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by E. Buddecke, I. Filipovic, B. Wortberg, and A. Seher

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# Long-Chain Mono-Unsaturated Fatty Acids and their Effect on the Energy Metabolism of the Heart*

by E. Buddecke, I. Filipović, B. Wortberg, and A. Seher**

From the University Physiologico-Chemical Institute and the Federal Institute for Fat Research, Münster (West Germany)

**Mechanism of Long Chain Monoenoic Fatty Acids Acting on the Energy Metabolism of Heart**

The oxidation of 1-$^{14}$C-erucic ($C_{22:1}$) and 1-$^{14}$C-nervonic ($C_{24:1}$) acids was studied in comparison to 1-$^{14}$C-palmitic and -oleic acids in isolated rat and pig heart mitochondria. After mitochondrial incubation with the albumin-bound fatty acids only small amounts of $^{14}CO_2$ developed from the oxidation of the long chain monoenoic acids as compared to palmitic or oleic acid. The slow down of the oxidation rate was more pronounced in rat than in pig heart mitochondria. The oxidation of palmitic or oleic acid was not found to be inhibited by the $C_{20}$–$C_{24}$-monoenoic acids, whereas palmitic or oleic acid inhibited the oxidation of erucic acid competitively. From present findings an idea may be developed of the interference on fatty acid metabolism in heart muscle by erucic and other long chain monoenoic acids.

## Introduction

The occurrence of erucic acid (cis $\Delta^{13}$docosenoic acid) and homologous $C_{20}$- $C_{24}$ mono-unsaturated fatty acids in cruciferous oils is held responsible for the nutritional damage observed after feeding these oils

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**Authors' addresses: Prof. Dr. *E. Buddecke* und Dr. *I. Filipović*, Physiologisch-Chemisches Institut der Universität Münster, Waldeyerstraße 15, 4400 Münster. Lebensm.-Chem. *Birgit Wortberg* und Prof. Dr. *A. Seher*, Bundesanstalt für Fettforschung, Piusallee 68/76, 4400 Münster.

to various animal species where this damage manifests itself mainly in pathological changes of the cardiac muscle[1,2].

In the organism, the degradation of long-chain mono-unsaturated fatty acids generally takes place over the β-oxidation[3]; however, it proceeds at different rates in the individual organs. According to "in vivo" investigations with 1-$^{14}$C-labelled erucic acid, the oxidation of the erucic acid proceeds clearly more slowly in the cardiac muscle than in the liver; in this process, eicosenoic acid and oleic acid accumulate in the heart lipids[4].

Also "in vitro" investigations of the fatty-acid degradation in (197)
mitochondria isolated from rat hearts showed a clearly lower oxidation rate for erucic acid when the fatty acids were used either as carnitine esters[5] or as free $^{14}$C-labelled fatty acids[6,7].

After the feeding of rapeseed oil to hogs, lesser changes were observed at the cardiac muscles of these animals than with rats[8]. In order to find out whether this lower sensitivity of hogs to erucic acid is due to better oxidation in the mitochondria of the cardiac muscle or whether there is another reason, we investigated the degradation of 1-$^{14}$C-labelled palmitic, oleic, erucic, and nervonic acids in mitochondria isolated from rat and hog hearts.

## Experimental Conditions

### 1. Mitochondrial Preparations

From the cardiac-muscle tissues of normally fed rats (about 300 g weight) and young hogs (10-12 weeks old), the mitochondria were isolated according to tried methods[7,9]. For the mitochondrial preparations from rat hearts, we homogenized about eight rat hearts in a 10% suspension in

0.25 M saccharose - 0.001 M NaEDTA - 0.01 M tris/HCl buffer (pH = 7.4)*, at 0°C. For mitochondrial preparations from hog heart muscle, with the same protein concentrations, we had to start from about 40 g of tissue.

The mitochondrial pellets obtained were suspended, at 0°C, in 2 ml of 0.25 M saccharose - 0.01 M tris/HCl buffer (pH = 7.4). The protein content was determined according to O.H. LOWRY[10]. The purity of the mitochondrial preparations was tested by electron-microscopic investigations**.

