Effect of butter compared with tallow consumption on postprandial oxidation of myristic and palmitic acids

Diane E MacDougall, Peter JH Jones, David D Kitts, and P Terrence Phang

ABSTRACT

To assess the influence of dietary fat composition on rates of oxidation of dietary myristic (MA) and palmitic (PA) acids, eight healthy males consumed prepared solid-food diets for 11 d with 40% of total energy as fat. Fifty-five percent of the energy obtained in the form of fat was provided as butter or beef tallow. On days 8 and 11 of each diet cycle, 20 mg/kg body wt of either [1-13C]MA or [1-13C]PA was ingested with breakfast. Hourly breath samples were collected over 9 h thereafter and 13CO2 enrichments were determined by using isotope-ratio mass spectrometry. The percentage of [1-13C]MA appearing in breath carbon dioxide over 9 h was more than twofold that of PA (P < 0.01). Diet fat composition did not influence the mean (± SEM) percentage 13C recovered over 9 h from either labeled MA (7.1 ± 1.0% compared with 8.6 ± 0.9% for butter and tallow, respectively) or PA (3.3 ± 0.7% compared with 3.0 ± 0.9% for butter and tallow, respectively). However, net MA oxidation, calculated as the percent recovery of fatty acids in the meal, was greater (P < 0.05) after the butter (329 ± 45 mg) than after the tallow (212 ± 25 mg) breakfast. In contrast, no difference was observed in net oxidation of dietary PA between butter (441 ± 99 mg) and tallow (348 ± 95 mg) meals. In conclusion, there was no effect of varying the dietary content of MA and PA on fractional oxidation; consequently, net oxidation of these fatty acids was proportional to their concentration within the diet. Am J Clin Nutr 1996;63:918–24.

KEY WORDS Fatty acid, oxidation, myristic acid, palmitic acid, butter, tallow, humans

INTRODUCTION

Discrimination of dietary fatty acids for oxidation compared with retention on the basis of chain length has been well documented. Rats fed medium-chain fatty acids (MCFAs) gained 20% less weight than those fed equivalent amounts of long-chain fatty acids (LCFAs) (1). Similarly, tracer-labeled dietary MCFAs undergo more rapid conversion to labeled carbon dioxide compared with LCFAs (2, 3). Correspondingly, storage of labeled dietary fatty acids in muscle, adipose tissue, and other tissues is reduced with shorter-chain fatty acid feeding (2, 3). In humans, the fractional oxidation rates of [1-13C]- and [1-13C]octanoate exceed those of palmitate (4, 5) and oleate (5–7) when administered orally in fat emulsions, lending further support to the view that fatty acid chain length is an important determinant in fatty acid partitioning toward immediate oxidation versus storage.

Although acyl chain length dependency of fatty acid oxidation has been described, how changes in dietary fat composition influence the partitioning of individual fatty acids for energy awaits better definition, particularly in humans. Whether changes in percentage (ie, fractional) and absolute (ie, net) oxidation rates for specific dietary fatty acids occur with shifts in the ratio of dietary polyunsaturated fat to saturated fat (P:S) has been examined only recently (8). However, this question has not been determined for shifts in the ratio of MCFAs to LCFAs in the saturated fat component of the diet. Increases in net dietary fatty acid oxidation rates could foreseeably result from either higher fractional oxidation, or unchanged fractional oxidation with increased dietary pool size. The extent to which dietary saturated fat composition and corresponding fatty acid pool size influences the oxidation of specific fatty acids remains to be clearly defined.

To better understand chain–dependent discrimination of fatty acid utilization, the objective of this study was to compare postprandial oxidation of the dietary LCFAs myristic acid (MA, 14:0) and palmitic acid (PA, 16:0) in healthy volunteers fed diets containing 55% of energy as fat, as butter or beef tallow. It was hypothesized that consumption of butter, which is higher in MCFAs, may result in altered oxidation of MA and PA compared with beef tallow, which is higher in LCFAs.

