In the present communication we report results which show that rat liver homogenate subfractions can catalyze the biosynthesis of palmitylcarnitine from free palmitate and carnitine. Table 1 shows that rat liver microsomes contain the enzyme system catalyzing this reaction. There is an absolute requirement for ATP and CoA, while the addition of albumin-complexed palmitate gives a 5-fold stimulation. The addition of magnesium chloride, glutathione and of sodium fluoride all have small, but significant stimulating effects. These results indicate that the following reactions take place:

(1) Palmitate + CoA + ATP $\xrightarrow{Mg^{2+}}$ Palmityl-CoA + AMP + pyrophosphate

(2) Palmityl-CoA + carnitine → palmityl-carnitine + CoA

Isolated mitochondria have also been found to catalyze the formation of palmitylcarnitine from carnitine.

These results confirm that fatty acid esters of carnitine are intermediates in the carnitine-stimulated oxidation of fatty acids. The results are also in agreement with the idea that carnitine functions as a carrier of activated fatty acids between metabolic compartments within the cell, as previously suggested [2].

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Studies on Sphingosines. The Isolation and Preliminary Identification of Some Preparative Byproducts of C_{18}-Sphingosine

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With an earlier described method for the dinitrophenylderivatives of sphingosines [1] in combination with silicic acid column and thin layer chromatography several hitherto unknown byproducts of the acid isolation of sphingosine have been prepared. Two seem to be results of an isomerisation of the allyl group in sphingosine and at least two seem to be dehydration products of sphingosine and of its allylic isomers.

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The Mechanism of Ethionine Antilipotropic Action

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Administration of ethionine to female rats results in a rapid and marked accumulation of triglycerides in the liver [1]. Concomitant with this accumulation, a pronounced drop in plasma lipoproteins, including the phospholipid lecithin, occurs. Thus, the accumulation of triglycerides in the liver may be due to an ethionine induced inhibition of plasma lipoprotein synthesis in the liver.

Two pathways for the biosynthesis of lecithin are known: (1) By incorporation of preformed, free choline *via* cytidinediphosphocholine [2], (2) By methylation of phosphatidylethanolamine [3].

In vitro experiments have shown that ethionine is an antimetabilite to methionine in protein biosynthesis [4], and to methionine as a methyl donor in lecithin biosynthesis from phosphatidylethanolamine [3].

Thus there are two possible mechanisms for the ethionine induced inhibition of plasma lipoprotein biosynthesis: (1) Interference with the incorporation of methionine into the protein of plasma lipoproteins, (2) Interference with the incorporation of the methionine-methyl group into the lecithin of plasma lipoproteins.

To test the relative importance of the two pathways of lecithin biosynthesis for plasma lipoprotein synthesis, and to test the relative importance of the two possible mechanisms of ethionine inhibition of plasma lipoprotein synthesis, the experiments shown in Table 1 have been performed.

The following points may be made: (1) The specific activity of blood lecithin compared to the specific activity of liver lecithin is nearly the same both when methyl-^{14}C-methionine and when hydroxyethyl-^{14}C-choline is injected, indicating that lecithin synthesized by both pathways enters a common pool in equilibrium with the plasma lecithin. (2) The inhibiting effect of ethionine on the incorporation of radioactivity into liver lecithin is more pronounced with methionine as the precursor. This may in part be due to an increased pool of free, unlabelled methionine in the liver of ethionine-treated rats, and in part to an inhibition of transmethylation [3]. (3) The specific activity of blood lecithin compared to the specific activity of liver lecithin in the ethionine-treated rats

Table 1. Female rats were injected subcutaneously at 0 and 1.5 h with 100 mg ethionine. Control animals were given corresponding injections of 0.9 % NaCl solution. At 2 h the rats were given an intraperitoneal injection of a tracer dose of either methyl-^{14}C-methionine or hydroxyethyl-^{14}C-choline. At 6 h the rats were killed by decapitation. The specific activity of choline in the choline-containing phospholipids in the liver and plasma was determined after extraction with chloroform-methanol (2:1) and subsequent hydrolysis of the phospholipids. The specific activities given as counts/min/mg choline represents the average of 5 animals.

Precursor	Source of choline isolated	Specific radioactivity of phospholipid choline isolated	
		Control	Ethionine-treated
^{14}C-choline	Liver phospholipid	19 300	15 000
	Plasma-phospholipid	14 670	7 570
$^{14}CH_3$-methionine	Liver phospholipid	33 500	17 600
	Plasma phospholipid	25 140	8 410

is the same with both precursors, although reduced as compared to this relation in the controls. This shows a delayed equilibration of the liver and plasma lecithins in the ethionine treated rats.

The plasmaphospholipid-choline content of the ethionine treated rats was reduced to approximately 50 % of the controls, while the liver phospholipid-choline content was unchanged (not shown). This shows an inhibited excretion of lecithin from the liver in the ethionine treated rats.

Altogether these results show that the antilipotropic action of ethionine mainly must be due to interference with the synthesis of the protein part of the plasmalipoproteins in the liver. This conclusion is in agrement with the fact that choline has no effect on the fatty liver in ethionine treated rats.

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Free Radicals in the Reaction of Alloxan with Glutathione and Ascorbic Acid

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It has been postulated [1] that the diabetogenic action of alloxan is produced by an inactivation of essential sulphydryl enzymes. This theory is based on the observation that the prior injection of large doses of either glutathione or cysteine protected animals from the action of alloxan. Further, *in vitro* experiments have shown that alloxan reacts with sulphydryl compounds [2]. These reactions are considered to involve a reduction of alloxan to dialuric acid.

With the technique of electron spin resonance (ESR) it has been found that free radicals are formed from alloxan in reactions with sulphydryl compounds and ascorbic acid.

Experimental. The ESR spectra were obtained by a Varian 100 kc. spectrometer. All experiments were performed with aqueous solutions at ambient room temperature.

Results. (1) Free radicals were obtained when alloxan (0.01 M) was dissolved in phosphate