activity factor was excluded. All mice were adult (about 90 days old). The result is shown in curve IV.

The spread in liver catalase activity factors per whole liver was thus reduced from 4–54 (curve I) to 8–22 (curve IV) or to 28 per cent of the original value.

With this method we tested substances separated from cancer tissue. When injected in mice the substances produce a marked decrease in liver catalase activity. Our results agree with those of Nakahara and Fukuoka with 'toxohormone'[6].

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Toxicity of a Single Subcutaneous Dose of Sodium Selenite in Pigs

SINCE the activity of 'factor 3' in liver necrosis in rats and exudative diathesis in chicks was shown to be dependent on selenium[1], compounds of this element have been shown to prevent other diseases induced by tocopherol-deficient diets. Sodium selenite (0·2 p.p.m.) as well as tocopherol acetate inhibit muscular degeneration in pigs fed grain, which causes field outbreaks[2]. Muscular degeneration in pigs is associated with a rise in plasma glutamic-oxaloacetic transaminase, but normal ornithine-carbamyl transferase[3]. When the disease is manifested, high glutamic-oxaloacetic transaminase levels rapidly return to normal after subcutaneous administration of 0·02 mgm. selenium (as Na_2SeO_3) per kgm. body-weight daily for three days (Orstadius *et al.*, unpublished work).

In veterinary medicine selenium has long since been known as a cause of poisoning in farm animals ('blind staggers', 'alkali disease'). 6–8 mgm. selenium per pound of body-weight as a single oral dose of sodium selenite is the minimum lethal dose for pigs[4]. Intoxicated animals showed emesis, diarrhœa, apathy, unwillingness to move, shivering and sometimes paresis before death. No thorough examination of the muscles was done.

Sodium selenite was given subcutaneously to pigs weighing 30–70 kgm. 2·0 mgm. and 1·2 mgm. selenium per kgm. body-weight caused death after 4 hr. and five days, respectively, during which time the pigs showed trembling, ataxia and paresis. Plasma glutamic-oxaloacetic transaminase was significantly increased (Fig. 1); but plasma ornithine-carbamyl transferase was normal, indicating that muscle but not the liver was affected[3]. At post-mortem examination the body musculature showed extensive dystrophy; but other organs, including the liver, showed no significant changes.

0·9–1·1 mgm. selenium per kgm. body-weight caused slight to moderate elevation of plasma glutamic-oxaloacetic transaminase in about half the number of pigs so treated, but otherwise no symptoms of disease. The ornithine-carbamyl transferase values were normal. The elevations of glutamic-oxaloacetic transaminase returned to the starting-level after a day or two. Biopsy specimens from the longissimus dorsi showed no changes at microscopical examination.

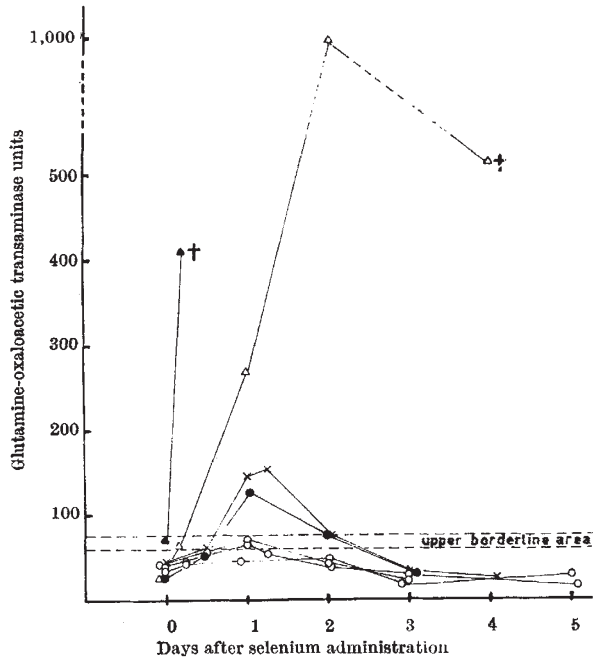

Fig. 1. ×, 0·9; ○, 1·0; ●, 1·1; △, 1·2; ▲, 2·0 mgm./kgm. body-weight

0·8 mgm. selenium per kgm. body-weight or smaller doses gave no increase of transaminase.

It is remarkable that the anti-muscular degeneration effect of low selenium doses corresponds to a high vulnerability of the muscles to increased selenium doses.

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Effects of γ-Aminobutyric Acid and Picrotoxin on Spontaneous Activity in the Central Nervous System of the Crayfish

SINCE the extraction of an inhibitory substance (factor I) from mammalian brain[1], one constituent of which was identified as γ-aminobutyric acid[2], this constituent has been tried on a number of invertebrate preparations[3]. It imitates the action of inhibitory neurones in crayfish and on injection into the animal results in flaccidity[2]. The action of γ-aminobutyric acid is very similar to that obtained on stimulation of the inhibitory fibres in crayfish stretch receptors[4]. The effects of γ-aminobutyric acid are counteracted by picrotoxin in this preparation[2] as well as in crayfish hearts[5]. Picrotoxin