SUBJECTS AND METHODS

Subjects

Informed, written consent was obtained from eight nonobese, free-living, healthy males aged 24.3 ± 1.4 y (x ± SEM) with a height of 176.0 ± 2.9 cm, an initial weight of 67.9 ± 2.1 kg, and an energy intake of 13.4 ± 0.8 MJ/d. Subjects had reportedly maintained stable weight for ≥4 wk before the study, were not taking any prescribed medications, did not smoke, had normal circulating lipid profiles, and were free of chronic disease. All subjects completed the study in its entirety.

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and were instructed to maintain typical and consistent sleep and exercise patterns during feeding periods. The protocol was approved by the Human Ethics Review Committee of the University of British Columbia. Volunteers provided informed written consent before the study began.

**Experimental diets**

Solid-food test diets providing 40%, 45%, and 15% of energy as fat, carbohydrate, and protein, respectively, were fed for 11 d in amounts calculated to satisfy individual energy requirements. Diets contained 55% of energy as fat, as either butter or beef tallow, and were otherwise identical. Diets were composed from regular foods and provided as a 2-d rotating menu of three meals per day. Meals were isoenergetic in total energy and fat. The rotating menu was modified to ensure that for all trials, day 1 of the rotating menu was used on pretest days whereas day 2 was used each test day. To minimize natural product variation, single homogenized batches of both butter (Dairy World, Vancouver, Canada) and tempered tallow (Cargill Foods, Edmonton, Canada) were obtained before study. Consistent brands of packaged food were purchased. Meats and vegetables were obtained in large batches. Carbohydrates from corn-based foods were avoided to minimize the contribution of $^{13}$C to breath carbon dioxide from sources other than the tracer. Ingestion of medication or energy-containing substances other than the prescribed diet was prohibited. All foods used for diets were measured to the nearest 0.5 g except for treatment fats, which were measured to the nearest 0.1 g. Nutrient contents of diets and component meals were calculated by using NUTRICOM (Robert Kok and Serge Trembley, version 1, 1984; Smart Engineering, Vancouver, Canada) a computerized Canadian nutrient composition program. Subjects' energy requirements were calculated by using the Harris-Benedict equation (9) with an adjustment factor of 1.7 for additional energy needs of young men (10) and an optional adjustment for strenuous athletics. Meals were prepared and consumed in a Metabolic Testing Unit at the University of British Columbia. Subjects unable to consume a meal at the Unit were given a packaged meal to be eaten at the regular meal time. Test-day breakfast meals were tailored to meet one-third of individual energy requirements (4.38 ± 0.1 MJ, 36 ± 1 g protein, 118 ± 3 g carbohydrate, 32.5 ± 0.9 g butter or 25.7 ± 0.7 g tallow, and 20.9 ± 0.6 g nontreatment fat). Fatty acid analyses of test breakfast meals are presented in Table 1. Fat composition differed slightly between the breakfast meal and the preceding diet, although the fat composition of diets was constant between and within subjects.

**Study protocol**

In a crossover design, each subject consumed both butter- and tallow-based diets for 11 d and received in each experimental diet both $[^{13}]$CMA and $[^{13}]$CPA, assigned in random order as established by using a table of random digits. Diet cycles were separated by ≥ 7 d of habitual eating. One tracer was provided on day 8 and the other on day 11 of each diet cycle. Subjects reported for testing at 0700 on days 8 and 11 of each diet cycle after a 12-h fast. After a 30-min relaxation period, breath-sample collection was performed before and hourly thereafter for 9 h after the breakfast meal began at 0800. The test meal was accompanied by a randomly assigned oral bolus administration of 20 mg/kg body wt of either $[^{13}]$CMA or $[^{13}]$CPA (Isotec Inc, Miamisburg, OH). Respiratory carbon dioxide excretion was measured almost continuously from 0720 to 1715. Subjects were permitted to consume a lunch meal at ≈1230 and to take washroom breaks as required throughout the day. Subjects reclined during all measurements while reading, listening to music, or watching television; sleeping and physical movement beyond minor positional changes were discouraged.

