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MODELS FOR THE PHYSIOLOGICAL EFFECTS OF SHORT O_3 EXPOSURES ON PLANTS

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ABSTRACT

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Some published effects of ozone on plant photosynthesis and evaporation are detailed, and attempts were made to develop explanatory models of increasing complexity. Stomatal regulation, which keeps the CO_2 concentration inside the leaf constant, is assumed. The O_3 concentration inside the leaf is assumed to be negligible, so O_3 uptake, CO_2 uptake and stomatal conductance must be proportional. The suppression of photosynthesis is assumed to be proportional to the integrated effective O_3 uptake. For a first model, a differential equation is derived from these assumptions and a simple analytical solution is found. A second model includes a threshold O_3 flux. The repair process is discussed and three models are investigated with a constant repair rate, a repair rate dependent on the stage of injury, and with a repair rate dependent on the photosynthetic rate. A comparison of the analytical solutions of the appropriate differential equations with literature data shows that the model with repair dependent on photosynthesis is the most successful one, but a constant repair rate also gives a fair approximation. The model with repair dependent on photosynthesis implies the existence of a threshold level for the suppression of photosynthesis which separates reversible and irreversible O_3 effects. With the assumption of an inhomogeneous leaf a model to predict visible leaf injury is derived. The possible existence of a maximum leaf injury index for one exposure concentration is explained from the properties of the models. The restrictions and possible extensions of the models are discussed. The concept of a threshold O_3 flux and of a critical suppression of photosynthesis are shown to give possible explanations of antagonistic and synergistic effects of O_3, SO_2 and NO_2.

INTRODUCTION

The injury to plants resulting from a range of ozone concentrations and exposure durations has sometimes been described by mathematical equations, called models. Heck et al. (1982) give dose–effect relations between O_3 concentration and yields of several crops exposed to O_3 in the growing season. Linear relations were used and for some plants a threshold con-

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centration was included to give a better fit to the experimental data. The results of Heck et al. (1966) showed that for short-term exposures the relation between leaf injury and dose, defined as the product of concentration and time, is not linear. Larsen and Heck (1976) gave a model for these results using logarithmic and probit scales. Logarithmic scales for concentration and time were also used by Nouchi and Aoki (1979) and their model also included the effect of previous O_3 exposures. However, none of the models mentioned so far includes a physiological mechanism that explains why dose–effect relations are not linear and the effects of other environmental factors are not included.

Several environmental factors have been found to modify the effects of air pollutants on plants. Black and Unsworth (1979a) showed that at a low SO_2 concentration of 50 $\mu g/m^3$ the effect of SO_2 on net photosynthesis can only be measured if the windspeed is high enough. At low windspeeds the boundary layer resistance restricts the exchange of gases between leaf and atmosphere. Also differences in light intensity may cause large differences in air pollutant sensitivity of plants. Black and Unsworth (1979a,b) used a high, but realistic light intensity to find effects at low SO_2 concentrations. Hill and Littlefield (1969) found a strong increase in plant response to O_3 when sunlight was used instead of the usual electric lighting. The stronger O_3 effect was correlated with a larger stomatal aperture, so the differences in O_3 effect might have been caused by differences in O_3 uptake through stomata. Also more recently it was realised by Krause et al. (1984) that differences in light intensity caused differences in plant sensitivity to air pollutants.

The large number of factors influencing the effect of O_3 is a good reason for the use of dynamic models to test hypotheses and predict effects. Dynamic models that predict the effects of variations in light intensity, windspeed and water availability, have already been developed (Penning de Vries and Van Laar, 1982). A first attempt to include O_3 effects in such a model to predict long-term effects on yields has been made by King et al. (1983). However, the model they propose only includes an irreversible, cumulative suppression of photosynthesis by O_3 uptake. But not all O_3 effects are irreversible and long O_3 exposures also cause other effects besides the suppression of photosynthesis. For example, the translocation of assimilates from the leaves may be reduced as a result of a long O_3 stress (Tingey and Taylor, 1982). A better start of modelling O_3 effects therefore may be to concentrate on short-term effects.

After developing a simple model that describes the measurable fluxes of gases during a short O_3 exposure, attention must be paid to the recovery period after the exposure. Recovery processes have received little attention in the research on air pollutant effects up to now, but is was realised that the amount of leaf injury after an O_3 exposure may increase if the plant is

subjected to O_3 a few days before the main O_3 exposure (Nouchi and Aoki, 1979; Mukammal et al., 1982; Steinberger and Naveh, 1982). This suggests the existence of an invisible kind of injury that may be measured as a suppression of photosynthesis as was found by Keller (1981) for SO_2 effects. But in contrast to the irreversible SO_2 effect observed by Keller, presently known O_3 effects on photosynthesis are only irreversible if visible leaf injury occurs (Hill and Littlefield, 1969; Black et al., 1982). This suggests that, for short-term O_3 exposures, visible leaf injury and suppression of photosynthesis are expressions of one and the same kind of injury. From this hypothesis it is possible to build one model for all kinds of short-term O_3 effects, which will be shown in this paper.

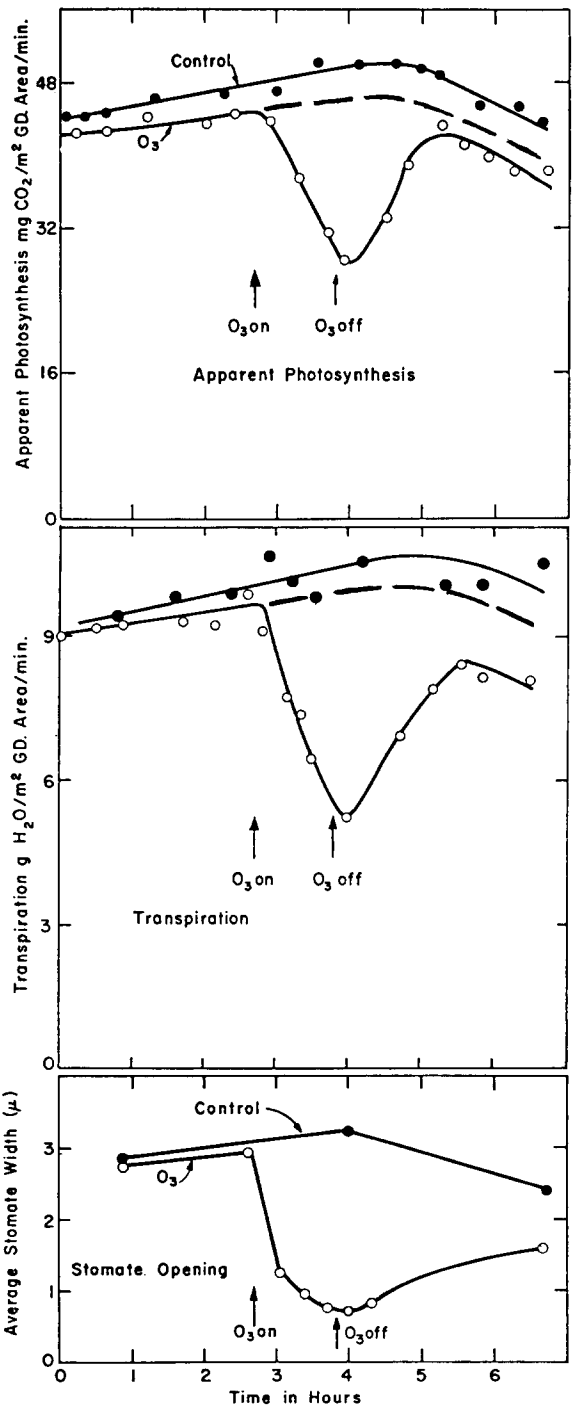
A NEW ANALYSIS OF OLD DATA

The model to be developed should describe the physical fluxes of O_3, CO_2 and water vapour, and the physiological reactions of the plant to O_3 and other environmental factors. To illustrate how these physical fluxes are involved, a description will be given of the most important data of Hill and Littlefield (1969) and Hill and Chamberlain (1974).

A small canopy of oat plants was put in a closed chamber to permit measurements of CO_2 uptake (net assimilation), O_3 uptake and transpiration. Concentrations in the chamber were kept at a constant level by removing or adding CO_2, O_3 or water vapour and so the exchange of these gases between air and canopy was measured. Stomatal width was measured by the epidermis stripping method. A windspeed of 2 miles/h (ca. 0.9 m/s) was applied and temperature and relative humidity were maintained at 24°C and 50%, respectively.

The fluxes of CO_2, O_3 and water vapour have to pass through two types of resistances: aerodynamic resistances governed by the windspeed, and a stomatal resistance governed by the physiological status of the plant. The calculation of the boundary layer resistance (using Monteith, 1973) and the relation between O_3 uptake and windspeed (Hill, 1967) both show that at 0.9 m/s the stomatal resistance is the most important one. This allows a simplification of the analysis: concentration differences within the canopy will be neglected.

Firstly the relation between transpiration and the sum of resistances will be analysed. The transpiration of the canopy E (g/min per m^2 ground area) is determined by the vapour pressure deficit vpd, i.e., the difference in vapour pressure between the air above the canopy and in the stomatal cavity (mbar), and the sum of resistance r (s/m) or conductivity $\sigma = r^{-1}$ (m/s). The vapour pressure in the leaf is assumed to be saturated and therefore only dependent on the leaf temperature. Differences in leaf temperature within


Apparent Photosynthesis mg CO_2/m^2 GD. Area/min.
48
32
16
0
Control
O_3
O_3on
O_3off
Apparent Photosynthesis
Transpiration g H_2O/m^2 GD. Area/min.
9
6
3
0
O_3on
O_3 off
Transpiration
Average Stomate Width (μ)
3
2
1
0
Control
O_3
Stomate. Opening
O_3on
O_3off
0
1
2
3
4
5
6
Time in Hours

the canopy will be neglected so r can be seen as the substitute resistance for all parallel resistances between the air above the canopy and the inside of the stomatal cavities.