## 2. Incubations

### 2.1. Incubation Solution

The radioactively labelled fatty acids, 1-$^{14}$C-palmitic and oleic acids (firm of AMERSHAM-BUCHLER), 1-$^{14}$C-erucic acid (firm of CEA-FRANCE), 1-$^{14}$C-nervonic acid (firm of NEN), were tested by thin-layer chromatography for purity[11]. With gas-chromatographically pure fatty acids (firm of NU CHEK PREP), they were diluted to a specific radioactivity of 0.2 $\mu$Ci/$\mu$mole, and saponified with ethanolic KOH. Subsequently, owing to the poor solubility of the potassium salts of saturated and long-chain unsaturated fatty acids in the aqueous incubation medium, all the fatty acids, before being added to the incubation solution, were combined with degreased beef serum albumin at the molar ratio of 7:1[12,13]. Besides, the addition of albumin eliminates the toxic soap effect of the fatty acids.

Composition of the incubation solution: 25 mmoles of K-phosphate buffer (pH = 7.4); 20 mmoles of KCl, 5 mmoles of $MgCl_2$; 25 mmoles of saccharose; 1 mmole of malate; 0.1 mmole of succinate; 2.5 mmoles of ATP;

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* Tris(hydroxymethyl)-aminomethane

**The authors are endebted to Dr. ROESSNER for his carrying out the investigations at the University of Münster Institute for Medical Cytology.

5 mmoles of d,l-carnitine, 0.05 mmole of cytochrome c; 0.05 mmole of coenzyme A. For the mitochondrial incubation, 1 ml of this freshly prepared solution was mixed with the desired concentration of the labelled, albumin-bound fatty acid.

2.2. Incubation Experiments

The incubation took place in 20-ml glass ampules (firm of MACHEREY & NAGEL); before the start, a glass tube with 0.3 ml of Hyamin solution (I M Hyamin hydroxide in methanol) was put into the incubation mixture to catch the $^{14}CO_2$. Of the mitochondrial suspension, a sample amount corresponding to about 1 mg of protein was added to the incubation solution. Subsequently, the ampules were closed gastight with rubber stoppers and Al caps, and incubated for 30 minutes in a 37°C agitated water bath. By injecting 0.5 ml of 2N $H_2SO_4$, the oxidation was terminated, and the sample containers were allowed to stand in the water bath for another 30 minutes, at 37°C, and for another 60 minutes at room temperature, to ensure the complete absorption of the $^{14}CO_2$ in the Hyamin solution. The radioactivity of the $^{14}CO_2$ was determined in 10 ml of scintillation solution (4.2 g of PPO (diphenyl oxazol) + 0.0525 g of POPOP (1,4-bis[2-(5-phenyloxazolyl)]-benzene) in 2 liters of toluene/ethyleneglycol-monomethyl ether (1:1)) in the Packard-Tricarb scintillation counter.

## Experimental Results

In the degradation of 1-$^{14}C$ oleic acid, a linear increase in the oxidation rate was obtained over a 50 minute incubation period with rat and hog heart mitochondria. The time dependence of the oxidation of erucic acid did not proceed linearly, since already after 20 minutes, an inhibition of the oxidation occurred (Fig. 1).

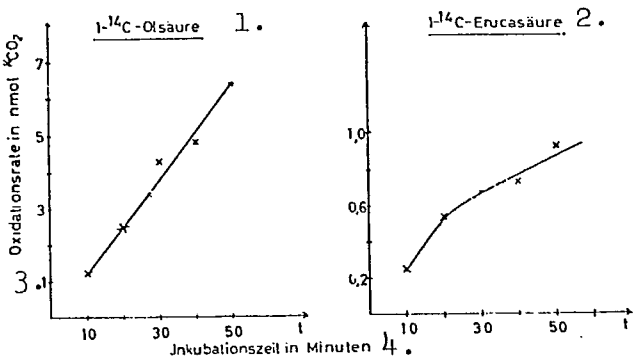



Fig. 1. Time dependence of the oxidation of 1-$^{14}$C-labelled fatty acids in mitochondria from rat hearts.

Investigations regarding the dependence of the oxidation on the fatty-acid concentration resulted, for oleic acid, in an optimum concentration of about 100 nmoles per mg of protein, for the heart mitochondria of rats, and about 300 nmoles per mg of protein for those of hogs. Higher concentrations caused increasing inhibition of the oxidation.

Erucic acid caused substrate inhibition, at concentrations in excess of 65 nmoles per mg of protein, in rat-heart mitochondria, and at concentrations in excess of 175 nmoles per mg of protein, in hog-heart mitochondria. Likewise, the altogether very low oxidation rate of nervonic acid was inhibited in the concentration range of $>65$ nmoles per mg of protein, in rat-heart mitochondria.

For palmitic acid, we determined at rising substrate concentration, (198) after the usual ascent, a constant oxidation rate, up to concentrations of over 700 nmoles per mg of protein.

