Regional uptake of meal fatty acids in humans

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Regional uptake of meal fatty acids in humans. Am J Physiol Endocrinol Metab 285: E1282–E1288, 2003. First published August 12, 2003; 10.1152/ajpendo.00220.2003.—Two protocols were performed to study meal fatty acid metabolism. In protocol 1, 14 patients scheduled for elective intra-abdominal surgery (11 undergoing bariatric surgery for severe obesity) consumed a meal containing [3H]triolein in the evening before surgery. This allowed us to measure adipose tissue lipid specific activity (SA) in mesenteric and omental, deep and superficial abdominal subcutaneous adipose tissue. Intra-abdominal adipose tissue lipid SA was greater than subcutaneous lipid SA. There were no significant differences between mesenteric and omental or between deep and superficial abdominal subcutaneous adipose tissue. Intra-abdominal adipose tissue lipid SA was greater than subcutaneous lipid SA. There were no significant differences between mesenteric and omental or between deep and superficial abdominal subcutaneous adipose tissue. In protocol 2, meal fatty acid oxidation and uptake into subcutaneous and omental adipose tissue ([3H]triolein) were measured in six normal, healthy volunteers. Meal fatty acid oxidation (3H2O generation) plus that remaining in plasma (~1%) plus uptake into upper body subcutaneous, lower body subcutaneous, and visceral fat allowed us to account for 98 ± 6% of meal fatty acids 24 h after meal ingestion. We conclude that omental fat is a good surrogate for visceral fat and that abdominal subcutaneous fat depots are comparable with regard to meal fatty acid metabolic studies. Using [3H]triolein, we were able to account for virtually 100% of meal fatty acids 24 h after meal ingestion. These results support the meal fatty acid tracer model as a way to study the metabolic fate of dietary fat.

[3H]triolein; body composition; adipose tissue; obesity

BODY FAT DISTRIBUTION is an important predictor of the metabolic complications of obesity (1, 13). A male-type or central fat distribution is associated with a greater risk of insulin resistance, dyslipidemia, hypertension, and diabetes mellitus, whereas a female fat distribution is associated with a lesser risk. Although a number of hormonal (2) and environmental (19) correlates of body fat distribution are known, there is less information regarding the basic, underlying mechanisms by which differences in body fat distribution develop in humans.

In principle, variations in body fat accumulation must be due to regional differences in the storage or release of fatty acids. Regional differences in adipose tissue lipolysis, or free fatty acid (FFA) release, between men and women could potentially account for the differences in body fat distribution. We tested the hypothesis that FFA release from leg adipose tissue is relatively reduced in women compared with men. Because men and women did not materially differ with respect to regional lipolysis under basal (9), meal (8), or exercise (4) conditions, we concluded that differences in fatty acid uptake would be the next logical explanation and began studies of adipose tissue meal fatty acid uptake (17, 18). The approach developed by Björntorp et al. (3) and Márin et al. (15) was used as a starting point as a means to test for sex-based differences in regional adipose tissue uptake of meal fatty acids. In addition to incorporating a radiolabeled fatty acid triglyceride tracer into a meal and performing adipose tissue biopsies 24 h after meal ingestion, we measured meal fatty acid oxidation by the generation of 3H2O from a [3H]triolein tracer (17, 18). The 24-h interval was chosen because meal absorption is complete and the clearance of meal fatty acids from the circulation is virtually complete. This approach allowed us to assess the fraction of meal fatty acids oxidized and stored in subcutaneous fat.

Despite assessing both oxidation and uptake into subcutaneous fat, we could not account for all meal fatty acids (18). Of interest, there was a positive correlation between visceral fat mass and the “unaccounted for” meal fatty acids (18), suggesting that significant portions of meal fatty acids are stored in visceral (omentale and mesenteric) fat. If the unaccounted for meal fatty acids are stored in visceral fat, it may be possible to understand the role of visceral adipose tissue in clearing dietary fat by measuring unaccounted for meal fatty acids. Correlation does not prove causation, however. For example, deep abdominal subcutaneous fat is thought to be different from superficial abdominal subcutaneous fat; the former is better correlated with both insulin resistance and visceral adiposity (12). The unaccounted for meal fatty acids we observed (18) could have been stored in deep subcutaneous fat (although the biopsy technique we use most
likely aspirates both deep and superficial adipose tissue in lean individuals). We also considered the possibility that mesenteric and omental adipose tissue may take up meal fatty acids differently. If so, this would have implications for sampling visceral fat, given the greater difficulty in sampling both depots as opposed to just omental fat.