**Collection, purification, and analysis of carbon dioxide in breath samples**

During respiratory gas-exchange measurement, a portion of exhaled gas flowing through the metabolic analyzer system was directed into a 100-cm spiral trap over 6 min. The trap contained 10 mL NaOH (1 mol/L), which enabled complete carbon dioxide collection (11) as verified in our laboratory by using a carbon dioxide analyzer. Collected carbon dioxide samples were stored in sealed evacuated tubes and frozen at −10 °C for later analysis.

To purify carbon dioxide, samples of the sodium hydroxide solutions were mixed with H$_2$PO$_4$. The carbon dioxide evolved was cryogenically separated from other gases by using liquid nitrogen and liquid nitrogen–methanol traps. This purified carbon dioxide was collected in a flame-sealed polytetrafluoroethylene tube before isotope-ratio mass spectrometric (VG 903; VG Isogas Limited, Cheshire, United Kingdom) analysis. The mass spectrometer was calibrated daily against reference standards. Sample $^{13}$C enrichments were corrected for $^{17}$O. Atom percent enrichment (APE) of predose and 1-h postdose breath samples (± SEM; n = 24: −0.024 ± 0.001 APE and −0.024 ± 0.001 APE, respectively) were similar and therefore pooled for analysis. Background $^{13}$CO$_2$ enrichment, based on predose and 1-h postdose breath samples, was subtracted from $^{13}$CO$_2$ enrichment of later samples (2–9 h postdose) to determine enrichment due to fatty acid oxidation. Samples were prepared and analyzed in duplicate. Mean internal and external precision ($n = 436$) levels were 0.014 ± 0.002% and 0.44 ± 0.04%, respectively, for mean enrichments of about −18%.

**Table 1**

<table>
<thead>
<tr>
<th>Fatty acid</th>
<th>Butter meal</th>
<th>Tallow meal</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>% by wt</td>
<td></td>
</tr>
<tr>
<td>8:0</td>
<td>1.2</td>
<td>0.6</td>
</tr>
<tr>
<td>10:0</td>
<td>2.1</td>
<td>0.5</td>
</tr>
<tr>
<td>12:0</td>
<td>2.4</td>
<td>tr</td>
</tr>
<tr>
<td>14:0</td>
<td>9.7</td>
<td>5.1</td>
</tr>
<tr>
<td>14:1</td>
<td>1.2</td>
<td>0.6</td>
</tr>
<tr>
<td>16:0</td>
<td>28.6</td>
<td>23.5</td>
</tr>
<tr>
<td>16:1</td>
<td>1.5</td>
<td>3.4</td>
</tr>
<tr>
<td>18:0</td>
<td>10.8</td>
<td>13.2</td>
</tr>
<tr>
<td>18:1</td>
<td>25.9</td>
<td>33.8</td>
</tr>
<tr>
<td>18:1t</td>
<td>3.9</td>
<td>2.3</td>
</tr>
<tr>
<td>18:2</td>
<td>12.7</td>
<td>11.5</td>
</tr>
</tbody>
</table>

1 Breakfast meals include $[^{13}]$C fatty acids. Fatty acid concentrations <0.5 % by wt were omitted. Fatty acid composition of diets differs slightly from that of test meals. tr, trace.
Measurement and analysis of whole-body carbon dioxide

Carbon dioxide production was measured with a Deltatrac Metabolic Monitor (Sensormedics, Anaheim, CA). A transparent, ventilated hood was placed over the subject’s head with a hose connecting the hood and analyzer system. After adequate warm-up, reference gas standards were used to calibrate the monitor. All measurements were corrected for ambient temperature, pressure, and humidity. The carbon dioxide production rate was measured each minute and analyzed in 60-min blocks. Initial validation of the specific monitor used against a lung model identified the accuracy of the carbon dioxide measurement within 1.5% (12).