The oxidation rates of the investigated 1-$^{14}$C-labelled fatty acids, after 30 minutes incubation time, are shown in Table 1.

In rat-heart mitochondria, less than 2 nmoles of erucic and nervonic acids per mg of protein were oxidized after 30 minutes. In hog-heart mitochondria, about 2 nmoles of erucic acid per mg of protein were degraded

Table 1

Oxidation rates of investigated 1-$^{14}$C-labelled fatty acids after 30 min. incubation time

| 1-$^{14}$C-markierte Fettsäuren 1. | Ratten- 2. Herzmitochondrien 100 nMol FS/mg Prot. | | Schweine-3. Herzmitochondrien 300 nMol FS/mg Prot. | |
|---|---|---|---|---|
| | 4. Oxidationsraten nach 30 Min. | | | |
| | 5. nMol FS/mg Pr. | % | nMol FS/mg Pr. | % |
| Ölsäure | 6. 78 | 100 | 35 | 100 |
| Palmitinsäure | 7. 69 | 88 | 33 | 94 |
| Erucasäure | 8. 1.4 | 1.8 | 2.1 | 5.7 |
| Nervonsäure | 9. 0.4 | 0.5 | — | — |

1. 1-$^{14}$C-labelled fatty acids
2. Rat-heart mitochondria, 100 nmoles FA/mg protein
3. hog-heart mitochondria, 300 nmoles FA/mg protein
4. Oxidation rates after 30 minutes
5. nmoles of FA/mg of protein
6. Oleic acid
7. Palmitic acid
8. Erucic acid
9. Nervonic acid

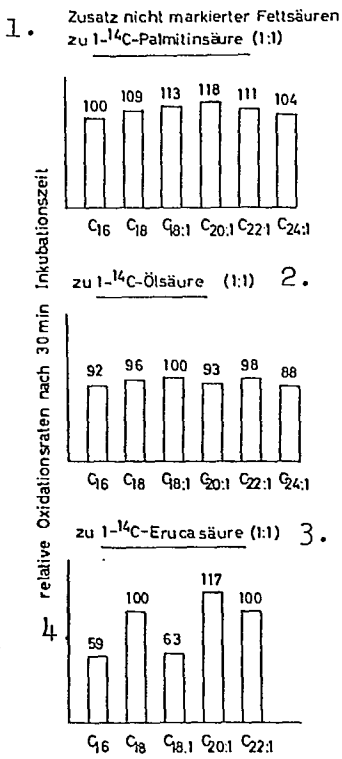



Fig. 2.
Relative oxidation rates of 1-$^{14}$C-labelled fatty acids after the addition of nonlabelled fatty acids at the ratio of 1:1

1. Addition of nonlabelled fatty acids to 1-$^{14}$C palmitic acid (1:1)
2. to 1-$^{14}$C oleic acid (1:1)
3. to 1-$^{14}$C erucic acid (1:1)
4. Rel. oxidation rates after 30 min. incubation time

after the same incubation time. For experiments for the degradation of nervonic acid in hog-heart mitochondria, unfortunately, no more mitochondrial material was available. In investigations on the effect of the fatty acids among one another, we determined the relative oxidation rates of the labelled fatty acids in the heart mitochondria, after the addition of nonlabelled, saturated $C_{18}$ fatty acid and $C_{20}$- $C_{24}$ mono-unsaturated fatty acids (Fig. 2).

Hence, long-chain mono-unsaturated fatty acids, added at the ratio of 1:1, caused no inhibition of the oxidation of palmitic or oleic acid. The low oxidation rate of erucic acid was inhibited competitively by oleic or palmitic acid. Experiments with hog-heart mitochondria provided results which are comparable to those with rat-heart mitochondria.

## Discussion of Experimental Results

The investigations for the degradation of long-chain mono-unsaturated fatty acids in heart mitochondria showed for both rats and hogs a clearly slower oxidation of erucic acid, compared to palmitic or oleic acid. However, the relative oxidation rate of erucic acid in hog-heart mitochondria is more than three times as high as in rat-heart mitochondria.

A better oxidation of erucic acid in the heart mitochondria of hogs might therefore be one of the reasons for the lower accumulation of erucic acid in hog-heart mitochondria[8].

Moreover, in these experiments, hog-heart mitochondria also proved to be less sensitive to substrate inhibition by high fatty-acid concentrations.

