violet irradiated RNA which had been recovered from the column was not significantly depolymerized. The small increase in ultra-violet absorbance in the first samples in the experiment presented in Fig. 1 *B* is the result of the breakdown of RNA, which in chromatograms of unirradiated RNA appears immediately after 23 *s* RNA and which contaminated our ribosomal fraction chosen for the described experiment. This breakdown is caused by factors other than irradiation[6].

One could suppose, therefore, that some irradiated RNA molecules are specifically retained on the column by binding sites which arose during ultra-violet irradiation.

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H. Kubinski

Laboratorium der Stiftung zur
Erforschung der spinalen
Kinderlähmung und der Multiplen Sklerose,
Universitäts Krankenhaus,
Hamburg-Eppendorf, Germany.

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BIOLOGY

An Effect of 'Smog' on Stomatal Behaviour

It was shown by Koritz and Went[1] that treatment with 'artificial smog' (hexene-ozone mixtures) could cause stomata to close, and also permanently destroy their capacity to re-open. We have recently observed an effect of 'natural' smog on stomata which does not appear to involve any permanent injury.

During the period December 2–7, 1962, this area was covered by a dense fog accompanied by unusually high air pollution, and we observed that opening of the stomata of *Xanthium pennsylvanicum* in light was slower, and the maximum degree of opening attained was less, than in normal weather. The effects are illustrated in Fig. 1. The experiments were carried out in a growth room ventilated by a small flow of air from outside. Some idea of the relative extent of pollution on the days in question is obtained from the results in Table 1, which are from a site in Reading 3 miles from these laboratories. These applied to the 24-h period 9 a.m.–9 a.m.; the measurements of stomatal opening were made starting at 9 a.m, so that polluted air drawn into the room before this would probably have been affecting the stomata, as well as that drawn in at the time; consequently figures for pollution on two consecutive days are given.

Table 1. Pollution in $\mu g/m^3$

Dates	Smoke	Sulphur dioxide
Nov. 29/30	147/152	214/296
Dec. 5/6	557/292	2,168/1,473
Dec. 6/7	292/242	1,473/322
Dec. 10/11	88/74	186/119

Nov. 29th = 9 a.m. on 29th to 9 a.m. on 30th, etc.

There appears to be a reasonable inverse correlation between the degree of pollution and the rate of opening as shown in the figure. We have no information as to which particular pollutants were affecting the stomata. It is not unlikely that both carbon dioxide and sulphur dioxide might be involved. Sulphur dioxide in smog is known to affect physiological processes in general[2], and

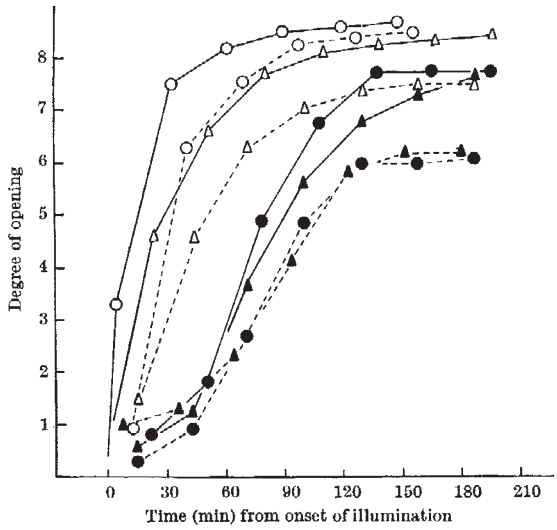

Fig. 1. Stomatal opening in 15,000 lux on four occasions: △, Nov. 30; ▲, Dec. 6; ●, Dec. 7; ○, Dec. 11. On each occasion there were measurements on a young leaf (broken lines) and an older leaf (continuous lines). The previous night length was 15 h in all cases. Ordinate scale in arbitrary units as defined elsewhere (ref. 3)

we have previously observed that small increases in carbon dioxide in growth rooms can cause a depression in stomatal opening[3].

There was no evidence of any permanent effect on the stomata, since measurements showed that they could open normally again after a return to normal weather (for example, the record for December 11 in Fig. 1).

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T. A. Mansfield
O. V. S. Heath

University of Reading
Horticultural Research Laboratories,
Shinfield Grange,
Shinfield, Berks.

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Chloramphenicol and Uptake of Salt in Plants

The antibiotic chloramphenicol inhibits the uptake of cations and anions by higher plant tissues when supplied at high concentrations (1–2 g/l.) (ref. 1). Since chloramphenicol specifically inhibits the synthesis of protein in bacteria[2], it has been suggested that this finding indicates a close connexion between salt uptake and protein synthesis in plants[1,3]. However, when precautions are taken to avoid the complication introduced by the inhibition by chloramphenicol of the uptake of amino-acids, no effect of chloramphenicol on the incorporation of L-leucine or L-threonine into the trichloroacetic acid-insoluble fraction of beet slices can be demonstrated, although salt uptake is inhibited under the same conditions[4]. These results suggest that the action of chloramphenicol on plant tissues may not be mediated in the manner anticipated from studies on bacterial systems.

The antibiotic chloramphenicol is the D-threo-isomer; the L-threo-isomer is not an antibiotic and is very much less active than the D-isomer at inhibiting the incorporation of amino-acids by bacterial extracts[5]. However, L-threo-chloramphenicol is metabolically active in plants, inhibiting the elongation of lupin roots even more effectively than the D-isomer[6]. Experiments were therefore performed to determine whether L-threo-chloramphenicol also inhibits the uptake of salt by higher plants.