optimum acetyl-CoA and malonyl-CoA concentrations both enzymes synthesized predominantly free palmitic acid as well as small proportions of C_{6:0}-C_{14:0} fatty acids. A significant proportion (10-15%) of butyric acid was also formed. Lowering the concentration of malonyl-CoA relative to acetyl-CoA decreased the rate of fatty acid synthesis and the proportion of palmitic acid formed. The proportion of medium-chain fatty acids synthesized increased slightly, whereas that of butyrate increased to about 50%. Hence the chain length of the fatty acids formed by these fatty acid synthetases can be controlled by the relative concentrations of acetyl-CoA and malonyl-CoA available. The enzyme from rabbit mammary gland does not possess a specific mechanism for the synthesis of medium-chain fatty acids. Alternative mechanisms of control of chain length, such as the interaction of fatty acid synthetase with other enzymes of lipogenesis, will be discussed.

- Burton, D. N., Haavik, A. E. & Porter, J. W. (1968). *Arch. Biochem. Biophys.* **128**, 141.
 Garton, G. A. (1963). *J. Lipid Res.* **4**, 237.
 Glass, R. L., Troolin, H. A. & Jenness, R. (1967). *Comp. Biochem. Physiol.* **22**, 415.
 Hsu, R. Y., Wasson, E. & Porter, J. W. (1965). *J. biol. Chem.* **240**, 3736.
 Smith, S. & Dils, R. (1964). *Biochim. biophys. Acta*, **84**, 776.
 Smith, S. & Dils, R. (1968). *Biochim. biophys. Acta*, **116**, 23.
 Smith, S., Watts, R. & Dils, R. (1968). *J. Lipid Res.* **9**, 52.
 Wakil, S. J. (1961). *J. Lipid Res.* **2**, 1.

The Polyenoic Acids and their Elongation Products in the Muscle Tissue of *Phacochoerus aethiopicus*: a Re-evaluation of 'Animal Fat'

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We have reported that the muscle tissue lipids from bovids free to select their own food were rich in polyenoic acids (30% of the total) when compared with those of domestic bovids, which may contain only 1-8% of the total (Crawford, 1968; Crawford, Gale & Woodford, 1969). The bovids are ruminants and we have now studied the muscle tissue from a non-ruminant, the wart-hog (*Phacochoerus aethiopicus*); we find an even higher proportion of polyenoic acids. Linoleate and linolenate alone account for 30-40% of the total, and the higher elongation products (eicosatetraenoate, eicosapentaenoate, docosapentaenoate and docosaheptaenoate) constitute another 10-15%. The C₁₈ saturated and monounsaturated acids, which characterize the fat

of domestic animals, are present in the proportion of only approx. 7-9% each.

Previously it has been considered that animal fats are predominantly of a saturated and monounsaturated nature (Hilditch & Williams, 1964; Hubbard & Pocklington, 1968; Crawford, 1968). The facts from free-living herbivores, both ruminants and non-ruminants, show that, on the contrary, animal 'tissue fats' contain appreciable amounts of polyunsaturated acids.

The higher polyenoic metabolic products such as eicosatetraenoate, eicosapentaenoate, docosapentaenoate and docosaheptaenoate are also present in tissues of domestic animals, but have been largely overlooked as the massive infiltration of saturated and monounsaturated fat makes them difficult to detect.

The higher elongation products are used for construction of phospholipids, prostaglandins and cerebral gangliosides. Their consistent presence in animal products along with the parent C₁₈ polyenoic acids indicates that a re-evaluation of the 'saturated' nature of 'animal fat' is needed. We have now examined 14 different species and the saturated and monounsaturated predominate only in animals living under domestic conditions.

- Crawford, M. A. (1968). *Lancet*, **i**, 1329.
 Crawford, M. A., Gale, M. M. & Woodford, M. H. (1969). *Biochem. J.* (in the Press).
 Hilditch, T. P. & Williams, P. N. (1964). *The Chemical Constitution of Natural Fats*, 4th ed. London: Chapman and Hall.
 Hubbard, A. W. & Pocklington, W. D. (1968). *J. Sci. Fd Agric.* **19**, 57.

Brain and Cerebellum Fatty Acid Pattern Changes in the Encephalomalacic Chick and the Presence of a Homeostatic Mechanism

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Several workers have reported changes in the fatty acid composition of various tissues in vitamin E deficiency (e.g. Bieri & Andrews, 1964; Witting & Horwitt, 1967; Lee & Barnes, 1969). Witting (1967), working with rat muscle tissue, postulated a homeostatic mechanism whereby the synthesis of certain polyunsaturated fatty acids is stimulated to make up for deficiencies in others. The tissues of the encephalomalacic chick were studied to discover if this homeostatic effect was observable in another tissue in which gross changes occur in vitamin E deficiency.

The total phospholipid from whole brain of encephalomalacic chicks showed significant rises in the relative percentages (expressed as means

$\pm$ 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.

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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

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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.