The Glycerol Phosphate, Dihydroxyacetone Phosphate and Monoacylglycerol Pathways of Glycerolipid Synthesis in Rat Adipose-Tissue Homogenates

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(Received 26 July 1976)

1. Fat-free homogenates from the epididymal fat-pads of rats were used to measure the rate of palmitate esterification with different substrates. The effectiveness of the acyl acceptors decreased in the order glycerol phosphate, dihydroxyacetone phosphate, 2-octadecenylglycerol and 2-hexadecylglycerol. 2. Glycerol phosphate and dihydroxyacetone phosphate inhibited their rates of esterification in a mutually competitive manner. 3. The esterification of glycerol phosphate was also inhibited in a partially competitive manner by 2-octadecenylglycerol and to a lesser extent by 2-hexadecylglycerol. However, glycerol phosphate did not inhibit the esterification of 2-octadecenylglycerol. 4. The esterification of dihydroxyacetone phosphate and 2-hexadecylglycerol was more sensitive to inhibition by clofenapate than was that of glycerol phosphate. Norfenfluramine was more effective in inhibiting the esterification of 2-hexadecylglycerol than that of glycerol phosphate or dihydroxyacetone phosphate. 5. It is concluded that rat adipose tissue can synthesize glycerolipids by three independent routes.

It has been suggested that triacylglycerol synthesis in adipose tissue can proceed via the esterification of three different precursors. These are respectively sn-glycerol 3-phosphate (Rose & Shapiro, 1960; Daniel & Rubenstein, 1968; Jamdar & Fallon, 1973), dihydroxyacetone phosphate (LaBelle & Hajra, 1972a,b) and monoacylglycerol (Schultz & Johnston, 1971). Little is known about the control of these pathways or their interrelationships. In fact, the relative activities of these three routes of metabolism measured simultaneously in the same tissue have not yet been reported. Neither is it known whether the incorporation of the three precursors into lipids is catalysed by the same or different enzymes.

There is considerable interest in the metabolism of adipose tissue both from the point of view of studying the normal processes of triacylglycerol storage and of investigating some of the events leading to obesity. The present study was designed to determine the relative activities of the three routes of glycerolipid synthesis, to characterise them, and to study their possible interrelationships. The experimental tissue chosen was the epididymal fat-pad of the rat.

Materials and Methods

Animals

Male Wistar rats (250–320g) were obtained from the University of Nottingham Joint Animal Breeding Unit, Sutton Bonington, Leics., U.K. They were allowed free access to the 41B diet (Brindley et al., 1976) and water before the experiments.

Materials

Unless stated to the contrary, materials were prepared or purchased as described previously (Sánchez et al., 1973; Mangiapane et al., 1973; Brindley & Bowley, 1975). Hepes [2-[(N-2-hydroxyethyl)piperazin-N'-y]ethanesulphonic acid] was obtained from Hopkin and Williams Ltd., Chadwell Heath, Essex, U.K., and octadec-cis-9-enol and dihydroxyacetone phosphate (dimethylketal, cyclohexylamine salt) were from Sigma (London) Chemical Co., Kingston-upon-Thames, Surrey, U.K. On the day before use, dihydroxyacetone phosphate was generated according to the manufacturer's instructions. The liberated methanol and the water from washings were removed by freeze-drying overnight. Immediately before use the solution was adjusted to pH 7–7.4 with solid KHCO3.

Preparation of alkylglycerols

2-O-Hexadecylglycerol was prepared by Mrs. V. J. Short (Short et al., 1974). A sample (1g) of this was labelled uniformly with 3H gas by The Radiochemical Centre, Amersham, Bucks., U.K., by using the TR2
(Wilzbach) method (Radiochemicals catalogue of The Radiochemical Centre). Excess of $^{3}$H and the catalyst were removed by The Radiochemical Centre and this product was then recrystallized twice from light petroleum (b.p. 40–60°C). The crystals were dissolved and stored in ethanol. Samples of this solution were purified by t.l.c. on plates (0.6 mm thick) of silica gel G by using two developments with chloroform/methanol (49:1, v/v). The product had a radiochemical purity of greater than 98% as determined by chromatography on t.l.c. plates of silica gel G in either chloroform/acetone (22:3, v/v) or chloroform/acetone/methanol (71:25:4, by vol.) as developing solvent. The $[U-^{3}H]2$-hexadecylglycerol (42 μCi/μmol) was diluted with non-radioactive 2-hexadecylglycerol to a specific radioactivity of 5 μCi/μmol as required. 2-$O$-Octadec-9-enyl$[1,3-^{3}H]$glycerol was prepared by the method of Bauman & Mangold (1964) by using $[1,3-^{3}H]$glycerol (1 μCi/μmol) to synthesize the intermediate benzyldiene-$[1,3-^{3}H]$glycerol. The 2-$O$-octadec-9-enyl$[1,3-^{3}H]$glycerol was finally purified by t.l.c. on plates (0.5 mm thick) of silica gel N (Macherey-Nagel, Düren, Germany) by using chloroform/methanol (49:1, v/v) for development (Thomas et al., 1965). The proton and $^{13}$C-n.m.r. spectra of the product were consistent with its being 2-$O$-octadecenyl$[3]^{3}H$glycerol and its radiochemical purity was greater than 99.6%.

