The patterns of the total fatty acids, i.e. the sum of the esterified and the free fatty acids, were also analysed by means of gas chromatography of the methyl esters after extraction and alkaline hydrolysis of the plasma lipids. When the linolenic acid percentage of the feed fat is increased from 24.1 to 51.1, the linolenic acid content in the total plasma fatty acid sample rises from 6.0 to 12.1%, whereas the free fatty acid fraction does not change significantly. Only massive amounts of unsaturated fat added to the diet could alter the free fatty acid pattern (Goosen & Mulder, 1970). This observation, and the fact that the diet had not yet been changed at parturition, rules out the possibility that the diet could have caused the change in free fatty acid composition. Probably the stimulated lipolysis is responsible for this change.

Bovine depot fat is very variable in fatty acid composition. The oleic acid/stearic acid ratio varies from 18.1 for ventral subcutaneous fat to 0.76 for pericardial fat (Mulder & Metz, 1970). It is possible, therefore, that the cow preferentially hydrolyses subcutaneous fats during periods with increased metabolic needs for fatty acids. A second possibility is that a preferential hydrolysis occurs of fat molecules which contain oleic acid. These possibilities are under investigation.

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The Effect of Adrenaline and of α - and β -Adrenergic Blocking Agents on ^{32}P Incorporation into Fat-Cell Phospholipids

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The effect of adrenaline on the incorporation of ^{32}P into the phospholipids of fat-cells has been studied. Fat-cells, prepared from rat epididymal fat-pad by the method of Rodbell (1964) were incubated in glucose-free Krebs bicarbonate buffer (1.3 mM- Ca^{2+}) containing $\text{KH}_2^{32}\text{PO}_4$ (10 $\mu\text{Ci/ml}$) P . Incubation with adrenaline (1 $\mu\text{g/ml}$) caused a significant increase in incorporation of ^{32}P into phospholipid. When adrenaline (1 $\mu\text{g/ml}$) was incubated together with the α -adrenergic blocking agent phentolamine (10 $\mu\text{g/ml}$) lipolysis was not impaired, and the increased uptake of ^{32}P into phospholipid promoted by adrenaline was similarly not impaired. When fat-cells were incubated with adrenaline (1 $\mu\text{g/ml}$)

together with the β -adrenergic blocking agent propranolol (10 $\mu\text{g/ml}$) the lipolytic response to adrenaline was abolished, but the increased incorporation of ^{32}P into phospholipid was not diminished. The work of Butcher, Ho, Meng & Sutherland (1965) and of Butcher & Sutherland (1967) has shown that β -adrenergic blocking agents, when incubated together with adrenaline, abolish the rise in 3':5'-cyclic AMP concentrations in rat fat-pad that would be elicited by incubation with adrenaline alone. Our results, in conjunction with this observation, indicate that the stimulation of uptake of ^{32}P into fat-cell phospholipids produced by adrenaline can be dissociated from the response of the adenylate cyclase system to adrenaline, and therefore does not appear to be effected through the β -receptors. The role of the α -receptors in this effect is not clear, since the α -adrenergic blocker phentolamine itself stimulated ^{32}P incorporation into the fat-cell phospholipids in the absence of adrenaline.

This work was supported by the Tobacco Research Council.

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The Distribution of C_{17} Fatty Acids in the Liver of a Child with Propionicacidemia

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The accumulation of $\text{C}_{17:0}$ and $\text{C}_{17:1}$ fatty acids in the tissues of children suffering from a defective propionyl-CoA carboxylase activity is a characteristic feature of this condition (Gompertz, Storrs, Bau, Peters & Hughes, 1970). The presence of approximately equal quantities of the $\text{C}_{17:0}$ and $\text{C}_{17:1}$ acids in the total liver lipids suggests that investigation of the distribution of these two acids between the various lipid classes and also between the acylation positions on individual lipids would help to demonstrate the effects of a single double bond on esterification patterns of fatty acids. Studies *in vitro* of the effect of chain length on desaturation and esterification have included odd-numbered fatty acids (Howling, Morris & James, 1968; Sanchez de Jimenez & Cleland, 1969; Abou-Issa & Cleland, 1969; Paulsrud, Stewart, Graff & Holman, 1970); however, the esterification of odd-numbered acids *in vivo* has not been investigated previously. In this study we have investigated the distribution of $\text{C}_{17:0}$ and $\text{C}_{17:1}$