The total transpiration also includes the transpiration from the ground, but extrapolation of transpiration data given in Hill and Chamberlain (1974) to dark periods with closed stomata indicates that the transpiration from the ground is small ($< 5\%$). Another possibly confusing factor in the relation between E and σ is the increase in leaf temperature caused by reduced transpiration during O_3 exposure. This temperature difference ΔT can be estimated from the change in the latent heat flux $\Delta\lambda E$ — where λ is the evaporation heat of water (J/g) — which equals the change in the sensible heat flux: $\Delta C = \rho c_p \Delta T$, in which ρc_p is the volumetric heat capacity of the air (1240 J m^{-3} K^{-1}) and r_b is the boundary layer resistance. If the ratio between leaf area and ground area is set to 4 m^2/m^2 and constants and formulae are taken from Monteith (1973) and Goudriaan (1977), the change in leaf temperature can be estimated at about 0.4 K. This implies that vpd changes about 5% during the O_3 exposure. So combining the effects of ground transpiration and the change in leaf temperature, we may assume that the change in σ is not more than 10% larger than the change in E. This means that as a first approximation a simple proportionality between E and σ can be assumed.

Now it is possible to compare the relative changes in CO_2 uptake, net photosynthesis P_n (mg/s per m^2 leaf area) and the conductance σ. The results given by Hill and Littlefield (Fig. 1) can be expressed as:

$$\frac{\Delta P_n}{P_n} = 0.40 \quad \text{and} \quad \frac{\Delta\sigma}{\sigma} = \frac{\Delta E}{E} = 0.48$$

If only the fast reversible parts of the O_3 effects (Fig. 1) are considered, these relations change into:

$$\frac{\Delta P_n}{P_n} = 0.36 \quad \text{and} \quad \frac{\Delta\sigma}{\sigma} = \frac{\Delta E}{E} = 0.38$$

This result provides a good starting point for modelling: a simple proportionality between CO_2 uptake and stomatal conductance for fast reversible

Fig. 1. Effects of O_3 on apparent photosynthesis, transpiration and stomatal width as found by Hill and Littlefield (1969) using 1200 $\mu g/m^3$ for 65 min. The broken lines are added as extrapolations of the no-effect lines. From the approximate equality of the relative suppression of photosynthesis and transpiration it can be deduced that the CO_2 concentration inside the leaf remains approximately constant during O_3 exposure. Reprinted with permission © 1969 American Chemical Society, Washington, DC, U.S.A.

effects can be concluded for this case. This implies that the CO_2 concentration inside the leaf C_{Ci} is kept constant by the plant if only fast reversible effects occur.

For the irreversible or slowly reversible part of the effect a rough estimate can be made:

$$\frac{\Delta P_n}{P_n} = 0.04 \quad \text{and} \quad \frac{\Delta \sigma}{\sigma} = \frac{\Delta E}{E} = 0.10$$

This indicates that the CO_2 concentration inside the leaf changes slightly during O_3 exposure. Consistently a change in C_{Ci} after repeated exposures to O_3 can be concluded from measurements of Coyne and Bingham (1978, 1981): for bean (*Phaseolus vulgaris*) C_{Ci} decreases and for Ponderosa pine C_{Ci} increases. This can be derived from the relative changes in P_n and σ as was just shown.

The influence of O_3 on C_{Ci} of bean plants during short-term exposures can again be derived from measurements of Hill and Littlefield (1969); they

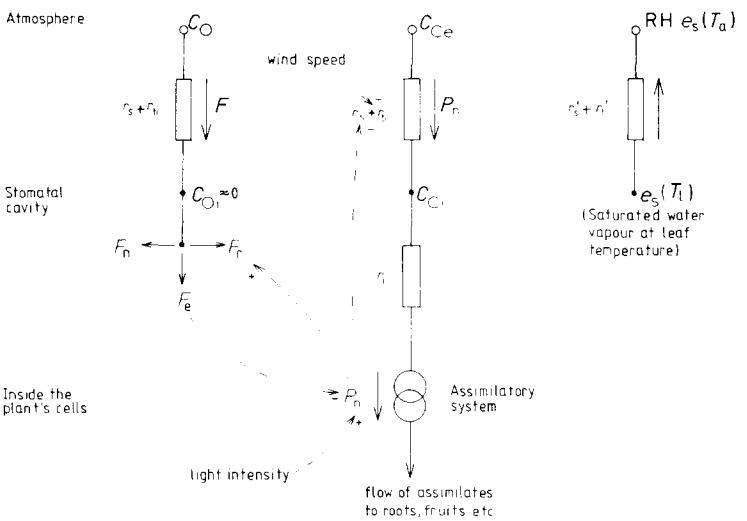


Fig. 2. Simplified representation of resistance and fluxes of O_3, CO_2 and water vapour. The broken lines show the causal relations that are assumed to derive the models. C_O and C_{Oi} are the O_3 concentrations outside and inside the leaf; C_{Ce} and C_{Ci} the CO_2 concentrations outside and inside the leaf; RH $e_s(T_a)$ and $e_s(T_l)$ water vapour pressures outside and inside the leaf. The fluxes through the stomata of O_3, CO_2 and water vapour are given by F, P_n and E. Inside the leaf the O_3 flux can be split into an effective part F_e and a non-effective part: $F_{ne} = F_n + F_r$; F_n causes no effect while the effect of F_r is not visible because of a repair process. The stomatal and boundary layer resistances r_s and r_b differ by constant factors from r_s' and r_b' because the diffusivity of gases changes with molecular weight.

report the change in stomatal width, w, and in net photosynthesis, P_n. The relation between w and σ is not linear but Kuiper (1972) gives an experimental relation between w and σ^{-1} for bean plants. If this relation is used, also for bean C_{Ci} appears to be approximately constant during a short O_3 exposure. For the small oats canopy O_3 uptake has been measured and related to stomatal width data (Hill and Chamberlain, 1974). If the change in w is chosen to be equal to the change in w after the O_3 exposure of Fig. 1, a change in the O_3 flux F can be compared with a change in the transpiration E:

$$\frac{\Delta F}{F} = 0.34 \quad \text{and} \quad \frac{\Delta E}{E} = 0.38$$

If this is compared with the previous results, the conclusion is that within an error of about 10% the relative changes in P_n, σ, E and F are equal so that a simple proportionality exists for these quantities during a short O_3 exposure. From the approximate proportionality between σ and F it can also be concluded that the O_3 concentration in the stomatal cavity is small. A similar conclusion for the CO_2 concentration inside the stomatal cavity cannot be drawn because it is a physiological process that adjusts the stomatal conductance so that C_{Ci} is constant with a value of at least 30% of the atmospheric CO_2 concentration (Goudriaan and Van Laar, 1978). Figure 2 illustrates the differences and similarities between the flows and resistances for O_3, CO_2 and water vapour. Also the causal relations and fluxes that have not yet been discussed are indicated to give an overview of the modelling plan.

MODELS

Model 1: cumulative O_3 effect and stomatal closure

The time-independent relations found in the previous section must firstly be summarised in terms of simple formulae. Then new relations will be introduced, resulting in a model with time dependence.

The O_3 flux into the leaf can be calculated with:

$$F = \sigma C_O \tag{1}$$

where F is the O_3 flux into the leaf (μg/s per m^2 leaf area), $\sigma = (r_b + r_s)^{-1}$, in which r_s, r_b stomatal and boundary layer resistance for O_3 (s/m), respectively, and C_O is the O_3 concentration outside the leaf (μg/m^3).

In relation (1) it is assumed that the O_3 concentration inside the leaf is negligible. The discussion of Hill and Littlefield's (1969) results showed that this is at least approximately true for oats and (1) was also found to hold by Rich et al. (1970), Turner et al. (1974) and Omasa et al. (1979).

In general Ohm's law can also be applied to the CO_2 flux through the boundary and stomatal resistance:

$$P_n = \sigma C_C \tag{2a}$$

or

$$\sigma = P_n / C_C \tag{2b}$$

where P_n is the CO_2 flux into the leaf or net assimilation (mg/s per m^2 leaf area), $C_C = C_{Ce} - C_{Ci}$, in which C_{Ce}, C_{Ci} the CO_2 concentrations outside and inside the leaf, respectively (mg/m^3), and σ is the same conductance as for O_3 (m/s) because CO_2 and O_3 have nearly equal molecular weights. In case of clean air, several plant species were found to regulate their stomata so that the CO_2 concentration inside the leaf, in the stomatal cavity, is constant (Goudriaan and Van Laar, 1978; Louwerse, 1980; Bell, 1982). In the previous section it was shown that C_{Ci} is also constant during short O_3 exposures on oats and bean plants. So, although the internal CO_2 concentration is not negligible, a simple linear relation between σ and P_n can be assumed.

To introduce a time dependence in the model, assumptions have to be made about the O_3 effect. Firstly O_3 is assumed to affect only P_n directly as is illustrated in Fig. 2. With this assumption O_3 might still injure the assimilatory system (enzymes, membranes) or react with the assimilates. The second assumption is that direct reactions with assimilates are not important. This implies the following first-order description of the O_3 effect:

$$P_n = P_0(1 - B) \tag{3}$$

where B is a measure for the O_3 effect to be defined more precisely.

