 1.5X UV-B (long dash line) and 2X UV-B (medium dash line).

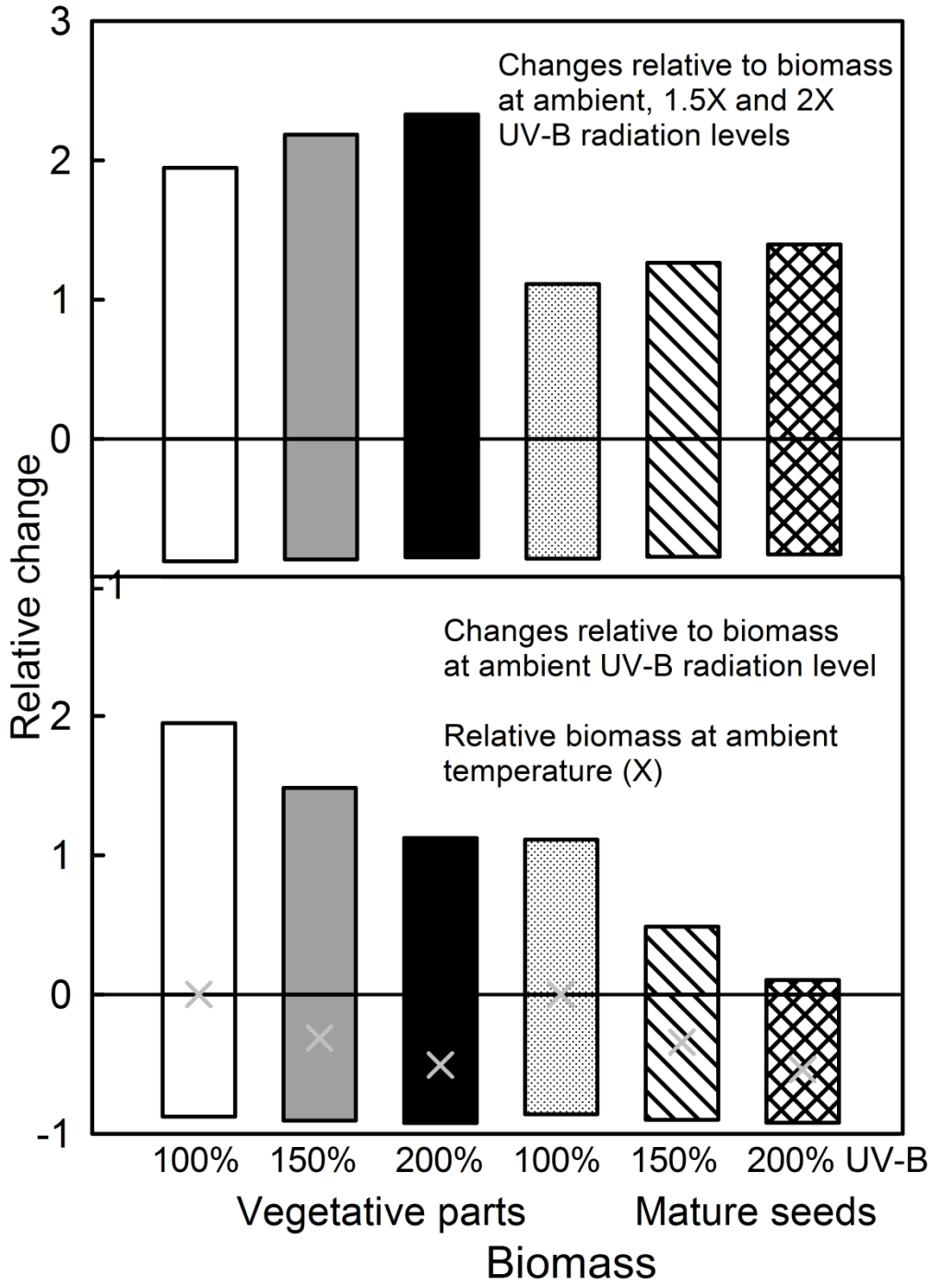

Figure 2.5: The effect of temperature on growth: relative growth under ambient, −5℃ and+5℃ temperatures.

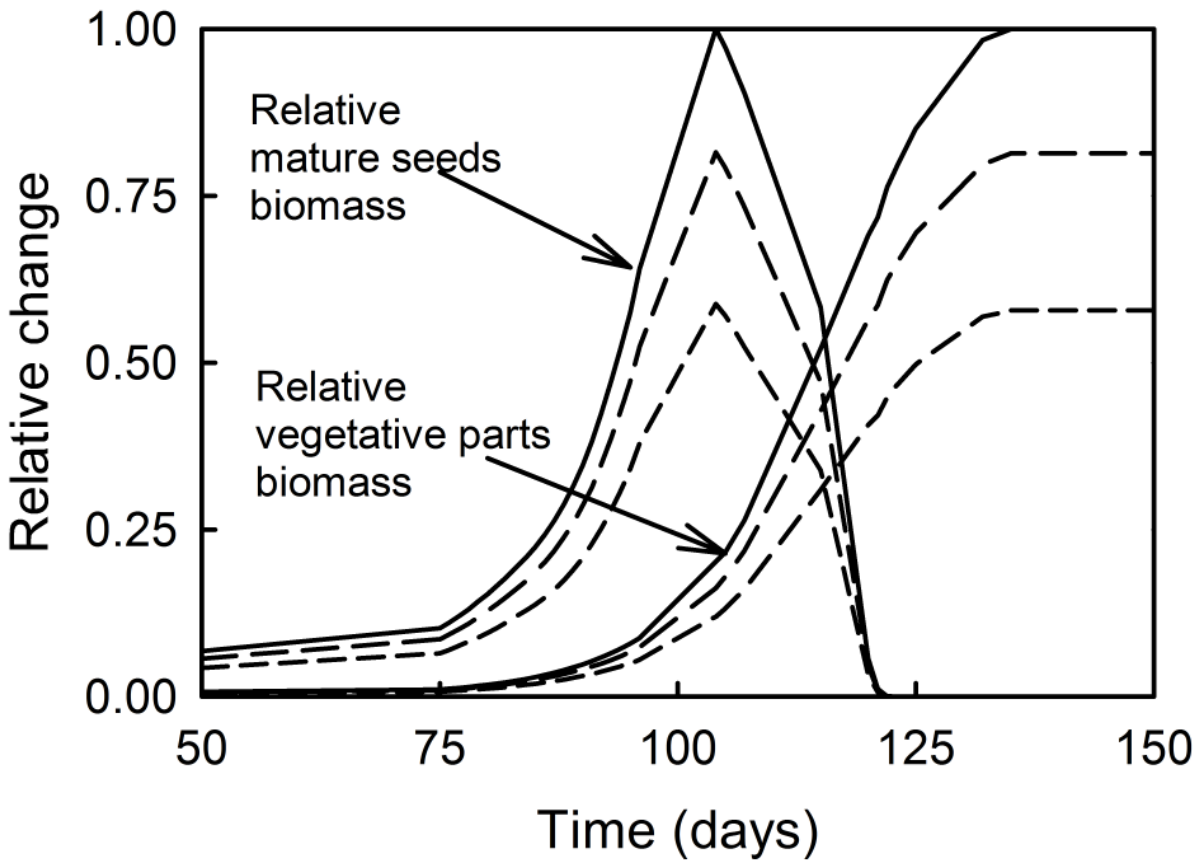

Figure 2.6: The effect of reproduction timing on maximum vegetative and mature seeds biomass for plants exposed to 100% UV-B (solid line), 150% UV-B (long dash line), and 200% UV-B (medium dash line) radiation.

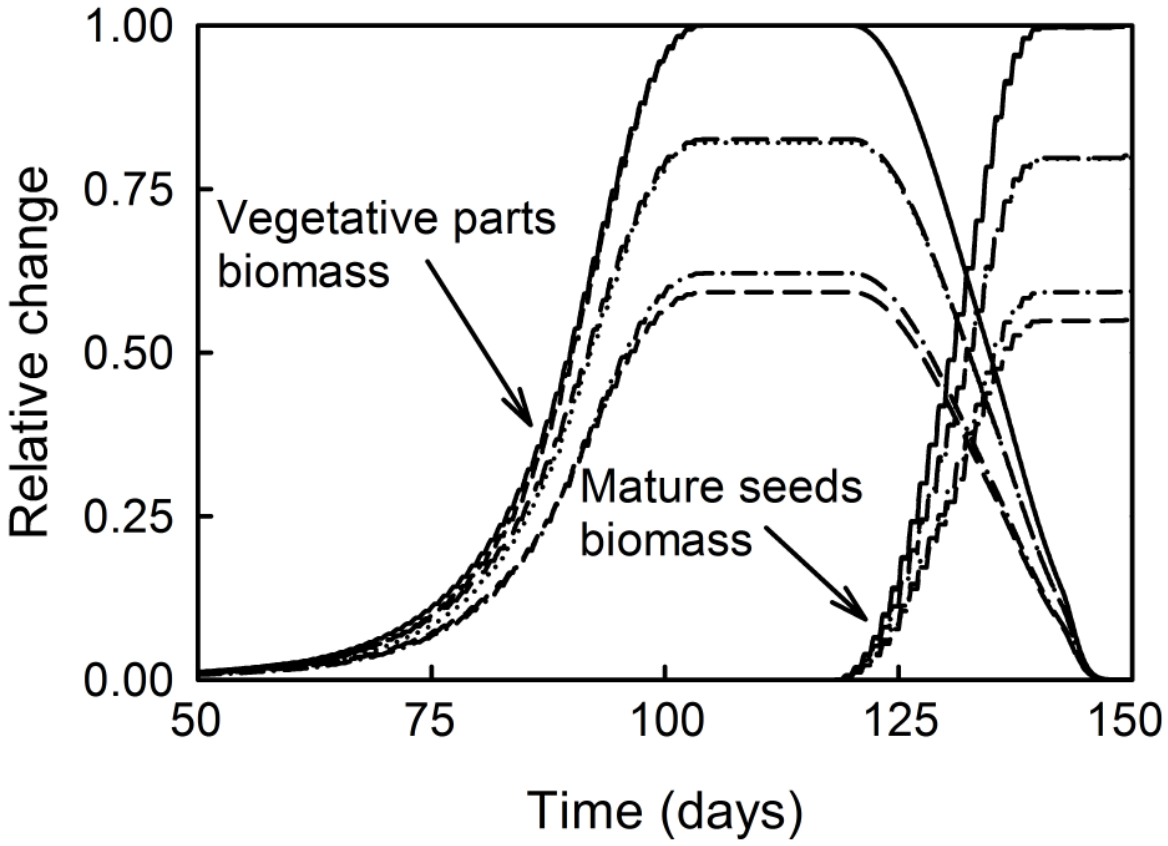

Figure 2.7: The effect of midday photosynthetic depression on maximum vegetative and mature seeds biomass. Plants without midday depression and exposed to 100% (solid line), 150% (long dash line) and 200% (medium dash line) UV-B radiation. Plants with midday depression and exposed to 100% (short dash line), 150% (dotted line) and 200% (dot and dash line) UV-B radiation.

Chapter 3

Integration and scaling of UV-B radiation effects on plants: the relative sensitivity of growth forms and community interactions

submitted to Ecological Modelling

3.1 Abstract

Our model is the first to integrate the effects of increased UV-B radiation from molecular level processes, whole plant growth and development, and community interactions. In the model simulations, species types exhibited different levels of sensitivity to increased UV-B radiation. Summer C3 and C4 annuals showed similar growth inhibition rates, while biennials and winter C3 annuals were the most sensitive. Perennials exhibited inhibitions in growth only if increased UV-B radiation results in increases in metabolic rates. In communities, species sensitive to UV-B radiation may have a competitive disadvantage compared to resistant plant species. But, sensitive species may have a wide array of responses that can increase their fitness and reproductive success in the community, such as, increased secondary metabolites production, changes in timing of emergence and reproduction, changes in seed size. While individual plants may exhibit significant inhibitions in growth and development, in communities, these inhibitions can be mitigated by small morphological and physiological adaptations. Infrequent or occasional increased UV-B radiation events should not have any lasting effect on the structure of the community, unless other environmental factors are perturbing the dynamic equilibrium.

3.2 Introduction

As an inherent natural environmental stress factor for organisms (Cockell and Horneck, 2001; Lowry et al., 1980; Rettberg et al., 1998), UV-B radiation can induce injuries to DNA, cause DNA mutations, inhibit photosynthetic processes, impair membrane function, and cause lethal cell damage (Britt, 1996; Rozema et al., 1999; Sancar and Sancar, 1988; Taylor et al., 1997; Weber, 2005). It may have been the catalysts for phylogenetic diversity through accelerated selection and evolution (Cockell, 2000; Sagan, 1973), and, at least partly,

responsible for the success of terrestrial plant species (Lowry et al., 1980; Rozema et al., 1999; Stafford, 1991).

Ultraviolet-B radiation-induced plant species changes may cascade across higher ecological scales and trophic levels (Bornman et al., 2015; Day and Neale, 2002; Searles et al., 2001), and interact with global warming effects (Andrady et al., 2015; Hartmann et al., 2000). Thus, understanding the effects of UV radiation environment of Earth may provide insights in its past contributions as a selection agent, and current and future influences across terrestrial communities, and towards global change.

Because of technological difficulties in simulating ambient and enhanced UV-B radiation environment due to stratospheric ozone depletion on a broad scale, experimental research on UV radiation effects on plants has been mostly limited to individual and sub-individual plant levels (DeLucia et al., 2001). A modeling research approach, which integrates and scales the effects of enhanced UV-B radiation on terrestrial plant communities, was therefore used to understand plant response mechanisms to UV-B radiation and their broader consequences, identify the processes insufficiently addressed by past research, as well as to investigate hypotheses that were untestable by experimental research.

We examined the relative plant type sensitivity to increased UV-B radiation and selected community interactions within a two annual species system with one sensitive to UV-B radiation species, and one resistant. The model follows previous work that modeled the molecular effects of increased UV-B radiation, the cellular responses to the molecular effects, and their potential consequences on leaf growth and development (Suchar and Robberecht, 2015b), and examined their consequences to whole plant growth and development (Suchar and Robberecht, 2015a). Our model integrated the molecular, organ, and whole plant processes, and showed how they can affect the competitive balances in plant communities. We were able to examine a variety of questions that were difficult to approach through experimental research, including: (1) are there differences among growth forms in regard to their sensitivity to UV-B radiation, (2) what are the changes in plant competitive balance in a community under increased UV-B radiation, and (3) what are the response mechanisms of the UV-B radiation-sensitive species that might keep them competitive in the plant community?

3.3 Model Framework

We previously modeled the function of an individual plant by integrating photosynthetic production, respiration, and resource allocation (Suchar and Robberecht, 2015a). Our research modeled the whole plant photosynthetic fixed carbon resource allocation towards short- or long- term carbohydrate plant needs (Smith, 2005), following a suggested similar isometric scale across many seed plant species (Enquist and Niklas, 2002; Niklas and Enquist, 2002b). To scale the effects of UV-B radiation in a plant, the whole plant model was augmented with the molecular effects of increased UV-B radiation, the cellular responses to the molecular effects, and their potential consequences on leaf growth and development (Suchar and Robberecht, 2015b).

To answer our research questions, first we modified the model rates for the plant growth forms considered (C3 and C4 summer annuals, C3 winter annuals, C3 biennials, C3 deciduous and evergreen perennials). Next, we considered a two C3 annual plant species system for community interactions. We recognize that communities have more than two species, but since we modeled generalized plants species with averaged parameter estimators, the focus of the model is to understand the dynamics of the species with relative different sensitivity to UV-B radiation, rather than predict the community structure. If intended, the models can be extended to more than two species, but, in the absence of species-specific parameter estimators, this may not lead to any additional conclusions.

Two species competition models suggest a few principles required for species to coexist. The growth of plant species can be limited by abiotic and biotic factors. In communities, species with higher growth rates under limiting conditions may competitively exclude the other species (Amarasekare, 2003; Holt et al., 1994). In order for two species to coexist, they have to interact differently with the external factors, resulting in a niche differentiation in which intraspecific competition is stronger than interspecific competition (Amarasekare, 2003; Chesson, 2000b). This differentiation can be achieved by specializing on different resources or different levels of resources, or if species avoid temporal and spatial interspecific competition conditions (Amarasekare, 2003; Chesson, 2000a, b; Levine and HilleRisLambers, 2009). Also, competition during seed emergence is based on seed size, while competition for light is a function of biomass and density (Levine and HilleRisLambers, 2009; Levine and Rees, 2002, 2004). However, it is possible that natural selection can result

in coexistence in situations where ecological dynamics may lead to competitive exclusion (Vasseur et al., 2011). Therefore, instead of focusing on temporal and spatial avoidance, we focused on what characteristics the UV-B radiation sensitive species may change in order to survive in a homogeneous resource system.

Ultraviolet-B radiation can interfere with the plant growth and development in several ways. Ultraviolet-B radiation induced DNA lesions (Britt, 1995, 1996; Sancar, 1994; Taylor et al., 1997), inhibition of cell division (Gonzalez et al., 1998; Rousseaux et al., 2004), and reduced cell expansion (Hectors et al., 2010; Wargent et al., 2009b), or both (Hoffman et al., 2003; Hopkins et al., 2002). These delays in cell division and expansion may result in significant reduction in leaf area (Suchar and Robberecht, 2015b). Plant protection against increased UV-B radiation requires investment of resources in metabolic processes, and generally results in changes in the quantity and quality of epidermal absorption (Dixon and Paiva, 1995; Li et al., 1993a; Schmelzer et al., 1988a; Winkel-Shirley, 2002). Field studies using modulated field radiation systems that supplement UV-B radiation proportionally to the ambient UV-B regimen show that enhanced UV-B radiation has no significant effects on the photosynthesis (Bassman et al., 2002; Bassman and Robberecht, 2006; Caldwell et al., 2007; Searles et al., 2001). Regardless, morphological changes such as reduced leaf area, shoot mass and plant height are often present (Caldwell et al., 2003; Caldwell et al., 2007; Searles et al., 2001). Changes in resource allocation and timing of reproduction has been observed (Demchik and Day, 1996; Koti et al., 2007; Koti et al., 2005), but it is not definitive that such changes are direct consequences of increased UV-B radiation or indirect effects caused by diminished carbohydrates production, or changes in nutrient uptake.

The effects of UV-B radiation on competitive interactions are largely unknown. While some algal communities were examined (Davidson et al., 1996; Zhang et al., 2013), very few field studies looked at increased UV-B radiation effects on multi-species plant communities. DeWit replacement series experiments on wheat-wild oats interactions, observed that, under increased UV-B radiation, wheat has a competitive advantage over wild oat, but without significant changes in the leaf area index (LAI) of plants (Barnes et al., 1990; Barnes et al., 1988; Ryel et al., 1990). The changes in the wild oats were morphological, i.e., less biomass was placed on the upper part of the plant. In canopies even small morphological changes can lead to significant modifications in light interception and photosynthetic production of

competing species. The resulting re-distribution of biomass in the plant canopy may result in shifts in competitive balance between plants (Barnes et al., 1990; Barnes et al., 1988; Ryel et al., 1990). Their studies also concluded that the response of species to increased UV-B radiation was different in monoculture stands than in mixed species stands, in monoculture stands increased UV-B radiation may not lead to decreases in overall production, and the shifts in competitive balance were significant in high precipitation years, but not in drought years (reduced sensitivity to UV-B due to drought-induced growth inhibition), result similar to what the whole plant model simulations indicated (Suchar and Robberecht, 2015a).

3.4 Conceptual Models

3.4.1 Relative sensitivity to enhanced UV-B radiation of annuals, biennials, and perennials plant species

Different plant species and growth forms respond differently to increased UV-B radiation (Rousseaux et al., 2004; Searles et al., 2001). For example, field studies showed that perennial species may exhibit little or no growth inhibition when exposed to enhanced UV-B radiation (Bassman et al., 2002, 2003; Bassman et al., 2001), while the effects on annuals might be significant (Hidema and Kumagai, 2006; Rousseaux et al., 2004) . In general, perennials produce secondary metabolites that are absorbing shortwave UV-B radiation wavelengths more effectively, thereby, in simulation models, significantly reducing the UV-B radiation growth inhibitions (Suchar and Robberecht, 2015b). Based on the timing of their growth and photosynthesis cycle used, the relative sensitivity to UV-B radiation of six plant types was compared: C3 and C4 summer annuals, C3 winter annuals, C3 biennials, deciduous and evergreen C3 perennials.

For generality, the model plants were considered to have the following organs: roots (R), aboveground structural organs (S), such as stems, or sheaths and stolons, leaves (L), reproductive organs (Ro), and seeds (Sd). Since the model considers only the plant function, only the carbon content and its use by different plant pools was considered (Haefner, 2005; Kerkhoff et al., 2005). For the model of how UV-B radiation effects plant growth and development, we used a generalized model that simulates the plant biomass changes under different radiation scenarios (Suchar and Robberecht, 2015a), which incorporates a previous

model that simulates relative leaf area for various UV-B radiation-induced DNA lesions and rates of photorepair and dark repair (Suchar and Robberecht, 2015b).