On similar experimental results, following the oxidation of 1-$^{14}$C-labelled fatty acids in rat-heart mitochondria, M.A. SWARTTOUW[7] reported recently. He did not find either an inhibition of the oxidation of palmitic or oleic acid by erucic acid. In contrast thereto, B.O. CHRISTOPHERSEN and J. BREMER[5] observed an inhibition of the oxidation of palmitic acid by

erucic acid when the fatty acids were used in the form of carnitine esters. In more recent investigations, B.O. CHRISTOPHERSEN[14] confirmed these findings. Owing to different experimental conditions, however, our own investigations are not directly comparable with B.O. CHRISTOPHERSEN's.

In the oxidation metabolism of long-chain mono-unsaturated fatty acids, all the enzyme systems involved must be taken into consideration, from the absorption in the myocardial cell, over the activation and the transport into the mitochondria, unto complete degradation to acetyl CoA. Accordingly, three essential sections are to be distinguished, as shown diagrammatically in Fig. 3:

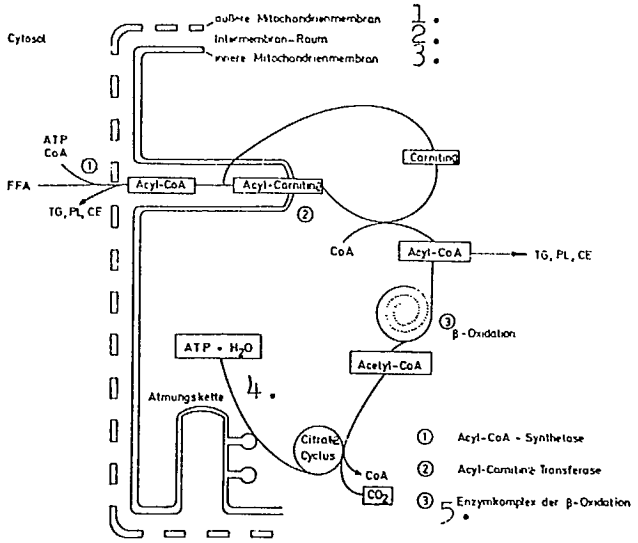
activation of the fatty acid by acyl CoA synthetase,
transport as acyl-carnitine ester through the inner mitochondrial membrane, catalyzed by acyl-carnitine transferase,
renewed activation to acyl CoA, and subsequent β-oxidation in the mitochondrial matrix to acetyl CoA.

The fatty acids activated as acyl-CoA esters may also enter the lipid synthesis both in the cytoplasm and in the mitochondria.

According to investigations carried out so far, the enzymatic action of the oxidation metabolism is clearly weaker on erucic acid than on palmitic or oleic acid. From the experiments described in the literature, the following may be seen:

During the feeding of erucic-acid-rich oils, there is a relatively (199) constant flow of long-chain mono-unsaturated fatty acids through the plasma lipids toward the myocardial cell [15]. A preferential absorption of erucic acid from the plasma lipids was not found[16].

Free fatty acids in the plasma are albumin-bound; however, the molar binding ratio of fatty acid to albumin varies, depending on chain length and number of double bonds. Table 2 shows the molar binding ratios between some long-chain fatty acids and beef serum albumin[13].




1. Outer mitochondrial membrane
2. Intermembraneous space
3. Inner mitochondrial membrane
4. Respiratory chain
5. Enzyme complex of β-oxidation

Fig. 3. Diagram of oxidation metabolism of fatty acids in the mitochondria

Table 2

Molar binding ratio between long-chain fatty acids (FA) and beef serum albumin (BSA)*

| Fatty acid | Moles FA/Moles BSA |
|---|---|
| $C_{16}$ | 7.0 |
| $C_{18:1}$ | 8.9 |
| $C_{18:2}$ | 10.6 |
| $C_{18}$ | 6.2 |
| $C_{20}$ | 2.3 |
| $C_{22}$ | 0.5 |

* nach *A. A. Spector* u. *J. C. Hoak*, Analyt. Biochem. 32, 297 [1969].

With increasing chain lengths, the binding of fatty acids to albumin decreases; by the introduction of double bonds, however, it is strengthened. Similar conditions exist for the distribution of long-chain fatty acids among lipoproteins and serum albumin (Table 3). From the data of Table 3[17], one may recognize a decrease in the binding of erucic and nervonic acids to serum albumin.

Consequently, the cytosol contains an increased portion of free long-chain mono-unsaturated fatty acids which interact with the mitochondria.

