ANIMAL PHYSIOLOGY

Comparative Adenylic Acid Deaminase Activity in Choroid Plexus and Ciliary Process Tissue

Analogies between the ciliary process production of aqueous humour and the choroid plexus production of cerebrospinal fluid have been reviewed quite extensively by Davson[1]. The presence of an adenosine triphosphate-activated 5-adenylic acid deaminase in purified preparations of hog and rabbit ciliary process tissue[2,3] led to an interest in a similar system that might be present in choroid plexus tissue. It has, in fact, been shown by other investigators that 5-adenylic acid deaminase activity is present in preparations of whole dog brain[4,5].

Analytical methods employed in this investigation were as reported previously[2]. Incubations were done under the conditions described in Table 1.

Table 1. Deaminase Activity in Whole-Tissue Homogenates
μMoles recovered after 40 min. incubation

Enzyme source	ATP	ADP	AMP	IMP	Adenosine	Total	μMoles NH_3
Dog choroid plexus	4·44	2·20	1·64	0·70	6·03	15·01	0·76
Hog ciliary processes	4·10	4·03	2·94	2·78	—	13·85	3·83

All vessels contained: 0·03 *M tris*-0·03 *M* succinate buffer, *p*H 7·4; 3·8 per cent fresh whole tissue homogenate; 7 μmoles each adenosine triphosphate and adenylic acid; 0·004 *M* magnesium chloride; 0·005 *M* potassium chloride; 0·025 *M* sodium chloride.
ATP, adenosine triphosphate; ADP, adenosine diphosphate; AMP, adenylic acid; IMP, inosinic acid.

Although the results indicate that choroid plexus tissue does have an active deaminase system, it is clear from the nucleotide recovery data that there are differences in the metabolic exchanges in the two types of tissue studied. We cannot as yet preclude the possibility that ammonia accumulation in the choroid plexus system could have resulted from deamination of any of the adenine derivatives recovered. The stoichiometry between the ammonia produced and inosinic acid accumulated is, however, strongly suggestive of the ammonia yield being the result of 5-adenylic acid deamination.

The fact that ammonia production in the ciliary process preparation was 5 times greater than in the choroid plexus preparation makes one question the activating influence of adenosine triphosphate as a deaminase co-factor in the latter tissue. This co-factor role of adenosine triphosphate has been reported for purified ciliary process preparations[6].

It has been shown previously that the deaminase of ciliary process is specific for 5-adenylic acid[2]. A similar specificity is seen in preparations of choroid plexus incubated under the same conditions described in Table 1 except substituting 14 μmoles of 3-adenylic acid as the nucleotide addition in some vessels and 14 μmoles of 5-adenylic acid as the nucleotide addition in other vessels. A yield of 0·2 μmole of ammonia was seen in the former case and 1·2 μmoles of ammonia in the latter—a 6-fold increase in activity with 5-adenylic acid.

Precise functional evaluation of the roles of these deaminase systems as regards actual aqueous humour and cerebrospinal fluid production must await further investigation. A role of adenine nucleotides in regulation of blood vessel patency[7,8] and roles of adenylic acid in many areas of metabolic interactions[9], either of which could invoke the need of an adenylic acid concentration-regulating deaminase, have already been suggested by others.

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Effect of Kinetin on Glucose Metabolism of Chicken Embryo Fibroblasts

Kinetin, a potent plant-growth hormone, was recently discovered and identified as 6-(furfuryl-amino) purine, by Miller and Skoog[1]. Orr and McSwain[2] found that kinetin, at rather low levels (0·015–0·06 μgm./ml.), stimulated growth of human tissue culture.

In the course of a study of the effect of various hormones on cultures of chick embryo fibroblasts[3,4], it was observed that graded amounts of kinetin within the range of 12–24 μgm./ml. produced a roughly linear increase of total glucose consumption and lactic acid production (Fig. 1).

The ratio between glucose and lactic acid was similar in hormone-treated and control cultures. A kinetin concentration which stimulated glucose metabolism caused only slight growth inhibition. Microscopic examination revealed no abnormalities in the morphological appearance of the cells grown in the presence of the hormone.

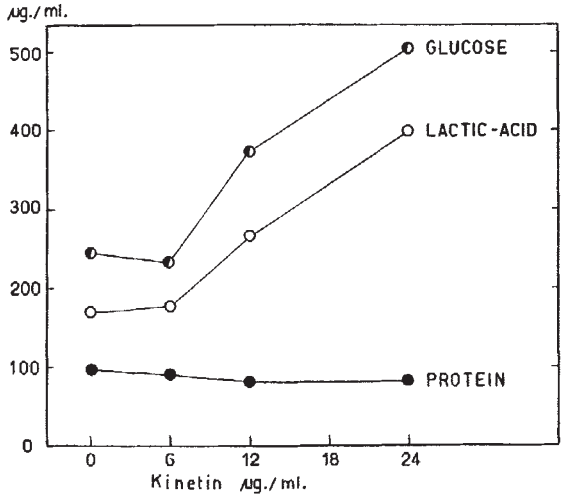

Fig. 1. The effect of kinetin on growth and glucose uptake of chick embryo fibroblasts and their lactic acid production. Procedures for cell cultivation and analytical methods were similar to those previously described (refs. 3 and 4). Cultures were incubated with hormone for 3 days prior to examination