It should be realised that (3) is not so trivial as it seems to be; if only assimilates would be affected (for example by autocatalytic reactions), (3) would have been:

$$P_n = P_0 - B' \tag{3'}$$

where B' should have been independent of P_0.

Before (3) can be tested, a relation between B and F has to be assumed. In contrast to the effect of SO_2, as described by Black and Unsworth (1979b), the net photosynthetic rate P_n usually continues to decrease during an exposure to O_3 (Hill and Littlefield, 1969; Black et al., 1982). This observation can be interpreted as a cumulative effect of O_3 uptake, so for model 1 we may try:

$$B = k \int_0^t F \, dt' \tag{4}$$

where k is a constant (m^2 leaf area per μg), and t is the time from the start of the exposure (s or h).

This relation (4) will be improved in the following models. A simple differential equation can be derived from (1), (2), (3) and (4) if all environmental factors are assumed to be constant except for switching the O_3 source on and off. Substitution of (3) in (2b), (2b) in (1) and (1) in (4) followed by differentiation to t yields:

$$\frac{dB}{dt} = (kP_0C_O/C_C)(1 - B) \tag{5a}$$

This expression can be abbreviated by substituting:

$$t_s = (kP_0C_O/C_C)^{-1} \tag{6}$$

Then (5a) becomes:

$$\frac{dB}{dt} = \frac{1 - B}{t_s} \tag{5b}$$

The solution of (5b) is given by:

$$B = 1 - \exp(-t/t_s) \tag{7}$$

Substitution of (7) into (3) and (3) in (2a) gives:

$$P_n = P_0 \exp(-t/t_s) \tag{8}$$

$$\sigma = \sigma_0 \exp(-t/t_s) \tag{9}$$

where

$$\sigma_0 = P_0/C_C \tag{10}$$

The model given by (8) and (9) gives a first description of the decrease of CO_2 uptake and of stomatal closure. Using the definition of t_s, (6), the dependence of t_s, P_n and σ on P_0, C_C and C_O can be investigated. As an example the effect of a change in light intensity will be treated. In (8) the exponential can be approximated for $t \ll t_s$; if (6) is also used this results in:

$$P_n(t) = P_0 - (kP_0^2C_O/C_C)t \tag{11}$$

Remarkable is the quadratic dependence of the factor before t on P_0. In the case of a canopy or low light intensity, P_0 is proportional to the light intensity, so in that case a quadratic increase of the effect for small t with light intensity can be expected. This was found by Hill and Littlefield (1969) for the miniature oats canopy described in the previous section. Their results are reproduced in Fig. 3. If the assumptions of the model are reviewed, it becomes clear that, physiologically, the strong dependence of the effect on light intensity can be explained as a combination of two factors: (a) the O_3 flux increases because the conductance increases due to the stomatal regulation (equations 1 and 2b); (b) O_3 injures the photosynthetic apparatus and

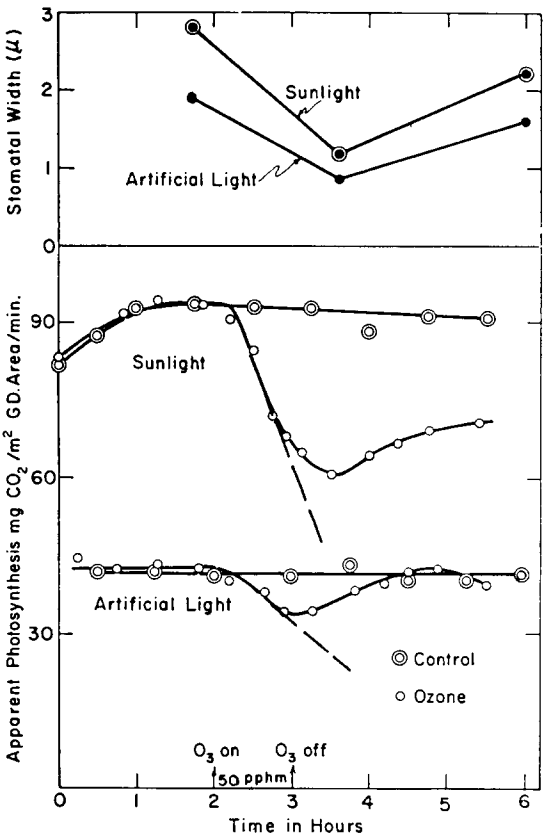


Fig. 3. Effect of O_3 on apparent photosynthesis and stomatal width of oats under sunlight and artificial lighting as found by Hill and Littlefield (1969) using 1000 $\mu g/m^3$ O_3 for 1 h. The broken lines are added. The slope of these lines, describing the first part of the effect, increases quadratically with the initial rate of photosynthesis. This feature can be explained with model 1. Reprinted with permission © 1969 Americal Chemical Society, Washington, DC, U.S.A..

not the assimilates, as was the motivation of applying (3) instead of (3′). If one of these points was not true, a linear P_0 dependence would have been found in (11) instead of a quadratic dependence.

For $t \gg t_s$ the model shows its limitations; a complete stomatal closure is predicted instead of a realistic partial stomatal closure. Also the existence of a threshold concentration and recovery processes are not yet described by model 1.

Model 2: a threshold for the O_3 *effect and partial stomatal closure*

The supression of P_n in *Vicia faba* after an O_3 exposure of 4 h was measured by Black et al. (1982). Variation of the exposure concentration C_O

showed that there exists a threshold concentration C_{Ot}: below this concentration no effects occur. Also earlier results from, for example Heck et al. (1966), indicated the existence of a threshold concentration. This feature can be included into the model by introducing a threshold flux F_t, C_{Ot} and F_t are related by:

$$F_t = \sigma_0 C_{Ot} \tag{12}$$

where F_t is the threshold O_3 flux into the leaf (μg/s per m^2 leaf area), C_{Ot} the threshold O_3 concentration ($\mu g/m^3$), and σ_0 the same conductance as in (10).

The O_3 flux into the leaf F can now be split into two components:

$$F = F_e + F_{ne} \tag{13}$$

where F_e is the part of F that causes a measurable effect, and F_{ne} the part of F that does not cause a measurable effect.

Because no effect is found below C_{Ot}, it may be assumed:

$$\begin{aligned} &\text{if } F > F_t, \quad F_{ne} = F_t \quad \text{so} \quad F_e = F - F_t \\ &\text{if } F < F_t, \quad F_{ne} = F \quad \text{so} \quad F_e = 0 \end{aligned} \tag{14}$$

Model 2 can now be derived by changing (4) into:

$$B = k \int_0^t F_e \, dt' \tag{15}$$

Similar to (5a) but now with (14) and with (15) instead of (4), the following differential equation can be found for $C_O > C_{Ot}$:

$$\frac{dB}{dt} = (kP_0 C_O / C_C)(1 - B) - kF_t \tag{16a}$$

The non-effective fraction of the initial O_3 flux can be defined as:

$$f = F_t / F_0 = C_{Ot} / C_O \tag{17}$$

where

$$F_0 = \sigma_0 C_O \tag{18}$$

Using (6) and (17), (16a) can be rewritten as:

$$\frac{dB}{dt} = \frac{1 - B - f}{t_s} \tag{16b}$$

After separating the variables, (16b) can be integrated giving:

$$B = (1 - f)(1 - \exp(-t/t_s)) \tag{19}$$

The time dependence of P_n and σ can be found from (19), (3) and (2a):

$$P_n = P_0(f + (1 - f) \exp(-t/t_s)) \tag{20}$$

$$\sigma = \sigma_0(f + (1 - f) \exp(-t/t_s)) \tag{21}$$

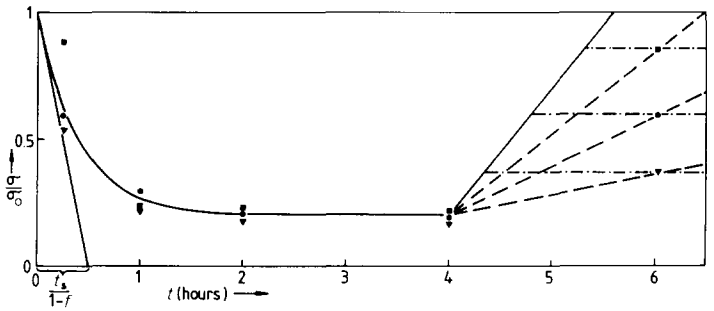


Fig. 4. Effect of O_3 on relative stomatal conductance σ/σ_0 according to models 2 and 3 with data points from Elkiey and Ormrod (1979). The assumed existence of a threshold O_3 flux gives an explanation for the incompleteness of stomatal closure. This is supported by the experimental data points derived from the results of Elkiey and Ormrod, obtained at 50% relative humidity with *Petunia*, prefloral stage (●, cv. Capri; ▼, cv. White Magic; ■, White Cascade). The theoretical curve (———) is given according to models 2 and 3 with $t_s = 0.4$ and $f = 0.2$ for $t < t_e$ and $f_r = 0.2$ for $t > t_e$. The tangent in $t = 0$ shows how $t_s/(1-f)$ can be estimated from measurements. The broken lines are given according to model 3 in the case of $f \neq f_r$ and $f_n > 0$ (— — —) and in the case of partially irreversible effects (— · —).

