the Vellar estuary (11°29' N; 79°46' E). The F/2 medium of Guillard[1] was used for culture of the diatom. The culture was maintained in 25‰ salinity at 29 ± 1°C under illumination of 4000 lux in a 12 h light and 12 h dark cycle. The cultured cells were harvested after they reached stationary phase (7th day). Cells (4.46×10^6) were extracted with hot methanol and the extractions were fractioned with diethyl ether, 1-butanol and water[2]. Only the water-soluble fraction, which was shown to be toxic[3], was used in the experiment. The water-soluble fraction was dialysed and the dialysate was evaporated to dryness in a vacuum evaporator.

Aqueous solution (10 mg/ml) of the substance was prepared for the isolated-heart experiment[4]. Frog's heart was isolated and perfused with Clark's frog Ringer solution[5] at pH 7.4. The effect of different concentrations of the test extract on the heart was recorded. The depressant effect of the test extract was compared with that of acetylcholine using atropine as the blocker. Similarly the depressant effect of the test extract was compared with that of propranolol, a beta-blocker, using adrenaline as the agonist. The experiment was repeated ten times. All the results were consistent.

The cardiac depressant effect of the test extract is dose-related (figure 1). The test extract produced cardiac depressant effect even after atropinization (figure 2), indicating a non-cholinergic nature of action. Figure 3 shows the effect of the test extract during the response to 2 μg of adrenaline. It is clear that the test extract produces initial inhibition followed by its own partial agonistic effect, and then blocks the effect of adrenaline-induced stimulation, unlike propranolol which has no partial agonistic action (figure 4). The test extract may be a beta-blocker with associated partial agonistic activity. Further investigation is in progress.

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MODULATION OF PROTECTIVE EFFECTS OF VITAMIN C IN AFLATOXICOSES

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WIDESPREAD infestation of food and feed by toxigenic strains of *Aspergillus flavus*[1,2] is known to cause mild to severe aflatoxicoses. The damage can be histopathologic[3,4], carcinogenic[5] or mutagenic[6]. In order to minimize the hazards of aflatoxins, a search was made for various drugs that could nullify toxin-caused damage. The well-known antitoxicant, L-ascorbic acid (vitamin C)[7,8], was tested and the results are presented here.

The rate of increase in body weight of weaning guinea pigs (*Cavea cavea*), was used as a parameter for adjudging the general physical profile of the animal. Six- to seven-week-old healthy animals (supplied by Central Drug Research Institute, Lucknow) were fed 50 ± 10 ppb of crude aflatoxin per day per animal along with their normal food for 20 weeks. The method of laboratory elaboration of toxin, maintenance of the experimental animals, and the mode of feeding have been described elsewhere[3,4]. The animals were divided into six groups: (i) no administration of vitamin C or toxin (control), (ii) administration of vitamin C only (AA), (iii) administration of toxin only (AFT), (iv) concurrent administration of toxin and vitamin C (AFT + AA), (v) toxin feeding for 10 weeks followed by exclusive feeding with vitamin C for 10 weeks (AFT → AA), and (vi) vitamin C feeding for 10 weeks followed by exclusive feeding with the toxin for 10 weeks (AA → AFT). The dose of vitamin C was 2 mg/kg body weight which is proportionate with the dose prescribed for humans.

To study the effect on body weight, a weekly mean weight was calculated for each group of animals ($n = 12$ in each group) for up to 20 weeks. From these weekly means, the relative increase in

body weight (RIW) was calculated as

$$\text{RIW} = \frac{W_i - W_0}{W_0} \times 100,$$

where W_i and W_0 are mean weight in the ith and zero week respectively.

In each group the trend of increase in body weight obtained as RIW was found to be rectilinearly dependent on duration of feeding. Regression equations were calculated and the lines of regression were drawn (figure 1). The results obtained show that the feeding with aflatoxin alone markedly retards the pace of RIW ($y = -1.95 + 4.31\,x$). This retardation is satisfactorily annulled in the group that received aflatoxin and vitamin C concurrently. Some improvement was also found in groups that received vitamin C before or after the toxin.

Though the exact mechanism of action of vitamin C in these situations is not yet known, it can. however, be inferred that a regular oral dose of vitamin C can, to a great extent, minimize the effects of aflatoxicoses.