3.4.2 Two annual species competition model

We considered a two C3 annual species system for community interactions. The mathematical and the conceptual principles that influence the coexistence among species in such communities were extensively investigated in a series of articles including, but not limited to Amarasekare (2003), Bolnick et al. (2011), Chesson (2000a, b), Holt et al. (1994), Levine and Rees (2002), and Vasseur et al. (2011). Therefore, the mathematical model and parameter estimators (presented in the following sections) follows the principles and structure proposed by this published research. The community dynamics under different levels of intra- and inter- competition, growth, mortality, predation (herbivory included) rates, etc., were extensively examined in these models. Instead, we focused on several specific traits that the UV-B radiation-sensitive species may change in order to survive in a homogeneous resource community.

A conceptual model of possible outcomes in a two species community, one resistant and one sensitive to UV-B radiation, is presented in Figure 1. Based on the principles laid out by previous research, it is reasonable to consider that rare to occasional increased UV-B radiation events may temporarily unbalance the community equilibrium. But, under the stress of UV-B radiation independent processes, the community may regain the initial dynamic equilibrium or reach a new dynamic equilibrium. High frequency or persistent increased UV-B radiation events may require acclimation of the sensitive species in order to coexist with the dominant species. Low phenotypic plasticity of the sensitive species may lead to competitive exclusion. The adaptive responses to increased UV-B radiation may include competitive selection towards qualitative and quantitative changes in secondary metabolites production to increase epidermal absorption, increase in the overall production of secondary metabolites, changes in the timing of seedling emergence or reproductive cycle, or changes in seed size.

3.5 Mathematical Models

3.5.1 Relative sensitivity to enhanced UV-B radiation of annuals, biennials, and perennials plant species

For the model of UV-B radiation effects on the plant growth and development we used a generalized model that simulates plant biomass changes under different radiation scenarios (Suchar and Robberecht, 2015a), which incorporates a previous model that simulates relative leaf area for various UV-B radiation-induced DNA lesions and rates of photorepair and dark repair (Suchar and Robberecht, 2015b).

3.5.2 Two annual species competition model

To model competition between two annual species with a seed bank we modified published models (Chesson, 2000a; Crawley, 2007; Levine and HilleRisLambers, 2009; Levine and Rees, 2002, 2004). These models consider the production, death, germination and predation rates of seed banks, survival rates of emerging seedlings, density- and biomass-dependent growth rates. We amended this model with the explicit generalized model that simulates plant biomass changes under different radiation scenarios (Suchar and Robberecht, 2015a), which incorporates a previous model that simulates relative leaf area for various UV-B radiation-induced DNA lesions and rates of photorepair and dark repair (Suchar and Robberecht, 2015b). We recognize that seed bank dynamics may not be directly relevant to the effects of UV-B radiation, but it is important in the evaluation of the community equilibrium.

The dynamics of seed banks are as follows:

$$S_{t+1,i} = (1 - g_i)(1 - d_i)(S_{t,i} + M_{t,i}n_{t,i}) \quad (1)$$

Where $S_{t,i}$ is the number of seeds in the annual population species i ($i = 1,2$) at the beginning of the growing season of year *t* before germination, g_i is the proportion of germinating seeds, d_i is the seeds death rate in the soil, $M_{t,i}$ is the number of mature plants at the end of the growing season of the year t, and $n_{t,i}$ is the number of seeds produced in average per mature plant. In the previous models (Chesson, 2000a; Levine and HilleRisLambers, 2009; Levine and Rees, 2002, 2004), the second term incorporated the

effects of competition on seed production, but since our model explicitly models single plant growth and development, these interactions were modeled separately.

If we consider the possibility of seed predation in our model (Chesson and Kuang, 2008; Kuang and Chesson, 2008, 2009), equation (1) becomes:

$$S_{t+1,i} = (1 - g_i)(1 - d_i)(S_{t,i} + s_i M_{t,i} n_{t,i}) \quad (2)$$

Where s_i is the proportion of seeds of species i ($i = 1,2$) surviving predation. Note that model considers only newly produced seeds to be predated, the un-germinated seeds being considered distributed within the soil layer (Kuang and Chesson, 2010).

The seed predation is considered frequency dependent in this model. For this we used a modified Kuang and Chesson model (2008, 2009, 2010) derived from Nicholson-Bailey formulation (Hassell 2000) and a special case of foraging model (McNair 1980). The proportion of seed surviving predation is

$$s_i = e^{-a\left((1-p_i)+p_i\frac{M_{t,i}n_{t,i}}{\sum_i M_{t,i}n_{t,i}}\right)P} \quad (3)$$

Where a is the maximum value of the attack rate, p_i proportion of predation that is frequency dependent, and P density of the predator (Kuang and Chesson, 2008, 2009, 2010).

The number of emerging seedlings at time t is a function of the number of seeds in the population at the beginning of the growing season t before germination, and the proportion of germinating seeds. But the survival and growth progression to maturity of the emerging seedling will depend on the number of seeds germinating in the neighborhood of the seedlings, or the density of the seedlings the competitive difference between seedlings resulting from potential seed-size, and the mature plant carrying capacity of the spatial environment (Amarasekare, 2003; Chesson, 2000a; Levine and Rees, 2002). Simplified, the number of surviving plants in a particular plot $P_{t,i}$ will follow a sigmoid density-dependent growth with the potential population size of:

$$M_{t+\Delta t} = \frac{g_i w_i S_{t,i}}{\sum_{i=1}^{2} g_i w_i S_{t,i}} K \quad (4)$$

Where Δt is the interval of time required for seedling to maturate, w_i's are the weighted values quantifying the seed-size dependent competition difference between seedlings, and K

is the carrying capacity of the plot. Note that when there is no seed-size induced competition, $w_1 = w_2 = 1$.

To model individual plants growth and development, and the effects of increased UV-B radiation, we augmented the whole plant model (Suchar and Robberecht, 2015a), with the intraspecific and interspecific exploitation competition effects on whole plant growth.

In annual plant communities, solar radiation is often not a limiting resource, and exploitative competition for other resources (i.e., water and soil nutrients) seem to interact with individual plant growth and development (Goldberg et al., 2001). Thus, we considered a growth inhibition coefficient, density and biomass dependent, modified from the two-species generalized linear model (Park et al., 2002, 2003; Watkinson, 1981).

$$c_i = \frac{1}{1+\propto_{ii}(B_i)\frac{M_i}{K}+\propto_{ij}(B_i,B_j)\frac{M_j}{K}} \quad (5)$$

Where c_i is the growth inhibition of a plant of species i, under competition conditions vs. its potential growth in isolation, and $\propto_{ii}$ and $\propto_{ij}$ are the per capita effects of intra- and interspecific competition coefficients, respectively.

Although solar radiation may not be a limiting resource (Goldberg et al., 2001), there can be significant differences in the solar radiation levels within the plant stands, and between different locations within individual plants, size and density dependent (MONSI and SAEKI, 2005). The light environment within an individual plant canopy is a result of self-shading and shading from the neighbors (both of the same species and of its competitors). Moreover, the relative size of the plant and its neighbors may be significant. The effect on individual plants of the photosynthetically active radiation (PAR) intercepted by the neighboring community is not the same as the effect of UV-B radiation intercepted by the neighboring community. For example, a reduction of 10% in UV-B radiation reaching the top of the plant may effectively result in no UV-B radiation induced growth inhibitions (Suchar and Robberecht, 2015a), while a reduction of 10% in the PAR radiation may have no effect on the plant productivity, since most plant species have light saturation points well under the ambient solar radiation regime (Mc Donald, 2003).

Since our approach modeled the effects of UV-B radiation on the function for a hypothetical generalized flowering plant with simple, planophyllic, glabrous, green leaves, for which we used averaged parameter estimators, we used two simple models for the intra-specific and inter-specific shading. First model is a simple linear model starting from 100% solar radiation reaching the plant at the beginning of the season and down to a fraction reaching the plant at the end of the vegetative growth period. The second model for inter-specific shading is a simple response surface of the size and density dependent relationship with 100% solar radiation reaching the plant when there are no neighboring plants, or there are neighboring plants but they are very small, or have a very low density. The maximum reduction in the solar radiation when the plant is surrounded by plants double in size and at maximum density.

3.6 Parameter Estimation

3.6.1 Relative sensitivity to enhanced UV-B radiation of annuals, biennials, and perennials plant species

Since we modeled hypothetical generalized plant growth forms, C3 and C4 summer annuals, C3 winter annuals, C3 summer biennials, deciduous and evergreen perennials, the parameter estimators considered were means of the minimum and maximum values calculated for a large array of species. Many of these values were obtained from the literature on comprehensive plant characteristics papers (Kattge et al., 2011; Poorter et al., 2009; Poorter and Remkes, 1990; Searles et al., 2001; Taiz and Zeiger, 2010; Wright et al., 2004). Table 1 shows the maximum values of the plant mass photosynthetic production rates and respiration rates at 20℃ used in the models (Larcher, 2003). The photosynthetic production rates extrapolated over the entire day follows a polynomial relationship as detailed in the previous model (Suchar and Robberecht 2015a). Respiration rates were inferred from (Larcher, 2003), which include data compiled from various authors. For the temperature-dependence of respiration, we considered a general Q10 value of 2.0, respiration doubles per 10℃ rise in temperature. While the Q10 respiration value is not constant and it is dependent on the temperature range used in its calculations and the temperature-response curve used (Atkin et al., 2005; Atkin and Tjoelker, 2003), it was considered a reasonable approximation since all

the other parameter estimators in the model are generalized values, averaged over a wide range of species. A 2.5% increase in metabolic rates under high UV-B radiation was considered in the simulations.

All the other parameter estimators used in the models were kept the same as in the previous models (Suchar and Robberecht, 2015a; Suchar and Robberecht, 2015b). Evergreen species and deciduous trees, shrubs and vines generally have higher relative UV-B epidermal absorptance at shorter wavelengths, while herbaceous and grass species have higher relative UV-B absorptance at longer wavelengths, or approximately equal relative absorptance across all UV-B wavelengths (Day et al., 1994; Lavola et al., 1997; Qi et al., 2003; Schmelzer et al., 1988b; Sisson, 1981). These absorptance trends were included in the simulations, since the trends can significantly affect the amount of DNA-weighted UV-B radiation reaching the species DNA (Suchar and Robberecht, 2015b). For all plant growth forms, average dark repair and photorepair rates were considered.

3.6.2 Two annual species competition model

The competition model for two annual species considers that both species are identical except for the sensitivity to increased UV-B radiation. One caveat of this model is that the UV-B radiation sensitive species will be competitively excluded from the system after a period of time, unless the density-dependent intraspecific competition rates exceed both the density-dependent interspecific competition rates and the increased UV-B effects on individual plants. The mature seed biomass is proportional to the maximum vegetative plant biomass, therefore this condition is necessary in order for the sensitive species to outgrow the resistant species, and produce proportionally more seeds per plant when is at the lower density in the community.

Rather than investigating the intra- and inter-specific competition rates that lead to a dynamic equilibrium in the community, we considered fixed values for these parameters in the model and evaluated what other changes may increase the fitness of the sensitive species. Thus, rather than following the relative frequency of species in the community across time, we used the competition model to evaluate the conditions that lead to relative seed productions that can allow the species to coexist. We investigated these scenarios at different initial relative plant densities. For simplicity, species one (Sp1) is the relatively UV-B

radiation resistant species, while species two (Sp2) is the UV-B radiation sensitive species. The simulations considered were: (1) vegetative and mature seed production of Sp1 and Sp2 without intra- and inter-specific competition , in monocultures with intra-specific competition, and at Sp1:Sp2 ratios in community of 90:10, 50:50, and 10:90 with both intra- and inter-specific competition, (2) effect of Sp2 early seed emergence in the growing season, at ratios in community of 90:10, 50:50, and 10:90, (3) effect of Sp2 doubling the seed size, at ratios in community of: 90:10, 50:50, and 10:90; (4) Effect of changes in Sp2 reproduction timing, at ratios in community of 90:10, 50:50, and 10:90, (5) effect of timing of the increased UV-B radiation event, at the beginning, middle and end of the growing season at Sp1:Sp2 ratio of 50:50 in the community, (6) effect of increased secondary metabolites in Sp2 at Sp1:Sp2 ratio of 50:50 in the community, and (7) effect of a combination of changes in Sp2 (emergence one day earlier, expansion of the growing season by one day, delays in reproduction by one day, and increases in cold tolerance by 1%) at ratios in community of 90:10, 50:50, and 10:90. While there are no known UV-B radiation effects on seed size, we wanted to investigate if increased seed size may provide a competitive advantage to the sensitive species.

For the germination and death rates we considered values of 90% and 70% per year ($g_i = 0.9$ and $d_i = 0.7$), values inferred by Levine and Rees (2004) and previous studies (Pavlik et al., 1993; Roberts and Neilson, 1981; Young et al., 1981). Levine and Rees (2004) provides a complete list of published research. For the density dependent seed predation, we considered values of $a = 0.2$ and $p_i = 0.4$ (Chase et al., 2002; Chesson and Kuang, 2008; Kuang and Chesson, 2010).

In the absence of UV-B radiation effects, we modeled a community in equilibrium, by considering the principle that the intraspecific competition exceeds the interspecific competition (Chesson and Kuang, 2008). Therefore, for estimators of the intra- and inter-specific competition effects, we considered that the maximum interspecific competition coefficient $\propto_{ii}$ is twice the intraspecific competition coefficient $\propto_{ij}$. To account for size dependence in the intensity of competition, a linear equation was considered: corresponding to the value of 0 for the inter- and intra-specific competition coefficients when seedlings emerge, and maximum considered values for inter- and intra- specific competition coefficients when plants reach their maximum potential size. For simplicity of the results visualization, the maximum values $\propto_{ii}$ and $\propto_{ij}$ were chosen through calibration process such,

that in the absence of UV-B radiation effects, the two species system reaches a stable equilibrium regardless of the initial species relative frequency in the community. The values considered were: $\propto_{11}=\propto_{22}= 0.2$ and $\propto_{12}=\propto_{21}= 0.1$.

The maximum intra-specific shading was considered about 20%, while the maximum inter-specific reduction of the solar radiation was considered to be about 20% too. These values are based on visual evaluations of graphs from various sources (Harper, 1977; MONSI and SAEKI, 2005) and by no means should be considered accurate. But, this 40% reduction in solar radiation reaching the plant is consistent with the values of photon flux density intercepted by foliage mixed canopy experiments of wheat and wild oats (Ryel et al., 1990). The reduction in UV-B irradiance was considered similar over the entire daytime period. The light saturation point of C3 grasses is a PAR level of about 500 μmol $m^{-2}s^{-1}$ (Mc Donald, 2003). Based on solar illumination on horizontal surface at various solar altitudes above the horizon values (Schlyter, 2015), a 50% reduction in PAR may lead to reductions in photosynthetic production only in the morning or afternoon. The reduction in the PAR radiation dose for individual plants was considered in the whole plant model for these periods of time. These parameter estimators allowed for a stable two-species community in the absence of UV-B radiation effects.

The enhanced UV-B radiation considered for all models was 1.5X the ambient UV-B radiation for this location. Previous results showed that the growth inhibitions at 2X ambient UV-B radiation were proportionally higher, and no interactions with the other parameters in the model were evident (Suchar and Robberecht, 2015a; Suchar and Robberecht, 2015b). Ultraviolet-B radiation data were obtained from the UV-B Monitoring and Research Program (UVMRP) for ten years 2000-2009, Pullman, Washington, which is a location that is representative of UV-B radiation for the northern temperate zone. We used UV-B Langley calibrated data, considered more appropriate than lamp calibrated data for sunny and dry locations (USDA, 2010). Ultraviolet-B radiation data were averaged for the 10-year period, and for each month of the year. Hourly temperature data was obtained for Spokane, Washington from National Oceanic and Atmospheric Administration - National Climatic Data Center (NOAA, 2011).

3.7 Modeling Methodology

The model was created in Vensim Systems software (2009). Data compilation, preparation, and analysis were done in various programs such as Microsoft Access, Excel, and R-language.

The models were verified for consistency and units, for correctness of the mathematics and for accuracy of the conceptual logic (Rykiel, 1996), calibrated and validated (Gardner and Urban, 2003; Rykiel, 1996; Shugart, 1984). Prior to this, sensitivity analysis procedures were performed (Aber et al., 2003; Plentinger and Penning de Vries, 1996; Rykiel, 1996).

3.8 Results

3.8.1 Relative sensitivity to enhanced UV-B radiation of annuals, biennials, and perennials plant species

Summer C3 and C4 annuals plants showed similar inhibition levels relative to the vegetative biomass (Figure 2). Summer C3 plants showed a 23% relative reduction in total biomass, while summer C4 plants showed a 25% relative reduction in total biomass. Plants with the C3 photosynthetic pathway exhibited a more rapid growth at the beginning of the growing season, while C4 plants had a faster growth rate during the midseason. Seeds production of C3 plant species exposed to increased UV-B radiation was slightly lower than the UV-B radiation exposed C4 plants. Winter C3 annuals and biennials showed the highest growth inhibition from all plant types considered (Figure 2). Winter C3 annuals showed 9% growth inhibition if there are no increased UV-B radiation effects on respiration, but 41% if increased UV-B radiation rise respiration by only 2.5%. Biennials C3 plants yearly growth inhibition was similar to that of C3 summer annuals (22% if exposed to increased UV-B radiation in the first year, and 26% if exposed to UV-B radiation in the second year). But the growth inhibition, when exposed to increased UV-B radiation for both years was 42% (Figure 2) similar to C3 winter annuals growth reduction of 41%.

Both deciduous and evergreen perennial species showed no growth inhibitions in the absence of increased metabolic rates. Increased metabolic rates only in leaves (plots 1 and 3 in Figure 3) resulted in growth inhibitions for deciduous species of 1%, 4% and 7.5%, for year one, two and three, respectively. For the same periods of time, evergreen growth inhibitions were 0%, 3%, and 6%. Increased metabolic rates in whole plant (plots 2 and 4 in Figure 3)

resulted in growth inhibitions for deciduous species of 3%, 14%, and 27% (years one, two and three, respectively), and 0%, 8%, and 16% for evergreen species, over the same time period. The plot shows the relative growth of perennials only for three years in the row, since the following years are just showing identical levels of relative growth inhibition induced by increased UV-B radiation

3.8.2 Two annual species competition model

First, vegetative and mature seed production for species relatively resistant (Sp1) and sensitive (Sp2) to UV-B radiation of Sp1 and Sp2 without competition, in monocultures, and at ratios in community of: 90:10, 50:50 and 10:90 were evaluated. Relative vegetative growth for Sp1 under increased UV-B radiation was about 98% under all scenarios considered (Figure 4). For Sp1, the mature seed production ranged from 83 to 98%. The highest mature seed biomass (98%) was shown when the resistant species was grown without competition, in monocultures, and Sp1:Sp2 ratio 90:10. The smallest mature seed production was 83%, observed in the Sp1:Sp2 plant ration of 50:50 communities. Ultraviolet-B sensitive species (Sp2) vegetative growth ranged from 84 to 89%.The mature seed production ranged from 26 to 88%. The lowest mature seed production was recorded for Sp1:Sp2 plant ratio of 90:10. For all scenarios considered, Sp2 vegetative growth and mature seed production were smaller than the vegetative growth and mature seed production of Sp1, but Sp2 vegetative growth was also comparable across all scenarios. The maximum vegetative biomass and mature seed production for the sensitive species (Sp2) was recorded in monocultures (Figure 4).

Model simulations of the effect of Sp2 early seed emergence in the growing season showed that if the seeds of Sp2 emerge earlier, Sp2 vegetative growth and seed production are significantly higher than those for Sp1 for all scenarios considered (Figure 5). Maximum vegetative growth for Sp1 was 35%, 53%, and 67% for the Sp1:Sp2 planting ratios 10:90, 50:50, and 90:10 respectively, and 99-100% for Sp2 for all scenarios considered. The mature seed production for Sp2 ranged from 71 to 100%, while Sp1 effectively produced mature seeds only when in Sp1:Sp2 planting ratio of 90:10.

The doubling of Sp2 seed size significantly increased both the vegetative biomass and seed production of Sp2 (Figure 6). Vegetative biomass and seed production of Sp1 was similar regardless of the scenarios considered, 97–100%, and 95–100% respectively. The

increase in the Sp2 seed size increased both vegetative and mature seed biomass significantly in all scenarios considered, from a range of 85-89% and 26-56%, to 98-99% and 69-97%, respectively. Model simulations showed that this increases may ensure the survival of Sp2 in the community.

Delays of three days in reproduction timing increased the seed production significantly, and may offer a competitive advantage to Sp2 (Figure 7). Vegetative biomass and seed production of Sp1 was similar regardless of the scenarios considered, 99–100%, and 88–89% respectively. The delay in the Sp2 reproduction timing increased mature seed biomass significantly in all scenarios considered, from a range of 26-49% to 44-100%. The values for the Sp2 vegetative growth were comparable 85-92%. Model simulations showed that there is an interaction between seed production and Sp1:Sp2 planting ratios.