Limit cases of (20) and (21) are useful to be considered. If $t \ll t_s$:

$$P_n = P_0(1-(1-f)t/t_s) \tag{22}$$

$$\sigma = \sigma_0(1-(1-f)t/t_s) \tag{23}$$

The conclusions drawn for (11) also hold for (22). If $t \gg t_s$:

$$P_n = fP_0 \tag{24}$$

$$\sigma = f\sigma_0 \tag{25}$$

Partial stomatal closure is explained by (25). Equations (17) and (25) also permit an estimation of the threshold concentration C_{Ot} from measurements at high C_O if $t \gg t_s$:

$$C_{Ot} = C_O\frac{\sigma}{\sigma_0} \tag{26}$$

The steady state condition $t \gg t_s$ is probably reached only in laboratory exposures to high O_3 concentrations; then C_O is large and hence, with (6), t_s is small enough to give $t \gg t_s$ in a few hours.

As an example of the use of (25) and as a test of the model, (21) is drawn in Fig. 4 with parameters derived from the results of Elkiey and Ormrod (1979). The parameters t_s and f have been fitted using (23) and (25). To represent the data from Elkiey and Ormrod (1979) in Fig. 4 as σ/σ_0, the

boundary layer resistance r_b had to be added to the measurements of the stomatal resistance r_s according to:

$$\sigma^{-1} = 1.65 r_s + 1.35 r_b \tag{27}$$

where the conversion factors depend on the diffusivities of water vapour and CO_2 (Black and Unsworth, 1979a; Monteith, 1973).

The value of r_b was not measured by Elkiey and Ormrod (1979), so an estimation for r_b had to be made. The air in the exposure chamber was not stirred so the air flow through the chamber may be assumed to determine r_b. Black and Unsworth (1979a) measured r_b in their chamber with and without stirring, and they found $r_b = 80$ s/m and $r_b = 120$ s/m, respectively. The air flow through the chamber of Elkiey and Ormrod (1979) was slightly higher, resulting in an estimate of $r_b = 100$ s/m. This is quite high compared to the open air situation where $r_b = 20$ s/m is a more normal value for petunia.

Now a quantitative comparison between experimental and model results can be made (Fig. 4). In general the theoretical curve fits the measurements fairly well; only for *Petunia* cv. 'White Cascade' the onset of stomatal closure seems to be delayed. This can be explained by a pool of scavenger material that is oxidised before any more harmful effects occur. Clearly the condition $t \gg t_s$ is reached after $t = 2$ h. To observe this effect, corrections for changes in control plants were necessary (σ is the conductance of exposed plants, σ_0 is taken as the conductance of control plants).

Equation (26) provides the possibility of estimating the threshold concentration from stomatal resistance measurements. Again using the data from Elkiey and Ormrod (1979), the result for *Petunia*, early vegetative stage, is:

$$f = 0.28, \quad C_{Ot} = 220\ \mu g/m^3$$

and for the prefloral stage:

$$f = 0.20, \quad C_{Ot} = 160\ \mu g/m^3$$

The differences between the varieties of *Petunia* after 4-h exposure are small and can be neglected. The threshold concentrations calculated for *Petunia* are larger than the 80 $\mu g/m^3$ found by Black et al. (1982) for *Vicia faba*, but the latter result was obtained at a light intensity about 4 times higher. If the light response curve of Black and Unsworth (1979b) can be used, P_0 must have been twice as high during the measurements of Black et al. (1982). With (10), (12) and (17) this implies that if the measurements of Elkiey and Ormrod (1979) had been carried out at the light intensity used by Black et al. (1982), the f-values and threshold concentrations for *Petunia*, early vegetative stage, would have been:

$$f = 0.14, \quad C_{Ot} = 110\ \mu g/m^3$$

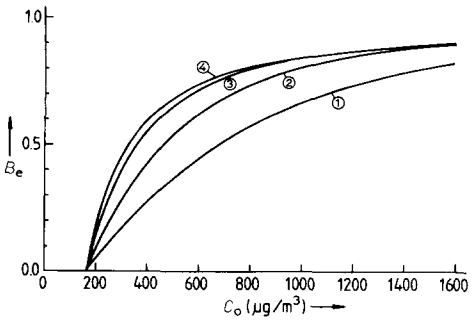


Fig. 5. Reduction of stomatal conductance and net assimilation at the end of O_3 exposure, B_e at a range of O_3 concentrations C_O and exposure durations t_e. Curves 1, 2, 3 and 4 are given according to model 2 for $t_e = 0.5$, 1, 2 and 4 h, respectively. The model parameters $t_s C_O$ and fC_O are derived from the *Petunia* parameters used in Fig. 4.

and for the prefloral stage:

$f = 0.10, \quad C_{Ot} = 80\ \mu g/m^3$

The corrected values for C_{Ot} are much below the exposure concentration of 800 $\mu g/m^3$ but remarkably close to the threshold concentration of *Vicia faba* as found by Black et al. (1982).

As another example of the possibilities of model 2 the dependence of B at the end of the exposure ($t = t_e = 4$ h) on the concentration C_O has been given in Fig. 5 for *Petunia*, prefloral stage. If t_{s0} and f_0 denote the parameter values at 800 $\mu g/m^3$, the parameters for any other concentration can be calculated from:

$$t_s = t_{s0}\frac{800}{C_O} \quad \text{and} \quad f = f_0\frac{800}{C_O}$$

These relations follow directly from the definitions (6) and (17) and substitution in (17) gives the relation shown in Fig. 2. No data were available to test this relation.

Model 3, 4 and 5: descriptions of the recovery process

The models treated up to now only gave a description of the processes for $t \leqslant t_e$. For a study of the recovery period it is necessary to put some more detail into the description of the flux F into the plant. This flux was already split into a part that causes a measurable effect F_e, and a non-effective part F_{ne} (equation 13). Now the interpretation of F_{ne} becomes important: F_{ne} is considered to have two components:

$$F_{ne} = F_n + F_r \tag{28}$$

where F_n is the part of F that is destroyed before causing any effects, and F_r

the part of F which compensates for the repair of O_3 effects. The effective flux into the plant is now given by (see also Fig. 2):

$$F_e = F - F_n - F_r \tag{29}$$

The O_3 destruction without effect described by F_n may be caused by the presence of insensitive surfaces such as cell walls, or by enzymes as was suggested from the results of Lee and Bennett (1982). Both surface and enzyme reactions have a limited capacity to destroy O_3, which is a possible explanation for the existence of a threshold concentration. Therefore F_n will be described similar to F_{ne} in (14):

$$\begin{aligned} &\text{if } F \geqslant F_{nt}, \quad F_n = F_{nt} \quad \text{so} \quad F_e = F - F_{nt} - F_r \\ &\text{if } F \leqslant F_{nt}, \quad F_n = F \quad \text{so} \quad F_e = -F_r \end{aligned} \tag{30}$$

where F_{nt} is a, constant, threshold flux. Similar to f in (17), f_n is defined as:

$$f_n = F_{nt}/F_0 \tag{31}$$

In (28) F_r is introduced as a real O_3 flux, which does not seem to have any effect because the effect of F_r balances with the repair of O_3 injury. But when the O_3 flux $F - F_n$ becomes zero at the end of the exposure, no more injuries are formed while the repair process continues. This can be described by leaving $F_r > 0$ if $t > t_e$, so that the net or effective flux F_e becomes negative. The repair process both during and after the exposure can now be included if a suitable expression for F_r can be found. Three possible definitions of F_r will be discussed in the following sections.

The models resulting from the various definitions of F_r can only be compared if detailed experimental data for $t > t_e$ are available. The results of Elkiey and Ormrod (1979) only include one point after the end of the exposure. More detail is given by Black et al. (1982): they give measurements of P_n suppression 2 and 20 h after an exposure of 4 h to various O_3 concentrations. These results are useful to get some understanding of the repair process. Only Hill and Littlefield (1969) gave enough measurements of the P_n suppression in the recovery period for an analysis of the shape of the curve for $t > t_e$. Their data given in Fig. 2 were redrawn in Fig. 6 as P_n/P_0 which is equal to $1 - B$ (equation 3). The measurements were corrected for controls and were displaced for 7 min along the t-axis. The delay, assumed with this procedure, may have been caused by an instrumental effect or a pool of oxidisable material inside or outside the plant, as was suggested for *Petunia* 'White Cascade'. The analysis of the time dependence of P_n/P_0 starts with an analysis of the recovery period from which the parameter determining F_r is estimated. Then this value and the demand $B(1.1) = 0.4$ is used to determine the parameters for the curve during the exposure.

Because there are so few useful measurements of P_n suppression, other kinds of data will be used to find hypotheses for F_r and to test the models.

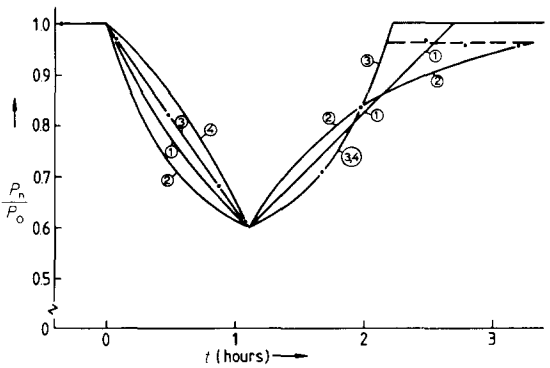


Fig. 6. Effect of O_3 on apparent photosynthesis of a small oats canopy. The data points from Hill and Littlefield (1969) given in Fig. 1 are redrawn on a relative scale to be able to chose the best model out of models 2 + 3, 4 and 5 represented by curves 1, 2 and 3. Curve 4 is also calculated with model 5 but with the condition $f_n = 0$. Clearly the best result is obtained with model 5 with $f_n \neq 0$. This suggests that repair depends on the rate of photosynthesis and the O_3 uptake without effect cannot be neglected. The broken line is given to indicate the incompleteness in short-term repair. For model parameters see Table 1.