Fatty acid composition of test breakfast meals

Replicate portions of test breakfast meals were homogenized with a commercial blender. The fatty acid content of homogenates was determined by gas-liquid chromatography after lipid extraction (13) and boron trifluoride methylation (14). The gas chromatograph (model 5890; Hewlett Packard Series II, Palo Alto, CA) was equipped with a flame-ionization detector. Separation of fatty acids was achieved on a Hewlett Packard-5, 30-m capillary column (0.2 mm internal diameter, 0.33 μm thickness) with a split ratio of 100:1. Running conditions were as follows: initial temperature of 180 °C, temperature increase of 1 °C/min, final temperature of 210 °C, hold for 30 min. The total run time was 60 min/sample.

Data analysis

Fractional oxidation rates of breakfast-meal MA and PA, based on the percentage [1-13]CMA or [1-13]CPA ingested appearing in breath as 13CO2, were determined by using the following equation (15):

\[
\text{Fractional oxidation (dietary 13C recovery/9 h)} = \frac{\text{mmol CO}_2/L \times f \times 13 \text{C APE} \times 1.25 \text{ mmol (l)}}{
\]

where \( f \) represents the fractional contribution of carbon mass to carbon dioxide, APE is the atom percent enrichment of 13CO2 over baseline, and the factor 1.25 corrects for labeled carbon dioxide dilution in bicarbonate pools (16).

Both MA and PA contents of test breakfast meals were calculated for individual subjects using the following formula:

\[
\text{Fatty acid content (g) = breakfast meal fat (g)} \times \text{concentration of corresponding fatty acid in the breakfast meal (% fatty acid by wt)} \quad (2)
\]

Net oxidation rates of dietary MA and PA were calculated as follows:

\[
\text{Net oxidation (mg/9 h) = breakfast meal fatty acid (mg)} \times \text{fractional oxidation (dietary 13C recovery/9 h)} \quad (3)
\]

Statistics

Data are expressed as means ± SEMs. Fractional oxidation rates over time were tested for effects of diet (butter compared with tallow) and fatty acid (MA compared with PA) by using repeated-measures analysis of variance (ANOVA). Cumulative net oxidation, cumulative and peak fractional oxidation, as well as time (h) of peak fractional oxidation of 13C fatty acids were analyzed by using a paired t test. The level of significance was \( P < 0.05 \).

RESULTS

During diet cycles, subjects consumed all meals provided. No alterations in sleep, activity patterns, or health were reported. Body weight change over the 11-d diet cycles ranged from 0.05 to 0.90 kg across subjects. Mean weight change was small for subjects fed butter (0.08 ± 0.17 kg) and tallow (0.13 ± 0.25 kg) diets, suggesting that the calculated energy requirements were consistent with actual energy expenditure. Weight fluctuation over the 11-d cycles did not differ significantly on the basis of fat treatment.

Fatty acid composition profiles of test breakfasts are shown in Table 1. Overall, 83% of fatty acids were shared across the butter and tallow breakfast meals. Thus, of the 46.5 ± 1.4 g fatty acids provided, 38.6 g were identical across test breakfasts. Both butter and tallow test-breakfast meals contained large quantities of PA and oleic acid, but lesser amounts of stearic and linoleic acids. The butter test meal contained more fatty acids ranging from 8:0 to 14:0 (15.4% and 6.6% in butter and tallow meal fat, respectively). PA was a major saturated fatty acid in both butter (28.6%) and tallow (23.5%) breakfast meals. The MA concentration in the meal containing butter (9.7%) exceeded that in the meal containing tallow (5.1%). The fatty acid compositions of test diets were similar to those of corresponding test meals. The butter test meal contained 4.67, 4.31, and 1.76 g/MJ, respectively, of saturated, monounsaturated, and polyunsaturated fat, whereas the butter test diet contained 5.62, 4.26, and 1.76 g/MJ, respectively, whereas the tallow test meal contained 3.86, 4.64, and 1.79 g/MJ, respectively, the tallow test diet contained 4.81, 4.76, and 0.86 g/MJ, respectively, as indicated by computerized nutrient composition analysis.