Model simulation of the increased UV-B radiation events at the beginning, middle and end of the growing season showed that, if the increased UV-B radiation event is early during the growing season, the vegetative growth and mature seeds biomass of Sp1 and Sp2 are comparable to the ones observed under increased UV-B radiation exposure for the entire season (results not shown). As the timing of the increased UV-B radiation event mover further during the growing season, the effect on the final growth of Sp2 is proportionally smaller. If the increased UV-B radiation event is in July, August, or September, neither species showed significant growth inhibitions.

The vegetative growth and seed production of Sp2 when exposed to increased UV-B radiation and with increased secondary metabolites production were almost identical to the ones recorded for Sp2 under ambient UV-B radiation, probably well within the natural variability of any plant species (results not shown).

Finally, the combination of small changes in Sp2 (emergence one day earlier, expansion of the growing season by one day, delays in reproduction by one day, and increases in cold tolerance by 1%) increased significantly the vegetative growth and seed production of Sp2 (Figure 8). The relative UV-B radiation-resistant species (Sp1) was outperformed by the relative UV-B radiation-sensitive species (Sp2) in all scenarios considered.

3.9 Discussion

3.9.1 Relative sensitivity to enhanced UV-B radiation of annuals, biennials, and perennials plant species

Summer C3 plants outgrew C4 plants at the beginning of the growing season, while C4 plants had a faster growth rate during the midseason. This was expected due to the quantum yield difference between C3 and C4 plants, and its temperature-dependence in C3 species (Ehleringer et al., 1997). However, since their overall growth inhibitions were similar, it suggests that both growth types exhibit similar sensitivity to the increased UV-B radiation. The lower C3 seed production for UV-B radiation exposed plants relatively to that for the UV-B exposed C4 plants is not a direct effect of increased UV-B radiation, but also a result of the physiological differences between C3 and C4 plants in the quantum yield efficiency and temperature-dependent photorespiration (Ehleringer et al., 1997). This suggests that in mixed annual communities, UV-B radiation may not affect the competitiveness of either plant types, and that the community dynamics may be influenced by other environmental factors and/or physiological and morphological characteristics.

The major source of growth inhibition in winter C3 annuals was due to the increase in metabolic rates (see Figure 2). Winter C3 annuals showed less than 10% growth inhibition if there are no increased UV-B radiation effects on respiration, but about 60% if increased UV-B radiation rises respiration by only 2.5%. The studies of the effects of increased UV-B radiation showed increases in respiration rates from 0 to 280% (Bassman et al., 2003; Gwynn-Jones, 2001). More research is necessary to measure species-specific metabolic rates in order to predict the effects of increased UV-B radiation in winter C3 annuals. When used in our model, the reported 280% increase of respiration rate significantly inhibited the growth of all annual species with or without increased UV-B radiation levels. Therefore, more research is necessary to also measure the species-specific net photosynthesis rates associated with these metabolic rates. We expected non-significant growth inhibition in winter annuals, since the UV-B radiation dose in the winter is significantly less than the summer doses. However, the model simulations suggested that this may be the case only in the absence of increases in metabolic rates, i.e., the molecular processes explicitly considered in the model were not responsible for the most winter C3 annuals growth inhibitions. Also, what the model did not considered were the increases in plant frost tolerance and survival due to increased UV-B

radiation, and the decreased UV-B radiation sensitivity of plants due to low temperatures (Bilger et al., 2007; Caldwell et al., 2007). Therefore, the UV-B radiation effect on winter annuals showed by the model simulations may be highly overestimated, and with a high degree of uncertainty.

Biennials exhibited the same level of yearly growth inhibition as the C3 summer annuals. The yearly growth inhibition, when exposed to increased UV-B radiation for both years, may accumulate (see Figure 2). Noteworthy is that a high UV-B radiation dose in the second year was more detrimental to plant growth than a high UV-B radiation dose in the first year. This was expected since in our model increased UV-B radiation inhibits leaf growth, and biennials exhibit significantly higher above-ground vegetative growth in the second year. This result is different than the response of summer annuals (Suchar and Robberecht, 2015a) which showed that plants were more sensitive to UV-B exposure during the early periods of growth and development. These differences in the responses of annuals and biennials to increased UV-B radiation may result in shifts in competitive balance in mixed growth form communities.

The increased UV-B radiation did not have any effect on the growth and development of perennial plants as long as it does not increase the metabolic rates (see Figure 3). As presented in (Suchar and Robberecht, 2015b), perennials generally have effective secondary metabolites that absorb UV-B radiation, thereby preventing most of the DNA damage, and preventing any delays in leaf growth. But, in model simulations, increased metabolic rates inhibited the growth of perennials. This inhibition is a result of lower levels of non-structural carbohydrates in storage, which may lead to smaller growth rates in the following year. This carbon balance deficit may accumulate over the years to cause a constant inhibition rate. Alternatively, increased metabolic rates that occur only in leaves seem to be more a plausible explanation of a moderate drop in the relative growth rate. Although the model showed that deciduous species might be more sensitive to increased UV-B radiation than evergreen species, the differences were small and may be well within the natural variability of the growth forms. The values for maximum photosynthetic and metabolic rates used in the model (see Table 1) may also have been a factor in our results. The ratio of relative metabolic rates to photosynthetic rates is higher in deciduous species than in evergreen species. This suggests that a larger percentage of gross production is used by metabolic processes in deciduous

species than in evergreen species. As a result, an increase of 2.5% in the metabolic rate is bound to have a higher effect in deciduous species than in evergreen species. Considering the UV-B radiation absorption efficiency of secondary metabolites, it is quite possible that the metabolic rate increases would be negligible or only limited to the leaf growth and expansion period. In such scenario, the reduction in the relative rate may be small, or even nonexistent.

The model simulations results were different from experimental results conducted at 200% UV-B radiation, that show significant decreases in total biomass in coniferous tree species, no significant differences or significant increases in total biomass of deciduous tree species, while no significant changes in metabolic rates were observed (Bassman et al., 2003). However, this study was conducted in a glasshouse, and in many cases glasshouse and growth chamber experiments show significantly increased UV-B effects that are unconfirmed by field studies (Bassman et al., 2002; Bassman and Robberecht, 2006; Caldwell et al., 2007; Searles et al., 2001). A similar study following the growth of *Pseudotsuga menziesii* (Douglas-fir) over three years in glasshouse and field experiments, showed that growth was not affected by increased UV-B radiation (Bassman et al., 2002). While the differences in the model results and experimental results may just be a result of simulating a different increased UV-B radiation rate, the difference may also be a result of model parametrization or failure to include some other UV-B radiation dependent processes.

3.9.2 Two annual species competition model

Community vegetative growth was not substantially affected by UV-B radiation exposure under the two-species competition scenarios considered. But, relatively small differences of vegetative growth of individual UV-B radiation sensitive plants were observed (see Figure 4). This result is supported by experiments on wheat-wild oat competition (Barnes et al., 1990; Barnes et al., 1988; Ryel et al., 1990). The major differences were in the mature seed production. Species that are relatively resistant to UV-B radiation (Sp1) always out-produced the UV-B radiation sensitive species (Sp2). The two annual species competition model indicated that Sp2 will be competitively excluded from the system, unless intra-specific competition outweighs both the UV-B radiation effects and interspecies competition. Considerably more sophisticated models that include a greater number of abiotic and biotic

factors and their interaction are needed to reliably determine the effect of UV-B radiation on complex communities.

Our model simulations showed that even relatively small morphological and/or physiological changes in Sp2 may not only allow it to survive in the community, but will even provide it a competitive advantage over Sp1. The simulations showed that if increases in the secondary metabolites production in Sp2 were at a level sufficient to prevent the effects of increased UV-B radiation, only small inhibitions in the overall vegetative and seed biomass will occur. These would probably be within the natural variability expected for any plant species. Also, the model simulations showed that if the seedlings of Sp2 emerge one week earlier, Sp2 vegetative growth and seed production would be higher than those for Sp1 for all scenarios considered (see Figure 5). Furthermore, the doubling of Sp2 seed size increased both the vegetative biomass and seed production of Sp2 (see Figure 6), even to the point where Sp2 has a competitive advantage over Sp1. Moreover, changes in reproduction timing may increase the Sp2 seed production (see Figure 7). These simulations showed an array of responses to UV-B radiation that can increase the fitness of the sensitive plant species (Sp2). It is unknown whether the selection of plants in the population with the seed twice the size of its competitors will occur. However, under constant increased UV-B radiation it is possible that the large seed size trait will become common in the population. The timing for germination may be similarly affected.

The timing for reproduction seems to have an effect on the fitness of the species. The model simulations show that a delay of only three days in reproduction, nearly doubles the seed production in Sp2, and even may even offer a competitive advantage over Sp1. Reproduction timing was determined from a wide range of studies, and the default values used in the model were not optimized for either maximum seed production or vegetative biomass. If the reproduction timing for both species modeled is optimized for maximum seed biomass production, any delays may actually result in decreases in mature seed biomass. However, if instead of considering a community of annual species, we examine o community of perennial grasses, for example, the trade-off between optimizing for sexual reproduction or clonal reproduction may lead to the niche differentiation in which intraspecific competition is stronger than interspecific competition, hypothesized to be essential for species coexistence (Amarasekare, 2003; Chesson, 2000b).

Ultraviolet-B radiation often increases both plant frost tolerance and survival (Bilger et al., 2007; Caldwell et al., 2007). Thus, it is also possible that Sp2 may exhibit greater growth at the beginning and end of the growing season, which are characterized by lower temperatures. Also, it may survive in the community for a longer period of time in the growing season. If there are small changes in the foliage distribution over the height of the plant, this can lead to changes in light interception and photosynthetic production of competing species which may result in shifts in competitive balance among species (Barnes et al., 1990; Barnes et al., 1988; Ryel et al., 1990), and changes in herbivory of the sensitive species. For example, if most biomass lost due to grazing is due to large herbivores, a lower plant profile in the community may result in decreased herbivory. Alternatively, if most biomass lost to grazing is due to insects or small mammalian herbivores, the sensitive species may experience increased herbivory.

While the changes in the vertical distribution of foliage in sensitive species may influence herbivory both ways, the changes in secondary metabolites production may result in increased herbivory. *Chrysomela scripta* (cottonwood leaf beetle) favored *Populus trichocarpa* (black cottonwood) leaves grown under increased UV-B radiation (i.e., with high secondary metabolites content) at the cost of diminished conversion efficiency of the digested food (Warren et al., 2002). While this decrease in conversion efficiency may result in reduced growth of the *Chrysomela scripta,* it is possible that the predation on this herbivore may also be reduced (Warren et al., 2002). In a different study, neither the *Brachylagus idahoensis* (pygmy rabbit) - a specialist feeder on sagebrush, or the *Sylvilagus floridanus* (eastern cottontail) – a generalist herbivore, showed any preference towards foliage grown under ambient or increased UV-B radiation, or differences in intake and digestion (Thines et al., 2007). Therefore, the changes in the UV-B radiation foliage structure and chemical composition may or may not change the herbivory on the plant species, but may have important consequences for some herbivore species composition and trophic structure of ecosystems where these processes may occur.

Occasional increased UV-B radiation events may have significant effects only if early the growing season. But, if it is a short term increased UV-B radiation event, the growth inhibitions in the UV-B radiation sensitive species should not have any long lasting effect on

the structure of the community, unless other environmental factors are perturbing the dynamic equilibrium. The community equilibrium may be regained in the following years.

One assumption used in the model was a community-wide environmental homogeneity. But this is not the case: more often than not, the environment is heterogeneous. This heterogeneity may affect the competitive advantage of the UV-B radiation resistant species (e.g. slightly northern exposure may reduce the UV-B radiation dose reaching the plant). Under persistent increased UV-B radiation, it is possible to have shifts in the relative abundance of the species across this heterogeneous environmental gradient, but our results does not suggest that competitive exclusions are likely.

In summary, under continuous increased UV-B radiation, species types have different levels of sensitivity to increased UV-B radiation. In communities, sensitive species may be at a competitive disadvantage compared to resistant plant species. But, sensitive species have a wide array of responses that can increase their fitness and reproductive success in the community (e.g., increased secondary metabolites production, changes in timing of emergence and reproduction, changes in seed size, etc.), even to a point of specialization for particular ecological niches, a key requirement for coexistence in plant communities (Chesson, 2000a; Crawley, 2007; Levine and HilleRisLambers, 2009; Levine and Rees, 2002, 2004). While individual plants may exhibit significant inhibitions in growth and development, in communities, these inhibitions can be answered with relatively small morphological and physiological changes. For example, the model simulations show that if the UV-B sensitive species (Sp2) emerges only one day earlier, expends its growing season by only one more day, delays reproduction by only one day, and increases its cold tolerance by only 1%, it may outperform the UV-B radiation resistant species, both in vegetative and mature seed biomass (see Figure 8). While these UV-B radiation-induced small morphological and physiological changes may not lead to significant changes in community's biomass and structure, they may result in significant changes in some herbivore species composition and trophic structure of ecosystems.

3.10 Conclusions

Our model is the first to integrate the effects of increased UV-B radiation through molecular level processes, whole plant growth and development, and community interactions.

We modeled the effects of UV-B radiation at molecular level, and proposed the possible mechanisms that lead to the observed whole plant dynamics. Then, we integrated these models in a two annual species competition model. The model showed differences in sensitivity to increased UV-B radiation between plant growth forms. In communities, the UV-B radiation sensitive species was constantly outcompeted by the resistant species. But small morphological and physiological changes can cancel the resistant species competitive advantage. A review of the relevant literature showed a wide range of values for the key parameters. Moreover, certain parameter values were inferred only from the calibration process. However our model allowed the testing of several to examine a variety of questions that were difficult to approach through experimental research.

3.11 Acknowledgments

We acknowledge the insightful comments of our reviewers.

3.12 References

Aber, J.D., Bernhardt, E.S., Dijkstra, F.A., Gardner, R.H., Macneale, K.H., Parton, W.J., S.T.A., P., Urban, D.L., Weathers, K.C., 2003. Standards of practice for review and publication of models: summary of discussion, in: Canham, C.D., Cole, J.J., Lauenroth, W.K. (eds.), Models in Ecosystem Science. Princeton University Press, Princeton, NJ, pp. 204-210.

Amarasekare, P., 2003. Competitive coexistence in spatially structured environments: a synthesis. Ecol Lett 6, 1109-1122.

Andrady, A.L., Aucamp, P.J., Austin, A., Bais, A.F., Ballare, C.L., Barnes, P.W., Bernhard, G.H., Bornman, J.F., Caldwell, M.M., De Gruijl, F.R., Erickson, D.J., Flint, S.D., Gao, K., Gies, P., Hader, D.P., Ilyas, M., Longstreth, J., Lucas, R., Madronich, S., McKenzie, R.L., Neale, R., Norval, M., Pandy, K.K., Paul, N.D., Rautio, M., Redhwi, H.H., Robinson, S.A., Rose, K., Shao, M., Sinha, R.P., Solomon, K.R., Sulzberger, B., Takizawa, Y., Tang, X., Torikai, A., Tourpali, K., van der Leun, J.C., Wangberg, S.A., Williamson, C.E., Wilson, S.R., Worrest, R.C., Young, A.R., Zepp, R.G., 2015. Environmental effects of ozone depletion and its interactions with climate change: 2014 assessment Executive summary. Photochemical & Photobiological Sciences 14, 14-18.

Atkin, O.K., Bruhn, D., Tjoelker, M.G., 2005. Response of plant respiration to changes in temperature: mechanisms and consequences of variations in Q_{10} values and acclimation, in: Lambers, H., Ribas-Carbo, M. (eds.), Plant Respiration: From Cell to Ecosystem. Springer, The Netherlands, pp. 95-135.

Atkin, O.K., Tjoelker, M.G., 2003. Thermal acclimation and the dynamic response of plant respiration to temperature. Trends Plant Sci 8, 343-351.

Barnes, P.W., Flint, S.D., Caldwell, M.M., 1990. Morphological Responses of Crop and Weed Species of Different Growth Forms to Ultraviolet-B Radiation. Am J Bot 77, 1354-1360.

Barnes, P.W., Jordan, P.W., Gold, W.G., Flint, S.D., Caldwell, M.M., 1988. Competition, Morphology and Canopy Structure in Wheat (*Triticum aestivum* L.) and Wild Oat (Avena fatua L.) Exposed to Enhanced Ultraviolet-B Radiation. Funct Ecol 2, 319-330.

Bassman, J.H., Edwards, G.E., Robberecht, R., 2002. Long-term exposure to enhanced UV-B radiation is not detrimental to growth and photosynthesis in Douglas-fir. New Phytol 154, 107-120.

Bassman, J.H., Edwards, G.E., Robberecht, R., 2003. Photosynthesis and growth in seedlings of five forest tree species with contrasting leaf anatomy subjected to supplemental UV-B radiation. Forest Science 49, 176-187.

Bassman, J.H., Robberecht, R., 2006. Growth and gas exchange in field-grown and greenhouse-grown *Quercus rubra* following three years of exposure to enhanced UV-B radiation. Tree Physiol 26, 1153-1163.

Bassman, J.H., Robberecht, R., Edwards, G.E., 2001. Effects of enhanced UV-B radiation on growth and gas exchange in *Populus deltoides* Bartr. ex Marsh. Int J Plant Sci 162, 103-110.

Bilger, W., Rolland, M., Nybakken, L., 2007. UV screening in higher plants induced by low temperature in the absence of UV-B radiation. Photochemical & Photobiological Sciences 6, 190-195.

Bolnick, D.I., Amarasekare, P., Araujo, M.S., Burger, R., Levine, J.M., Novak, M., Rudolf, V.H.W., Schreiber, S.J., Urban, M.C., Vasseur, D.A., 2011. Why intraspecific trait variation matters in community ecology. Trends Ecol Evol 26, 183-192.

Bornman, J.F., Barnes, P.W., Robinson, S.A., Ballare, C.L., Flint, S.D., Caldwell, M.M., 2015. Solar ultraviolet radiation and ozone depletion-driven climate change: effects on terrestrial ecosystems. Photochemical & Photobiological Sciences 14, 88-107.

Britt, A.B., 1995. Repair of DNA damage induced by ultraviolet radiation. Plant Physiol 108, 891-896.

Britt, A.B., 1996. DNA damage and repair in plants. Annual Review of Plant Molecular Biology 47, 75-100.

Caldwell, M.M., Ballare, C.L., Bornman, J.F., Flint, S.D., Bjorn, L.O., Teramura, A.H., Kulandaivelu, G., Tevini, M., 2003. Terrestrial ecosystems increased solar ultraviolet radiation and interactions with other climatic change factors. Photochemical & Photobiological Sciences 2, 29-38.

Caldwell, M.M., Bornman, J.F., Ballare, C.L., Flint, S.D., Kulandaivelu, G., 2007. Terrestrial ecosystems, increased solar ultraviolet radiation, and interactions with bother climate change factors. Photochemical & Photobiological Sciences 6, 252-266.

Chase, J.M., Abrams, P.A., Grover, J.P., Diehl, S., Chesson, P., Holt, R.D., Richards, S.A., Nisbet, R.M., Case, T.J., 2002. The interaction between predation and competition: a review and synthesis. Ecol Lett 5, 302-315.

Chesson, P., 2000a. General theory of competitive coexistence in spatially-varying environments. Theor Popul Biol 58, 211-237.

Chesson, P., 2000b. Mechanisms of maintenance of species diversity. Annu Rev Ecol Syst 31, 343-+.

Chesson, P., Kuang, J.J., 2008. The interaction between predation and competition. Nature 456, 235-238.

Cockell, C.S., 2000. The ultraviolet history of the terrestrial planets - implications for biological evolution. Planetary and Space Science 48, 203-214.

Cockell, C.S., Horneck, G., 2001. The history of the UV radiation climate of the Earth - theoretical and space-based observations. Photochem Photobiol 73, 447-451.

Crawley, M.J., 2007. Plant population dynamics, in: May, R.M., McLean, A.R. (eds.), Theoretical ecology: principles and applications. Oxford University Press, Great Britain, pp. 62-83.

Davidson, A., Marchant, H., De la Mare, W., 1996. Natural UVB exposure changes the species composition of Antarctic phytoplankton in mixed culture. Aquatic Microbial Ecology 10, 299-305.

Day, T.A., Howells, B.W., Rice, W.J., 1994. Ultraviolet absorption and epidermal-transmittance spectra in foliage. Physiol Plantarum 92, 207-218.

Day, T.A., Neale, P.J., 2002. Effects of UV-B Radiation on Terrestrial and Aquatic Primary Producers. Annu Rev Ecol Syst 33, 371-396.

DeLucia, E.H., Coleman, J.S., Dawson, T.E., Jackson, R.B., 2001. Plant physiological ecology: linking the organism to scales above and below - Ecological Society of America Meeting Snowbird, UT, USA, August 2000. New Phytol 149, 12-16.