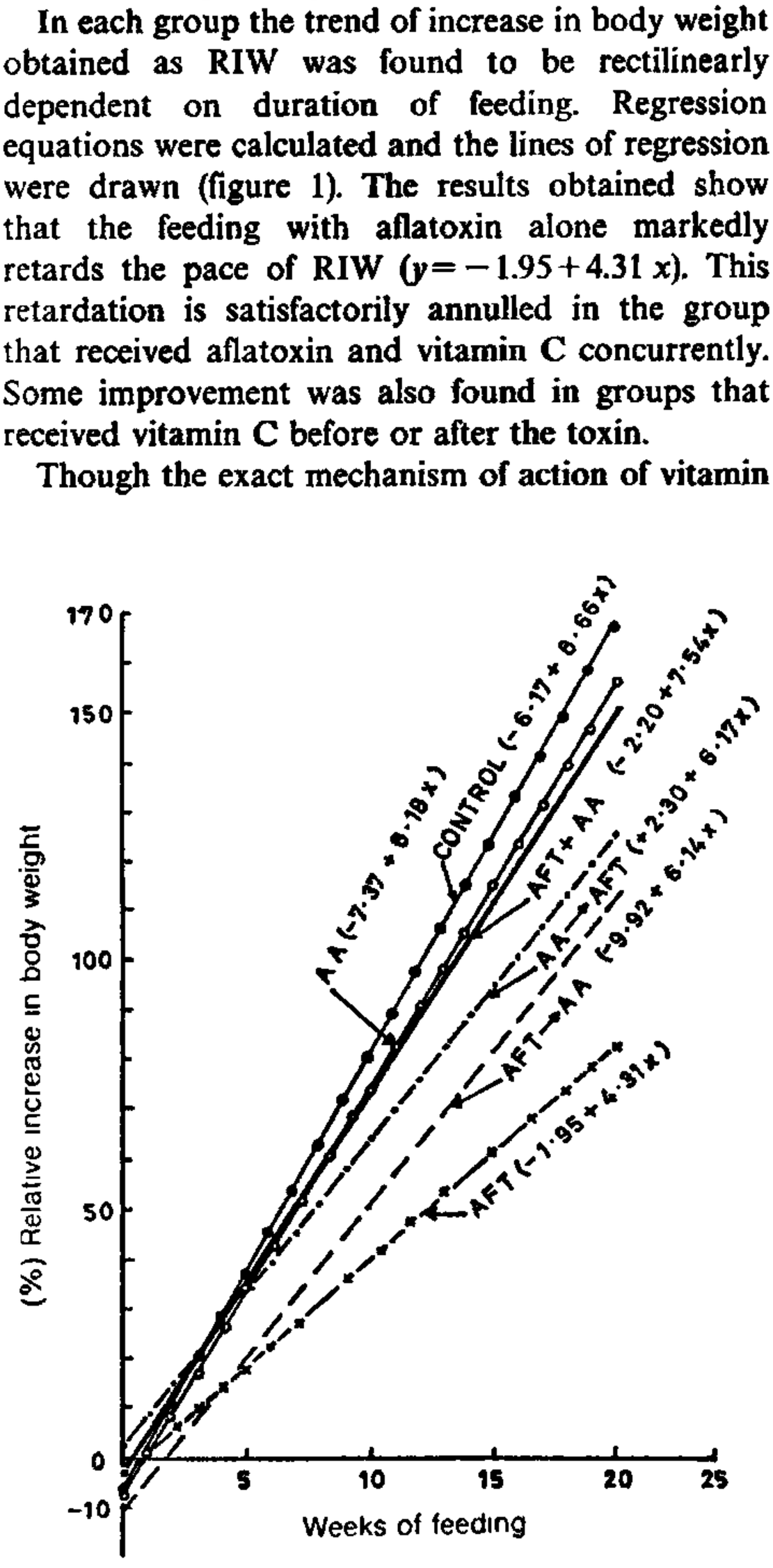

Figure 1. Lines of regression showing weekly relative increase in body weight after toxin and vitamin C treatments. ●———●, Control; O———O, ascorbic acid only (AA); ———, aflatoxin + ascorbic acid (AFT + AA); ·–·–·–·, ascorbic acid followed by aflatoxin (AA→AFT); – – –, aflatoxin followed by ascorbic acid (AFT→AA); – + – + –, aflatoxin only (AFT).

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PARASITES OF UZI FLY, *EXORISTA SORBILLANS* WIEDEMANN (DIPTERA: TACHINIDAE): A NEW RECORD

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In recent years five hymenopteran parasites, viz. *Brachymeria lugubris* (Walker), *Dirhinus himalayanus* (Chalcididae), *Nesolynx thymus* (Girault) (Eulophidae), *Spilomicrus karnatakensis* Sharma (Diapriidae), and *Exoristobia philippinensis* Ashmead (Encyrtidae), have been recorded as pupal parasites of uzi fly[1–4], a serious endoparasite of silkworm larvae *Bombyx mori* L.[5] These parasites were earlier reported as primary parasites of Calliphoridae, Muscidae, Sarcophagidae and Tachinidae, and secondary parasites of many lepidopterans through dipterans[6–10].

Recently two more hymenopteran parasites, viz. *Spalangia cameroni* Perkins and *Pachycrepoideus vindimae* (Rondani) (Pteromalidae), were tried on uzi fly maggots and pupae with a view to evaluate their usefulness in biological control of uzi fly[11]. These hymenopterans are usually predominant as house fly