Similar hourly recoveries of 13CO2 from [1-13]CMA were observed after consumption of both butter and tallow breakfast meals (Figure 1). The most rapid increase in [1-13]CMA recovery occurred from 3 to 6 h postdose. Individual recovery of [1-13]CMA peaked at 3.15 ± 2.31% of the administered dose per hour 6.00 ± 0.37 h after the butter breakfast meal, compared with 2.44 ± 0.15% of the administered dose per hour 5.83 ± 0.31 h after consumption of the tallow breakfast meal. Cumulative recovery rates of 13CO2 from [1-13]CMA over 9 h postdose are shown in Figure 2. Cumulative recoveries of 13CO2 from [1-13]CMA at 4 h postdose were 1.26 ± 0.36% for butter and 1.95 ± 0.38% for tallow meals. There was a steady increase in cumulative recovery from [1-13]CMA 6 h postdose (4.60 ± 0.87% of administered [1-13]CMA for butter and 5.74 ± 0.66% for tallow meals) and 9 h postdose (7.13 ± 1.04% of administered [1-13]CMA for butter and 8.61 ± 0.86% for tallow meals). There was no significant effect of the source of dietary fat on cumulative 13CO2 recovery from [1-13]CMA.

The hourly recovery of 13CO2 from [1-13]CPA was similar in subjects consuming the butter and tallow breakfast meals (Figure 1). For each treatment, individual recovery of 13CO2 from [1-13]CPA proceeded slowly, peaking at 0.83 ± 0.16% of the
administered dose per hour at 5.50 ± 0.67 h in subjects fed the butter meal, and 0.70 ± 0.28% of the administered dose per hour at 6.67 ± 0.62 h in subjects fed the tallow meal. Cumulative recoveries of label from [1-13C]PA over 9 h postdose are shown in Figure 2. Cumulative recovery of [1-13C]PA 4 h postdose (0.97 ± 0.47% of administered [1-13C]PA for butter and 0.60 ± 0.18% for tallow), 6 h postdose (2.08 ± 0.48% of administered [1-13C]PA for butter and 1.53 ± 0.45% for tallow), and 9 h postdose (3.26 ± 0.73% of administered [1-13C]PA for butter and 3.01 ± 0.86% for tallow) did not differ significantly as a function of dietary fat treatment.

Fractional 13CO2 recovery rates from dietary [1-13C]MA were different from those of [1-13C]PA from 4 to 7 h postdose (P < 0.001). Individual peak recovery of dietary [1-13C]MA exceeded that of [1-13C]PA (P < 0.01), although individual peak recovery occurred at a similar time for both dietary [1-13C]fatty acids. Over the 9-h period, the fractional recovery of dietary [1-13C]MA significantly exceeded that of dietary [1-13C]PA for both fat treatments (P < 0.01).

Net oxidation rates of MA and PA within the test breakfast meals are shown in Table 2. Net oxidation of dietary MA from the butter meal (329 ± 45 mg) exceeded that from the tallow meal (212 ± 25 mg) over the 9-h postprandial period (P <
TABLE 2

Net oxidation of breakfast meal fatty acid over 9 h

<table>
<thead>
<tr>
<th></th>
<th>Butter-MA</th>
<th>Tallow-MA</th>
<th>Butter-PA</th>
<th>Tallow-PA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Meal fatty acid (g)</td>
<td>4.6 ± 0.1</td>
<td>2.4 ± 0.1</td>
<td>13.6 ± 0.4</td>
<td>11.2 ± 0.3</td>
</tr>
<tr>
<td>Oxidized meal fatty acid (mg)</td>
<td>329 ± 45</td>
<td>212 ± 25</td>
<td>441 ± 99</td>
<td>348 ± 95</td>
</tr>
</tbody>
</table>

\(^1\) \(±\) SEM. MA, myristic acid; PA, palmitic acid.
\(^2\) Significantly different from tallow-MA, \(P < 0.05\).

0.05). In contrast, net dietary PA oxidation over 9 h was not different between butter (441 ± 99 mg) and tallow (348 ± 95 mg) meals.