Demchik, S.M., Day, T.A., 1996. Effect of enhanced UV-B radiation on pollen quantity, quality, and seed yield in *Brassica rapa* (Brassicaceae). Am J Bot 83, 573-579.

Dixon, R.A., Paiva, N.L., 1995. Stress-Induced Phenylpropanoid Metabolism. Plant Cell 7, 1085-1097.

Ehleringer, J.R., Cerling, T.E., Helliker, B.R., 1997. C4 photosynthesis, atmospheric CO2, and climate. Oecologia 112, 285 - 299.

Enquist, B.J., Niklas, K.J., 2002. Global allocation rules for patterns of biomass partitioning in seed plants. Science 295, 1517-1520.

Gardner, R.H., Urban, D.L., 2003. Model validation and testing: past lessons, present concerns, future prospects, in: Canham, C.D., Cole, J.J., Lauenroth, W.K. (eds.), Models in Ecosystem Science. Princeton University Press, Princeton, NJ, pp. 184-203.

Goldberg, D.E., Turkington, R., Olsvig-Whittaker, L., Dyer, A.R., 2001. Density dependence in an annual plant community: Variation among life history stages. Ecol Monogr 71, 423-446.

Gonzalez, R., Mepsted, R., Wellburn, A.R., Paul, N.D., 1998. Non-photosynthetic mechanisms of growth reduction in pea (*Pisum sativum* L.) exposed to UV-B radiation. Plant Cell Environ 21, 23-32.

Gwynn-Jones, D., 2001. Short-term impacts of enhanced UV-B radiation on photo-assimilate allocation and metabolism: a possible interpretation for time-dependent inhibition of growth. Plant Ecol 154, 65-73.

Haefner, J.W., 2005. Modeling biological systems: principles and applications, 2 ed. Springer Science+Business media, New York, NY.

Harper, J.L., 1977. Population Biology of Plants. Academic Press, London.

Hartmann, D.L., Wallace, J.M., Limpasuvan, V., Thompson, D.W.J., Holton, J.R., 2000. Can ozone depletion and global warming interact to produce rapid climate change? Proceedings of the National Academy of Sciences 97, 1412-1417.

Hectors, K., Jacques, E., Prinsen, E., Guisez, Y., Verbelen, J.P., Jansen, M.A., Vissenberg, K., 2010. UV radiation reduces epidermal cell expansion in leaves of Arabidopsis thaliana. J Exp Bot 61, 4339-4349.

Hidema, J., Kumagai, T., 2006. Sensitivity of rice to ultraviolet-B radiation. Ann Bot-London 97, 933-942.

Hoffman, R.W., Campbell, B.D., Bloor, S.J., Swinny, E.E., Markham, K.R., Ryan, K.G., Fountain, D.W., 2003. Responses to UV-B radiation in Trifolium repens l. - physiological links to plant productivity and water availability. Plant, Cell and Environment 26, 603-612.

Holt, R.D., Grover, J., Tilman, D., 1994. Simple Rules for Interspecific Dominance in Systems with Exploitative and Apparent Competition. Am Nat 144, 741-771.

Hopkins, L., Bond, M.A., Tobin, A.K., 2002. Ultraviolet-B radiation reduces the rates of cell division and elongation in the primary leaf of wheat (Triticum aestivum L. cv Maris Huntsman). Plant, Cell and Environment 25, 617-624.

Kattge, J., Diaz, S., Lavorel, S., Prentice, C., Leadley, P., Bonisch, G., Garnier, E., Westoby, M., Reich, P.B., Wright, I.J., Cornelissen, J.H.C., Violle, C., Harrison, S.P., van Bodegom, P.M., Reichstein, M., Enquist, B.J., Soudzilovskaia, N.A., Ackerly, D.D., Anand, M., Atkin, O., Bahn, M., Baker, T.R., Baldocchi, D., Bekker, R., Blanco, C.C., Blonder, B., Bond, W.J., Bradstock, R., Bunker, D.E., Casanoves, F., Cavender-Bares, J., Chambers, J.Q., Chapin, F.S., Chave, J., Coomes, D., Cornwell, W.K., Craine, J.M., Dobrin, B.H., Duarte, L., Durka, W., Elser, J., Esser, G., Estiarte, M., Fagan, W.F., Fang, J., Fernandez-Mendez, F., Fidelis, A., Finegan, B., Flores, O., Ford, H., Frank, D., Freschet, G.T., Fyllas, N.M., Gallagher, R.V., Green, W.A., Gutierrez, A.G., Hickler, T., Higgins, S.I., Hodgson, J.G., Jalili, A., Jansen, S., Joly, C.A., Kerkhoff, A.J., Kirkup, D., Kitajima, K., Kleyer, M., Klotz, S., Knops, J.M.H., Kramer, K., Kuhn, I., Kurokawa, H., Laughlin, D., Lee, T.D., Leishman, M., Lens, F., Lenz, T., Lewis, S.L., Lloyd, J., Llusia, J., Louault, F., Ma, S., Mahecha, M.D., Manning, P., Massad, T., Medlyn, B.E., Messier, J., Moles, A.T., Muller, S.C., Nadrowski, K., Naeem, S.,

Niinemets, U., Nollert, S., Nuske, A., Ogaya, R., Oleksyn, J., Onipchenko, V.G., Onoda, Y., Ordonez, J., Overbeck, G., Ozinga, W.A., Patino, S., Paula, S., Pausas, J.G., Penuelas, J., Phillips, O.L., Pillar, V., Poorter, H., Poorter, L., Poschlod, P., Prinzing, A., Proulx, R., Rammig, A., Reinsch, S., Reu, B., Sack, L., Salgado-Negre, B., Sardans, J., Shiodera, S., Shipley, B., Siefert, A., Sosinski, E., Soussana, J.F., Swaine, E., Swenson, N., Thompson, K., Thornton, P., Waldram, M., Weiher, E., White, M., White, S., Wright, S.J., Yguel, B., Zaehle, S., Zanne, A.E., Wirth, C., 2011. TRY - a global database of plant traits. Global Change Biol 17, 2905-2935.

Kerkhoff, A.J., Enquist, B.J., Elser, J.J., Fagan, W.F., 2005. Plant allometry, stoichiometry and the temperature-dependence of primary productivity. Global Ecol Biogeogr 14, 585-598.

Koti, S., Reddy, K.R., Kakani, V.G., Zhao, D., Gao, W., 2007. Effects of carbon dioxide, temperature and ultraviolet-B radiation and their interactions on soybean (*Glycine max* L.) growth and development. Environ Exp Bot 60, 1-10.

Koti, S., Reddy, K.R., Reddy, V.R., Kakani, V.G., Zhao, D., 2005. Interactive effects of carbon dioxide, temperature, and ultraviolet-B radiation on soybean (Glycine max L.) flower and pollen morphology, pollen production, germination, and tube lengths. J Exp Bot 56, 725-736.

Kuang, J.J., Chesson, P., 2008. Predation-competition interactions for seasonally recruiting species. Am Nat 171, E119-E133.

Kuang, J.J., Chesson, P., 2009. Coexistence of annual plants: Generalist seed predation weakens the storage effect. Ecology 90, 170-182.

Kuang, J.J., Chesson, P., 2010. Interacting coexistence mechanisms in annual plant communities: Frequency-dependent predation and the storage effect. Theor Popul Biol 77, 56-70.

Larcher, W., 2003. Physiological plant ecology, 4 ed. Springer-Verlag, Berlin 513 pp.

Lavola, A.N.U., Julkunen-Tiitto, R., Aphalo, P., De La Rosa, T., Lehto, T., 1997. The effect of u.v.-B radiation on u.v.-absorbing secondary metabolites in birch seedlings grown under simulated forest soil conditions. New Phytol 137, 617-621.

Levine, J.M., HilleRisLambers, J., 2009. The importance of niches for the maintenance of species diversity. Nature 461, 254-257.

Levine, J.M., Rees, M., 2002. Coexistence and relative abundance in annual plant assemblages: The roles of competition and colonization. Am Nat 160, 452-467.

Levine, J.M., Rees, M., 2004. Effects of temporal variability on rare plant persistence in annual systems. Am Nat 164, 350-363.

Li, J., Ou-Lee, T., Raba, R., Admundson, R.G., Last, R.L., 1993. Arabidopsis flavonoids mutants are hypersensitive to UV-B irradiation. The Plant Cell 5, 171-179.
Lowry, B., Lee, D., Hebant, C., 1980. The origins of land plants: a new look at an old problem. Taxon 29, 183-197.

Mc Donald, M.S., 2003. Photobiology of higher plants. John Wiley & Sons, England 354 pp.

MONSI, M., SAEKI, T., 2005. On the Factor Light in Plant Communities and its Importance for Matter Production. Ann Bot-London 95, 549-567.

Niklas, K.J., Enquist, B.J., 2002. On the vegetative biomass partitioning of seed plant leaves, stems, and roots. Am Nat 159, 482-497.

NOAA, 2011. National Climatic Center. http://www.ncdc.noaa.gov/. Accessed May 2011.

Park, S.E., Benjamin, L.R., Watkinson, A.R., 2002. Comparing biological productivity in cropping systems: a competition approach. J Appl Ecol 39, 416-426.

Park, S.E., Benjamin, L.R., Watkinson, A.R., 2003. The Theory and Application of Plant Competition Models: an Agronomic Perspective. Ann Bot-London 92, 741-748.

Pavlik, B.M., Ferguson, N., Nelson, M., 1993. Assessing Limitations on the Growth of Endangered Plant-Populations .2. Seed Production and Seed Bank Dynamics *of Erysimum-Capitatum* Ssp *Angustatum and Oenothera*-Deltoides Ssp Howellii. Biol Conserv 65, 267-278.

Plentinger, M.C., Penning de Vries, F.W.T. (eds.) 1996. CAMASE register of agro-ecosystems models, http://library.wur.nl/way/bestanden/clc/1763788.pdf electronic ed 420 pp.

Poorter, H., Niinemets, U., Poorter, L., Wright, I.J., Villar, R., 2009. Causes and consequences of variation in leaf mass per area (LMA): a meta-analysis. New Phytol 182, 565-588.

Poorter, H., Remkes, C., 1990. Leaf-Area Ratio and Net Assimilation Rate of 24 Wild-Species Differing in Relative Growth-Rate. Oecologia 83, 553-559.

Qi, Y., Bai, S., Heisler, G.M., 2003. Changes in ultraviolet-B and visible optical properties and absorbing pigment concentrations in pecan leaves during a growing season. Agr Forest Meteorol 120, 229-240.

Rettberg, P., Horneck, G., Strauch, W., Facius, R., Seckmeyer, G., 1998. Simulation of planetary UV radiation climate on the example of the early Earth. Advances in Space Research 22, 335-339.

Roberts, H.A., Neilson, J.E., 1981. Seed Survival and Periodicity of Seedling Emergence in 12 Weedy Species of Compositae. Ann Appl Biol 97, 325-334.

Rousseaux, M.C., Flint, S.D., Searles, P.S., Caldwell, M.M., 2004. Plant responses to current solar ultraviolet-B radiation and to supplemented solar ultraviolet-B radiation simulating ozone depletion: an experimental comparison. Photochem Photobiol 80, 224-230.

Rozema, J., Van De Staaij, J., Bjorn, L.O., De Bakker, N., 1999. Depletion of stratospheric ozone and solar UV-B radiation: evolution of land plants, UV-screens and function of polyphenolics, in: Rozema, J. (ed.), Stratospheric ozone depletion: the effects of enhanced UV-B radiation on terrestrial ecosystems. Backhyn Publishers, Leiden, The Netherlands.

Ryel, R.J., Barnes, P.W., Beyschlag, W., Caldwell, M.M., Flint, S.D., 1990. Plant Competition for Light Analyzed with a Multispecies Canopy Model .1. Model Development and Influence of Enhanced Uv-B Conditions on Photosynthesis in Mixed Wheat and Wild Oat Canopies. Oecologia 82, 304-310.

Rykiel, J.E.J., 1996. Testing ecological models: the meaning of validation. Ecol Model 90, 229.

Sagan, C., 1973. Ultraviolet Selection Pressure on Earliest Organisms. J Theor Biol 39, 195-200.

Sancar, A., 1994. Structure and function of DNA photolyase. Biochemistry-Us 33, 2-9.

Sancar, A., Sancar, G.B., 1988. DNA-Repair Enzymes. Annu Rev Biochem 57, 29-67.

Schlyter, P., 2015. How bright are natural light sources?

Schmelzer, E., Jahnen, W., Hahlbrock, K., 1988a. In situ localization of light-induced chalcone synthase mRNA, chalcone synthase, and flavonoid end products in epidermal cells of parsley leaves. Proceedings of the National Academy of Sciences 85, 2989-2993.

Schmelzer, E., Jahnen, W., Hahlbrock, K., 1988b. In situ localization of light-induced chalcone synthase mRNA, chalcone synthase, and flavonoid end products in epidermal cells of parsley leaves. P Natl Acad Sci USA 85, 2989-2993.

Searles, P.S., Flint, S.D., Caldwell, M.M., 2001. A meta analysis of plant field studies simulating stratospheric ozone depletion. Oecologia 127, 1-10.

Shugart, H.H., 1984. A Theory on Forest Dynamics. The Ecological Implications of Forest Succesion Models. Springer-Verlag, New York, NY 278 pp.

Sisson, W.B., 1981. Photosynthesis, Growth, and Ultraviolet Irradiance Absorbance of Cucurbita pepo L. Leaves Exposed to Ultraviolet-B Radiation (280-315 nm). Plant Physiol 67, 120-124.

Smith, B.N., 2005. Photosynthesis, respiration, and growth, in: Pessarakli, M. (ed.), Handbook of Photosynthesis. 2 ed. Taylor & Francis Group, Boca Raton, FL, pp. 671-677.

Stafford, H.A., 1991. Flavonoid Evolution - an Enzymatic Approach. Plant Physiol 96, 680-685.

Suchar, V.A., Robberecht, R., 2015a. Integration and scaling of UV-B radiation effects on plants: from molecular interactions to whole plant responses. under review.

Suchar, V.A., Robberecht, R., 2015b. Integration and scaling of UV-B radiation effects on plants: from DNA to leaf. Ecology and Evolution, (in press).

Systems, V., 2009. Vensim: Ventana Simulation Environment, 5.6 ed, http://www.vensim.com.

Taiz, L., Zeiger, E., 2010. Plant Physiology, 5 ed. Sinauer Associates 782 pp.

Taylor, R.M., Tobin, A.K., Bray, C.M., 1997. DNA damage and repair in plants, in: Lumsden, P.J. (ed.), Plants and UV-B Responses to Environmental Change. Cambridge University Press, Cambridge, UK, pp. 53-76.

Thines, N.J., Shipley, L.A., Bassman, J.H., Fellman, J.K., Mattison, D.S., Slusser, J.R., Gao, W., 2007. Effects of enhanced UV-B radiation on plant chemistry: nutritional consequences for a specialist and generalist lagomorph. J Chem Ecol 33, 1025-1039.

USDA, 2010. UV-B Monitoring and Research Program. http://uvb.nrel.colostate.edu/UVB/. Accessed January 2010.

Vasseur, D.A., Amarasekare, P., Rudolf, V.H.W., Levine, J.M., 2011. Eco-Evolutionary Dynamics Enable Coexistence via Neighbor-Dependent Selection. Am Nat 178, E96-E109.

Wargent, J.J., Moore, J.P., Roland Ennos, A., Paul, N.D., 2009. Ultraviolet Radiation as a Limiting Factor in Leaf Expansion and Development. Photochem Photobiol 85, 279-286.

Warren, J.M., Bassman, J.H., Eigenbrode, S., 2002. Leaf chemical changes induced in *Populus trichocarpa* by enhanced UV-B radiation and concomitant effects on herbivory by *Chrysomela scripta* (Coleoptera: Chrysomidae). Tree Physiol 22, 1137-1146.

Watkinson, A.R., 1981. Interference in Pure and Mixed Populations of *Agrostemma-Githago*. J Appl Ecol 18, 967-976.

Weber, S., 2005. Light-driven enzymatic catalysis of DNA repair: a review of recent biophysical studies on photolyase. Bba-Bioenergetics 1707, 1-23.

Winkel-Shirley, B., 2002. Biosynthesis of flavonoids and effects of stress. Curr Opin Plant Biol 5, 218-223.

Wright, I.J., Reich, P.B., Westoby, M., Ackerly, D.D., Baruch, Z., Bongers, F., Cavender-Bares, J., Chapin, T., Cornelissen, J.H.C., Diemer, M., Flexas, J., Garnier, E., Groom, P.K., Gulias, J., Hikosaka, K., Lamont, B.B., Lee, T., Lee, W., Lusk, C., Midgley, J.J., Navas, M.L., Niinemets, U., Oleksyn, J., Osada, N., Poorter, H., Poot, P., Prior, L., Pyankov, V.I., Roumet, C., Thomas, S.C., Tjoelker, M.G., Veneklaas, E.J., Villar, R., 2004. The worldwide leaf economics spectrum. Nature 428, 821-827.

Young, J.A., Evans, R.A., Raguse, C.A., Larson, J.R., 1981. Germinable Seeds and Periodicity of Germination in Annual Grasslands. Hilgardia 49, 1-37.

Zhang, Y., Jiang, H.-B., Qiu, B.-S., 2013. Effects of UVB Radiation on competition between the bloom-forming cyanobacterium *Microcystis aeruginosa* and the *Chlorophyceae Chlamydomonas microsphaera*1. Journal of Phycology 49, 318-328.

Table 3.1: Summary of the photosynthesis, respiration and temperature dependence for the growth forms considered

		Assigned values					
		Annuals			**Biennial**	**Perennial**	
Parameter definition	**Unit**	*C3 summer*	*C4 summer*	*C3 winter*		*Deciduous*	*Evergreens*
Photosynthesis							
Maximum plant mass net photosynthetic production rate multiplier (at 20℃)	$hour^{-1}$	0.045	0.100	0.045	0.045	0.018 (sun)	0.011
Temperature dependence of photosynthesis							
Minimum	℃	-1.0	2.5	-3.5	-1.0	-2.0	-4.0
Optimum		25.0	35.0	15.0	25.0	22.5	17.5
Maximum		45.0	55.0	42.5	45.0	42.5	38.5
Respiration							
Maximum plant mass respiration rate multiplier (at 20℃)	$hour^{-1}$	0.005	0.005	0.005	0.005	0.0035 (sun)	0.0007 (sun)
Temperature dependence of respiration					Q10 value of 2 for all plant types		

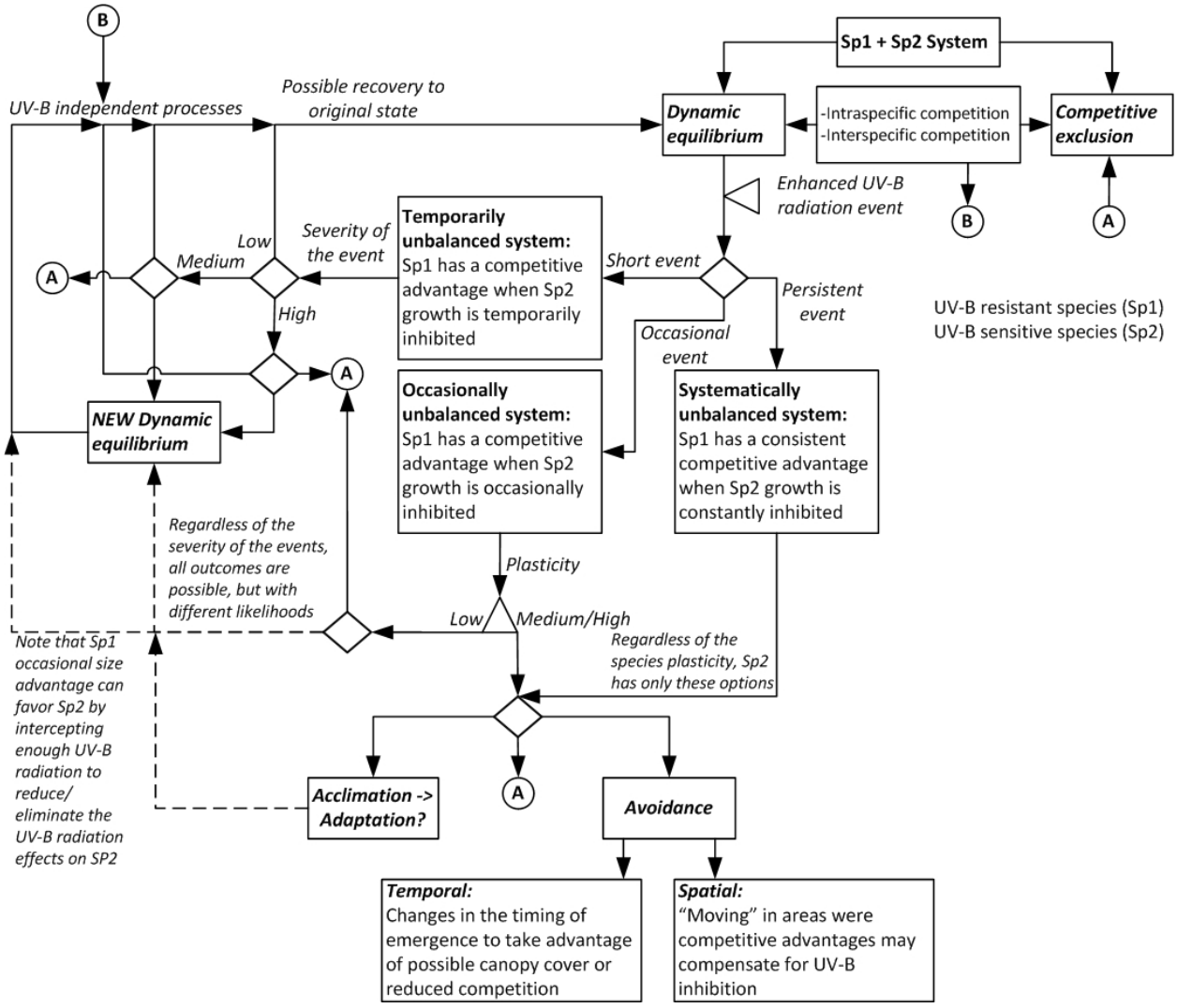

Figure 3.1: Conceptual model of UV-B radiation effects on a plant community.

