stability of the enzyme. Experiments are in progress to elucidate the factors controlling ornithine decarboxylase turnover in these cells.

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Creatine Kinase Activity in Cultures of Differentiating Myoblasts

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Differentiation of myoblasts *in vitro* results in the fusion of single cells to form multinucleate syncytia and an increase in the activity of creatine kinase (Hauschka, 1968). The analogous increase in creatine kinase activity associated with muscle development *in vivo* is accompanied by a change in isoenzyme pattern (Eppenberger *et al.*, 1964), and preliminary findings suggest a similar change *in vitro* (G. Morris, A. Cooke & R. J. Cole, personal communication).

This abstract is concerned with the appearance of creatine kinase in primary cultures of chick myoblasts and the relationship between changes in the enzyme and cell fusion.

A burst of fusion (measured as the percentage of nuclei in multinucleate syncytia) began after 45h in culture, giving rise to 50% multinucleate syncytia at 69h. In contrast there was relatively little increase in creatine kinase activity/mg of DNA until 69h in culture, but by 96h the creatine kinase activity/ μ g of DNA was 20-fold higher than its initial value at 33h. Creatine kinase activity was assayed by measuring fluorimetrically the creatine produced by the back-reaction of the enzyme (Koedam, 1969).

The isoenzyme compositions of creatine kinase in cell culture were obtained from the ratios of enzyme activity at high and at low concentrations of creatine phosphate and ADP (Eppenberger *et al.*, 1967). Two forms of the enzyme from chick heart and breast muscle gave values of 2.1 and 5.0 respectively. In cell culture a constant ratio of approx. 2.4 was obtained at 33–58h and this then increased to 3.3 at 69h and to 4.0 at 96h. Thus in newly fused myoblasts the 'heart'-type isoenzyme characteristic of embryonic muscle is predominant, and a change in isoenzyme composition occurs coincidental with the

marked increase in creatine kinase activity rather than fusion of the myoblasts.

Cells grown with 0.1mM-dibutylryl cyclic AMP (6-*N*,2'-*O*-dibutylryladenine 3':5'-cyclic monophosphate) fused 10h later than their controls, but the increase in creatine kinase activity was not delayed. This result contrasts with that obtained by Shainberg *et al.* (1971), who found that the inhibition of fusion caused by low Ca^{2+} concentrations also prevented the increase in creatine kinase activity.

It therefore seems that, although cell fusion is necessary for the observed increase in creatine kinase activity and appearance of the new isoenzyme, the two processes are not as tightly linked as previously suggested (Shainberg *et al.*, 1971).

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The Effect of Aflatoxin B₁ on Ribonucleic Acid Synthesis in Control and Cortisol-Stimulated Rat Liver

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The hepatocarcinogenic fungal toxin aflatoxin B₁ is a potent inhibitor of the induction by cortisol of tryptophan pyrrolase activity in rat liver (Clifford & Rees, 1967). The increased Mg^{2+} -stimulated RNA polymerase activity resulting from the administration of cortisol has been found to be strongly inhibited by aflatoxin B₁ *in vivo*, as is the increased incorporation *in vivo* of labelled orotate. The increase in the incorporation of labelled leucine *in vivo*, which results from the administration of cortisol, like the induction of tryptophan pyrrolase, is also strongly inhibited by aflatoxin B₁. The control tryptophan pyrrolase activity and leucine incorporation are not inhibited by aflatoxin B₁, whereas the control rate of RNA synthesis is strongly inhibited. Mg^{2+} -stimulated RNA polymerase activity in hepatic nuclei is decreased by prior adrenalectomy of the rats, but if these rats are also treated with aflatoxin B₁ the enzyme activity observed is no lower than that in intact animals treated with the toxin.

These findings suggest a close connexion between the stimulatory action of cortisol and the inhibitory