Nouchi and Aoki (1979) measured the increase of leaf injury as a function of the time between pre- and main O_3 exposure. A linearly decreasing relation was found and 5 days after the pre-exposure the effect of the pre-exposure had disappeared. Sutton and Ting (1977) used another measure for the O_3 injury. After a short-term exposure of Pinto bean plants, leaf discs punched from the primary leaves showed an enhanced uptake of labeled sugar. This was assumed to be a measure for the O_3 injury to membranes. In the normal alteration of light and dark periods the increased sugar uptake reduced to control after 5 days. In continuous dark less than half of the effect was repaired. In continuous light the repair rate increased until complete recovery was observed on the 3rd day after O_3 exposure. The repair rate was also found to increase if the temperature increased or if glucose was applied directly after the exposure. From these results Sutton and Ting (1977) concluded that repair is mainly dependent on the availability of energy in the form of assimilates. This conclusion will be tested again in comparing the performance of the following three models.

Model 3: repair at a constant rate

The results of Nouchi and Aoki (1979) suggest that the repair rate is constant as long as there is any injury, so F_r can be defined as:

$$\begin{aligned} F_r &= F_{rc} \quad \text{if} \quad B \neq 0 \\ F_r &= 0 \quad \text{if} \quad B = 0 \end{aligned} \tag{32}$$

TABLE 1

Parameters for curves in Figs. 6, 7 and 8 for oats

Parameter	Curve number in Figs. 6 and 7			
	1	2	3	4
	Model number			
	3	4	5	5
t_s (h)	1.26	1.17	0.55	0.55
f_n	0 [a]	0 [a]	0.34	0
t_s/f_r (h)	4.0	–	–	–
t_s/a' (h)	–	1.0	–	–
t_s/b' (h)	–	–	0.55	0.55
B_C	–	–	0.46	0.46

[a] Any value of $f_n > 0$ would give a worse fit to the data points in Fig. 6.

where

$$F_{rc} = F_t - F_{nt} \text{ (which is constant)} \tag{33}$$

Similar to f and f_n, a fraction f_r can be defined as:

$$f_r = F_{rc}/F_0 \tag{34}$$

It can easily be shown that:

$$f = f_n + f_r \tag{35}$$

The processes during the O_3 exposure were described by model 2; now F_{ne} is interpreted according to (28). A study of the recovery period may reveal whether F_n or F_r is the most important component of F_{ne}. The recovery period can be described after substituting (30) in (15) by:

$$B = k\int_0^{t_e}(F - F_n - F_r)\,dt' + k\int_{t_e}^{t} - F_r\,dt'$$

$$= B(t_e) - kF_r(t - t_e)/t_s \tag{36}$$

while $B \neq 0$. The definitions of t_s and f_r, (6) and (34) can be used to rewrite (36):

$$B = B(t_e) - f_r(t - t_e)/t_s \tag{37}$$

The expressions for P_n and σ are, if $t \gg t_e$ and $B \neq 0$:

$$P_n = P_0(1 - B(t_e) + f_r(t - t_e)/t_s) \tag{38}$$

$$\sigma = \sigma_0(1 - B(t_e) + f_r(t - t_e)/t_s) \tag{39}$$

Complete recovery at $t = t_r$ corresponds to $B = 0$ in (37), so with (19):

$$\frac{t_r - t_e}{t_s} = \frac{1 - f}{f_r}(1 - \exp(-t_e/t_s)) \tag{40}$$

If $t_e \gg t_s$, then this reduces to:

$$t_r - t_e = \frac{1-f}{f_r} t_s \tag{41}$$

Because $f = f_n + f_r$ and $f_n > 0$, f_r cannot be larger than f. This implies the existence of a minimum value for $t_r - t_e$:

$$(t_r - t_e)_{min} = \frac{1-f}{f} t_s \tag{42}$$

The data of Elkiey and Ormrod (1979) for the recovery period were not detailed enough to provide a complete test of the model given with (39). The incomplete recovery after 2 h at $t = 6$ h, may be explained as an irreversible or incomplete reversible effect, as is indicated in Fig. 4. However, it can be deduced from (42) that the experimental data do not contradict the model; the recovery period of about 2 h is larger than the minimum recovery period demanded by (42), $(t_r - t_e)_{min} = 1.6$ h. If the difference between the minimum and the observed recovery period of about 2 h is significant, the existence of an O_3 flux without effect can be concluded ($F_n \neq 0$, $f_n \neq 0$), but this analysis also shows that F_r is the largest component of F_{ne}.

The data of Hill and Littlefield (1969) and the line according to (38) given in Fig. 6 show that (38) is acceptable as a first approximation. The value of the parameter t_s/f_r can be derived from (38) and the data for $t > t_e$ (Table 1). If this value is inserted in (20) with $f_n = 0$, the best curve for $t > t_e$ can be found. Figure 6 shows that also for $t < t_e$ the model curve only approximates the shape of the curve through the data points.

Model 4: repair rate only dependent on B

In an attempt to improve model 3, the amount of the injury, B, may be assumed to be the limiting factor for the repair process. For small B the repair process is trivially determined by B; if $B = 0$ then the repair process stops. But for high B the repair process may be determined by B or by the repair capacity of the plant. Firstly, in model 4, B will be assumed to be always the limiting factor. This can be expressed by:

$$F_r = aB \tag{43}$$

where a is a constant (s^{-1}). Because now F_r is not constant during the exposure, (16b) no longer holds and a new differential equation has to be derived for $t < t_e$. Therefore (43) must be substituted in (29) and the result in (15); then it can be derived that:

$$\frac{dB}{dt} = t_s^{-1}((1-f_n) - (1+a')B) \tag{44}$$

where

$$a' = a/F_0 = akt_s \tag{45}$$

The solution of (44) for $t < t_e$ is given by:

$$B = \frac{1 - f_n}{1 + a'}(1 - \exp(-(1 + a')t/t_s)) \tag{46}$$

Similar to (36) the solution for $t > t_e$ can be found from:

$$B = B(t_e) - k\int_{t_e}^{t} F_r(t')\,dt' \tag{47}$$

Differentiation of (45) gives:

$$\frac{dB}{dt} = -kF_r = -kaB \tag{48}$$

$$B = B(t_s)\exp(-a't/t_s) \tag{49}$$

The data of Hill and Littlefield (1969) and curve 2 in Fig. 6 show that model 4, that is (46) and (49), do not describe these data very well. However, Mukammal et al. (1982) used with success a relation like (47) with $t_s/a' \approx 4$ days to predict leaf injury in bean plants. The data of Sutton and Ting (1977) help to explain this apparent contradiction of experiments. If long dark periods are included the experimental curve may indeed have a shape like (47) but this can be explained from a decrease in pools of assimilates so the hypothesis (43) is not necessary to explain the results of Mukammal et al. (1982). So from the bad fit of model 4 in Fig. 6 it can be concluded that at normal B-values B is not limiting the repair process and hence, if $B \neq 0$, the B-dependence of F_r can be neglected.

Model 5: repair dependent on P_n

The negative result of testing model 4 suggests that the repair capacity of the plant is the limiting factor in the repair process. In view of the results of Sutton and Ting (1977) it is reasonable to assume that the availability of sugars determines the repair rate. The strong dependence of the repair rate on light intensity indicates that the photosynthetic production of sugars is more important for the repair rate than the existence of carbohydrate pools. In the following models the repair rate will be assumed to be dependent only on the photosynthetic rate P_n according to:

$$F_r = b(P_n - P_c) \quad \text{if} \quad 0 < B < 1 \tag{50}$$

where b is a constant (μg/mg), and P_c is the assimilate flow used for other processes than repair of O_3 injury. The introduction of P_c was found to be necessary to obtain a good fit of the model to the data of Hill and Littlefield

(1969); with $P_c = 0$ only a slight improvement of model 3 can be obtained. The introduction of $P_c \neq 0$ in (50) has an important consequence. If O_3 causes a suppression of P_n below P_c then F_r changes sign and (48) describes increasing injury instead of repair. So, as a result of fitting curve shapes, irreversible effects are introduced. The threshold O_3 concentration for irreversible effects found by Black et al. (1982) is now replaced by a critical P_n level which can be given a physiological interpretation. The new parameter P_c may still depend on P_0 but this could not be checked. For further derivations (50) can be written as:

$$F_r = bP_0(B_c - B) \quad \text{if} \quad 0 < B < 1 \tag{51}$$

where $B_c = P_c/P_0$. Expression (51) can be substituted in (29) and a differential equation for $t < t_e$ can be derived, but care should be taken for the possibility that now $F < F_{nt}$ may occur for large B and hence small σ, then $F_e = -F_r$. So if $B < (1 - f_n)$, which is equivalent to $F < F_{nt}$, and $(1 - f_n - b'B_c) > 0$ for $B > 0$ from $t = 0$, then:

$$t_s \frac{dB}{dt} = (1 - f_n - h'B_c) - (1 - b')B \tag{52}$$

where

$$b' = bP_0kt_s = bC_C/C_O \tag{53}$$

But if $1 > B > (1 - f_n)$ or $t > t_e$ then:

$$t_s \frac{dB}{dt} = -(B - B_c)b' \tag{54}$$

If (52) applies then for $b' < 1$ feedback is negative and for $b' > 1$ feedback is positive. In the previous models there was only negative feedback caused by stomatal closure while in (52) there is a balance between stomatal closure and the collapse of F_r. If (54) applies the criterion for positive or negative feedback is different; if $B < B_c$ feedback is negative and if $B > B_c$ then feedback is positive. For $b' = 1$ the net feedback is (52) is zero and the solution is:

$$B = (1 - f_n - B_c)t/t_s \tag{55}$$

For $b' \neq 1$ the solution of (52) is:

$$B = \frac{1 - f_n - b'B_c}{1 - b'}(1 - \exp(-(1 - b')t/t_s)) \tag{56a}$$

$$= \left(1 + \frac{b' - b'B_c - f_n}{1 - b'}\right)(1 - \exp(-(1 - b')t/t_s)) \tag{56b}$$