Table 3

Distribution of long-chain, saturated and unsaturated fatty acids (FA) among human serum albumin and lipoproteins, after ultracentrifuging*

| Fatty acid | % FA in albumin fraction |
|---|---|
| $C_{18}$ | 78.7 |
| $C_{18:1}$ | 85.2 |
| $C_{18:2}$ | 91.3 |
| $C_{18:3}$ | 96.2 |
| $C_{20}$ | 59.3 |
| $C_{20:4}$ | 77.4 |
| $C_{22}$ | 27.0 |
| $C_{22:1}$ | 36.0 |
| $C_{24}$ | 9.0 |
| $C_{24:1}$ | 18.5 |

* nach[10] *E. Shafrir, S. Gatt* u. *S. Khasis*, Biochim. biophysica Acta [Amsterdam] 98, 365 [1965].

M.A. SWARTTOUW[7] reports on these interactions. After the incubation of rat-heart mitochondria with the 1-$^{14}$C-labelled fatty acids, in the presence of beef serum albumin and buffer, he separated the suspension by ultracentrifuging, and determined the radioactivity of the separate fractions. Oleic and palmitic acids then were more strongly bound to albumin than to the mitochondria. In the incubations with erucic acid, the mitochondria (pellets) contained 3 to 7 times more of this acid than in the experiments with oleic or palmitic acid.

The activation of erucic acid by acyl CoA synthetase and the transfer to carnitine by means of acyl carnitine transferase proceed at a fifth of the rate at which palmitic acid is converted[7,18].

R.O. VLES et al.[19] therefore consider the transport through the inner mitochondrial membrane as a limiting factor for the oxidation in the mitochondria. However, an inhibition of the ß-oxidation, in the opinion of these authors, is hardly probable. Model experiments with pure acyl CoA dehydrogenase might clarify which step is rate-determinative in the slow oxidation of long-chain mono-unsaturated fatty acids.

An inhibitory effect of long-chain mono-unsaturated fatty acids on the oxidation metabolism of heart mitochondria was shown, even in a different manner, by R.O. VLES and U.M.T. HOUTSMULLER[19,20]. After a three-day feeding of rapeseed oil or synthetic triglycerides with various homologous and isomeric $C_{20}$ to $C_{24}$ mono-unsaturated fatty acids to rats, they isolated the heart mitochondria of the animals, and determined therein the formation of ATP after the oxidation of glutamate. At increasing chain length of the fatty acids, they then found an increasing inhibition, up to almost 50 %, of the ATP formation. Position-isomeric mono-unsaturated (200) acids inhibited to a similar extent; hence, the chain length is of decisive importance.

An oxygen absorption, decreased by about 10 %, in isolated heart mitochondria of rats, fed with rapeseed oil for three days[18] or several weeks[21], was observed also with the use of fatty acid carnitine esters as substrates for the oxidation. According to more recent investigations, higher portions of erucyl carnitine and erucyl CoA as inhibitory substrates of the acyl carnitine transferase or of the acyl CoA dehydrogenase might be responsible for the inhibitory effect on the oxidation of other fatty acids[14].

The accumulation of erucyl CoA in the mitochondria would cause a deficiency in free coenzyme A for the oxidation of CoA-dependent substrates[18].

How the lipid accumulation, the introduction of long-chain mono-unsaturated fatty acids into the triglycerides, phospholipids and cholesterol esters, the low albumin binding, yet increased binding to the mitochondrial membrane, affect the oxidation metabolism of the heart mitochondria, remains to be elucidated.

More recent reports[22,23] provide some indications to the effect that a changed phospholipid composition of the inner mitochondrial membrane may affect, particularly, the membrane-bound enzymes of the respiratory chain. The exchange of the linoleic and arachidonic acids in 2-position of the phospholipids changes the physical properties of the mitochondrial membrane. A changed fatty-acid composition in the phospholipids with a specific function on lipid-dependent enzymes of the respiratory chain would suggest an influence on the enzyme activity.

Model experiments with phospholipids of known composition of long-chain mono-unsaturated fatty acids on the effect on various phospholipid-dependent mitochondrial enzymes might contribute to the research of possible specific effects of erucic acid and other long-chain mono-unsaturated fatty acids on the oxidation metabolism of the cardiac muscle.

The experiments we have carried out so far with hog-heart mitochondria point to the fact that also for this animal species, basically similar conditions exist in the degradation of long-chain mono-unsaturated fatty acids in the cardiac muscle as for the rat. However, the changed quantitative results make it clear that in an attempt to estimate the nutrition-physiological hazard for man that may result from erucic acid and corresponding mono-unsaturated fatty acids from hydrogenated fish fats, additional factors must be taken into consideration.

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