DISCUSSION

Despite observations suggesting that labeled MCFAs are more rapidly oxidized to respiratory carbon dioxide than are LCFAs (2–7), there have until recently (8) been no reports on the response of \(^{13}\text{CO}_2\) liberated from ingested tracer-labeled fatty acid in individuals fed a changing dietary fat source. The present study is the first to follow the oxidation of two labeled saturated fatty acids to carbon dioxide in response to feeding diets varying in saturated fatty acid composition. Dietary fatty acids were compared in terms of the percentage oxidized, referred to as fractional or proportional oxidation, as well as the milligrams oxidized, referred to as net oxidation, over the 9-h postprandial period. Clearly, if fractional oxidation of a dietary fatty acid from two different meals is the same, net oxidation will correspond to dietary content. Results show that over a 9-h postprandial period, very similar but small proportions of dietary [\(1\)^\(13\text{C}\)MA (\(≈8\)% and [\(1\)^\(13\text{C}\)PA (\(≈3\)% were recovered as respiratory \(^{13}\text{CO}_2\). This observation occurred independently of the amount of either fatty acid in the test meal and the preceding diet. Although remaining independent of fat treatment, the fractional oxidation rates of MA and PA were markedly different because of the relatively small difference in acyl chain length. The greater net oxidation of MA with butter feeding can be attributed to the higher proportion of MA in the fat. In contrast, the similar net oxidation of PA with butter or tallow feeding can be attributed to dietary PA pool sizes that were similar across dietary treatments. Shifts in the P:S of dietary fat have been shown to alter the utilization of individual \(^{13}\text{C}\)-labeled fatty acids (8); however, the influence of saturated fatty acid chain length has not been investigated.

The present demonstration of acyl chain–dependent differences in the percentage of label liberated in breath carbon dioxide is not inconsistent with results obtained from previous experiments in humans in which a single (5, 6, 15) and multiple (8) diet approach were used. However, although former tracer studies have compared oxidation of various labeled fatty acids by using olive (3, 17), corn (18), or soybean (19) oils, systematic comparisons of saturated fats varying in fatty acid chain length have not been done. Whether a diet higher in MCFAs might increase or decrease the oxidation rate of dietary MA or PA, possibly because of enzyme induction and competition, respectively, or alternatively have no effect on dietary MA or PA oxidation, is unknown. Our finding that fractional oxidation of dietary MA was independent of meal MA content is novel and affords some insight into mechanisms governing the relative partitioning of fats varying in chain length through processes of absorption, transport, and oxidation. The consistent proportion of label from fatty acid appearing in breath carbon dioxide, regardless of diet pool size, suggests independent movement of individual fatty acids through each of these metabolic processes. Thus, if for PA a much larger pool size at some stage of metabolism results in greater retention than oxidation of this fatty acid compared with a shorter-chain fatty acid, then the fraction of uptake of PA into this pool remains fixed, regardless of flux through the pool. However, because the extent of discrimination appears to vary with different dietary fats, changing relative amounts of fat in the diet will produce an overall effect of shifting the net partitioning toward or away from oxidation. The present findings argue that the concentration of rapidly oxidized fatty acids within a given fat source therefore becomes the primary determinant of its net oxidation potential.

