ference maintained by us between opposite poles of the nucleus was of the order of 10 mV., and caused the contents to pile up against the membrane in a few seconds. Chromosome migration is slower than this, and the potential difference needed for it should be easily maintainable biologically.

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Participation of Pyridoxal Phosphate in the Enzymic Synthesis of Serine

Evidence that pyridoxal phosphate is involved in the synthesis of serine by micro-organisms has been provided by Lascelles and Woods[1], who found that incorporation of pyridoxal phosphate in the medium stimulated serine synthesis by washed cells of *Streptococcus faecalis* R. and enabled *Leuconostoc mesenteroides* to grow in the presence of glycine and a CO_2-rich atmosphere instead of serine. Participation of pyridoxal phosphate in serine synthesis in chickens was suggested by the observation that liver extracts from pyridoxine-deficient chickens showed a reduced ability to incorporate [^{14}C]formate into the β-position of serine[2]; but hitherto attempts to demonstrate a direct activation by pyridoxal phosphate of serine synthesis by liver extracts have been unsuccessful[3].

The role of pyridoxal phosphate in serine synthesis has been further studied with an enzyme preparation which was extracted from acetone-dried rabbit liver powder and partially purified by heat treatment and ammonium sulphate fractionation at *p*H 7·5. The enzyme was incubated with glycine, formaldehyde, tetrahydropteroyl glutamic acid (tetrahydroPGA) and bicarbonate buffer, *p*H 7·2, at 37° for 1 hr. Serine was determined by manometric estimation of the carbon dioxide produced by periodate oxidation. A control in which tetrahydroPGA was omitted provided an adequate correction for small amounts of carbon dioxide produced by periodate oxidation of material other than serine.

Addition of pyridoxal phosphate to the otherwise complete system appreciably increased the rate of serine synthesis, increments of more than 100 per cent being sometimes obtained in the presence of 5×10^{-4} *M* pyridoxal phosphate. Although added pyridoxal phosphate consistently increased serine synthesis by these enzyme preparations, they nevertheless exhibited considerable activity in the absence of added pyridoxal phosphate. Prolonged dialysis, even after subjecting the enzyme to high salt concentrations at temperatures up to 60°, did not significantly reduce the activity in absence of added pyridoxal phosphate, provided the *p*H was kept within the limits 5·0–7·5. After the enzyme had been incubated for 30 min. at *p*H 8·6 and 37°, however, it was almost inactive for serine synthesis. An enzyme preparation so treated could be largely reactivated for serine synthesis by incubation with pyridoxal phosphate at *p*H 7·2 and at 37° prior to addition of substrates. The degree of reactivation increased as the period of incubation of the enzyme with pyridoxal phosphate was increased up to 2 hr. Under these conditions serine synthesis with the pyridoxal phosphate-reactivated enzyme was up to one hundred times greater than that with the inactivated enzyme.

Similar though less striking effects of pyridoxal phosphate on serine breakdown were also observed, both with normal and inactivated enzyme preparations. The reaction was in this case followed by measuring formaldehyde produced by the enzyme from DL-serine[4].

A more detailed report of this work will be published elsewhere.

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The Precursor of 2-Carboxy-Pyrrole in Mucoproteins

We have previously reported[1] the isolation of a pyrrole derivative from the alkaline hydrolysate of bovine submaxillary gland mucoprotein and its identification as 2-carboxy-pyrrole (III). On reduction it yielded 2-carboxy-pyrrolidine (proline), chromatographically it showed the same behaviour as synthetic III, and it gave in the cold a purple colour with Ehrlich's reagent. Recently, the ultra-violet absorption spectra of both the substance prepared from mucoprotein and synthetic III were found to be identical.

The presence of about 4 per cent of the Ehrlich-reacting substance in the mucoprotein together with the high molecular extinction coefficient of III ($\varepsilon_{max.}$ 256 mμ = 11,118 in phosphate buffer *p*H 7·0, ionic strength 0·05) prompted us to carry out a spectroscopical analysis of the untreated mucoprotein. Contrary to expectation, no peak at 256 mμ was exhibited, though admixture of III corresponding to 2·6 per cent of the dry weight of the mucoprotein present gave rise to an easily detectable peak at 256 mμ.

Like many other mucoproteins, the bovine submaxillary gland mucoprotein inhibits influenza virus hæmagglutination. Interaction between inhibitory mucoproteins and purified influenza virus (or the receptor-destroying enzyme of *Vibrio cholerae*) was shown[2] to result in the liberation of a dialysable compound consisting of a non-amino sugar and a nitrogenous component. The compound decomposed on treatment with dilute mineral acid. It coupled with *p*-dimethylaminobenzaldehyde to give a purple colour even after prolonged treatment with alkali,