The condition for a non-negative solution given before (52) implies the existence of a threshold concentration, for this model given by:

$$C_{Ot} = C_O(f_n + b'B_c) \tag{57}$$

The concentration C_{Ot} does not depend on C_O because f_n and b' are both inversely proportional to C_O. Another critical O_3 concentration can be defined, the O_3 concentration for which $b' = 1$:

$$C_{Ob} = bC_C = b'C_O \tag{58}$$

Also C_{Ob} is independent of C_O. From (58) it follows directly that if $C_O > C_{Ob}$ then $b' < 1$ and if $C_O < C_{Ob}$ then $b' > 1$. But the condition $C_O < C_{Ob}$ is only meaningful if $C_{Ob} > C_{Ot}$ and this is equivalent to:

$$f_n < b' - b'B_c \tag{59}$$

The validity of (59) is independent of C_O so if (59) holds for one C_O then there are C_O for which $b' > 1$ and if (59) is false for one C_O then $b' < 1$ for all C_O. If (59) holds it can also be shown from (56b) that a steady state solution, with $B(t) < B_{lim} < 1$ for large t, is not possible. Equation (56b) only yields curves similar to those found in models 2 and 3 if (59) if false and $B_c > B_{lim}$. In practical terms this implies that models 2 and 3 can be used only if repair rates are low enough or B_c is high enough.

The solution of (54) still has to be given; if $t > t_e$ or $1 > B > (1 - f_n)$ then:

$$B = B_c - (B_c - B_e)\exp(b'(t - t_e)/t_e) \tag{60}$$

where $B_e = B(t_e)$.

With (60) it is possible to get a good fit of the recovery period in Hill and Littlefield (1969) data for oats (Figs. 1 and 6), and the parameters t_s/b' and B_c can be determined (Table 1). The data points for P_n during O_3 exposure are best fitted with a straight line, which implies $b' = 1$ and (55) can be used. The correct inclination for the line can be found only if $f_n > 0$. The curve according to (56) with $f_n = 0$ is also given in Fig. 6 but does not fit so well as the line according to (55). This indicates that O_3 destruction without effect cannot be neglected.

The concentration dependence of $B_e(t_e = 1.1$ h) according to models 3, 4 and 5 with best fitting parameters for oats (from Fig. 6) is given in Fig. 7; the curve numbers correspond to those in Fig. 6. The absence of any effect below the threshold concentrations is probably not fully realistic because of the assumptions in (30) and the ignorance of any B dependence in (50). The real $B_e(C_O)$ curve probably increases slowly up to C_{Ot} and increases much faster for $C_O > C_{Ot}$. This is also indicated from the fact that Hill and Littlefield (1969) report effects on P_n only for $C_O > 1000$ $\mu g/m^3$ but their more sensitive measurements of stomatal width show effects for $C_O > 100$ $\mu g/m^3$ within a few hours. (For large stomatal width w a decrease of w causes a relatively small decrease in stomatal resistance, σ and w are not simply proportional!)

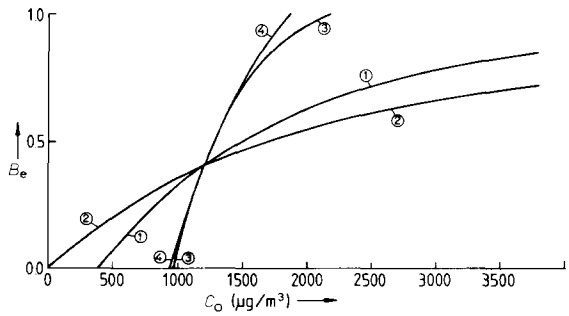


Fig. 7. Reduction of net assimilation and stomatal conductance at the end of O_3 exposure; B_e as a function of the O_3 concentration C_O. Models 2, 4 and 5 are represented by curves 1, 2 and 3 respectively. Curve 4 is the result for model 5 with $f_n = 0$. The exposure duration is fixed at $t_e = 1.1$ h. For the other model parameters see Table 1.

Figure 8 illustrates the response of oats to O_3 according to model 5 for a larger range of t and C_O. Condition (59) holds for oats, so solutions with a positive exponent in (56) are possible and Fig. 8 shows that this does not have to cause an irrealisticly fast increase of $B(t)$. This figure also illustrates that the change from equation (56) to (60) at $B(t_e) = (1 - f_n)$ only causes a discontinuity in the second derivative of $B(t_e)$. For the highest C_O this discontinuity causes a minimum in the slope of the curve. This minimum would be more pronounced if (59) held; B_c would be just below $1 - f_n$, and

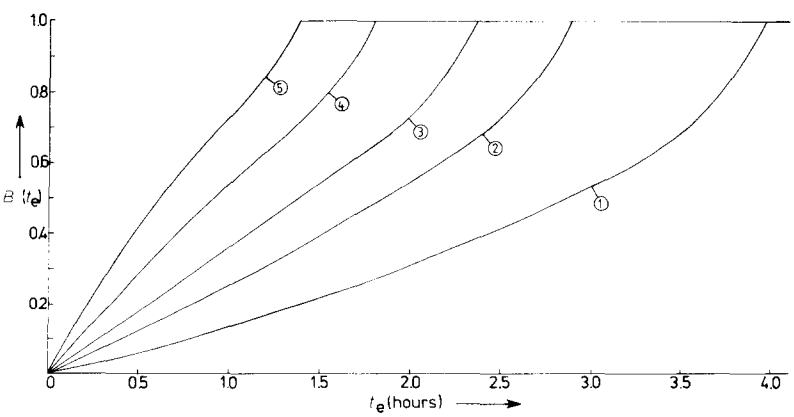


Fig. 8. Reduction of net assimilation and stomatal conductance at the end of O_3 exposure; B_e as a function of the exposure duration t_e. All curves are calculated using model 5, with $C_O = 1040$, 1120, 1200, 1360 and 1600 $\mu g/m^3$ for curves 1, 2, 3, 4 and 5, respectively. For the other model parameters see Table 1. The lack of a steady state value for B_e is typical for the class of plants to which oats belong.

a near steady-state situation might occur. This is a possible explanation of the 'hesitation' in the P_n suppression measurements of Black et al. (1982).

Model 6: Visible leaf injury

Visible leaf injury was found to be associated with an irreversible suppression of P_n (Black et al., 1982), so as a first approximation the fraction of the leaf that is injured can be set equal to $B(t)$ for large t. But for large t model 5 does not give a realistic prediction; only $B = 0$ or $B = 1$ are possible results, so a more detailed model is needed.

Injury on leaves can be seen as spots, which indicates that the leaf surface is not homogeneous. This may be explained by differences in stomatal density and stomatal aperture but biochemical factors will also vary over the leaf surface. The leaf will now be considered as a sum of leaf parts for which (60) applies after the O_3 exposure. The whole plant parameters are considered as averages of their values in leaf parts. Examination of the percentage visible leaf injury usually occurs quite a long time after exposure at $t = t_v$. Then (60) reduces to a simple relation independent of b':

$$\begin{aligned} B_v &= 0 \quad \text{if} \quad D < 0 \\ B_v &= 1 \quad \text{if} \quad D > 0 \end{aligned} \tag{61}$$

where $D = B_e - B_c$, $B_e = B(t_e)$, $B_v = B(t_v)$ and $t_v \gg t_e$.

To include the differences in sensitivity of leaf parts into the model, a probability density $p(D)$ is introduced. If $p(D)$ is assumed to be Gaussian:

$$p(D) = \frac{1}{\sqrt{2\pi}\, s} \exp\left(-(D - \bar{D})^2/(2s^2)\right) \tag{62}$$

where $\bar{D}$ is the mean value of D which is equal to the actual measurement or calculation of $B_e - B_c$ for the whole leaf, and s the standard deviation of D representing the inhomogeneity of the leaf, which is assumed to be small.

The expectation value of B_v and $\bar{B}_v$, which gives the percentage of leaf injury, can now be calculated:

$$\begin{aligned} \bar{B}_v &= \int_{-\infty}^{+\infty} B_v(D)\; p(D)\; \mathrm{d}D \\ &= \tfrac{1}{2} + \frac{1}{\sqrt{2\pi}} \int_0^{\bar{D}/s} \exp(-x^2/2)\; \mathrm{d}x \\ &= \tfrac{1}{2}(1 + \phi(\bar{D}/s)) \end{aligned} \tag{63}$$

The function $\phi(z)$ is tabulated, for example in Squires (1972). If $z < 0$ then $\phi(z) = -\phi(-z)$ can be used. Up to now the percentage of irreversible suppression of P_n has been assumed to be equal to the percentage of visible

leaf injury which may be unrealistic. The results of Black et al. (1982) for *Vicia faba* indicate an overestimation of the leaf injury index by using $\overline{B}_v$, especially for $\overline{B}_v > 0.3$. In the next section it will be assumed that this can be compensated for by a linear correction on $\overline{B}_v$.