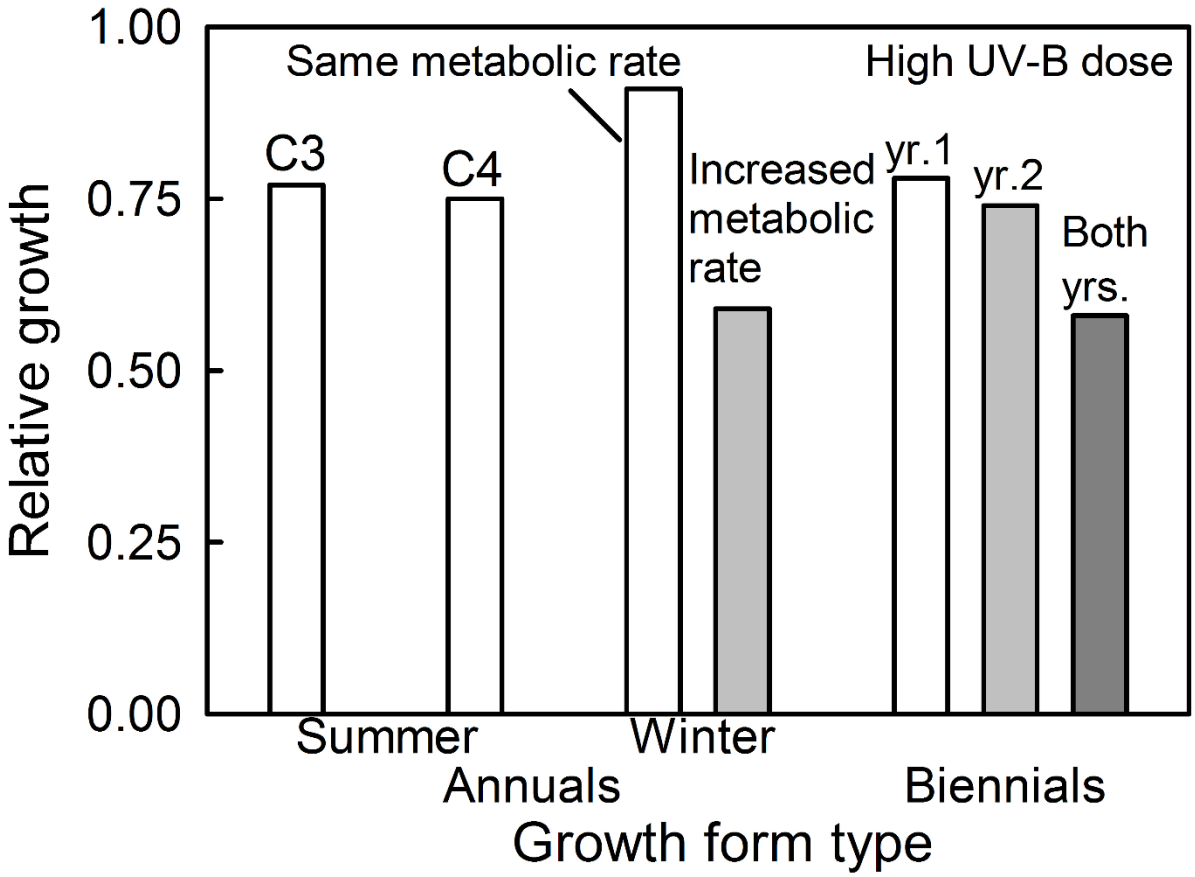

Figure 3.2: The effect of increased UV-B radiation on annuals and biennials.

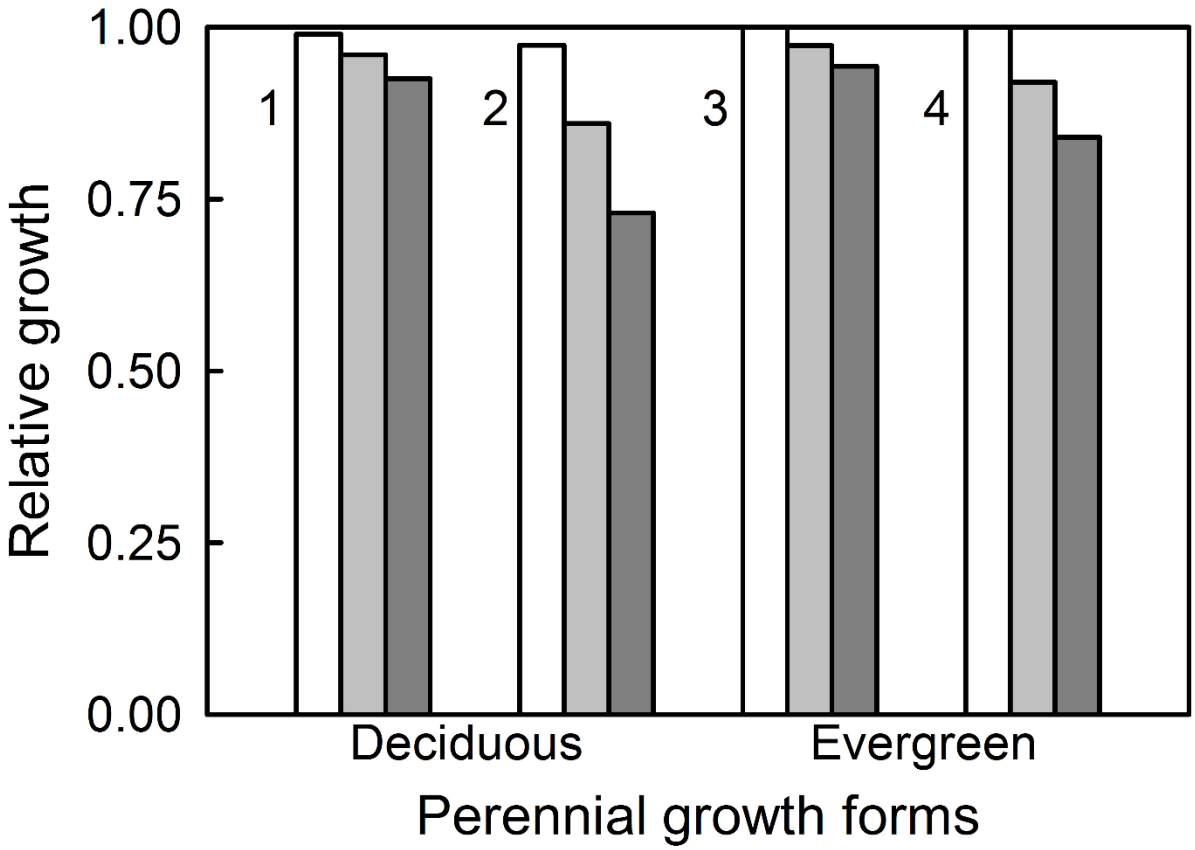

Figure 3.3: The effect of increased UV-B radiation on perennials: biomass year 1 (no fill bar), biomass year 2 (light gray bar), biomass year 3 (dark gray bar). Plot 1 and 3: increased metabolic rates only in leaves. Plot 2 and 4: Increased metabolic rates in whole plant. Note: plots for no increase in metabolic rates are not shown, since there were no effects.

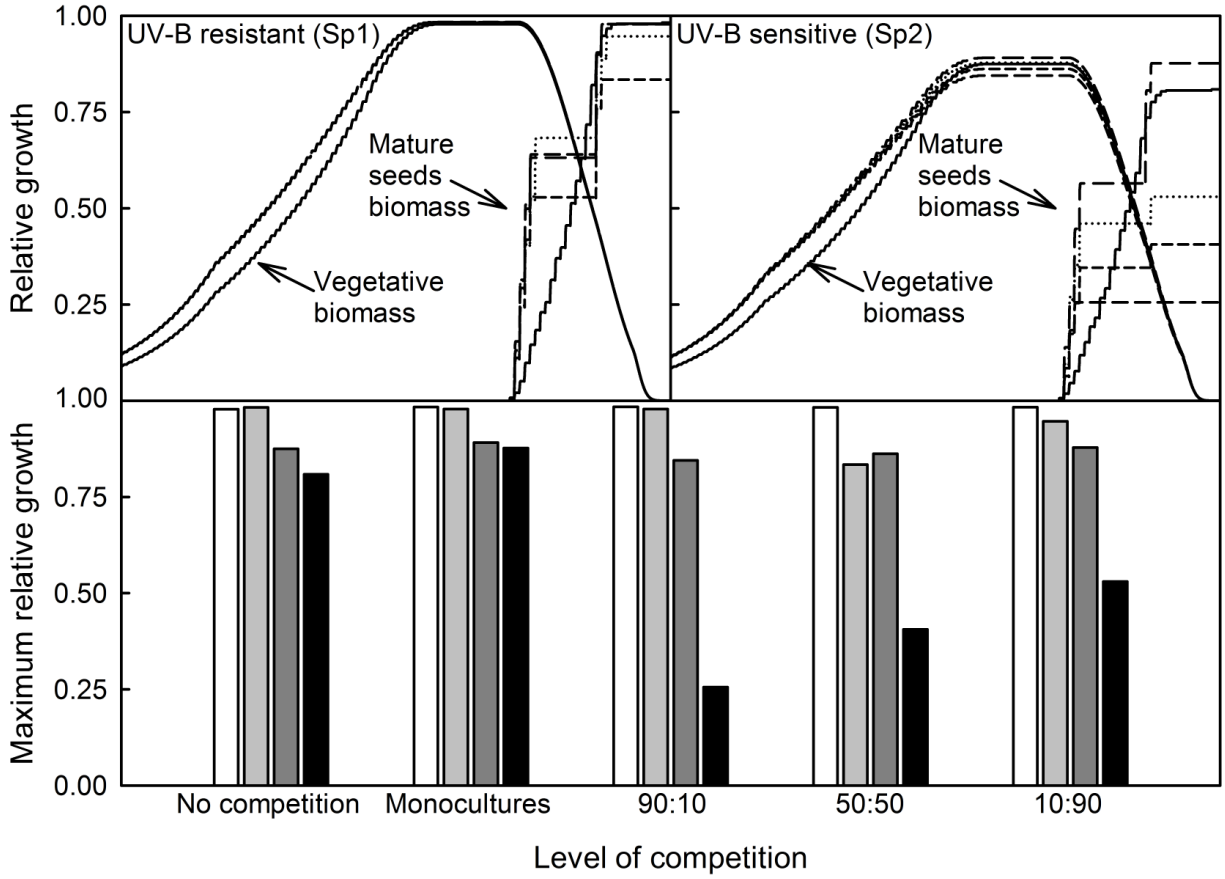

Figure 3.4: Effect of UV-B radiation in plant communities: no competition (solid line), monocultures (long dash line), Sp1:Sp2 ratio 90:10 (medium dash line), Sp1:Sp2 ratio 50:50 (short dash line), Sp1:Sp2 ratio 10:90 (dotted line). In bottom picture: Sp1 vegetative biomass (no fill bar), Sp1 mature seed biomass (light gray bar), Sp2 vegetative biomass (dark gray bar), Sp2 mature seeds biomass (black bar).

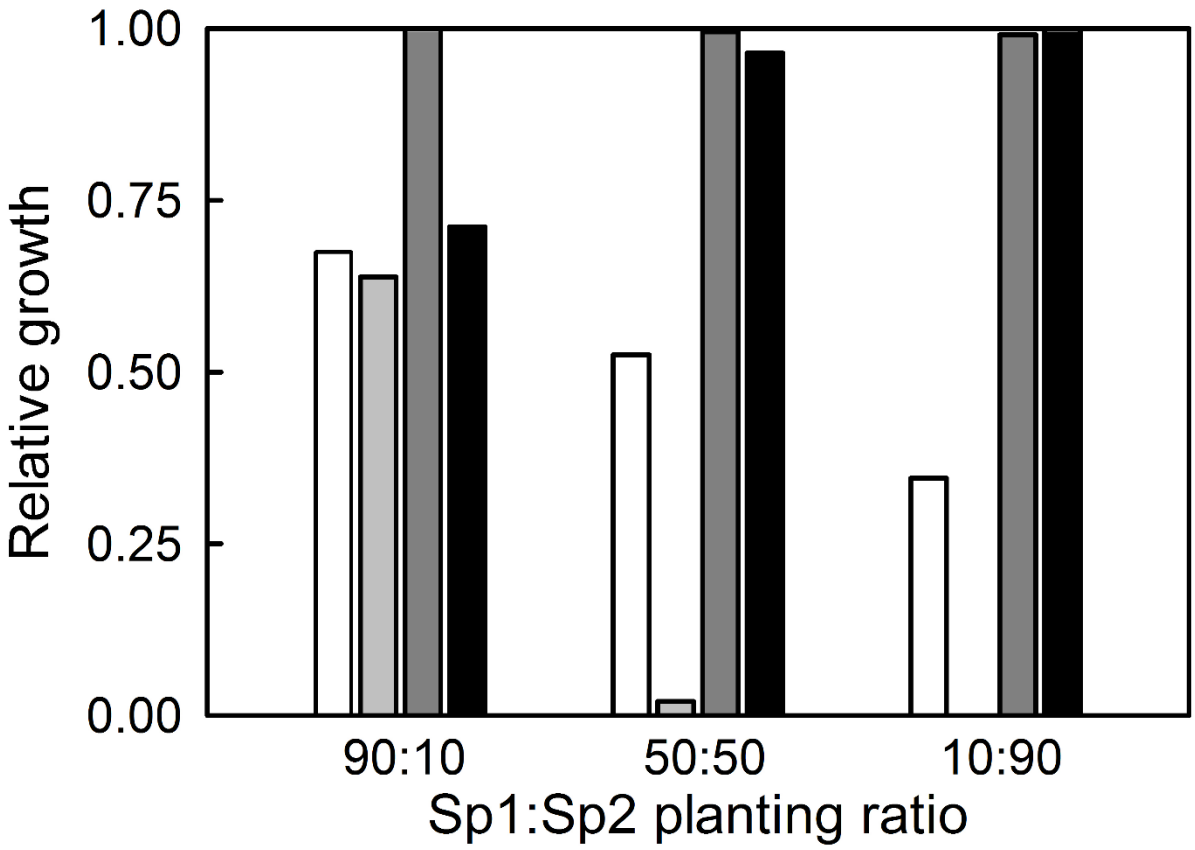

Figure 3.5: Effect of species 2 early seedling emergence: Sp1 vegetative biomass (no fill bar), Sp1 mature seed biomass (light gray bar), Sp2 vegetative biomass (dark gray bar), Sp2 mature seeds biomass (black bar).

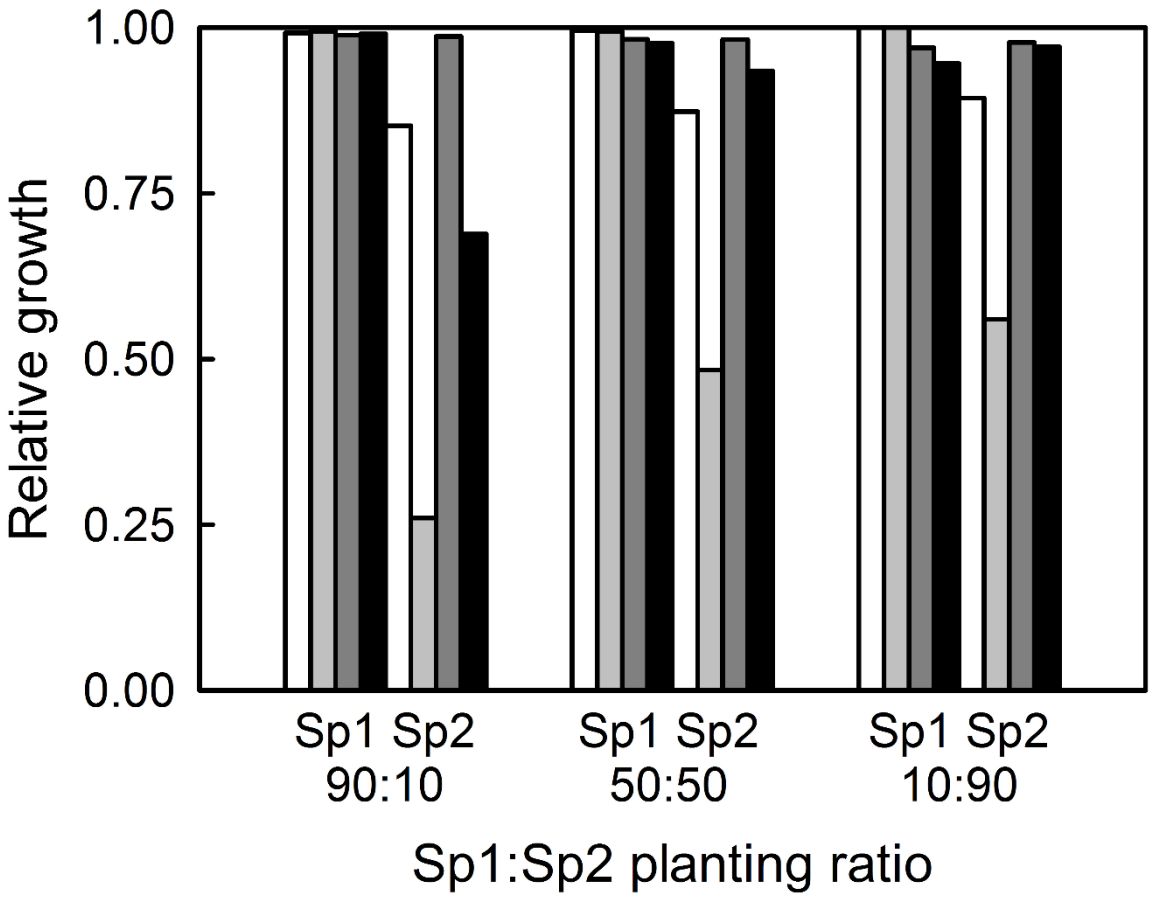

Figure 3.6: Effect of species 2 doubling in seed size: Sp1/Sp2 vegetative biomass for seed size ratio 1:1 (no fill bar), Sp1/Sp2 mature seed biomass for seed size ratio 1:1 (light gray bar), Sp1/Sp2 vegetative biomass for seed size ratio 1:2 (dark gray bar), Sp1/Sp2 mature seeds biomass for seed size ratio 1:1 (black bar).