**Preparation of fat-free homogenates**

These were prepared essentially by the method of Jamdar & Fallon (1973), except that the epididymal fat-pads of single rats were homogenized in 0.25M-sucrose containing 1 mM-Hepes buffer, adjusted to pH7.4 with KOH.

**Enzyme assays**

The concentration of substrates and cofactors described below were those that gave maximum reaction rates except where specifically stated to the contrary. The rates of reaction were constant throughout the 30 min period of the incubations.

**Determination of the rate of glycerol lipid synthesis from sn-[1,3-3H]glycerol 3-phosphate**

Each assay contained in a volume of 0.25 or 0.50 ml: 25 mM-Hepes buffer, adjusted to pH7.4 with KOH, 4 mm-dithiothreitol, 10 mM-MgCl2, 1.8 mm-ATP, 50 μM-CoA, 0.8 mM-potassium palmitate, 13.6 mm-sn-[1,3-3H]glycerol 3-phosphate (1 μCi/μmol) and 6 mg of fatty acid-poor bovine serum albumin/ml (approx. 90 μg). The reaction was started with about 200 μg of homogenate protein/ml of assay medium. After incubation for 30 min at 37°C the reaction was stopped with 1.88 ml of chloroform/methanol (1:2, v/v). Lipids were extracted and analysed on t.l.c. plates as described by Brindley & Bowley (1975).

**Measurement of dihydroxyacetone phosphate acyltransferase activity** (EC 2.3.1.42)

The reaction conditions were identical with those used for measuring the esterification of glycerol phosphate, except that 0.8 mM-potassium [1-$^{14}$C]-palmitate (0.5 μCi/μmol) was used and glycerol phosphate was replaced by 5 mM-dihydroxyacetone phosphate. The activity was defined as the dihydroxyacetone phosphate-dependent incorporation of [1-$^{14}$C]-palmitate into acylglycerol. After incubation at 37°C for 30 min, reactions were stopped with 1.88 ml of chloroform/methanol (1:2, v/v) and lipids extracted by the method of Hajra et al. (1968). Samples (100 μl) of the bottom phase were applied to t.l.c. plates of Kieselgel 60/Kieselguhr F254 (Merck, Darmstadt, Germany) and developed for 60% of the length with chloroform/methanol/acetic acid/10% (w/v) sodium metabisulphite (25:10:3:1, by vol.). This was followed by two developments with hexane/diethyl ether/acetic acid (60:40:1, by vol.) to remove [14C]-palmitate from the phospholipid areas. Lipids were detected with I2 vapour and spark-chamber photography. Acylglycerol was taken as the band with Rf 0–0.1.

**Measurement of monoacylglycerol acyltransferase activity** (EC 2.3.1.22)

The activity was determined by using 2-alkylglycerols as model substrates for the reaction (Short et al., 1974). These were prepared by sonication (Short et al., 1974) in 0.6 mg of fatty acid-poor bovine serum albumin/ml. The assays contained in a final volume of 0.25 or 0.5 ml: 25 mM-Hepes buffer, adjusted to pH7.4 with KOH, 4 mM-dithiothreitol, 10 mM-MgCl2, 1.8 mM-ATP, 50 μM-CoA, 1 mM-potassium palmitate (0.5 μCi/μmol), 6 mg of fatty acid-poor bovine serum albumin/ml and either 2-2-monoacylglycerol or 2-[2-octadecenyl$[3]^{3}H$]glycerol (1 μCi/μmol). Reactions were stopped after 30 min at 37°C with 1.88 ml of chloroform/methanol (1:2, v/v) and the water content was adjusted to 0.5 ml. Lipids were extracted by the method of Bligh & Dyer, (1959). Neutral lipids were isolated by column chromatography with 8 g of aluminium oxide by elution with 12 ml of chloroform. The products were also analysed on t.l.c. plates of silica gel G by using hexane/diethyl ether/acetic acid (60:40:1, by vol.) as a developing solvent.
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Measurement of the competition for palmitate esterification by glycerol phosphate, dihydroxyacetone phosphate and 2-alkylglycerols

