30% in perinephric adipose tissue (Duncan & Garton, 1967). Although the high proportion of stearate in perinephric triglycerides is probably associated with the incorporation of exogenous stearate derived from hydrogenation of dietary unsaturated C₁₈ acids in the rumen (Duncan & Garton, 1967), some of the acid is evidently of endogenous origin, as evidenced by the observation that the perinephric triglycerides of sheep reared on a lipid-free diet contained 24% stearate compared with 11% in their subcutaneous triglycerides (Duncan *et al.*, 1971), suggesting that desaturase activity may be greater in subcutaneous tissue.

Accordingly, the desaturation of [1-¹⁴C]stearate was studied with microsomal preparations from both subcutaneous and perinephric adipose tissue of five individual sheep; the method used was that of Bickerstaffe & Annison (1969), except that stearate was added in solution in propylene glycol (Gellhorn & Benjamin, 1964). Desaturase specific activities, expressed as nmol of stearate converted into oleate/30min per mg of microsomal protein, were 9.9 and 15.0 (mean values) respectively for perinephric and subcutaneous tissue; the difference was statistically significant ($P < 0.05$).

Corresponding mean values for liver microsomal preparations from sheep (10), rats (4) and hens (3) were 1.5, 13.0 and 26.5 respectively, showing that marked differences exist between species (cf. Cook & Reiser, 1968; Thompson & Allen, 1969). Whereas lactating mammary tissue of the rat (Lossoy & Chaikoff, 1958) and the rabbit (Bu'lock & Smith, 1965) shows no capacity to desaturate stearate, that of the cow (Laurysens *et al.*, 1961), goat and pig (Bickerstaffe & Annison, 1970) has considerable desaturase activity. Sheep mammary tissue obtained from animals in full lactation was found to be as active as subcutaneous adipose tissue, although, as the gland regressed, desaturase specific activities fell to less than 1.0.

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Uptake of Vitamin E by Rat Small-Intestinal Slices

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Previous reports have suggested that vitamin E (α -tocopherol) is absorbed more efficiently by the intestine from a micellar dispersion than from an emulsion (Thompson, 1969; MacMahon & Thompson, 1970). Bile salts are known to be necessary for absorption (Gallo-Torres, 1970). They are required for the formation of mixed micelles with the products of fat digestion, namely β -monoglycerides and free fatty acids (Hofmann & Borgström, 1962). These results implicate the need for a micellar dispersion of α -tocopherol before its absorption from the intestinal lumen. We have examined the effect of systematically varying the composition of the incubation medium on uptake of tritiated α -tocopherol by slices of rat small intestine, to obtain more information about molecular events relating to absorption.

About 82% of the radioactivity recovered from tissue total lipids was associated with the mucosal layer, and was shown by t.l.c. to be mostly unchanged α -tocopherol. Uptake of α -tocopherol was just as effective from dispersion in 10mM-sodium taurodeoxycholate alone, and when 5mM-mono-olein was present, or even when 5mM-oleate was added to the system. Uptake was also as effective from 10mM- and 30mM-sodium taurodeoxycholate. Changing the pH of the medium from 5.3 to 7.2 did not alter uptake from dispersions in 10mM-sodium taurodeoxycholate and 5mM-mono-olein. When an oil phase, 5mM-glycerol trioleate, was present in equilibrium with a micellar phase, uptake was decreased from 8.96 to 3.90nmol/g of tissue. When α -tocopherol was dispersed with triolein in Krebs-Ringer phosphate buffer, pH 6.3, i.e. in the complete absence of a micellar phase, uptake was decreased considerably from 6.48 to 2.17nmol/g of tissue. When the concentration of α -tocopherol in the incubation medium, consisting of 10mM-sodium taurodeoxycholate with or without 5mM-mono-olein, was increased beyond the micellar solubilizing capacity of the systems (i.e. up to 200 μ g/ml), no change in slope of a graph of uptake against α -tocopherol concentration was observed.

It appears that a micellar phase is probably the preferred form from which uptake of vitamin E occurs, although alteration of the composition of the micelles did not affect the uptake. There is some evidence to suggest that the emulsifying properties of bile salts are also important.

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The Intestinal Absorption of 3-*sn*-Phosphatidylethanolamine in the Rat

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1,2-Di[1-¹⁴C]palmitoyl-*sn*-glycero-3-[³²P]phosphoryl[2-³H]ethanolamine (Owen *et al.*, 1971) was fed in olive oil by gastric intubation to albino rats with thoracic-duct fistulae.