Models 2 and 6: dose and dose–effect relations

The prediction of leaf injury with model 6 needs only one parameter from the model describing the plants' reaction during O_3 exposure: $\overline{B}_e$, which is equal to B_e for the whole leaf as considered in models 1 to 5. This parameter can be interpreted as a new definition of dose, which can also be justified

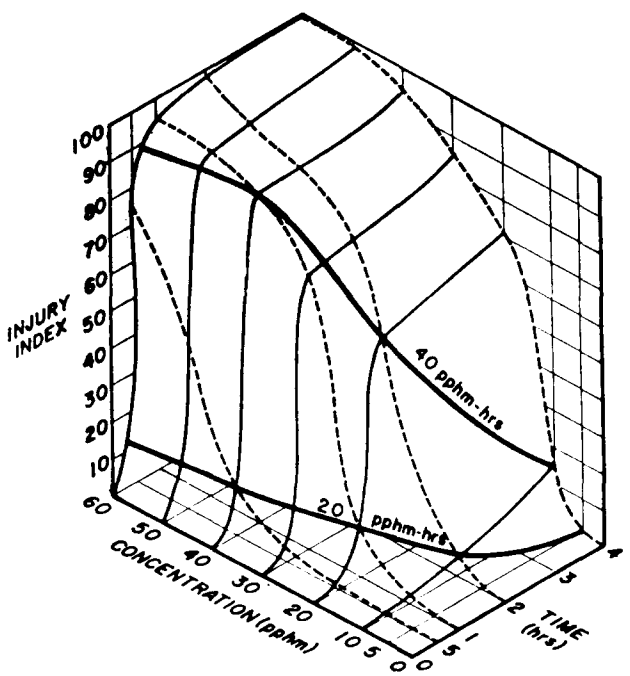


Fig. 9. Relation of the leaf injury index, exposure duration (time = t_e) and O_3 concentration (C_O) as found by Heck et al. (1966) for Pinto bean; 1 pphm (parts per hundred million) $O_3 \triangleq 20\ \mu g/m^3$ O_3. For the fat lines the product of concentration and time is constant. The corresponding injury index is not constant, so a better definition of dose is needed.

It is remarkable that for O_3 concentrations between 400 and 1000 $\mu g/m^3$ the injury index does not change after $t_e = 2$ h. This can be explained by partial stomatal closure (model 2); see Fig. 4. It is also remarkable that the maximum leaf injury index is only 100% for very high O_3 concentrations. This can be explained by differences in O_3 sensitivity of leaf parts (model 6) and the capacity of leaf parts to recover from a small amount of O_3 injury (model 5). Reprinted with permission © 1966 American Association for the Advancement of Science, Washington, DC, U.S.A.

with (15) and (29); B_e/k is the effective total uptake of O_3 at the end of the exposure. The definition of dose as B_e has an advantage above other definitions because it includes, to a certain extent, the effect of environmental and plant parameters. There are, however, some problems in applying model 6 to B_e values calculated with model 5. If the repair rate (at $t = 0$) is high then $B > (1 - f_n)$ may occur for $t < t_e$ and the different functions for $B < (1 - f_n)$ and $B > (1 - f_n)$ may both apply to leaf parts and the distribution $p(D)$ will not be Gaussian. Therefore only low repair rates will be considered from now on. Then model 5 can be approximated by model 2 for $t \leqslant t_e$ which is also much simpler to use; for model 2 only t_s and f_n have to be known.

For Pinto bean it can be verified that repair is slow compared to the suppression of σ (Sutton and Ting, 1977; Rich and Turner, 1972). This makes the set of leaf injury data for Pinto bean, reported by Heck et al. (1966) and Larsen and Heck (1976) suitable to be used as a test of model 6 in combination with model 2. Especially the presentation of the Pinto bean

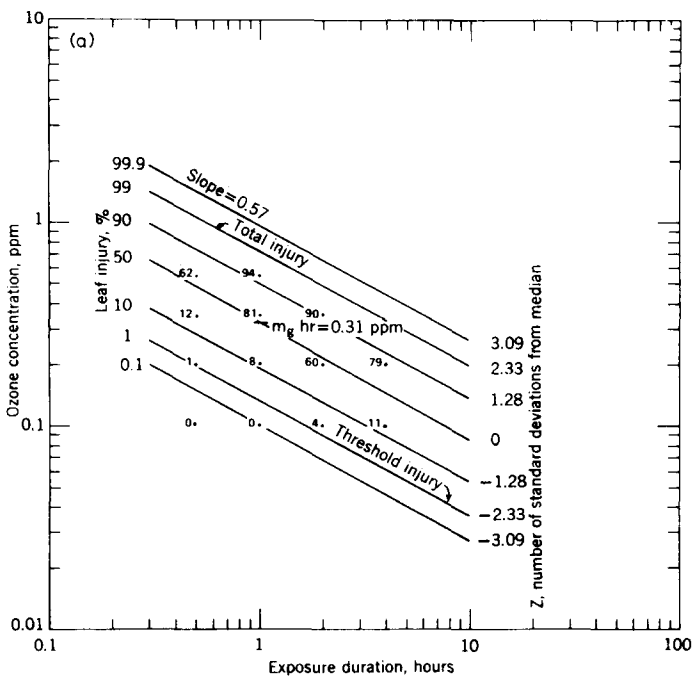


Fig. 10a. Percentage of leaf injury in Pinto bean plants exposed to various ozone concentrations for various durations, concentration versus exposure duration, as given by Larsen and Heck (1976), 1 ppm $O_3 = 2000\ \mu g/m^3$ O_3. The straight lines give the best fit to the data points according to the model of Larsen and Heck. Reprinted with permission © 1976 Air Pollution Control Association, Pittsburgh, PA, U.S.A.

data in Heck et al. (1966) permits an interesting test of the new definition of dose (Fig. 9). The leaf injury index (LI) increases rapidly for exposure durations $t_e < 2$ h, but for $t_e > 2$ h LI is nearly constant if $400 < C_O < 1200$ $\mu g/m^2$. For $C_O < 400$ $\mu g/m^3$ the increase of LI with time is much slower and at about 100 $\mu g/m^3$ no effect occurs, corresponding to a threshold concentration close to that found for *Vicia faba* and *Petunia* (at high light intensity, see model 2). Most remarkable is the nearly constant level of LI below 100% for $t_e > 2$ h and $C_O > 400$ $\mu g/m^3$. Then LI only depends on C_O which has not been explained up to now. Models 2 and 6 do explain a constant level of B_e and LI ($= c\bar{B}_v$) for large t, which can be illustrated with the curves given for *Petunia* in Figs. 4 and 5. Physiologically this can be explained by partial stomatal closure and a decrease of the O_3 flux to the threshold flux F_t. This implies that a stomatal reaction should occur for $t < 2$ h if $C_O > 400$ $\mu g/m^3$ and this can be checked. Rich and Turner (1972) have measured the stomatal resistance r_s before and after an exposure to

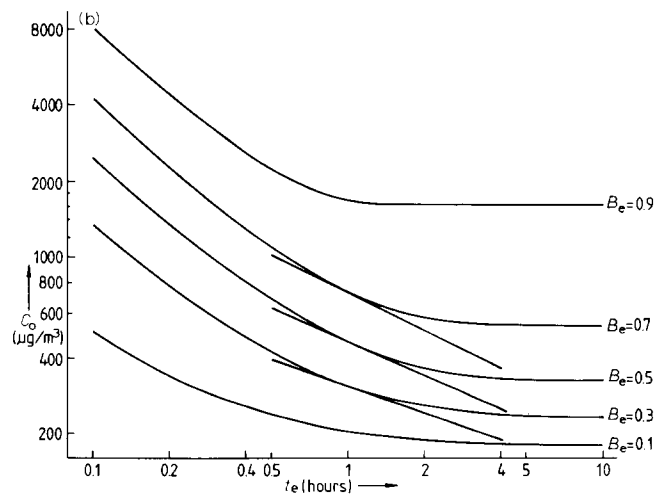


Fig. 10b. Relation between the O_3 concentration C_O and the exposure duration t_e on logarithmic scales for constant B_e calculated with model 2. B_e/k (k is a constant) is the effective total uptake of O_3 per unit of leaf area. Only a one-to-one relation between leaf injury and effective O_3 uptake has to be assumed to see that these curves also represent the relation between log C_O and log t_e for a constant leaf injury index. The model parameters $t_s = 0.4$ h and $f = 0.2$ were also shown to be useful for Pinto bean so Fig. 10a can be compared with Fig. 10b. Especially the points near the 50% leaf injury line show that the curves in Fig. 10b give a better fit to the experimental data. The tangents to the curves in Fig. 10b are approximately parallel to the lines in Fig. 10a, which supports the choice of the model and the parameters; the lines of Larsen and Heck (1976) are a first approximation of the real curves.