In these experiments the conditions were essentially the same as in the assays described above, except that two acyl acceptors were added at the concentrations indicated in Figs. 2–4. Antimycin A (4 μg/ml) was included in the assays that contained both glycerol phosphate and dihydroxyacetone phosphate in order to inhibit any glycerol phosphate dehydrogenase (EC 1.1.99.5) activity. Incubations involving alkylglycerols and glycerol phosphate were extracted by the method of Bligh & Dyer (1959) and those involving alkylglycerols and dihydroxyacetone phosphate by the method of Hajra et al. (1968). Samples of the bottom phase (100 μl) in the latter system were analysed as above by t.l.c. to detect acylaldihydroxyacetone phosphate. The bottom phase was then washed twice with 2 ml of synthetic top phase made by mixing 1 litre of chloroform, 1 litre of methanol, 400 ml of water and 500 ml of 100 mm-Tris buffer, adjusted to pH 8 with HCl. Portions of the bottom phase were then applied to an alumina column as above to determine the radioactivity in neutral lipids.

Radioactivity

Radioactivity was determined by liquid-scintillation counting (Brindley et al., 1976).

Protein

Protein was determined by a biuret method (Hübscher et al., 1965).

Enzyme kinetics

Lineweaver & Burk (1934) analysis was performed by using a weighted regression procedure (Wilkinson, 1961).

Experimental and Results

Characterization of the glycerol phosphate, dihydroxyacetone phosphate and monoacylglycerol acyltransferase activities of rat adipose tissue

The mean specific activity of glycerol phosphate acyltransferase activity was 9.1 ± 5.5 (s.d. from ten independent experiments) nmol/min per mg of homogenate protein. The molar composition of the products was approx. 15% lysophosphatidate, 61% phosphatidate, 6% diacylglycerol and 18% triacylglycerol. This product composition indicates that phosphatidate phosphohydrolase activity is rate-limiting in the synthesis of triacylglycerol under these conditions. The assay system had been specifically optimized for the acylation of glycerol phosphate rather than for the synthesis of neutral lipids.

Fig. 1. Effect of NADPH on the esterification of dihydroxyacetone phosphate with palmitate

The rate of esterification of dihydroxyacetone phosphate was measured as described in the Materials and Methods section, except that NADPH was added to some incubations. The rates of synthesis of acyl-dihydroxyacetone phosphate (A), phosphatidate (B) and total glycerolipid (C) were calculated by assuming that 1 mol of palmitate incorporated is equivalent to 1 mol of dihydroxyacetone phosphate and 0.5 mol of phosphatidate. Negligible amounts of neutral lipid were synthesized. The rate of synthesis of acyl-dihydroxyacetone phosphate was 2.3 ± 0.4 (s.d. from three independent experiments) nmol/min per mg of homogenate protein, and this is shown as 100 on the graph. The other rates of incorporations are expressed relative to this and s.d. values are indicated by the error bars.

The rate of esterification of glycerol phosphate was significantly greater than that for dihydroxyacetone phosphate (P < 0.001). The latter activity was 2.2 ± 1.6 (s.d. from 14 independent experiments) nmol of dihydroxyacetone phosphate esterified/min per mg of homogenate protein. The product was almost entirely palmitoyl-dihydroxyacetone phosphate. As reported for liver (LaBelle & Hajra, 1974), the addition of NADPH resulted in the formation of phosphatidate (Fig. 1). Optimum concentrations (0.2–0.4 mm) of NADPH did not alter the rate of esterification of dihydroxyacetone phosphate (Fig. 1), but, as expected, resulted in a doubling of the rate of palmitate incorporation. The rate of esterification of dihydroxyacetone phosphate was greater than that of 2-hexadecylglycerol (P < 0.01), but not significantly different from the rate of 2-octadecenylglycerol esterification.

