fragments of glass did not produce resorption, and in one specimen the glass became closely embedded in the bone surface without perforating it. The local reaction of the bone to the pressure of a grafted fragment may, of course, be modified if the fragment also exerts a physiological effect by virtue of its chemical character. Grafts performed with carotene, made up in fibrinogen–thrombin clot, showed no resorption or other noteworthy effect. It appears that the fractures observed by Moore and Wang[4] and others in rats given massive doses of vitamin A may be attributable to local action on the bones, and the results are interesting in relation to Mellanby's[5] findings on the effect of vitamin A deficiency on osteoclasis. As might be expected, carotene is evidently not converted to vitamin A by bone tissue, at least to any considerable extent.

Ten grafts (five into male and five into female hosts), using small single chips from a fused œstradiol tablet, showed no evidence of either local resorption or increased local bone deposition. Increased deposition might, perhaps, have been anticipated in view of the known action of injected œstrogens in promoting bone-formation in the medullary cavities of the long bones. Sutro[6] claims that osteosclerosis of the calvarium also occurs in young mice of either sex given prolonged œstrogen treatment. The character and degree of the response to œstrogens apparently varies in different regions of the skeleton: thus the pubic bone undergoes resorption; in the long bones only endosteal bone formation occurs; and the magnitude of the effect depends on dose – time relations. Further graft experiments, taking these factors into account, will be needed before the local action of œstrogens can be satisfactorily assessed.

The detailed results, including histological findings, will be reported elsewhere at a later date.

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## Effect of Pantothenic Acid on the Infection of *Trypanosoma evansi* in Rats

Withholding of pantothenic acid from the diet has been shown by Becker, Mansera and Johnson[1] to interfere with the production of ablastin, the trypanosome-reproduction inhibiting antibody, formed by rats infected with *Trypanosoma lewisi*. An investigation of exploratory nature was undertaken to ascertain if pantothenic acid had a similar effect on the course of *T. evansi* infection in rats.

*Experiment* 1. Six rats, all kept on bread and milk, were used in this experiment. The diet of three of these was supplemented by pantothenic acid at the rate of 200 mgm. a rat daily for five days prior to infection and continued until the end. All the rats were infected on the same day with 3,000 trypanosomes each by the subcutaneous route. On the fifth day after infection, two out of three rats of the control batch showed trypanosomes in their peripheral circulation, while all three of the acid group remained negative. On the sixth day, only two rats of the acid group showed trypanosomes in the tail blood, while the third remained negative; in all the controls the parasites were swarming in the blood, and one died during that night. On the seventh day one rat of the acid group died, while two others had a rise in the number of parasites; controls showed an even greater number and died during the night; the remaining two of the acid group, on the other hand, died twenty-four hours later.

*Experiment* 2. The dietary and other conditions were the same as above; but the dose of the trypanosomes was increased to 46,000 a rat. The rats of both groups became positive on the third day; but the number of parasites in the controls was appreciably larger than those receiving pantothenic acid. One control rat died on the fourth day and the remaining two during the night; in the acid group, on the other hand, all three showed swarming parasites on the fourth day, survived the fifth day and died during the night.

*Experiment* 3. The conditions were slightly varied. A batch of ten rats was kept for fifteen days on a vitamin-free diet consisting of casein (vitamin-free), 20 parts; coco-nut oil, 5 parts; dextrinized starch, 71 parts; salt mixture (Osborn and Mendel), 4 parts. Five of these, in addition, received pantothenic acid as above. On the sixteenth day the rats received a dose of 2,500 trypanosomes each subcutaneously. In the control batch two rats were positive on the fifth day and all on the sixth day; while in the acid group one became positive on the sixth day, and with an addition of one on each day to the positive group, all were positive by the tenth day. Commencing from the night of the tenth day, all the controls died within the next forty-eight hours; whereas in the acid group the first death occurred on the twelfth day and within the next forty hours all were dead.

Though further experimental observations have yet to be made before any generalization can be attempted on the subject, the results presented here are interesting enough to merit publication. The results perhaps justify a tentative suggestion that pantothenic acid exerts an influence on the rate of multiplication of *T. evansi* in rats, although finally the host is overwhelmed by parasitæmia.

Bovines in India are considered to be 'carriers' of *T. evansi*, and the pantothenic acid reserve in these animals may play the important part of maintaining the 'carrier' condition. Sporadic fatal outbreaks of surra in bovines are, however, not uncommon in India, and the cause for this may perhaps be found to be correlated with the interference of pantothenic acid balance in these animals.

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