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A Cephalinase in Nervous Tissue

The phospholipids were regarded by the older investigators as relatively inert, and for this reason they have been classed among the 'permanent' or structural elements of the cell. Experiments with the radioactive isotope, phosphorus-32, have shown, however, that the phospholipids of the brain have a significant phosphorus turnover-rate[1,2], and the turnover is greater for cephalin than lecithin.

When sterile rat brain tissue, or a homogenate containing buffer (*p*H 7·5), is incubated at 37° C. in the presence of 1/2,000 merthiolate to prevent bacterial growth, there is a fall in the phospholipid content. Thus in eight experiments the mean phospholipid content of the whole rat brain fell from 4·92 per cent tissue weight to 4·30 per cent in twenty-four hours at 37° C. Determination of the different phospholipid fractions, by methods similar to those described by Hack[3], showed no significant change in the sphingomyelins: the lecithin content fell only from 1·44 to 1·29 per cent, and the main change was in the cephalin fraction, which showed a significant fall from 2·84 to 2·40 per cent.

The decomposition of cephalin is apparently due to an enzyme which splits off the phosphate-containing residue; this is then further broken down by phosphatase action to liberate inorganic phosphate, which could be shown to be formed. The cephalinase has relatively little or no action on lecithin in brain homogenates; for this reason it apparently differs from the lecithinase previously described by King[4].

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Effects of 'Aminopterin' on the Male Sex Organs of the Guinea Pig

Following the interesting communication by Hertz[1] on the decreased growth response of the oviduct to œstrogens in chicks fed on a purified diet without folic acid intake, many reports have appeared lately showing that, in the animals studied, either folic-acid deficiency[2,3] or treatment with 'Aminopterin'[4–7] were able to interfere with the growth response to œstrogens of the female genital tract.

On the basis of these experiments and because of the fact that 'Aminopterin' has been shown to interfere with the ordinary depressive influence of œstrogens on the rat prostate[8], it has been reasonably assumed that folic acid is a prerequisite for the proper utilization of œstrogens. Whether folic acid might also be a prerequisite for the proper utilization of androgens has also been studied; but the results have been discordant. Breudler[8] reported that 'Aminopterin' did not interfere with androgen stimulation of tissue growth in castrated adult and immature rats. On the other hand, Goldsmith and others[9] reported that, in mice fed a crude folic acid antagonist, a reduced response to androgen was evident in the vesicular and coagulating glands.

In the following experiments the effects of 'Aminopterin' on the sex organs of the intact adult guinea pig are studied. The guinea pig was chosen because this animal has no rutting season. Twenty guinea pigs (median weight 600 gm.) were divided into two groups of ten each. Each animal of the first group received 1 mgm. of 'Aminopterin' (kindly supplied by Dr. J. M. Ruegsegger, of the Lederle Laboratories Division of the American Cyanamid Co., Pearl River, New York) intraperitoneally every day for eight days. The animals of the second group received the same amount of 'Aminopterin' for fifteen days.

All the animals in the first group were killed on the eighth day, and the testicles, epididymis, prostate and vesicular glands were studied comparatively with the same organs belonging to five guinea pigs of the same weight used as controls. Grossly, the epididymis, the testicle and the prostate of the guinea pigs receiving 'Aminopterin' did not show any appreciable damage. The vesicular glands, on the other hand, did show an evident decreased size in comparison with the controls. The histological study of the material revealed no apparent change in the testicle; the tubules of the epididymis of some of the animals were almost devoid of spermatozoa, while the epididymis of the controls was overloaded with spermatozoa. In the vesicular glands of the treated animals, the natural infolding of the mucosa was clearly less accentuated than in the controls. Slight signs of atrophy were present in the prostate.

In the second group, although the 'Aminopterin' was given for a period twice as long, the same findings were noticed as in the first group. Hence it may be assumed that the effects of 'Aminopterin' on the male sex organs are not striking if we consider the high doses of the drug being used. In any event, the most evident changes are on the epididymis, which contains less spermatozoa, and on the vesicular glands, which are clearly reduced in size.

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