The rate of glycerol phosphate esterification was also greater (P < 0.005) than the rates of esterification of 2-hexadecylglycerol and 2-octadecenylglycerol. These were respectively 0.8 ± 0.3 (s.d. from 11 independent experiments) and 1.1 ± 0.5 (s.d. from seven independent experiments) nmol of alkylglycerol esterified/min per mg of homogenate protein. For both precursors the products were about 98% acylalkylglycerol, with the remainder being diacylalkylglycerol. Similar product compositions were obtained.
with preparations from guinea-pig intestinal mucosa (Short et al., 1974). However, Schultz & Johnston (1971) obtained relatively more diacylalkylglycerol (50%) by using enzymes derived from hamster adipose tissue. In the present work the rate of diacylalkylglycerol synthesis might have been greater if the ionic strength of assay system had been increased and if an unsaturated fatty acid had been used as acyl donor (Coleman, 1976).

**Interaction between the three pathways of glycerolipid synthesis**

These experiments examine whether glycerol phosphate, dihydroxyacetone phosphate and 2-hexadecylglycerol are mutually competitive for the esterification of palmitate.

Fig. 2 shows that glycerol phosphate and dihydroxyacetone phosphate inhibit the esterification of each other in a competitive manner. The apparent $K_m$ value for glycerol phosphate based on the incorporation of $[14C]$glycerol 3-phosphate into lipid was $0.6 \pm 0.2$ mM (s.d. of three independent experiments), whereas that based on the incorporation of $[14C]$-palmitate into phosphatidate was $0.09 \pm 0.02$ mM (s.d. of three independent experiments). These two methods for determining the requirement for glycerol phosphate measure slightly different parameters. The incorporation of palmitate is affected by a contribution from the endogenous fatty acids of the preparation and by the fatty acid specificities of the three enzymes responsible for phosphatidate synthesis. The apparent $K_m$ for dihydroxyacetone phosphate was $2.9 \pm 0.9$ mM (s.d. from four independent experiments) and the respective $K_i$ values for glycerol phosphate and dihydroxyacetone phosphate were $0.16$ and $0.24$ mM. There was a residual activity of dihydroxyacetone phosphate acyltransferase activity which could not be further inhibited by concentrations of glycerol phosphate between 2.75 and 15 mM (Fig. 2b). Since homogenates were used in these studies the $K_m$ and $K_i$ values should be treated with caution. However, the results do show the competitive interactions between glycerol phosphate and dihydroxyacetone phosphate.

Schultz et al. (1971) and Polheim et al. (1973) have shown that octadecenoylglycerol and octadecenylglycerol inhibit the esterification of glycerol phosphate in preparations of hamster adipose tissue and small-intestinal mucosa. In our hands the addition of
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Fig. 3. Effect of 2-hexadecylglycerol on the rate of glycerol phosphate esterification

The Figure shows the effect of varying the concentration of 2-hexadecylglycerol in incubations containing either 1 (●) or 13.5 (○) mm-sn-glycerol 3-phosphate. The results are expressed relative to the maximum rate of glycerol phosphate esterification which was taken as 100 and which was 8.2 ± 2.1 (s.d. from four independent experiments) nmol/min per mg of homogenate protein. The error bars at 13.5 mM-glycerol phosphate represent the s.d. from the four experiments, whereas those at 1 mm-glycerol phosphate indicate the ranges from two experiments.

increasing concentrations of 2-hexadecylglycerol to the incubations, which were optimum for glycerol phosphate esterification, produced up to a twofold stimulation of lipid synthesis from glycerol phosphate (Fig. 3). It was expected that an inhibition of glycerol phosphate esterification might be observed if the concentration of enzyme protein was increased to make it rate-limiting in the assay (W. C. Hülsmann, personal communication). Such an inhibition was not observed in the present work, but an inhibition of up to 35% was obtained when the concentration of glycerol 3-phosphate was made suboptimum in the assay system (Fig. 3).

This result indicates that the inhibition of glycerol phosphate esterification by alkylglycerols might be of a competitive nature. This possibility was tested extensively by using 2-octadeceny[2H]glycerol, which was expected to be a more potent inhibitor. When this unsaturated analogue was used, consistent inhibition of glycerol phosphate esterification was observed (Fig. 4). The kinetics were not of the normal hyperbolic type, which makes conventional analysis impossible. However, Fig. 4 demonstrates the partially competitive nature of the inhibition, which was more pronounced at low concentrations of glycerol phosphate. The esterification of 2-octadeceny[2H]glycerol, which was measured simultaneously, was not significantly affected by varying the concentration of glycerol phosphate.