Low fractional oxidation rates of dietary fatty acids as seen in the present study have been reported in other experiments (5, 8, 15) using similar time periods. Watkins et al (5) reported that 27%, 11%, and 6.6% of labeled tricosanoic, triolein, and PA, respectively, as components of Lipomul (The Upjohn Co, Kalamazoo, MI) were recovered in the breath of resting, healthy children after 6 h. Cumulative fractional oxidation rates of \(≈8\)% and 50% for [\(1\)^\(13\text{C}\)PA and decanoic acids, respectively, were reported when mixed with different diets (8). Jones et al (15) found that \(≈15\)% and \(≈10\)% of total [\(1\)^\(13\text{C}\)PA and oleic acid, linoleic, and -stearic acids, respectively, shared a meal containing largely saturated fat, were recovered in breath \(^{13}\text{CO}_2\) after 9 h in resting, healthy males. Thus, except for shorter-chain saturated fatty acids (8), the available data indicate that the majority of dietary fatty acids within a meal is directed toward storage relative to the amount consumed over the initial 6–9-h period. In turn, most fat actually oxidized after a meal derives from endogenous fat sources. This high proportion of fat partitioned for deposition is reflected in the ability of the blend of fatty acids consumed to readily influence storage pool fatty acid composition (20–23) and labeled fatty acid accumulation in adipose tissue (2, 24); however, adipose tissue composition is also dependent on other factors such as energy intake (21, 23). Our results suggest that the body does not alter the percentage of dietary MA oxidized in response to dietary content. The body does respond to changing dietary fat source by increasing or decreasing the extent to which desaturation and retroconversion of saturated fatty acids occurs (22). Furthermore, except for selective depletion of linoleate and \(\alpha\)-linolenate during weight cycling, major fatty acids appear to be oxidized in proportion to their concentration in adipose tissue (23). Whether dietary fat composition influences the oxidation of specific endogenous fatty acids beyond that due to altered endogenous availability remains to be determined.

Measurement of dietary fatty acid oxidation based on the evolution of \(^{13}\text{CO}_2\) after consumption of meals containing \(^{13}\text{C}\)-labeled fatty acids depends on several assumptions, involving digestion and absorption of meal fat and clearance of \(^{13}\text{CO}_2\) from tissues of oxidation to expelled breath. Prefeeding is critical because it ensures that preoxidative pools qualitatively and quantitatively reflect the new dietary fat source. Differences in substrate utilization due to dietary fat treatment are detectable in 7 d (25, 26), which allows sufficient turnover of
fatty acids in preoxidative pools. Thus, for the present study, 
\(^{13}\text{C}\)-labeled fatty acid oxidation was determined on day 8 and 
was repeated on day 11 to allow sufficient washout between 
\(^{13}\text{C}\)-labeled fatty acid administration. In the present study, fat 
digestion and absorption were assumed to be similar between 
diet fat treatments. Additionally, it was assumed that tracer and 
tracee absorption would be equivalent, despite the provision of 
tracee in the free fatty acid form. In support of the former 
assumption, Denke and Grundy (27) reported similar fat ab-
sorption efficiencies in 10 healthy males consuming butter or 
tallow liquid-formula diets containing 40% of energy as fat. 
The absorption of PA (97% and 96% for butter and tallow 
treatment, respectively), stearic acid (90% and 94% for butter 
and tallow treatment, respectively), and oleic acid (99% and 
99% for butter and tallow treatment, respectively) did not differ 
as a result of fat treatment, as determined by using 3-d fecal 
collections (27).

Of concern also was the potential for discrepancy between 
digestion and absorption of tracer compared with tracee fatty 
acids in the test meals. \(^{13}\text{C}\)-labeled fatty acids were provided 
in the free form, whereas corresponding unlabeled dietary fatty 
acids were primarily esterified as triacylglycerols. It has been 
suggested that absorption of esterified saturated fatty acids may 
be greater than those in the free form because of the lack of 
monoacylglycerols, which promote fatty acid emulsification 
and micellar solubilization during digestion (28–30). Because 
monoacylglycerols were in ample supply from the almost ex-
clusive consumption of triacylglycerol fatty acids in the test 
breakfast meal, saturated fatty acids in the free and esterified 
form should be equally absorbed in the present study. The fact 
that \(^{13}\text{C}\)-triacylglycerols produce a similar cumulative oxidative 
response when administered either parenterally or orally 
(6) and the previously reported similarity in absorption of free 
compared with esterified fatty acids (30) suggest comparable 
absorption efficiency of tracer and tracee in the present study.

