fin fiber support pads used under the membrane. This agent also accounts for the foaming of redistilled water after passage through the filter, as observed by Simpson. Others have reported to us that certain tissue cultures were similarly affected.

Immediately after the discovery of the inhibitory property, we recalled and destroyed about 10,000 suspect units. All Nalgene Filter Units now have a support pad of an entirely different material (cellulose fibers treated with an inert resin), and extensive tests show that no inhibitory effects are produced. Each lot is tested for toxicity to *Pseudomonas* spp. and other sensitive organisms.

We can furnish a reasonable number of sample Filter Units at the request of any laboratory or investigator who may wish to verify the nontoxicity of the present Nalgene Disposable Membrane Filter Units.

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Ozone Dose and Plant Injury

In a report on nonlinear responses of pinto bean and tobacco plants to ozone, Heck, Dunning, and Hindawi (*1*) took issue with our "empirical exposure factor" and with our calling our damaging oxidant "ozone." The exposure factor made a linear relation between data for ozone dose and injury to tobacco in the field (*2*), in accordance with a linear relationship described by Middleton (*3*).

In heavy experimental fumigations similar to those of Heck *et al.*, one of us had previously noted that interference by stomatal closure limited sensitivity at high doses (*4*, table 9). Increased environmental stress in a greenhouse fumigation chamber may be partly responsible, but ozone itself is known to close leaves' stomata, for example within 1 hour in 80 pphm ozone (*5*), and this effect persists (*6*). There is another reason for nonlinearity at high concentrations. The visual rating of injury presumably done by Heck *et al.* bears a nonlinear (logarithmic) relation to actual injury, as it is insensitive at the upper end of the range (*7*).

However, in the case of weather fleck in tobacco (*2*), we are concerned with the less severe responses of well-adapted plants in the field to low concentrations of ozone. The maximum concentrations of ozone on the 52 damage-inflicting days of 1960 averaged 3.9 pphm, and the average was 5.8 pphm for the 37 days that damaged plants in 1961. Over the effective 15 hours of daylight on these particularly polluted days, the average hourly concentrations were 1.8 pphm in 1960 and 3.4 pphm in 1961, and the average ozone doses were about 29 and 52 pphm-hours, respectively. The threshold dose was 200 pphm-hours under natural conditions, interrupted natural conditions, and artificial fumigations in the field (*2*). Reference to the curves of Heck *et al.* shows agreement with our threshold dose at low concentrations and suggests that we were operating largely in a linear range of dose response. The responses we observed in the field, especially at the lower doses, were neither linear nor nonlinear but random in relation to ozone dose (pphm-hours) so that injury clearly depended on additional factors. Even at frequently occurring higher ozone concentrations (5 to 15 pphm), which tended to be accompanied by more humid air masses and relatively low wind speeds, a given dose of ozone did not produce a given amount of injury. In the absence of continuous measurements of physiological factors, micrometeorological factors were examined, and all anomalous plant responses, could be accounted for in terms of those having a particular physiological significance. The "empirical exposure factor" which gave dose-response linearity was obtained in the form of the coefficient of evaporation derived from the ratio of actual evapotranspiration to the product of wind speed and vertical water-vapor gradient (*8*). It was postulated that this coefficient indirectly reflects among other factors the degree of exchange of gases between air and leaf and thus determines the downward ozone flux actually available for absorption. The well-known inverse relation between sensitivity and moisture stress (*2, 4, 6*) is also handled by the expression. For example, under conditions of strong advection, when the actual evapotranspiration is less than the wind and moisture gradient would indicate, the development of moisture stress is favored and the coefficient of evaporation is relatively low, resulting in a low modified ozone dose and less flecking.

No significant amounts of competitive reducing agents, spent or active, were present in the air during our field studies. Organic oxidants were not detected in toxic quantities, and the amount of air-polluting ozone determined by several analytical methods, including rubber cracking, accounted for all routinely measured oxidation of potassium iodide. Field plants responded to fumigation with carbon-filtered air containing artificially generated ozone, both qualitatively and quantitatively, as they did to equal doses of the air-polluting ozone.

In conclusion, the relation of ozone dose to injury of susceptible mesophyll tissue under natural conditions has not been shown to be nonlinear. Apparent nonlinearity in the relation may be a consequence of not measuring ozone uptake, of ignoring the concentration- and time-dependent limitation imposed by stomatal closure, of disregarding the meaning of threshold dose, and of using a nonlinear method of rating injury. We expect that a constant related to the rate of absorption is the governing parameter under a given set in environmental conditions (*9*). Where the latter are varying, as in the field, it is necessary to apply to the ozone dose, computed from measurements taken at a given point, a correction factor such as one derivable from prevailing micrometeorological conditions, in order to arrive at an expression of effective ozone dose.

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