Experiments were also performed to test the interactions between the dihydroxyacetone phosphate and monoacylglycerol pathways. Varying the concentration of dihydroxyacetone phosphate (0–5 mM) and 2-hexadecylglycerol (0–4 mM) had no significant effect on the rate of the esterification of the other acyl acceptor.

Effects of clofenapate and norfenfluramine on glycerolipid synthesis

Clofenapate [4-(4′-chlorophenyl)phenoxysisobutyrate] and norfenfluramine [1-(m-trifluoromethylphenyl)-2-amino propane] are two drugs that interfere with lipid metabolism. They were added to assay systems containing the three different precursors for glycerolipid synthesis to see whether they could be used to detect differences in properties between the respective acyltransferase activities (Table 1). Clofenapate, an amphiphilic anion, inhibited all three acyltransferase activities. By analogy with p-chloro-
Table 1. Effects of clofenapate and norfenfluramine on the activities of the acyltransferases which act on glycerol phosphate, dihydroxyacetone phosphate and 2-hexadecylglycerol

The mean concentrations of clofenapate and norfenfluramine that were required to produce a 50% inhibition of the enzyme activities are listed. The values were obtained from three independent preparations with at least five different concentrations of the drugs for each enzyme. The combined results were plotted as ln concentration (abscissa) against the percentage inhibition (ordinate). An inhibition of 50% was arbitrarily given a value of 0 and the results were subjected to linear regression analysis. The mean concentration of drug giving a 50% inhibition was defined as the intercept on the abscissa. The values in parentheses show the range of ±1 s.d. from these means. Some compounds produced no significant inhibition up to the concentration shown and this is indicated by *.

<table>
<thead>
<tr>
<th>Concentration (mM) of drug required to produce a 50% inhibition of enzyme activity</th>
<th>Clofenapate</th>
<th>Norfenfluramine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glycerol phosphate acyltransferase</td>
<td>1.5</td>
<td>20*</td>
</tr>
<tr>
<td>(1.4–1.6)</td>
<td></td>
<td></td>
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<tr>
<td>Dihydroxyacetone phosphate acyltransferase</td>
<td>0.91</td>
<td>16*</td>
</tr>
<tr>
<td>(0.84–1.00)</td>
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<tr>
<td>Monoacylglycerol acyltransferase</td>
<td>0.87</td>
<td>15</td>
</tr>
<tr>
<td>(0.78–0.98)</td>
<td></td>
<td>(7–30)</td>
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</tbody>
</table>

Phenoxyisobutyrate, this effect is probably non-competitive with respect to the acyl acceptor (Lamb & Fallon, 1972). The activity of the glycerol phosphate acyltransferase appeared to be less sensitive to inhibition (P < 0.001) than the activity of dihydroxyacetone phosphate acyltransferase, which resembles the situation in rat liver (Bowley et al., 1973; Bowley & Brindley, 1976). Monoacylglycerol acyltransferase activity was also more sensitive to inhibition (P < 0.001) than was glycerol phosphate acyltransferase. The clofenapate inhibition of monoacylglycerol acyltransferase has not been reported before, but clofenapate does appear to be a general inhibitor of acyltransferase activities (Brindley & Bowley, 1975).

In contrast with clofenapate, norfenfluramine is an amphiphilic cation. It was a poor inhibitor of acyltransferase activities (Table 1). Dannenburg et al. (1973) reported that norfenfluramine inhibited the activity of monoacylglycerol acyltransferase in the small intestine by 50% at about 1.7 mM. The equivalent inhibition was not obtained until about 15 mM in the present work (Table 1). Wilson & Galton (1971) reported an inhibition of neutral-lipid synthesis by human adipose tissue by 2–3 mM-fenfluramine, which they attributed to an inhibition of glycerol phosphate acyltransferase. In the present work concentrations of norfenfluramine in this range stimulated glycerol phosphate acyltransferase activity by up to 2.5-fold. However, the proportion of neutral lipid synthesized relative to phosphatidate was decreased. This indicates that an inhibition of phosphatidate phosphohydrolase is the likely explanation for the decreased rate of synthesis of neutral lipids. This conclusion is supported by work with rat liver (Brindley & Bowley, 1975).