The question of whether labeled fatty acids should be ad-
ministered via constant infusion or pulse-chase approaches to 
measure fatty acid oxidation has also been disputed. In recent 
years, assumptions inherent in the constant infusion technique 
regarding the attainment of isotopic equilibrium within preoxi-
dative fatty acid pools have been criticized (31). Estimation of 
intracellular premitochondrial fatty acid kinetics within organs 
such as heart, muscle, or liver in humans may be in error 
because isotopic equilibrium must be indirectly determined 
through the measurement of plasma substrate enrichment. Be-
cause a significant portion of oxidized fat appears to be derived 
from intracellular lipids, possibly not in equilibrium with cor-
responding plasma pools (31–33), constant infusion ap-
proaches to measurement of fat oxidation may have limited use 
under certain circumstances. The noncompartmental analysis 
of respiratory \(^{13}\text{CO}_2\) evolution after a nonpruned oral bolus of 
\(^{13}\text{C}\) fatty acids in the present study represents a stochastic 
approach in which \(^{13}\text{C}\) input and output are monitored without 
the complex analyses of whole-body fatty acid metabolism. 
Although the method used in this study is free of potentially 
incorrect assumptions concerning isotopic equilibrium and 
dependent fatty acid kinetics, other factors may limit the 
effectiveness of its approach. Notably, our method provides no 
insight into endogenous fatty acid pool size, distribution and 
turnover, or the relative amount of endogenous or exogenous 
fatty acid oxidized. However, regardless of the stage of me-
tabolism at which labeled fatty acid is retained in vivo, the 9-h 
fractional oxidation rate provides a useful indicator of fatty 
acid partitioning at the whole-body level.

In conclusion, we report that in healthy, normally fed humans, 
postprandial oxidation of dietary saturated fatty acids is markedly 
influenced by fatty acid chain length. Further, fractional oxidation 
of dietary fatty acids is constant and independent of the mixture 
of dietary fatty acids consumed. Lastly, the higher net oxidation 
of dietary MA occurring with butter consumption appears to result 
from a larger diet pool size of MA in butter. It is suggested that for 
mixed meals differing only in fat composition, the concentration 
of rapidly oxidized compared with low-oxidized dietary fatty 
acids is a primary determinant of the net contribution of each to 
total fat oxidation.

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REFERENCES

Overfeeding with medium-chain triglyceride diet results in diminished 

2. Johnson RC, Young SK, Cotter R, Lin L, Rowe WB. Medium-chain 
triglyceride lipid emulsion: metabolism and tissue distribution. Am J 

3. Leyton J, Drury PJ, Crawford MA. Differential oxidation of saturated 
93.

Metabolic and hormonal responses to lipid and carbohydrate diets during exercise 

JA. Diagnosis and differentiation of fat malabsorption in children using 
\(^{13}\text{C}\)-labeled lipids: triocitoin, troitein, and palmitic acid breath 

6. Metges CC, Wolfarm G. Medium- and long-chain triglycerides labeled with 
\(^{13}\text{C}\): a comparison of oxidation after oral or parenteral adminis-

7. Paust H, Keles T, Park W, Knoblich G. Fatty acid metabolism in 
Stable isotopes in paediatric nutritional and metabolic research. 

8. Cladnin MT, Wang LCH, Rajotte RV, French MA, Goh YK, Kielo 
ES. Increasing the dietary polyunsaturated fat content alters whole-

Washington, DC: Carnegie Institute, 1911. (Publication no. 279.)

10. Bell L, Jones PJH, Telch J, Cladnin MT, Pencharz PB. Prediction of 

11. Klein PD. Nutrition applications of \(^{13}\text{C}\): strategic considerations. In: 
Whitehead RG, Prentice A, eds. New techniques in nutrition research. 

12. Phang PT, Rich T, Ronco J. A validation and comparison study of two 

13. Folch J, Lees M, Sloane SGH. A simple method for the isolation 
and purification of total lipids from animal tissues. J Biol Chem 1957:226: 
497-509.

Faucon G. Assay of lipids in dog myocardium using capillary gas 
chromatography and derivatization with boron trifluoride and metha-

15. Jones PJH, Pencharz PB, Cladnin MT. Whole body oxidation of 
dietary fatty acids: implications for energy utilization. Am J Clin Nutr 