$\pm$ S.E.M., with numbers of samples in parentheses) of eicosa-5,8,11,14-tetraenoic ($C_{20:4\omega6}$) acid from 5.65 ± 0.29 (6) in control chicks to 6.71 ± 0.27 (6), in docosa-7,10,13,16-tetraenoic ($C_{22:4\omega6}$) acid from 1.42 ± 0.07 to 1.75 ± 0.10 and in docosa-10,13,16,19-tetraenoic ($C_{22:4\omega3}$) acid from 0.75 ± 0.11 to 1.33 ± 0.14 ; that of docosa-7,10,13,16,19-pentaenoic ($C_{22:5\omega3}$) acid fell from 4.11 ± 0.12 to 3.62 ± 0.17 . Total phospholipid from cerebellum showed rises in $C_{20:4\omega6}$ acid from 3.47 ± 0.18 (5) to 5.01 ± 0.28 (6), in $C_{22:4\omega6}$ acid from 1.11 ± 0.14 to 1.77 ± 0.22 and in $C_{20:1\omega9}$ acid from 1.16 ± 0.03 to 1.54 ± 0.13 ; hexadecanoic ($C_{16:0}$) acid fell from 20.03 ± 0.84 to 17.73 ± 0.41 .

Deficiency of vitamin E did not produce any changes in the relative proportions of the major phosphatides, which could have contributed to the observed phospholipid fatty acid changes.

Lung, heart and striated muscle phospholipid fatty acids of vitamin E-deficient chicks showed significant decreases in incorporation of [^{14}C]-acetate into fatty acids. Decreases in incorporation were also found in brain tissue, but these were not statistically significant. When the radioactivity in each fatty acid group was expressed as a percentage of the total radioactivity, lung and heart phospholipid showed a significantly increased incorporation into the polyunsaturated fatty acids and a decrease in incorporation into saturated and monoenoic fatty acids compared with control birds. Brain and striated muscle showed the same changes, but the differences were not significant.

It would appear that there is a homeostatic response in the vitamin E-deficient chick whereby the rates of synthesis of polyunsaturated fatty acids rise relative to those of saturated and monoenoic acids despite an overall decrease in fatty acid synthesis.

We are grateful to the Science Research Council for a Research Studentship for P. M. P.

Bieri, J. G. & Andrews, E. L. (1964). *Biochem. biophys. Res. Commun.* **17**, 115.

Lee, D. J. W. & Barnes, M. McC. (1969). *Brit. J. Nutr.* **23**, 289.

Witting, L. A. (1967). *Lipids*, **2**, 109.

Witting, L. A. & Horwitt, M. K. (1967). *Lipids*, **2**, 89.

Antiketogenic Action of Glucocorticoids in the Cow

By G. D. BAIRD and R. J. HEITZMAN. (*Agricultural Research Council Institute for Research on Animal Diseases, Compton, Newbury, Berks.*)

At 48 hr. after administration of 10 mg. of Voren (dexamethasone 21-pyridine-4-carboxylate; kindly

given by Abbott Laboratories Ltd., Agro-Vet Division, Queenborough, Kent) to healthy lactating cows, the steady-state concentrations of the citrate-cycle intermediates citrate, α -oxoglutarate and malate had increased in the liver by about 200% above control values. At the same time the hepatic activities of fructose 1,6-diphosphatase and glucose 6-phosphatase were essentially unchanged, whereas that of phosphopyruvate carboxylase was slightly depressed.

Further analysis revealed that the hepatic concentrations of intermediates of the Embden-Meyerhof pathway were either unchanged or were decreased below control values. The increase in citrate-cycle intermediates seemed therefore to be selective, with a 'crossover point' between malate and phosphoenolpyruvate, since the latter compound was present at only 60% of the control value.

That glucocorticoid treatment does in fact increase the rate of gluconeogenesis in bovine liver under these circumstances is suggested by the marked hyperglycaemia and by the accumulation of glycogen, the concentration of which had doubled over the 48 hr. period. However, these changes may in part have been occasioned by the concomitant depression of milk yield, which is a feature of the action of glucocorticoids in the cow.

There may possibly be an inverse relationship between the ability of glucocorticoids to increase citrate-cycle components and their ability to increase the enzymic capacity for gluconeogenesis. Thus, in the cow, if glucocorticoids increase the gluconeogenic flux into the citrate cycle, the capacity of the reverse Embden-Meyerhof pathway may be exceeded and this in turn may lead to an accumulation of material in the citrate cycle. A correlation between the concentration of citrate-cycle constituents and gluconeogenic enzyme capacity is also suggested by the situation in the untreated non-lactating cow. Here the concentrations of citrate and α -oxoglutarate were found to be higher than in the untreated lactating cow, whereas the activity of phosphopyruvate carboxylase was significantly lower.

Glucocorticoid treatment is an effective therapy for bovine lactational ketosis. The findings suggest that one aspect of the antiketogenic activity of glucocorticoids in the cow may be the ability to accumulate those citrate-cycle intermediates that are known to be depleted in ketosis (Baird *et al.* 1968; Ballard, Hanson, Kronfeld & Raggi, 1968).

Baird, G. D., Hibbitt, K. G., Hunter, G. D., Lund, P., Stubbs, M. & Krebs, H. A. (1968). *Biochem. J.* **107**, 683.
Ballard, F. J., Hanson, R. W., Kronfeld, D. S. & Raggi, F. (1968). *J. Nutr.* **95**, 160.