about 400 $\mu g/m^3$ O_3 for 30 min using well watered Pinto bean plants. In a dry atmosphere r_s changed from 370 to 670 s/m but in a moist atmosphere r_s did not change at all. So the leaf injury and stomatal data are consistent only if the results of Heck et al. (1966) were obtained in a dry atmosphere. This is probable because without artificial humidification the atmosphere in exposure chambers as used by Heck et al. is usually dry (Tonneijk, personal cummunication, 1984). The data for Pinto bean treated up to now indicate a stomatal reaction close to that of *Petunia*, so model 2 with $t_s = 0.4$ h and $f_n = 0.2$ will be used for $t \leqslant t_e$ to analyse the Pinto bean data as presented by Larsen and Heck (1976). The relation between exposure concentration C_O and duration t_e for a constant leaf injury index can easily be given because only a one-to-one relation between dose (B_e) and the injury index has to be assumed. The C_O–t_e relation for constant B_e according to model 2 is given in Fig. 10b. In contrast to the model of Larsen and Heck (1976), the relations are not straight lines but have a curved shape, more like the envelopment curves used by Posthumus et al. (1983). A comparison of the

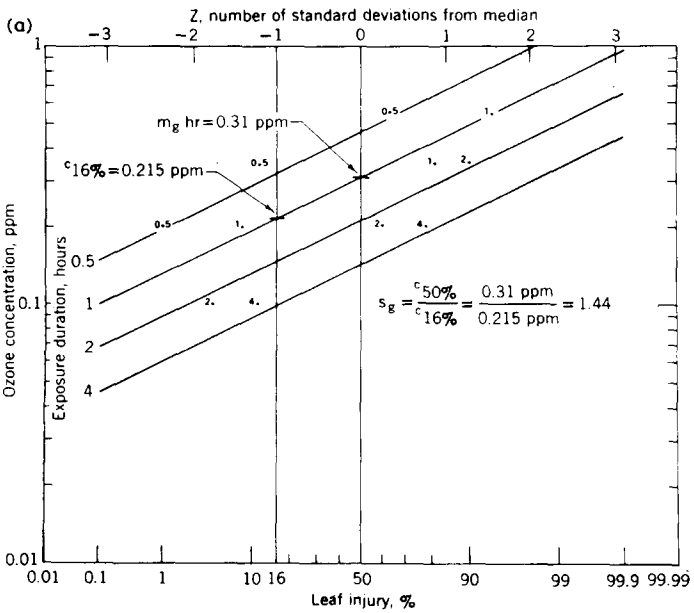


Fig. 11a. Percentage of leaf injury in Pinto bean plants exposed to various ozone concentrations for various durations, concentration versus percentage leaf injury, as given by Larsen and Heck (1976); 1 ppm $O_3 \triangleq 2000$ $\mu g/m^3$ O_3. The straight lines give the best fit to the data points according to the model of Larsen and Heck. Reprinted with permission © 1976 Air Pollution Control Association, Pittsburgh, PA, U.S.A.

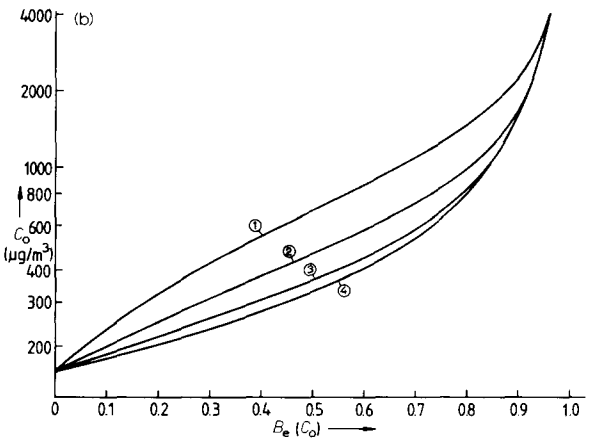


Fig. 11b. Relation between B_e and the O_3 concentration C_O for various exposure durations calculated with model 2 and model parameters for Pinto bean. Curves 1, 2, 3 and 4 give $B(C_O)$ for $t_e = 0.5$, 1, 2 and 4 h respectively. The linear B_e scale corresponds to the probability scale in Fig. 11a (using model 6). Comparison of Figs. 11a and 11b shows that model 2+6 gives a better description of the experimental data than the lines on equal distances used by Larsen and Heck (1976).

data given in Fig. 10a with the curves in Fig. 10b shows that the model curve gives a better fit to the data than do the straight lines used by Larsen and Heck (1976), but the inclination of these lines is nearly equal to the inclination of the tangents to the model curves. The deviation of a straight

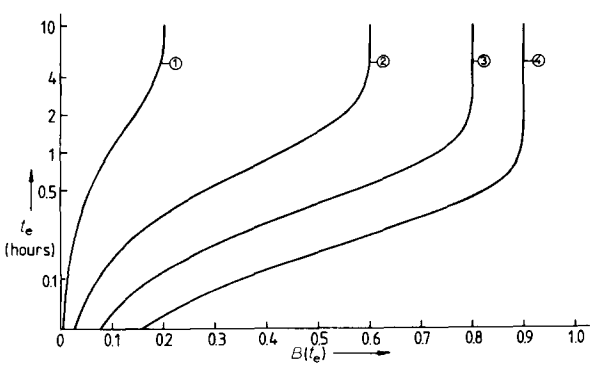


Fig. 12. Relation between B_e and the exposure duration t_e for various O_3 concentrations C_O calculated with model 2 and model parameters for Pinto bean. Curves 1, 2, 3 and 4 are given for $C_O = 200$, 400, 800 and 1600 $\mu g/m^3$ O_3, respectively. The linear B_e scale corresponds to a probability scale for the leaf injury index (model 6). Note that the range for which $B(t_e)$ is linear is very limited if $t_e > 0.5$ h.

line in $\log(C_O) - \log(t_e)$ plots is also reported by Larsen and Heck: they notice a break in the curve between 1 and 2 h. This corresponds to the effect of stomatal closure as was already discussed.

Also for the relation between O_3 concentration and injury index a comparison between experimental data and model curves can be made. For this purpose Fig. 4 has been redrawn on scales comparable with those used by Larsen and Heck (1976) (Figs. 11a and b). According to model 6 a linear B_e scale corresponds to the probability scale used by these authors. Even in deviations of the approximate straight lines the model curve describes the experimental data. Curves for the relation between exposure duration and injury index also have been given (Fig. 12) but Larsen and Heck did not give the data points to test this relation.

DISCUSSION

In the models given in this paper all relevant processes have been assumed to be coupled through the rate of CO_2 assimilation P_n. This probably holds only if the light availability determines CO_2 uptake. If other factors, such as water or nutrients are limiting CO_2 uptake adapted models will be needed. Also a high humidity of the air may cause a failure of the assumptions used to derive the models. At high relative humidity stomatal closure was found to be inhibited (Rich and Turner, 1972; Elkiey and Ormrod, 1979) and probably C_{Ci} in equations (2a) and (2b) is not constant. This is consistent with the absence of stomatal regulation as found in hot and humid environments (Goudriaan, personal communication, 1984). There are even plants, for example sunflower, for which stomatal regulation is absent at any air humidity (Louwerse, 1980).

To extend the models to periods longer then 1 day, several kinds of processes have to be included. For dark periods, pools of assimilates have to be considered in a description of the repair process. As was noticed before, C_{Ci} may change after O_3 exposure on subsequent days, which may be associated with the same hormonal processes that cause enhanced leaf ageing (Tingey et al., 1982; Mooi, 1983; Reich, 1983). Also changes in assimilate translocation have to be included in long term models (Tingey et al., 1971; Arndt et al., 1982).

Effects of other pollutants in combination with O_3 can be included only if the mechanisms of their interactions are specified. For example, if O_3 and NO_2, which are both oxidants, react with the same kind of chemical bonds, the O_3 concentration C_O can be replaced by: $C_{Ox} = C_O + gC_{NO2}$ where C_{NO2} is the NO_2 concentration and g indicates the toxicity of NO_2 relative to O_3. This may explain the synergistic effect on photosynthesis that was observed by Furukawa and Totsuka (1979) because the weighted sum of the O_3 and

NO_2 flux may exceed the threshold flux F_n while the separate components are still below F_n. Also synergistic, irreversible effects of O_3 and NO_2 (Omasa et al., 1979) can be explained, the individual components may not cause a suppression of P_n below P_c while the combination of O_3 and NO_3 does. Other effects of NO_2, which may interact with the SO_2 effects are described by Mansfield and Freer-Smith (1981) with possible interaction mechanisms.

For simultaneous exposures to SO_2 and O_3 Black et al. (1982) report the effects to be additive which indicates independent mechanisms for the suppression of P_n by SO_2 and O_3. For high O_3 concentrations an antagonistic effect was found which may partly be explained by stomatal closure.

Modelling the effect of O_3 after pre-exposure to SO_2 may be easier and also more useful because a photochemical period may be preceeded by a long exposure to background SO_2. Hofstra and Beckerson (1981) report a decrease of O_3 sensitivity for white bean and an increase of O_3 sensitivity for cucumber caused by SO_2 pre-exposure. A decrease of O_3 sensitivity can be explained by an increase of F_n and the increased sensitivity for cucumber can be explained by a decrease of F_n or an increase of P_c. The changes in these parameters should be related to SO_2 uptake and S-metabolism (Keller, 1978; Priebe et al., 1978; Hälgrenn and Fredriksson, 1982; Sekiya et al., 1982; Schut, 1984, 1985).

This kind of reasoning in the comparison of literature results shows how a simple quantitative model for O_3 effects can be helpful to explain experimental facts. Without treating biochemical details it can be shown that these facts are not contradictory if typical plant properties and environmental conditions are taken into account.

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