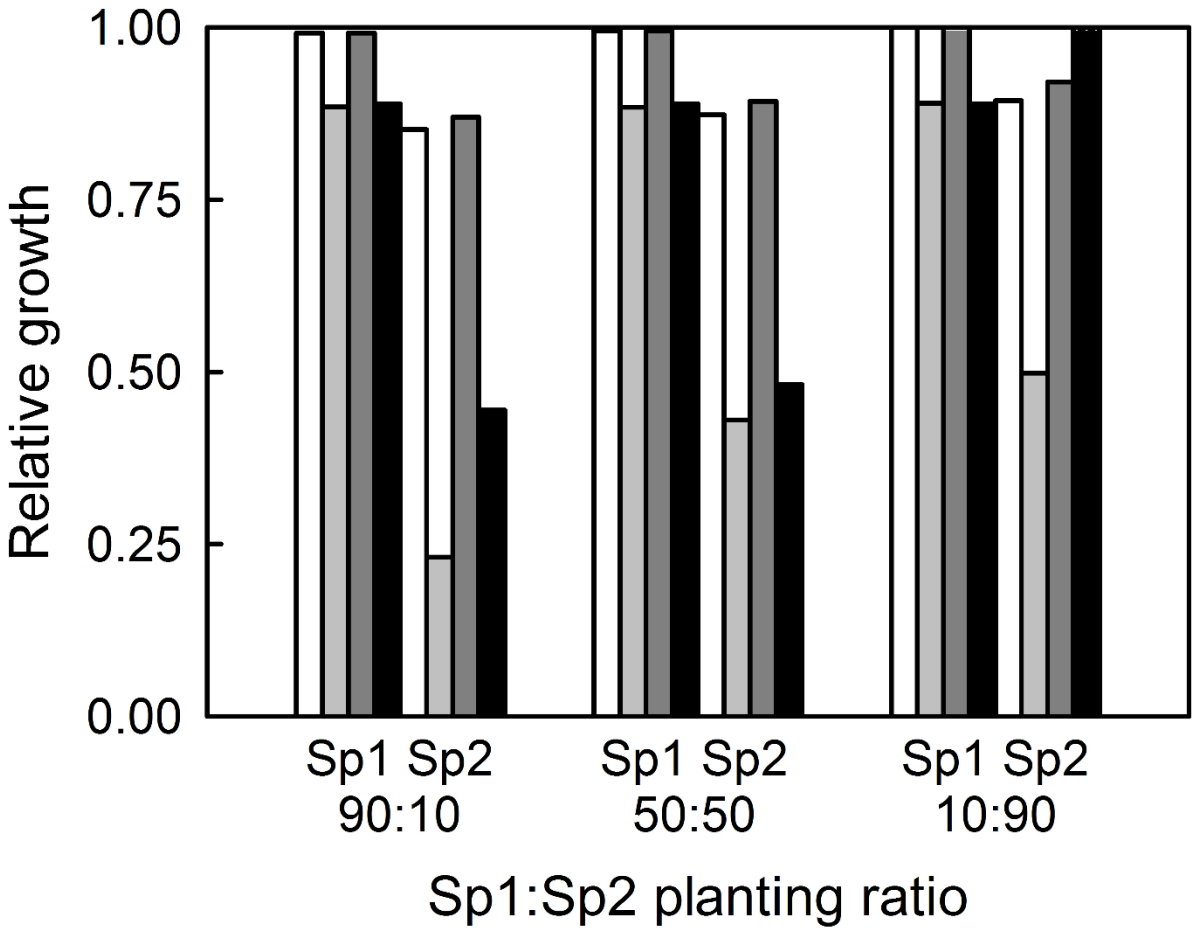

Figure 3.7: Effects of reproduction timing: Sp1/Sp2 vegetative biomass for same reproduction time (no fill bar), Sp1/Sp2 mature seed biomass for same reproduction time (light gray bar), Sp1/Sp2 vegetative biomass for Sp2 delay in reproduction (dark gray bar), Sp1/Sp2 mature seeds biomass for Sp2 delay in reproduction (black bar).

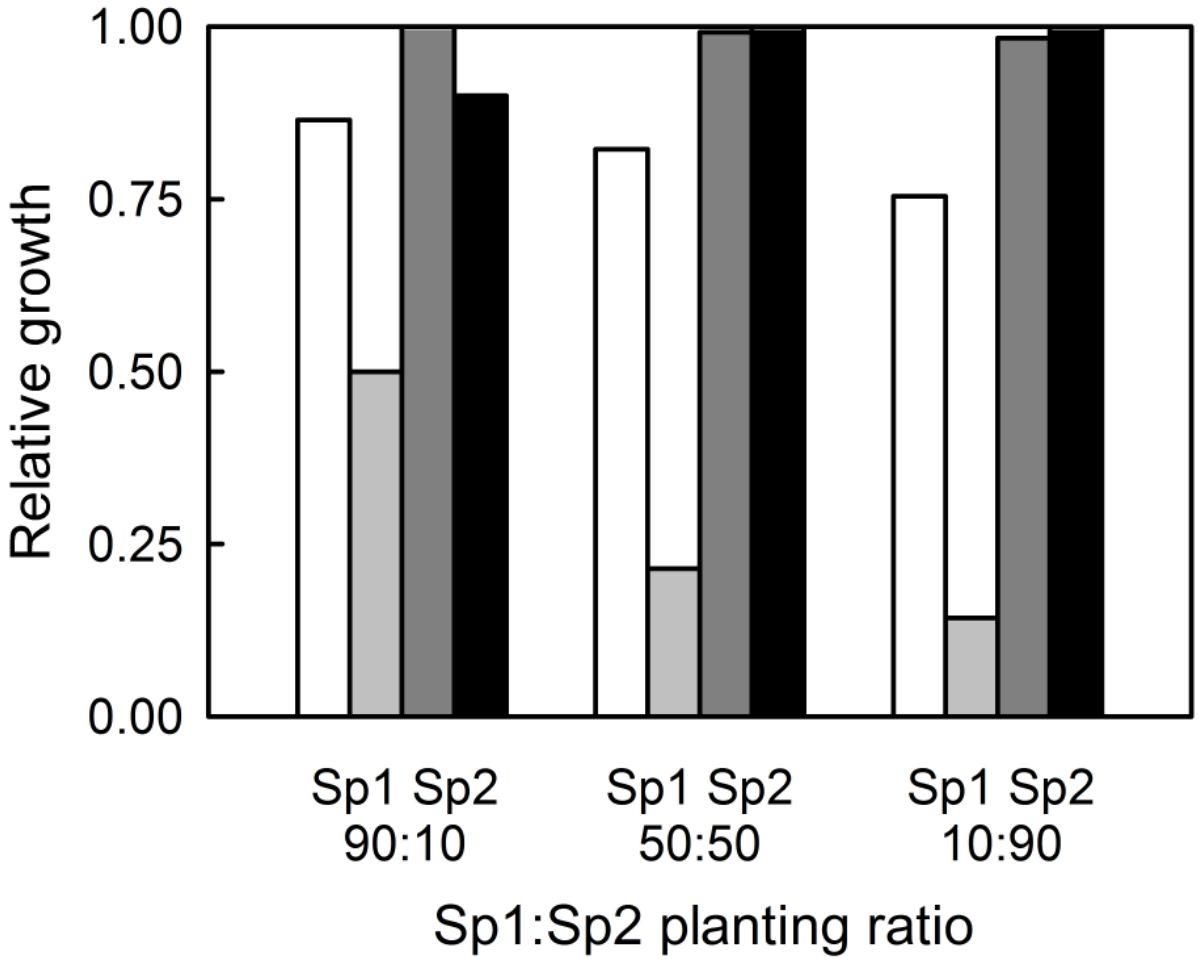

Figure 3.8: Effect of combination of changes in Sp2: Sp1 vegetative biomass (no fill bar), Sp1 mature seed biomass (light gray bar), Sp2 vegetative biomass (dark gray bar), Sp2 mature seeds biomass (black bar).

Appendices

Appendix A: Supporting information

A.1 Mathematical model

The dynamics of the UV-B radiation pathway in the leaf, the consequences for cell processes, and leaf morphology were expressed mathematically as follows.

A.1.1 UV-B radiation

Ultraviolet-B radiation data were obtained from the UV-B Monitoring and Research Program (UVMRP) over the period 2000-2009, for nearest location, Pullman, Washington. We used UV-B Langley calibrated data, considered more appropriate than lamp calibrated data for sunny and dry locations (USDA, 2010). Ultraviolet-B radiation data were averaged for the 10-year period, and for each month of the local growing season (May-September). Averaged hourly temperature data were obtained for Spokane, Washington from National Oceanic and Atmospheric Administration - National Climatic Data Center (NOAA, 2011).

A.1.2 Leaf optical properties

UV-B radiation reaching a leaf reflected, absorbed, or transmitted.

$$E = E_R + E_A + E_T \quad (1)$$

Where, E is the total solar UV-B radiation incident to the leaf, E_R total solar UV-B radiation reflected by the leaf, E_A total solar UV-B radiation absorbed by the leaf, and E_T total solar UV-B radiation transmitted through the leaf.

Fractions of the total solar UV-B radiation incident on the leaf are reflected and transmitted:

$$E_R = k_R E \quad (2)$$

$$E_T = k_T E \quad (3)$$

Where k_R and k_T are the total solar UV-B radiation incident on the leaf reflected and transmitted multipliers.

Solar UV-B radiation is absorbed by secondary metabolites, DNA and other leaf structures. The current model assumes that the fraction of the UV-B radiation not absorbed by

secondary metabolites is entirely absorbed by DNA. Although other leaf structures and cell components are important receptors of UV-B radiation, in the absence of quantitative evaluations of their relative absorptance, we made the assumption that DNA is the major recipient because of its key role in the sensitivity of plant species to UV-B radiation.

$$E_A = E_{A,SM} + E_{A,DNA} \quad (4)$$

Where, $E_{A,SM}$ is the UV-B radiation absorbed by secondary metabolites, and $E_{A,DNA}$ is the UV-B radiation absorbed by DNA.

The UV-B radiation absorbed by secondary metabolites was expressed as:

$$E_{A,SM} = k_{A,SM} E_A \quad (5)$$

Where $k_{A,SM}$ is the UV-B radiation absorbed by the secondary metabolites multiplier.

The radiation absorbed by secondary metabolites $E_{A,SM}$ is proportional to the quantity of secondary metabolites, and changes accordingly.

A.1.3 UV-B radiation induced DNA damage and repair

The general model for UV-B radiation induced damage in a leaf cell is as follows:

$$D_{DNA,CPD/6-4PP} = D_{I,CPD/6-4PP} - D_{PR,CPD/6-4PP} - D_{ER,CPD/6-4PP} \quad (6)$$

Where D_{DNA} represent the CPD/6-4PPs frequency present in the DNA, D_I are the CPD/6-4PPs frequencies induced by the UV-B radiation reaching the DNA, D_{PR} and D_{ER} are the CPD/6-4PPs frequencies photorepaired and excision repaired, respectively (CPD/6-4PPs Mb^{-1}).

Since the induced CPD/6-4PPs frequencies are UV-B radiation dose dependent, and spectra dependent, the CPD/6-4PPs frequency induced D_I becomes:

$$D_I = k_{A,DNA} k_c E_{A,DNA} \quad (7)$$

Where, $k_{A,DNA}$ is the UV-B radiation reaching the DNA - CPD/6-4PPs frequency conversion factor, and k_c is a correction factor multiplier due to differences in absorption spectra of epidermal secondary metabolites.

To evaluate k_c the DNA weighted UV-B radiation relationships (Caldwell et al., 1983; Setlow, 1974), were used for various absorption scenarios (Day et al., 1994; Lavola et al., 1997; Qi et al., 2003; Schmelzer et al., 1988b; Sisson, 1981).

The total DNA weighted UV-B exposure is given by

$$E_{A,DNA,weighted} = \int_{T_1}^{T_2} \int_{280}^{320} S_\lambda E_{A,DNA,\lambda} d\lambda dt \quad (8)$$

and the action spectra for DNA damage was given by (Caldwell et al., 1983; Setlow, 1974).

$$S_\lambda = e^{13.82\left(\frac{1}{1+e^{\frac{\lambda-310}{9}}}-1\right)} \quad (9)$$

Where, S_λ is the action spectra for DNA damage (Caldwell et al., 1983; Setlow, 1974), $E_{A,DNA,\lambda}$ (Wm^{-2} nm^{-1}) is the radiant flux density incident on the surface per unit of wavelength interval reaching the DNA, λ (nm) is the wavelength, T_1 and T_2 is the time interval the total exposure is calculated.

Both the CPD/6-4PPs frequencies photorepaired (D_{PR}), and excision repaired (D_{ER}) are proportional to the level of damage induced (Hidema et al., 2001; Hidema et al., 1997; Taylor et al., 1997).

$$D_{PR/ER,CPD/6-4PP} = rD_{I,CPD/6-4PP} \quad (10)$$

Since photorepair and excision repair mechanisms are enzyme mediated, the rates of repair were considered to follow a basic Michaelis-Menten model (Lodish et al., 2008) until the CPD/6-4 PP photolyase reach a level of saturation, followed to a decline in rates to zero, which is the instant cell apoptosis corresponding to the level of damage that disturbs instantaneous the cell activity (Figure A1).

This relationship is adjusted accordingly for photorepair: in the absence of PAR radiation the rate of repair is zero.

$$r = \begin{cases} \frac{r_{max}D_{I,CPD/6-4PP}}{k_m+D_{I,CPD/6-4PP}} & 0 \le D_{I,CPD/6-4PP} \le k_s \\ b_0 - b_1 D_{I,CPD/6-4PP} & k_s < D_{I,CPD/6-4PP} \le k_a \end{cases} \quad (11)$$

Where, r_{max} is the maximum rate of repair, k_m is the Michaelis constant (the concentration of substrate that gives exactly a rate half of r_{max}), k_s is the enzyme saturation point, k_a is the level of DNA damage that causes instant cellular apoptosis , b_0 and b_1 are the linear regression parameters for repair rate decline (Figure A1).

The temperature dependence of both CPD/6-4 PP induction and repair were considered to follow a polynomial relationship of the form:

$$r(\%) = b_{\circ\mathrm{C},0} + b_{\circ\mathrm{C},1}\,℃ + b_{\circ\mathrm{C},2}\,℃^2 \quad (12)$$

Where, $r(\%)$ is the CPD/6-4 PP induction/repair rates, $b_{\circ\mathrm{C},0}$, $b_{\circ\mathrm{C},1}$, and $b_{\circ\mathrm{C},2}$ the coefficients of the polynomial relationship, and ℃ is the temperature (℃).

A.1.4 Leaf growth and development

The processes governing leaf progression were grouped in three major stages: expansion, longevity, and senescence. Leaf expansion refers to the period when leaf increases its surface from the leaf primordium to the maximum area of the leaf. Longevity refers to the period beginning with leaf expansion until complete senescence. Leaf senescence refers to the period when the leaf starts to exhibit chlorophyll loss until cell-leaf death (Nooden, 2004; Srivastava, 2002).

To model the leaf growth, we chose the beta sigmoid function, which has few, unique, and readily interpretable parameters (Muller et al., 2006; Yin et al., 2003). In the beta function the starting and ending times of growth and senescence are clearly defined, and it is a function of seven biologically relevant parameters (Figure A2).

Thus, leaf area dynamics (Figure A2) were simulated as follows:

$$A = \begin{cases} 0 & if\ t < t_{b,g} \\ A_{max}\left(1 + \frac{t_{e,g} - t}{t_{e,g} - t_{m,g}}\right)\left(\frac{t - t_{b,g}}{t_{e,g} - t_{b,g}}\right)^{\frac{t_{e,g} - t_{b,g}}{t_{e,g} - t_{m,g}}} & if\ t_{b,g} \le t \le t_{e,g} \\ A_{max} & if\ t_{e,g} \le t \le t_{b,s} \\ A_{max}\left[1 - \left(1 + \frac{t_{e,s} - t}{t_{e,s} - t_{m,s}}\right)\left(\frac{t - t_{b,s}}{t_{e,s} - t_{b,s}}\right)^{\frac{t_{e,s} - t_{b,s}}{t_{e,s} - t_{m,s}}}\right] & if\ t_{b,s} \le t \le t_{e,s} \\ 0 & if\ t > t_{e,s} \end{cases} \quad (13)$$

Where, $t_{b,g}$, $t_{m,g}$ and $t_{e,g}$ are time when growth begins, time of inflection, and time of cessation of growth, respectively; $t_{b,s}$, $t_{m,s}$ and $t_{e,s}$ are time when senescence begins, time of inflection, and time of cessation of senescence, respectively; A_{max} is the maximum relative leaf area.

Leaf growth was expressed as a discrete process:

$$A_{t+1} = \lambda(t)\, A_t \quad (14)$$

Where, A_t and A_{t+1} are leaf area at time t and t+1, respectively; $\lambda(t)$ is the time-dependent rate of increase, derived from Equation S13. These rates of increase were corrected according to the level of DNA damage.

Leaf growth process was considered to be driven initially by active cell division, followed by a decrease in the number of dividing cells, active cell expansion and differentiation, and leaf maturity (Beemster et al., 2005). Thus, increased UV-B radiation was considered to cause delays in cell division and expansion, during the leaf growth process (i.e., reduced $\lambda(t)$, time-dependent rates of leaf increase).

A.2 Parameter estimation

A.2.1 UV-B radiation

The ten year averaged UV-B radiation for the Pullman, Washington station of the UV-B Monitoring and Research Program (UVMRP) was considered the baseline UV-B radiation environment. Increases of 100% in UV-B radiation scenarios were considered in our simulations. The parameter estimates are presented in Table 1.1.

A.2.2 Leaf optical properties

The range for the leaf reflectance was considered $k_R = 0.05 - 0.7$ of the incident solar UV-B radiation, while the one for transmittance was $k_T = 0.01 - 0.1$ (Gausman et al., 1975; Robberecht and Caldwell, 1978; Robberecht et al., 1980).

The epidermal pigments absorption was considered $k_{A,SM} = 0.94$ (Robberecht and Caldwell, 1978). Changes in epidermal pigments absorption with increased UV-B radiation were considered to range between $k^*_{A,SM} = -0.2 - 1$ per kJ m^{-2} d^{-1} (Bornman et al., 1997; Day and Demchik, 1996; de la Rosa et al., 2001; Kolb et al., 2001; Li et al., 1993b; Liu et al., 1995; Meijkamp et al., 1999; Olsson et al., 1998; Sheahan, 1996; Tegelberg et al., 2003; Tevini et al., 1981; Tevini et al., 1982, 1983; Vandestaaij et al., 1995).

A.2.3 UV-B radiation induced DNA damage and repair

Since the UV-B radiation induced damage to DNA is a photochemical process, the rate of CPD induction should be similar for most species. Studies on rice varieties cultivated under laboratory conditions indicate that a dose of unweighted UV-B radiation of 1 kJ m^{-2} at

the leaf surface induces approximately 4 CPDs Mb^{-1} (Hidema and Kumagai, 1998; Hidema et al., 2000; Hidema et al., 1997; Takeuchi et al., 1996), depending on the growth conditions and UV-B action spectra. To quantify the rate of CPD induction as a function of the dose of UV-B radiation reaching the DNA, we considered two extreme scenarios regarding the UV-B absorptance of epidermal secondary metabolites. Firstly, an epidermal absorptance of 0.94 leads to a $k_{A,DNA} = 74$ CPD Mb^{-1} kJ^{-1} m^2 h. We consider this value as an overestimation of the true value, since plants in these studies were cultivated without UV-B radiation exposure, and the doses used to induce CPDs were over 10-20 times greater than the ambient conditions. Secondly, if we consider an epidermal absorptance of about 0.03 – resulting from quantifications of secondary metabolites in rice species grown with and without UV-B radiation supplementation, and the expected epidermal absorptance under ambient UV-B radiation conditions (Hidema et al., 1997; Kang et al., 1998; Kon et al., 2004; Robberecht and Caldwell, 1978), we come with a value of $k_{A,DNA} = 5$ CPD Mb^{-1} kJ^{-1} m^2 h. The second value we believe to be an underestimation of the true value due to the poor understanding of the dynamics of secondary metabolites in epidermis, at different UV-B radiation exposures. The range considered was $k_{A,DNA} = 5 - 74$ CPD Mb^{-1} kJ^{-1} m^2 h.

We assumed that low UV-B radiation produces CPD to 6-4PP ratio of 9:1, and high UVB doses produce ratios of 6:4 (Sancar, 2003), since no published data were available. This model used the following arbitrary rule: UV-B radiation induced CPD to 6-4PP ratio is 9:1 for the 1st quartile of the overall UV-B radiation for the growing season, 8:2 for the 2nd quartile, 7:3 for the 3rd quartile, and 6:4 for the 4th quartile.

Species with maximum absorption at shorter wavelengths (Figure A3) had up to 70% less DNA weighted UV-B radiation reaching the DNA than the species exhibiting equal absorptance across wavelengths, while species with maximum absorption at longer wavelengths (Figure A3) had up to 70% higher DNA weighted UV-B radiation reaching the DNA than the species exhibiting equal absorptance across wavelengths. These translates for initial values for k_c values of 0.3 to 1.7 depending on the absorption trend considered, with $k_c = 1$ for species with equal epidermal absorptance across all UV-B wavelengths.

The Michaelis-Menten photorepair model parameters could not be inferred from the studies considered (Hidema et al., 2001; Hidema et al., 1997; Hidema et al., 2007; Iwamatsu et al., 2008; Kang et al., 1998; Quaite et al., 1994), since in most of these studies the enzyme

saturation was not reached. Therefore, the Michaelis-Menten photorepair model was approximated with a linear rate of repair increase as a function of CPD concentration followed by maximum rate of repair (corresponding to enzyme saturation). Thus, equation S11 becomes:

$$r = \begin{cases} aD_{I,CPD/6-4PP} & 0 \le D_{I,CPD/6-4PP} \le r_{max}/a \\ r_{max} & r_{max}/a \le D_{I,CPD/6-4PP} \le k_s \\ b_0 - b_1 D_{I,CPD/6-4PP} & k_s < D_{I,CPD/6-4PP} \le k_a \\ 0 & k_s < D_{I,CPD/6-4PP} \end{cases} \quad (15)$$

The proposed values for each CPD photorepair and dark repair mechanisms are presented in Table 1.1 (Hidema et al., 2001; Hidema et al., 1997; Hidema et al., 2007; Iwamatsu et al., 2008; Kang et al., 1998; Quaite et al., 1994). The estimation of k_s (enzyme saturation point), and k_a (level of DNA damage that causes instant cellular apoptosis) was more difficult. In *Oryza* and *Medicado* varieties, the rate of CPD repair was not inhibited at induced levels of $50 - 70\ CPD\ Mb^{-1}$, and no instantaneous apoptosis was observed (Hidema et al., 2001; Hidema et al., 1997; Hidema et al., 2007; Iwamatsu et al., 2008; Kang et al., 1998; Quaite et al., 1994). Based on the efficiency of protection and repair mechanisms, some bacterial species can recover from DNA damage induced-levels up to $400\ CPD\ Mb^{-1}$ (Zenoff et al., 2006). Thus, we considered arbitrary $k_s = 300\ CPD\ Mb^{-1}$ and $k_a = 500\ CPD\ Mb^{-1}$ for both light and dark repair mechanisms (see Table 1.1).