Analysis of the intestinal contents 1 or 2 h after feeding showed that at least 15% of the fed compound had been degraded to monoacyl-*sn*-glycero-3-phosphorylethanolamine and palmitate. Up to 90% of the ³²P and ³H radioactivities in the clear aqueous supernatant, after centrifugation of the intestinal contents, was recovered as monoacyl-*sn*-glycero-3-phosphorylethanolamine.

Up to 4.5% of the administered ¹⁴C, 6% of the administered ³²P and 6% of the administered ³H were recovered in the lymph phospholipids. Of the administered ¹⁴C 52% was recovered in the lymph diacylglycerols and triacylglycerols. ³²P and ¹⁴C were recovered in the lymph ethanolamine phosphoglyceride (3-*sn*-phosphatidylethanolamine and monoacyl-*sn*-glycero-3-phosphorylethanolamine), choline phosphoglyceride, phosphoinositide and sphingomyelin fractions. ³H was recovered in the lymph ethanolamine phosphoglyceride fractions only.

The ¹⁴C, ³²P and ³H relative specific radioactivities of the lymph 3-*sn*-phosphatidylethanolamine fraction rose to maxima of 25, 50 and 50 respectively when the specific radioactivities of the administered trilabelled 3-*sn*-phosphatidylethanolamine were 100 d.p.m./μg of lipid P.

The isotopic proportions ¹⁴C:³²P:³H of the lymph 3-*sn*-phosphatidylethanolamine fraction remained constant at 1.0:1.0:1.0 throughout lymph collection when the administered trilabelled compound had isotopic proportions 2.0:1.0:1.0. The ¹⁴C:³²P ratio of the major lymph phospholipid fraction, 3-*sn*-phosphatidylcholine, rose to a maximum of 7.0:1.0.

These results suggest that the administered 3-*sn*-phosphatidylethanolamine was absorbed from the intestinal lumen as monoacyl-*sn*-glycero-3-phosphorylethanolamine and in part subsequently reacylated (Stein & Stein, 1966) within the mucosa. A small fraction of both the ¹⁴C- and ³²P-labelled metabolites of the administered compound were incorporated into other phospholipid species.

When 1,2-di[1-¹⁴C]palmitoyl-*sn*-glycero-3-[³²P]-phosphorylcholine was fed to rats under similar con-

ditions to those described above, ¹⁴C and ³²P radioactivities were recovered in lymph choline phosphoglycerides only; no detectable label was found in other lymph phospholipid species. This latter result is in agreement with previous work (Scow *et al.*, 1967; Nilsson, 1968).

The generous financial support of the Medical Research Council and the Wellcome Trust are acknowledged.

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The Effect of Polyoxin D on Morphogenesis in *Coprinus cinereus*

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Polyoxin D, a nucleoside antibiotic (Isono *et al.*, 1967), is a powerful competitive inhibitor of chitin synthetase (UDP-*N*-acetylglucosamine–chitin *N*-acetylglucosaminyltransferase, EC 2.4.1.16) in *Neurospora crassa* (Endo *et al.*, 1970). It has little effect on respiration or on synthesis of DNA, RNA and protein in test fungi (Sasaki *et al.*, 1968).

Polyoxin D inhibited the chitin synthetase activity in the microsomal preparation from homogenized stalks of fruit-bodies of the toadstool *Coprinus cinereus* (Schaeff. ex Fr.) S. F. Gray (= *Coprinus lagopus sensu* Buller). In an enzyme assay with 400 μM-substrate (UDP-*N*-acetylglucosamine), 5 μM-polyoxin D gave 50% inhibition.

Young excised stalks of the toadstools, with or without their caps, elongated *in vitro* when kept in a moist atmosphere overnight. The extent of the elongation was predictable and depended on the original size, with the larger stalks elongating as much as they would have done *in vivo*, and the smaller ones progressively less. When the cap was left on, normal morphogenesis occurred, with cap expansion, autolysis and release of viable spores. The active nature of the elongation and subsequent development was confirmed, as they could be prevented by dipping the basal cut ends of the stalks into solutions of metabolic inhibitors in water (e.g. NaCN, NaN₃, cycloheximide, 2,4-dinitrophenol, all at 50 μg/ml). This system has been used *in vitro* to study the effect of polyoxin D on morphogenesis.

The elongation and development of the excised fruit-bodies was totally prevented by 190 μM-polyoxin D and considerably diminished by 19 μM-polyoxin D. This inhibition suggests an intimate involvement of chitin synthetase in these processes.