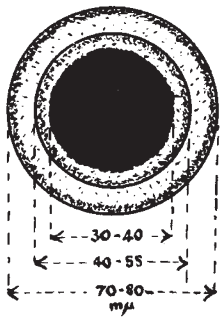

Fig. 2

The occurrence of these bodies in various amounts in other chicken neoplasms such as the Murray-Begg tumour[6], the erythroblastosis[7] and their presence even in normal young chickens[7] is an important fact, but for the moment its interpretation remains hypothetical: there may exist either an inactive ubiquitous agent in all chicken tissues which can become differentiated and oncogenic in an appropriate environment; or, the particles in normal controls could represent the widespread lymphomatosis virus, although the strains used for this study were clinically free of this disease.

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Intestinal Lecithinase

Schmidt, Bessman and Thannhauser[1] described a phospholipase in mitochondria of rat intestinal mucosa which is highly active towards cephalin but relatively inactive towards lecithin. A possible explanation of this lack of activity towards lecithin was found in our studies of a similar enzyme preparation from rat intestine. It was found that the breakdown of lecithin to glycerophosphorylcholine depends on the concentration of enzyme used (see Fig. 1, (I)–(III)). With low enzyme concentrations a considerable lag period was observed followed by an autocatalytic rate of reaction. The lag period could be abolished by the addition of degradation products of lecithin, fatty acids (palmitic, oleic, stearic) and lysolecithin.

It seems likely that the presence of fatty acids is obligatory for lecithinolysis. Small amounts of fatty acids (6 μgm./ml.) were found to be present in the enzyme preparations. Lysolecithin also furnishes fatty acids due to the active lysolecithinase present. Similarly, the activation by cephalin reported by

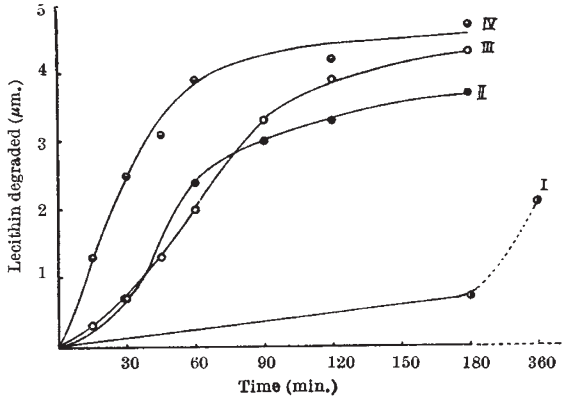

Fig. 1. (I) With 0·3 ml. enzyme; (II) with 0·3 ml. enzyme plus 4 μgm. palmitic acid; (III) with 0·6 ml. enzyme; (IV) with 0·6 ml. enzyme plus 4 μgm. palmitic acid.

Rat intestinal mucosa was ground with equal weight of sand, extracted with water (2 ml./gm. mucosa) and centrifuged at 800*g* at 4° C. The supernatant was brought to *p*H 5·5 with 0·1 *N* acetic acid and the precipitate was resuspended in 0·02 *M* phosphate buffer *p*H 6 (2 ml./gm. of original tissue weight). 5 μgm. egg lecithin (ref. 3)+40 μgm. phosphate buffer *p*H 6 with the indicated enzyme amounts made up to 1 ml. was incubated at 37° C. The rate of the reaction was determined by following the decrease in ester content according to Stern and Shapiro (ref. 4)

Schmidt *et al.*[1] may be due to the fatty acids liberated from this substrate.

The intestinal lecithinase described differs from generally known lecithinases[2], being bound to particles and inhibited by Ca^{++} at 20 μgm./ml. and Cd^{++} 0·5 μgm./ml. as well as by cholic acid. Lecithinase activity could not be separated from lysolecithinase by any purification procedure so far tried.

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Sensitivity of Pancreas Hexokinase towards Alloxan and its Modification by Glucose

THE administration of glucose, mannose or fructose, but not galactose or several other sugars tested, can prevent the effect of a normally diabetogenic dose of alloxan. This protection was found by Bhattacharya[1,2] in the rat. From the relative efficiencies of these sugars he concluded that the diabetogenic action of alloxan could depend on an inactivation of the hexokinase of the beta cells of the pancreas. Arteta and co-workers have confirmed the protective effect of glucose in the dog[3]. They have also observed that insulin[3] and floridzin[4] can increase, and adrenalin[5] and glucagon[6] can decrease, the sensitivity towards alloxan, apparently in relation to their effect on the blood sugar-level. The work described here on the effect of alloxan on pancreas hexokinase *in vitro* does not support the hexokinase inhibition theory of alloxan action.