Since photorepair of 6-4 photoproducts is 70% more efficient that CPD photorepair (Chen et al., 1994; Jiang et al., 1997), and NER repair of 6-4PP is approximately 10-fold faster than NER repair of CPDs (de Lima-Bessa et al., 2008; Lo et al., 2005), we adjusted the values accordingly (Table 1.1). Since the reviewed literature did not even hint at the k_s (enzyme saturation point), and k_a (level of DNA damage that causes instant cellular apoptosis) for 6-4PP repair, we considered the same values as for CPD repair. Parameters b_0 and b_1 were calculated for each r_{max}, k_a, and k_s combinations.

The model coefficients for the temperature dependence of the DNA damage induction and repair (Figure A4, see Table 1.1) were inferred from (Li et al., 2002; Takeuchi et al., 1996; Waterworth et al., 2002).

A.2.4 Leaf expansion, longevity, and senescence

Three leaf expansion parameters sets were considered: fast growing leaves (growth completed in seven days), medium growing leaves (growth completed in 15 days), and slow growing leaves (growth completed in 30 days). The corresponding estimates for the equation 13 parameters are presented in Table 1.1.

Published studies did not provide sufficient data for a quantitative relation between the levels of photoproducts and the percent of apoptotic cells (Figure A5).

Instead, a linear equation inferred from Lo et al. (Lo et al., 2005) was used (see Table 1.1), with the warning that percent apoptosis predictions for CPD and 6-4PP levels above 55 CPD Mb^{-1} and 12 6-4PP Mb^{-1} are probably erroneous. To link the UV-B radiation induced DNA damage to leaf expansion, we used the following causal loop: if DNA damage is lower than 10 $CPD\ Mb^{-1}$, then cell division and cell expansion is unaffected; else if DNA damage is higher than 10 $CPD\ Mb^{-1}$, but lower than 500 $CPD\ Mb^{-1}$, cell division is delayed for 8-16 hours; if, after 8-16 hours, DNA damage is lower than 10 $CPD\ Mb^{-1}$, then cell division and cell expansion is resumed; if DNA damage is lower than 10 $CPD\ Mb^{-1}$ sooner then 8-16 hours, then cell division and cell expansion is resumed; if after 8-16 hours, DNA damage is higher than 10 $CPD\ Mb^{-1}$, or if the DNA damage is higher than 500 $CPD\ Mb^{-1}$, cells undergo apoptosis (de Lima-Bessa et al., 2008; Lo et al., 2005; Zenoff et al., 2006).

Although the leaf growth process is driven initially by active cell division, followed by, a decrease in the number of dividing cells, active cell expansion and differentiation, and leaf maturity (Beemster et al., 2005), the leaf expansion delays were not considered in this model. It has been showed, in both laboratory and field studies, that either processes, or either one, is responsible for leaf expansion inhibitions (González et al., 1998; Hofmann et al., 2003; Hopkins et al., 2002; Wargent et al., 2009b). Moreover, there are differences in the processes responsible for the cell expansion inhibitions for leaves from different locations on the same plant (González et al., 1998). Some of these studies are comparing no UV-B radiation treatments with ambient UV-B radiation treatments, or apply the supplemental UV-B radiation for only brief periods of time. It is possible that, similar to the pigment content, solar UV-B radiation might have a greater influence on the epidermal pigments content than the increased UV-B radiation (Ryan et al., 1998; Ryan et al., 2002). We recognize that the photomorphogenic responses are important, and in some species may be the primary process

leading the observed phenotypic plant responses to enhanced UV-B radiation. Since the rates for cell expansion inhibition are unclear at this time, all delays during the leaf growth were approximated by delays in cell division. This approximation may reduce the predictive power of the model.

A.3 References

Beemster GT, De Veylder L, Vercruysse S et al. (2005) Genome-wide analysis of gene expression profiles associated with cell cycle transitions in growing organs of *Arabidopsis*. Plant Physiology, 138, 734-743.

Bornman JF, Reuber S, Cen Y-O, Weissenbock G (1997) Ultraviolet radiation as a stress factor and the role of protective pigments. In: Plants and UV-B: responses to environmental change. (ed Lumsden PJ) pp Page. Cambridge, UK, Cambridge University Press.

Caldwell MM, Gold WG, Harris G, Ashurst CW (1983) A Modulated Lamp System for Solar Uv-B (280-320 Nm) - Supplementation Studies in the Field. Photochemistry and Photobiology, 37, 479-485.

Chen JJ, Mitchell DL, Britt AB (1994) Light-Dependent Pathway for the Elimination of Uv-Induced Pyrimidine-(6-4) Pyrimidinone Photoproducts in *Arabidopsis*. Plant Cell, 6, 1311-1317.

Day TA, Demchik SM (1996) Influence of enhanced UV-B radiation on biomass allocation and pigment concentrations in leaves and reproductive structures of greenhouse-grown *Brassica rapa*. Vegetatio, 127, 109-116.

Day TA, Howells BW, Rice WJ (1994) Ultraviolet absorption and epidermal-transmittance spectra in foliage. Physiologia Plantarum, 92, 207-218.

De La Rosa TM, Julkunen-Tiitto R, Lehto T, Aphalo PJ (2001) Secondary metabolites and nutrient concentrations in silver birch seedlings under five levels of daily UV-B exposure and two relative nutrient addition rates. New Phytologist, 150, 121-131.

De Lima-Bessa KM, Armelini MG, Chigancas V, Jacysyn JF, Amarante-Mendes GP, Sarasin A, Menck CF (2008) CPDs and 6-4PPs play different roles in UV-induced cell death in normal and NER-deficient human cells. DNA Repair (Amst), 7, 303-312.

Gausman HW, Rodriguez RR, Escobar DE (1975) Ultraviolet Radiation Reflectance, Transmittance, and Absorptance by Plant Leaf Epidermises1. Agron. J., 67, 720-724.

González R, Mepsted R, Wellburn AR, Paul ND (1998) Non-photosynthetic mechanisms of growth reduction in pea (*Pisum sativum* L.) exposed to UV-B radiation. Plant, Cell & Environment, 21, 23-32.

Hidema J, I.-K. S, Sato T, Kumagai T (2001) Relationship between ultraviolet-B sensitivity and cyclobutane pyrimidine dimer photorepair in rice. Journal of Radiation Research, 42, 295-303.

Hidema J, Kumagai T (1998) UV-B induced cyclobutil pyrimidine dimer and photorepair with progress of growth and leaf age in rice. Journal of Photochemistry and Photobiology B: Biology, 43, 121-127.

Hidema J, Kumagai T, Sutherland BM (2000) UV radiation-sensitive Norin 1 rice contains defective cyclobutane pyrimidine dimer photolyase. Plant Cell, 12, 1569-1578.

Hidema J, Kumagai T, Sutherland JC, Sutherland BM (1997) Ultraviolet B - sensitive rice cultivar deficient in cyclobutyl pyrimidine dimer repair. Plant Physiology, 113, 39-44.

Hidema J, Taguchi T, Ono T, Teranishi M, Yamamoto K, Kumagai T (2007) Increase in CPD photolyase activity functions effectively to prevent growth inhibition caused by UVB radiation. Plant Journal, 50, 70-79.

Hofmann RW, Campbell BD, Bloor SJ, Swinny EE, Markham KR, Ryan KG, Fountain DW (2003) Responses to UV-B radiation in *Trifolium repens* L. - physiological links to plant productivity and water availability. Plant Cell and Environment, 26, 603-612.

Hopkins L, Bond MA, Tobin AK (2002) Ultraviolet-B radiation reduces the rates of cell division and elongation in the primary leaf of wheat (*Triticum aestivum* L. cv Maris Huntsman). Plant, Cell and Environment, 25, 617-624.

Iwamatsu Y, Aoki C, Takahashi M et al. (2008) UVB sensitivity and cyclobutane pyrimidine dimer (CPD) photolyase genotypes in cultivated and wild rice species. Photochem Photobiol Sci, 7, 311-320.

Jiang C-Z, Yee J, Mitchell DL, Britt AB (1997) Photorepair mutants of *Arabidopsis*. Proceedings of the National Academy of Sciences, 94, 7441-7445.

Kang HS, Hidema J, Kumagai T (1998) Effects of light environment during culture on UV-induced cyclobutyl pyrimidine dimers and their photorepair in rice (Oryza sativa L.). Photochemistry and Photobiology, 68, 71-77.

Kolb CA, Kaser MA, Kopecky J, Zotz G, Riederer M, Pfundel EE (2001) Effects of natural intensities of visible and ultraviolet radiation on epidermal ultraviolet screening and photosynthesis in grape leaves. Plant Physiology, 127, 863-875.

Kon H, Ichibayashi R, Matsuoka N (2004) Changes of Diffuse UV-B Radiation on Clear Sky Days. Journal of Agricultural Meteorology, 60, 285-290.

Lavola ANU, Julkunen-Tiitto R, Aphalo P, De La Rosa T, Lehto T (1997) The effect of u.v.-B radiation on u.v.-absorbing secondary metabolites in birch seedlings grown under simulated forest soil conditions. New Phytologist, 137, 617-621.

Li J, Ou-Lee TM, Raba R, Amundson RG, Last RL (1993) *Arabidopsis* Flavonoid Mutants Are Hypersensitive to UV-B Irradiation. The Plant Cell Online, 5, 171-179.

Li SS, Paulsson M, Bjorn LO (2002) Temperature-dependent formation and photorepair of DNA damage induced by UV-B radiation in suspension-cultured tobacco cells. Journal of Photochemistry and Photobiology B-Biology, 66, 67-72.

Liu L, Gitz DC, Mcclure JW (1995) Effects of Uv-B on Flavonoids, Ferulic Acid, Growth and Photosynthesis in Barley Primary Leaves. Physiologia Plantarum, 93, 725-733.

Lo HL, Nakajima S, Ma L, Walter B, Yasui A, Ethell DW, Owen LB (2005) Differential biologic effects of CPD and 6-4PP UV-induced DNA damage on the induction of apoptosis and cell-cycle arrest. BMC Cancer, 5, 135.

Lodish H, Berk A, Kaiser CA et al. (2008) Molecular Cell Biology, New York, NY, W.H. Freeman and Company.

Meijkamp B, Aerts R, Van Der Staaij J, Tosserams M, Ernst W, Rozema J (1999) Effects of UV-B on secondary metabolites on plants. In: Stratospheric Ozone Depletion: The Effects of Enhanced Uv-B Radiation on Terrestrial Ecosystems. (ed Rozema J) pp Page. Leiden, The Netherlands, Backhuys Publishers.

Muller J, Behrens T, Diepenbrock W (2006) Use of a new sigmoid growth equation to estimate organ area indices from canopy area index in winter oilseed rape (*Brassica napus* L.). Field Crops Research, 96, 279-295.

NOAA (2011) National Climatic Center. http://www.ncdc.noaa.gov/. Accessed May 2011. pp Page.

Nooden LD (2004) Introduction. In: Plant Cell Death Processes. (ed Nooden LD) pp Page. San Diego, CA, Academic Press.

Olsson LC, Veit M, Weissenbock G, Bornman JF (1998) Differential flavonoid response to enhanced UV-B radiation in *Brassica napus*. Phytochemistry, 49, 1021-1028.

Qi Y, Bai S, Heisler GM (2003) Changes in ultraviolet-B and visible optical properties and absorbing pigment concentrations in pecan leaves during a growing season. Agricultural and Forest Meteorology, 120, 229-240.

Quaite FE, Takayanagi S, Ruffini J, Sutherland JC, Sutherland BM (1994) DNA damage levels determine cyclobutil pyrimidine dimer repair mechanisms in alfalfa seedlings. The Plant Cell, 6, 1635-1641.

Robberecht R, Caldwell MM (1978) Leaf Epidermal Transmittance of Ultraviolet-Radiation and Its Implications for Plant Sensitivity to Ultraviolet-Radiation Induced Injury. Oecologia, 32, 277-287.

Robberecht R, Caldwell MM, Billings WD (1980) Leaf ultraviolet optical properties along a latitudinal gradient in the arctic-alpine life zone. Ecology, 61, 612-619.

Ryan KG, Markham KR, Bloor SJ, Bradley JM, Mitchell KA, Jordan BR (1998) UVB radiation induced increase in quercetin: Kaempferol ratio in wild-type and transgenic lines of Petunia. Photochemistry and Photobiology, 68, 323-330.

Ryan KG, Swinny EE, Markham KR, Winefield C (2002) Flavonoid gene expression and UV photoprotection in transgenic and mutant Petunia leaves. Phytochemistry, 59, 23-32.

Sancar A (2003) Structure and Function of DNA Photolyase and Cryptochrome Blue-Light Photoreceptors. Chemical Reviews, 103, 2203-2238.

Schmelzer E, Jahnen W, Hahlbrock K (1988) In situ localization of light-induced chalcone synthase mRNA, chalcone synthase, and flavonoid end products in epidermal cells of parsley leaves. Proceedings of the National Academy of Sciences, 85, 2989-2993.

Setlow RB (1974) Wavelengths in Sunlight Effective in Producing Skin Cancer - Theoretical Analysis. Proceedings of the National Academy of Sciences of the United States of America, 71, 3363-3366.

Sheahan JJ (1996) Sinapate esters provide greater UV-B attenuation than flavonoids in *Arabidopsis thaliana* (Brassicaceae). American Journal of Botany, 83, 679-686.

Sisson WB (1981) Photosynthesis, Growth, and Ultraviolet Irradiance Absorbance of *Cucurbita pepo* L. Leaves Exposed to Ultraviolet-B Radiation (280-315 nm). Plant Physiology, 67, 120-124.

Srivastava LM (2002) Plant growth and development: hormones and environment, San Diego, CA, Academic Press.

Takeuchi Y, Murakami M, Nakajima S, Kondo S, Nikaido O (1996) Induction and repair of damage to DNA in cucumber cotyledons irradiated with UV-B. Plant Cell Physiology, 37, 181-187.

Taylor RM, Tobin AK, Bray CM (1997) DNA damage and repair in plants. In: Plants and UV-B Responses to Environmental Change. (ed Lumsden PJ) pp Page. Cambridge, UK, Cambridge University Press.

Tegelberg R, Veteli T, Aphalo PJ, Julkunen-Tiitto N (2003) Clonal differences in growth and phenolics of willows exposed to elevated ultraviolet-B radiation. Basic and Applied Ecology, 4, 219-228.

Tevini M, Iwanzik W, Thoma U (1981) Some Effects of Enhanced Uv-B Irradiation on the Growth and Composition of Plants. Planta, 153, 388-394.

Tevini M, Thoma U, Iwanzik W (1982) Effect of enhanced UV-B radiation on development and composition of plants. In: Biological Effects of UV-B Radiation: Workshop: Papers. (eds

Bauer H, Caldwell MM, Tevini M, Worrest RC) pp Page. Munich, Germany, Gesellschaft fur Strahlen- und Umweltforschung.

Tevini M, Thoma U, Iwanzik W (1983) Effects of Enhanced Uv-B Radiation on Germination, Seedling Growth, Leaf Anatomy and Pigments of Some Crop Plants. Zeitschrift Fur Pflanzenphysiologie, 109, 435-448.

USDA (2010) UV-B Monitoring and Research Program. http://uvb.nrel.colostate.edu/UVB/. Accessed January 2010. pp Page.

Vandestaaij JWM, Ernst WHO, Hakvoort HWJ, Rozema J (1995) Ultraviolet-B (280-320 Nm) Absorbing Pigments in the Leaves of *Silene Vulgaris* - Their Role in Uv-B Tolerance. Journal of Plant Physiology, 147, 75-80.

Wargent JJ, Moore JP, Roland Ennos A, Paul ND (2009) Ultraviolet Radiation as a Limiting Factor in Leaf Expansion and Development. Photochemistry and Photobiology, 85, 279-286.
Waterworth WM, Jiang O, West CE, Nikaido M, Bray CM (2002) Characterization of Arabidopsis photolyase enzymes and analysis of their role in protection from ultraviolet-B radiation. Journal of Experimental Botany, 53, 1005-1015.

Yin XY, Goudriaan J, Lantinga EA, Vos J, Spiertz HJ (2003) A flexible sigmoid function of determinate growth. Annals of Botany, 91, 361-371.

Zenoff VF, Sineriz F, Farias ME (2006) Diverse responses to UV-B radiation and repair mechanisms of bacteria isolated from high-altitude aquatic environments. Applied and Environmental Microbiology, 72, 7857-7863.

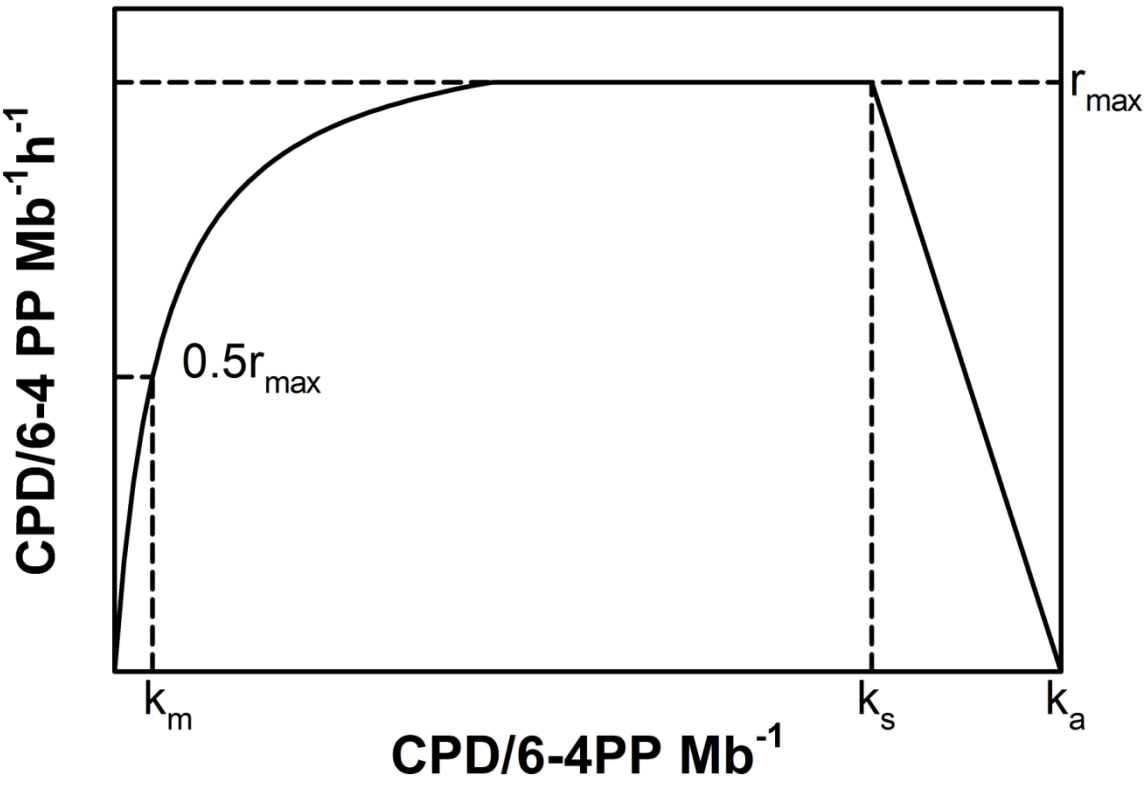

Figure A1: Conceptual model of DNA repair rate as a function of concentration of CPD/6-4PP concentration. Repair rates follow a basic Michaelis-Menten model (Lodish et al., 2008) until the photolyase reach a level of saturation, followed to a decline in rates to zero, corresponding to the level of damage that disturbs instantaneous the cell activity. Note: the processes expressed are not at real scale.