Discussion

Palmitate esterification by homogenates of rat adipose tissue was measured by using four precursors for lipid glycerol. The rates of esterification obtained with these precursors decreased in the order glycerol phosphate, dihydroxyacetone phosphate, 2-octadecenoylglycerol and 2-hexadecylglycerol. These rates were dependent on the presence of the other precursors in the assay system. For instance, glycerol phosphate and dihydroxyacetone phosphate were mutually competitive (Fig. 2). A similar effect has been demonstrated with the microsomal fraction of guinea-pig liver (Hajra, 1968). However, these precursors were not mutually competitive with respect to their esterification by the mitochondrial fractions of guinea-pig (Hajra, 1968) and of rat liver (Bowley et al., 1973). Hajra (1968) also showed that the fatty acid specificity of the mitochondrial dihydroxyacetone phosphate acyltransferase differed from that of the microsomal enzyme. Schlossmann & Bell (1976) reported that more than 85% of the activity of dihydroxyacetone phosphate acyltransferase in rat adipose cells was microsomal. LaBelle & Hajra (1972b) have shown that the specific activity of dihydroxyacetone phosphate acyltransferase in the microsomal fraction of rat adipose tissue exceeds that in the mitochondrial fraction by a factor of 2.5. The failure of glycerol phosphate (2.75–15 mM) to inhibit dihydroxyacetone phosphate acyltransferase completely is compatible with there being a dual localization of this enzyme in adipose tissue, the glycerol phosphate-insensitive activity being located in the mitochondria. Schlossmann & Bell (1976) concluded from studies with N-ethylmaleimide, trypsin, Triton X-100 and deoxycholate that the
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estification of glycerol phosphate and dihydroxyacetone phosphate is catalysed in the microsomal fraction of rat adipocytes by a single enzyme. Although a mutual competition for palmitate esterification was also demonstrated between glycerol phosphate and dihydroxyacetone phosphate (Fig. 2; Schlossmann & Bell, 1976), the observation that the acyltransferase activities towards these substrates varied independently of each other (Dodds et al., 1976) indicates that there are two different enzymes. This is also confirmed by the greater sensitivity of the dihydroxyacetone phosphate acyltransferase activity to inhibition by clofenapate.

2-Octadecenylglycerol also inhibited the activity of glycerol phosphate acyltransferase. The inhibition appeared to be of a competitive type (Fig. 4), but the effect of this lipid substrate cannot be described in terms of conventional kinetics (Brindley & White, 1974). 2-Hexadecylglycerol only produced relatively small inhibitions of glycerol phosphate acyltransferase when the concentration of glycerol phosphate in the assay system was suboptimum (Fig. 3). At optimum concentrations of glycerol phosphate a stimulation was observed (Fig. 3) and this has not been reported before. In previous work with the microsomal fraction of guinea-pig intestinal mucosa, 1-monopalmitoylglycerol did not significantly affect the rate of glycerol phosphate esterification (Brindley, 1973). Schultz et al. (1971) proposed that the inhibition of glycerol phosphate acyltransferase by monoaoylglycerol, as demonstrated with their ether analogues, might constitute a control mechanism in the cell. This hypothesis is very attractive, but the inhibition might be caused by a non-specific detergent effect.

The following paper (Dodds et al., 1976) shows a close correlation between the activities of glycerol phosphate acyltransferase and monoaoylglycerol acyltransferase. This may mean that the activities are catalysed by one enzyme. However, the inability of glycerol phosphate to inhibit the esterification of octadecenylglycerol and the greater sensitivity of the monoaoylglycerol acyltransferase to inhibition by norfenfluramine indicate that the two reactions are catalysed by separate enzymes as they are in the enterocyte.

The present paper demonstrates that adipose tissue, like the small intestine (Brindley, 1974), can synthesise glycerolipids from glycerol phosphate, dihydroxyacetone phosphate and monoaoylglycerols. The relative specific activities of the three acyltransferases have been measured and their interactions described. Evidence has been presented to show that these acyltransferases are separate enzymes. The following paper (Dodds et al., 1976) shows how these activities in adipose tissue vary when rats are fed on different diets and discusses the possible physiological significance of these different routes of glycerolipid synthesis.

We thank Dr. H. Booth for advice on n.m.r. spectroscopy, Dr. R. Watts and Miss H. Glenny for help in the synthesis of alkylglycerols, Miss S. Burditt for technical assistance and the Science Research Council and Unilever Research for providing a CASE award to P. F. D.

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