may be an isomer of phosphoglycolaldehyde[8]. The quantitative yield of phosphoserine is shown in Table 1.

The individual reactions involved in the synthesis of phosphoserine :

D-3-Phosphoglycerate + DPN$^+$ ⇌
phosphohydroxypyruvate + DPNH + H$^+$

Phosphohydroxypyruvate + L-glutamate ⇌
phosphoserine + α-oxo-glutarate

have been demonstrated, and the specificity and properties of the enzymes are under investigation.

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Effect of Copper on Growth and Catalase Levels of *Corcyra cephalonica* St. in Zinc Toxicity

The work of Smith and Larson[1], van Reen[2] and van Reen and Pearson[3] has revealed that the dietary ingestion of toxic levels of zinc results in a marked inhibition of growth and deranged iron metabolism in rats. The latter effect was reflected in anæmia and decreased levels of liver catalase and cytochrome oxidase. Liver extract partially counteracted the growth inhibition, while minute supplements of copper to the toxic diet reversed the anæmic condition[1] and restored the levels of the iron enzymes[2]. From such results it has been concluded that the inhibition of growth is unrelated to the enzymic changes observed, and that the two phenomena are distinct and different effects of zinc toxicity in the animal organism.

In view of our earlier studies on zinc toxicity in the larvæ of the rice moth, *Corcyra cephalonica* St.[4], it was thought of interest to investigate the zinc–copper relationship from this point of view. For this purpose, groups of 10–15 day-old larvæ, weighing between 7·0 and 9·0 mgm. per ten, were grown for a period of three weeks on the following diets : (1) control, basic diet of sieved wheat flour ; (2) zinc toxic-basal diet plus 0·4 per cent $ZnSO_4,7H_2O$; (3) zinc toxic diet as in (2) with graded amounts of $CuSO_4,5H_2O$; and (4) zinc toxic diet supplemented with 1·0 per cent liver extract. The last group, namely, that with liver extract, was included for purposes of comparison with earlier work[1–4]. The experimental procedure involved in the preparation of the liver extract and the rearing of the larvæ has been described in detail elsewhere[4]. Growth was recorded at weekly intervals. At the end of three weeks, larvæ from each group were weighed and homogenized in *M*/15 phosphate buffer (*p*H 7·0). Catalase activity was determined by allowing aliquots of the buffer extract to act upon 0·05 *M* hydrogen peroxide and titrating the unreacted hydrogen peroxide, at the end of the reaction period, with *N*/100 potassium permanganate, according to the procedure of Ramachandran and Sarma[5]. Catalase activity was expressed as ml. of *N*/100 permanganate consumed per gm. of larval tissue per minute. The results obtained in a typical experiment are shown in Table 1.

Table 1. Influence of Copper on Zinc Toxicity in Rice Moth Larvæ

No.	Supplements to basal diet (10 gm. sieved wheat flour)*	Weight of 10 larvæ (mgm.) At transfer	1st week	2nd week	3rd week	Catalase units
1	Control (no zinc)	7·7	38	105	226	493·2
2	None	8·3	22	57	100	309·1
3	0·005 per cent copper sulphate	7·0	23	56	103	393·6
4	0·01 per cent copper sulphate	8·2	22	59	98	540·6
5	1·0 per cent liver extract	6·0	31	94	194	315·5

* Unless otherwise stated, all diets also contained 0·4 per cent zinc sulphate.

It can be seen that the intake of toxic amounts of zinc brings about not only an inhibition of growth but also a pronounced fall in tissue catalase activity. 0·01 per cent copper sulphate, added to the zinc-toxic diet, restores the enzyme levels without, however, improving growth to any extent. Sivarama Sastry, Radhakrishnamurty and Sarma[4] have recently shown that this growth inhibition can be considerably reversed by the inclusion of liver extract in the toxic diet. The results presented in Table 1 show that liver extract has little effect on catalase activity, however.

These results indicate that the inhibition of growth and in catalase activity are separate effects of zinc toxicity in the insect organism, as in the rat[1,2], and point to the probable multiplicity of metabolic disturbances involved in zinc toxicosis. Since it is well known that catalase level in animal tissues is decreased by either an iron or copper deficiency[6] and that copper influences iron metabolism[7], it is likely that the antagonistic behaviour of zinc and copper brought out by the present investigation is a reflexion of an adverse effect of zinc on iron metabolism in the larvæ. It is also possible that such an interference with iron metabolism is a common phenomenon in zinc toxicosis.

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