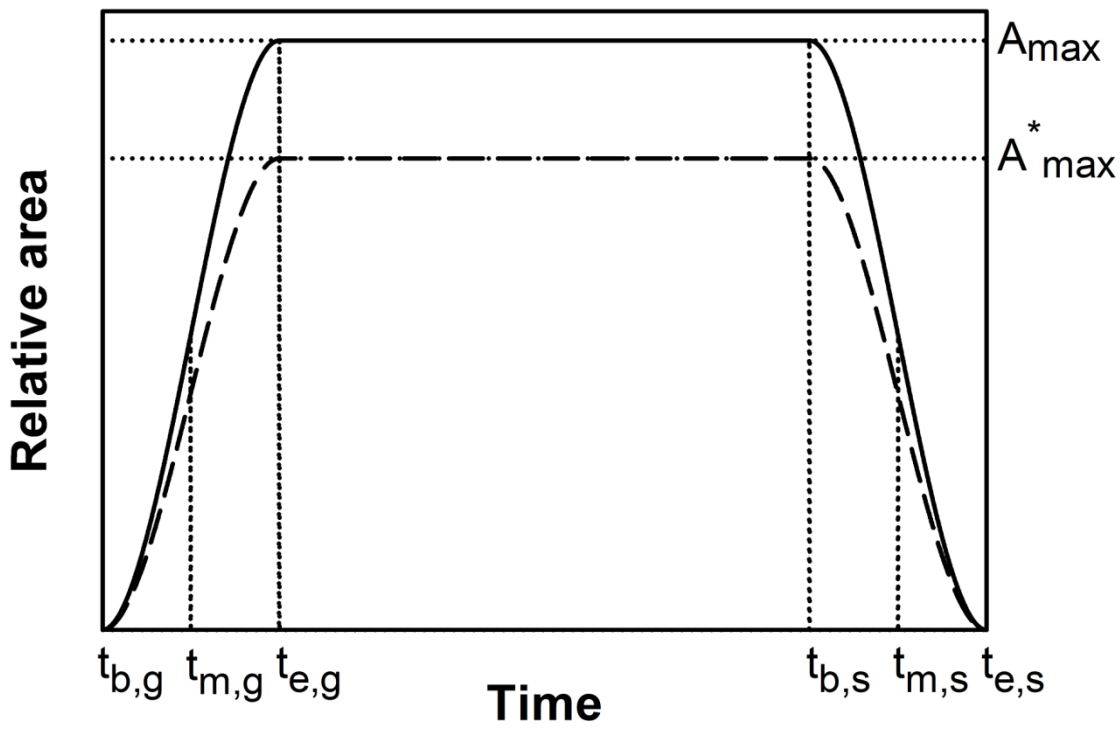

Figure A2: Dynamics of leaf area using the beta sigmoid function: normal leaf – solid line (with maximum area A_{max}); hypothetical leaf with UVB-induced DNA damage during growth – dashed line (with maximum area A^*_{max}). Note: the chlorophyll loss during the senescence period is expressed as effective loss of leaf area (Muller et al., 2006; Yin et al., 2003).

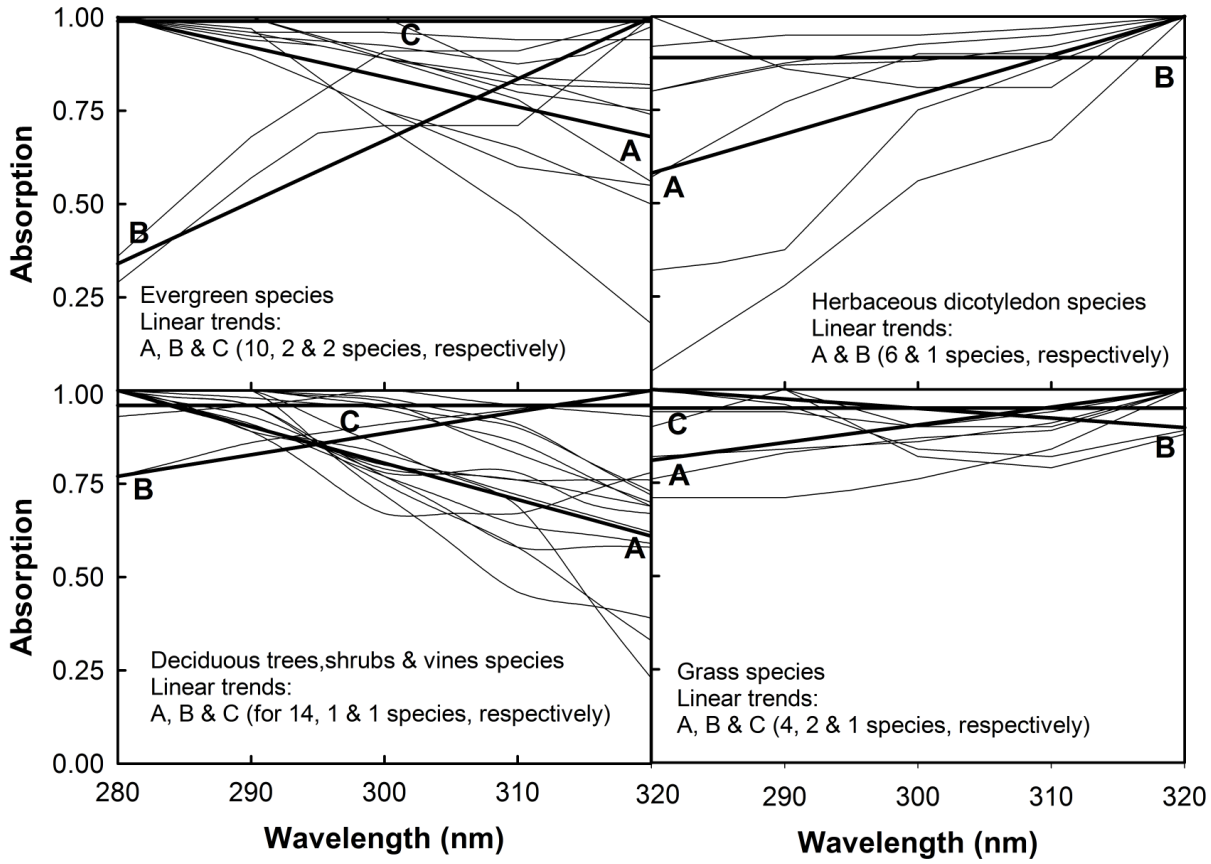

Figure A3: Relative absorption of secondary metabolites for evergreens, deciduous trees, shrubs, vines, herbaceous dicotyledons and grass species. The thin lines indicate the relative absorptance of individual species, while the bold lines (A, B, and C) indicate the general linear trends derived from the relative absorptance for individual species. Inferred from (Day et al., 1994; Lavola et al., 1997; Qi et al., 2003; Schmelzer et al., 1988; Sisson, 1981).

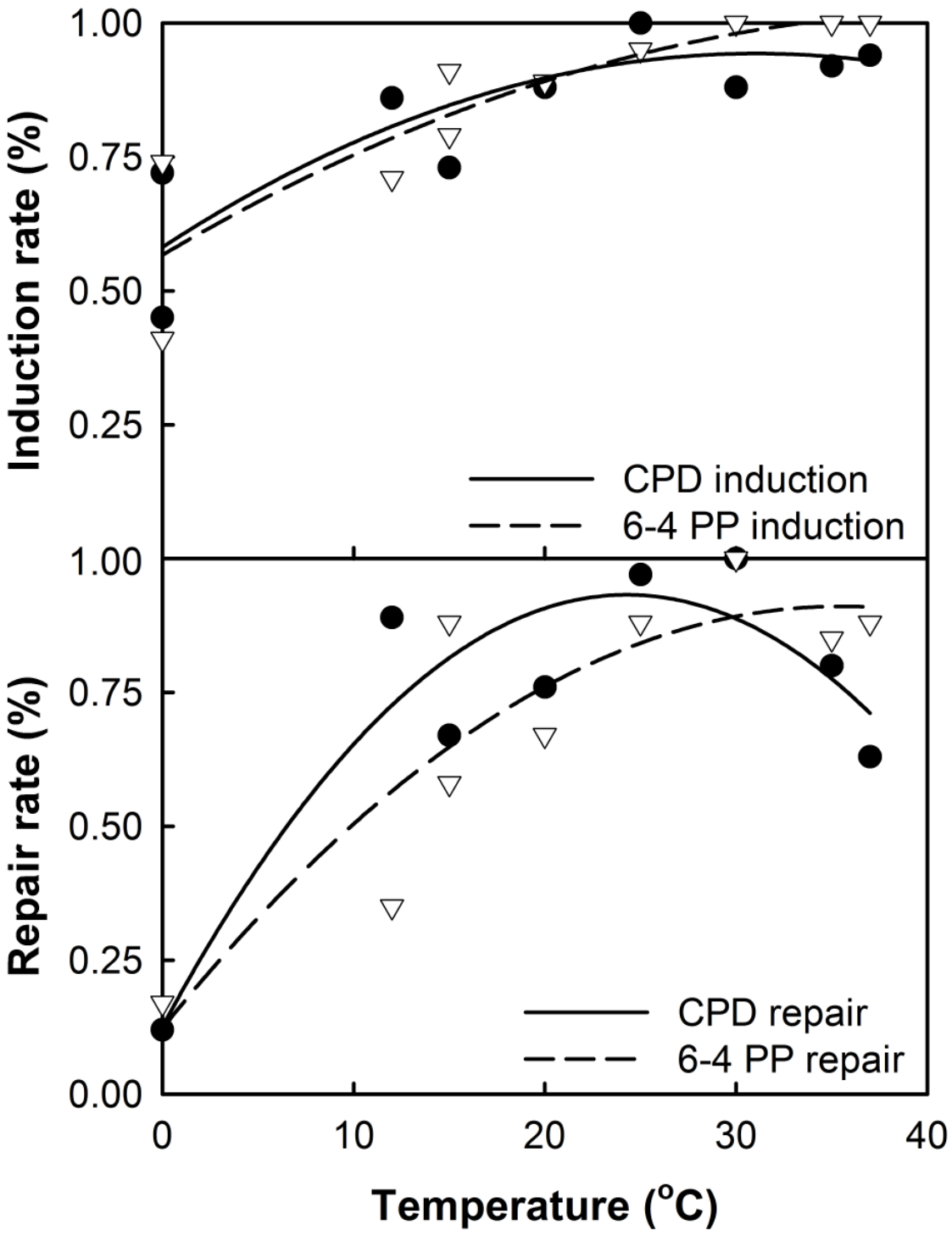

Figure A4: Temperature-dependent relative photoproducts induction and repair rates (Li et al., 2002; Takeuchi et al., 1996; Waterworth et al., 2002).

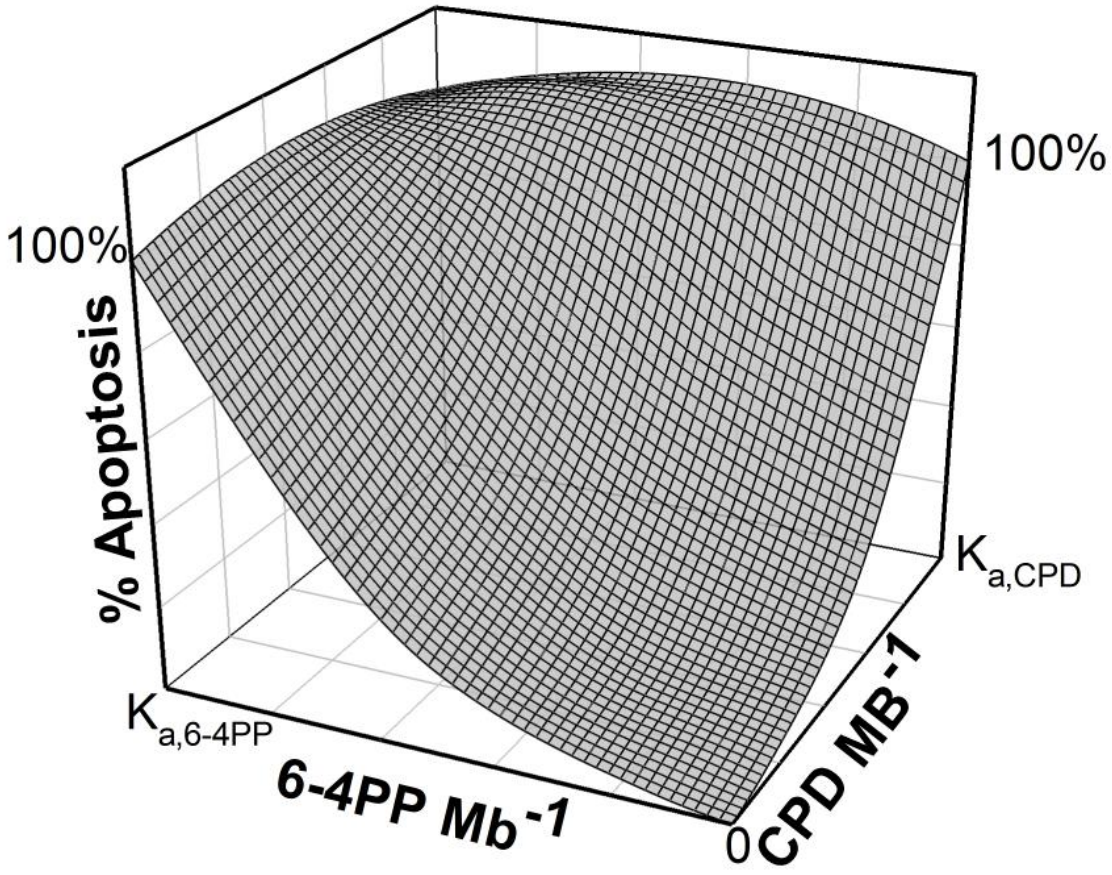

Figure A5: Theoretical model of the percent of apoptotic cells as a function of CPD/6-4PPs Mb^{-1}.

Appendix B: Selected Vensim models

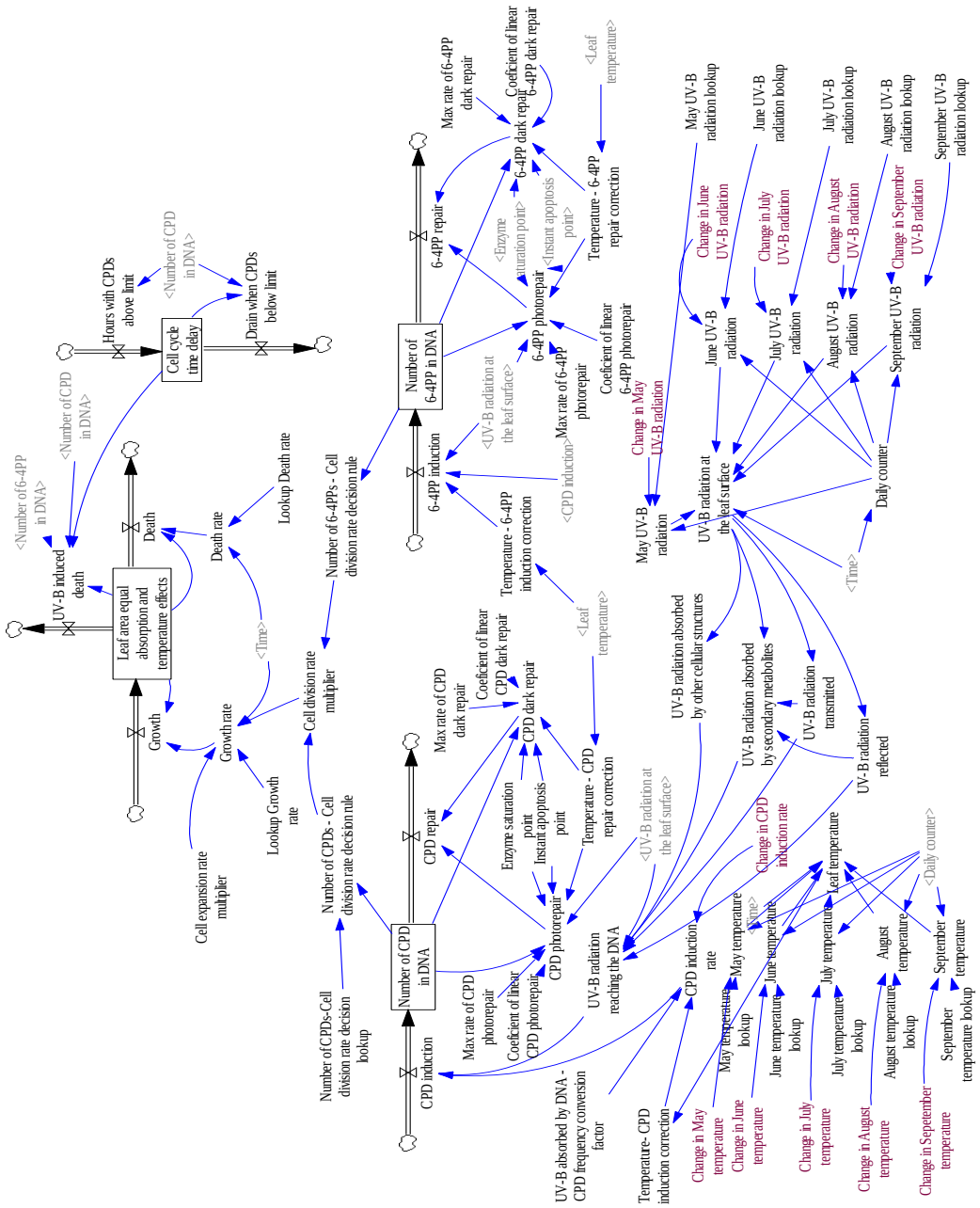

Figure B1: Vensim model of increased UV-B radiation induction of DNA lesions and effects on leaf area.

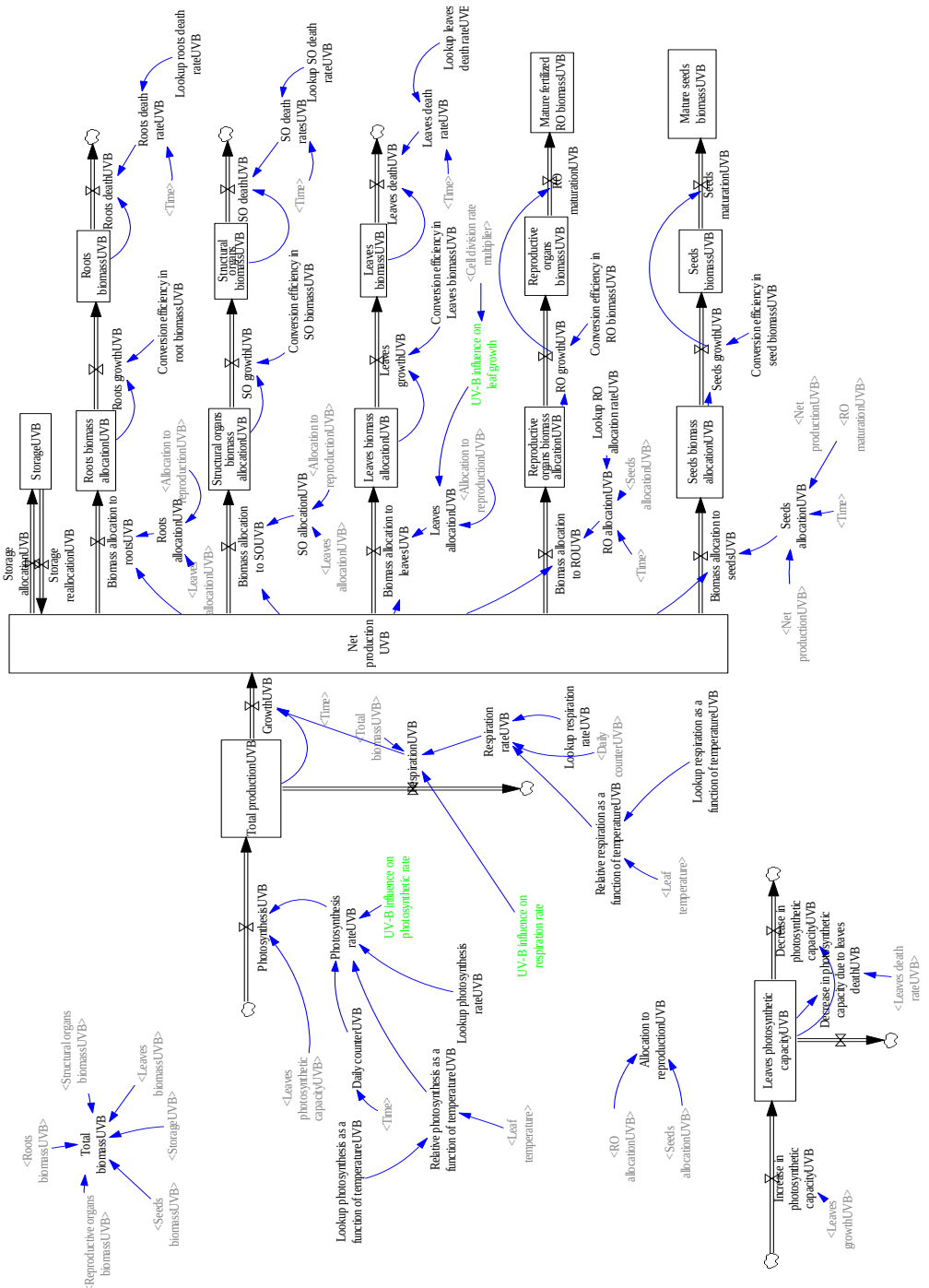

Figure B1: Vensim model of increased UV-B radiation effects on whole plant growth and development.