means did not rise above 9 p.p.hm. at any time.

Bleasdale's data appear to show that S23 ryegrass is more sensitive to SO_2 than is apparent from the study by Bell and Clough. This discrepancy may be the result of their having performed experiments in different conditions, but it may equally have resulted from Bell and Clough's simulated SO_2 pollution rather than polluted air from outside. In Bleasdale's experiments other air pollutants would also have been present. One such is NO_2. This is found in polluted atmospheres because NO is produced by combination when fossil fuels are burnt, and is quickly oxidised in air to NO_2. We have recently completed some experiments in which the combined effects of SO_2 and NO_2 pollution on photosynthesis in the pea,

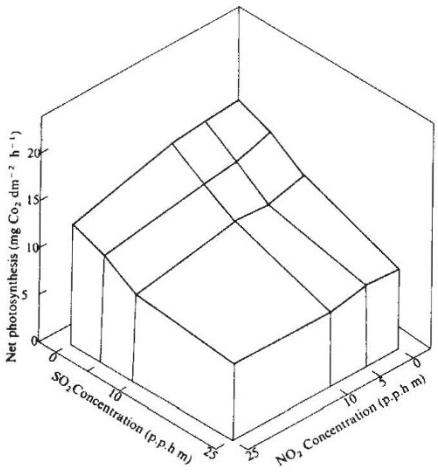

Fig. 1 Effects of simulated SO_2 and NO_2 pollution on net photosynthesis in *Pisum sativum*. Fumigation conditions: 1.20 air changes min^{-1}; temperature, 21° C; light intensity, 73 J^{-2} s^{-1}; water vapour pressure deficit, 995 N m^{-2}.

Pisum sativum, were examined over a concentration range of 0–25 p.p.hm. for each gas. Initially, exposure to these pollutants increased net photosynthesis, but the effect was short lived and a substantial inhibition soon followed. The magnitude of this inhibition for 15 different treatments is shown in Fig. 1, with the control included for comparison. Analysis of this factorial experiment revealed statistically significant effects of SO_2 and NO_2 ($P<0.001$ in both cases) but no significant $SO_2 \times NO_2$ interaction. It is clear, however, that the effects of the two pollutants are at least additive. These observations were made on detached leaves and no visible injury accompanied the depression of net photosynthesis, but more prolonged exposures to concentrations of 10 p.p.hm. and above of both SO_2 and NO_2 were found to produce visible lesions on intact plants. Visible injury to crop plants after simultaneous exposure to SO_2 and NO_2 has been reported previously[3].

Our data suggest that combined effects of SO_2 and NO_2 might explain the growth inhibition caused by urban pollution[2], and that this possibility should be considered in future studies.

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Yours faithfully,

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Regulation of albumin-bound tryptophan

Sir—Madras *et al.*[1] recently reported on the effect of tryptophan concentrations in serum and brain concerning its conversion to serotonin in rats. This and related work have been summarised by Fernstrom and Wurtman[2] who suggest that albumin-bound tryptophan is the precursor of serotonin. These authors[1,2] have found that decreased nonesterified fatty acid content (albumin-bound) in the serum caused by administration of carbohydrate or insulin results in higher concentrations of albumin-bound tryptophan, while fasting (high fatty acid content in serum) results in lower albumin-bound tryptophan levels, suggesting that fatty acids competitively prevent the binding of tryptophan by albumin.

If one assumes a serum albumin concentration of 4 g per 100 ml, the data of Madras *et al.*[1], on the concentrations of serum-bound tryptophan and fatty acids can be converted into moles of tryptophan or fatty acid bound moles of serum albumin (Table 1). Thus conditions which decrease concentrations of fatty acids in the serum increase concentrations of albumin-bound tryptophan. McMenamy and Oncley[3] found that bovine albumin bound 1 mol of L-tryptophan per mol of albumin. Addition of fatty acid decreased the binding capacity of the albumin for tryptophan by 0.1 mol for each mol of fatty acid. When 2.0 mol of oleate was present the albumin bound 0.75 mol of tryptophan.

Since 2.0 mol of fatty acid per mol of albumin represents the largest fatty acid ratio in Table 1, I assume that a minimum of 0.75 mol of tryptophan could be bound to the albumin rather than the maximum of 0.135 mol observed if fatty acid competition were the limiting factor.

The free energy (−7.2), enthalpy (−14.5) and entropy (−24) changes determined by Fairclough and Fruton[4] for the binding of L-tryptophan to serum albumin are similar to those observed for binding of iodide[5], lysolecithin[6] and probably dodecyl sulphate[5] to the high energy site of albumin. Thus such a material, which is strongly bound to the albumin, might decrease the amount of this site available for tryptophan and so function, along with fatty acids, to regulate the amount of tryptophan bound.

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Table 1 Effects of glucose and diet on bound tryptophan and fatty acids

	Effect of glucose			Effect of diet		
	Control	Glucose 1 h	Glucose 2 h	Fasted control	Carbohydrate + fat	Carbohydrate
Serum bound tryptophan mol per mol albumin	0.091	0.13	0.135	0.09	0.11	0.135
Non-esterified fatty acids mol per mol albumin	2.0	1.14	1.05	1.46	1.08	0.53

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