In the process of refining methods to assess regional meal fatty acid uptake, we undertook these two additional studies. These experiments were designed to address three questions with respect to meal fatty acid metabolism. 1) Because omental fat is more accessible to biopsy than mesenteric fat, is omental fat a reasonable marker for visceral meal fatty acid uptake? 2) Are there systematic differences in the uptake of meal fatty acids between deep and superficial abdominal subcutaneous adipose tissue? 3) If visceral meal fatty acid uptake can be assessed, will inclusion of this depot allow us to account for all meal fatty acids 24 h after the ingestion of a labeled meal?

METHODS AND MATERIALS

Subjects

No subjects or data in this report are from previously published work. For protocol 1, written, informed consent was obtained from 14 patients (4 men and 10 women) scheduled for elective intra-abdominal surgery. Eleven of the patients were undergoing bariatric surgery for severe obesity and three were undergoing other elective surgical procedures. None of the three nonbariatric surgery patients was acutely or chronically ill, was taking medications that could alter fatty acid metabolism, or had lost or gained body weight before surgery. Seven of the bariatric surgery patients had type 2 diabetes (average fasting plasma glucose 9.3 mmol/l). No patients were taking thiazolidinediones. For protocol 2, written, informed consent was obtained from six volunteers who participated in a more comprehensive study of regional meal fatty acid uptake. All of the latter volunteers were weight stable (±1 kg/wk variation) for ≥2 mo before the study.

Materials

[9,10-3H]triolein (DuPont-NEN Research Products) was sonicated into a liquid meal.

Assays and Methods

Adipose tissue and meal lipids were extracted using standard (7) procedures, and the triglyceride (TG) specific activity (SA) was measured as previously described (18). In protocol 2, O2 consumption and CO2 expiration were measured by indirect calorimetry by use of a DeltaTrac Metabolic Cart (SensorMedics, Yorba Linda, CA). Plasma glucose concentrations were measured with a Beckman glucose analyzer (Beckman Instruments, Fullerton, CA). Plasma insulin concentrations were measured using a ch Emiliuminescence method with the Access Ultrasensitive Immunoenzymatic assay systems (Beckman, Chaska, MN). To separate chylomicrons, 1.25 ml of 1.006 g/ml density solution was placed into open-top ultracentrifuge tubes under which 750 µl of plasma were carefully pipetted. This was further topped up with an additional 0.45 ml of the same solution. The tube was placed in a Beckman (Beckman Instruments, Spino Divi-

sion, Palo Alto, CA) ultracentrifuge at 30,000 rpm for 20 min using a 50.3 Ti rotor. The chylomicron fraction was removed by slicing the top of the tube with a tube slicer and aspirating the chylomicron layer into a separate glass tube with a Pasteur pipette. The tube slicer and pipette were rinsed with a small amount of the density solution and added to the glass tube. The TG concentrations were measured on a small single-sliced computed tomography (CT) at the L2–3 level (10). Total body water was measured with 2H2O (20). The triolein tracer was assayed for radiochemical purity by measuring the radioactivity in the TG and the non-TG fractions by HPLC (17). Briefly, the FFA (if any) and TG fractions were isolated using a modified (8) HPLC (5) procedure. The TG fraction was hydrolyzed, and the component fatty acids were converted to their phenylacetyl derivative (16) and analyzed by HPLC again (16) to determine isotopic purity. Any FFA collected from the initial isolation procedure were directly derivatized and analyzed by HPLC (16).

Protocols

Protocol 1. Protocol 1 included participants scheduled for elective intra-abdominal surgery. After signing informed consent, these volunteers were given an aliquot (50 ml) of Ensure Plus (Ross Laboratories) containing 57% of calories as carbohydrate, 27% as fat (16% saturated, 27% monounsaturated, and 57% polyunsaturated), and 15% as protein into which ~50 µCi of [3H]triolein had been sonicated. Volunteers were given instructions to consume this with their evening meal between 6 and 7 PM on the day before surgery. None of the volunteers changed their usual eating habits before surgery. At the time of surgery the next morning, biopsies from mesenteric fat, omental fat, and subcutaneous fat were obtained. For the last 11 participants in the study, biopsies were taken from two sites, superficial and deep, in the abdominal subcutaneous adipose tissue bed. The subcutaneous fat biopsies were taken from the incision site used to perform the surgical procedures in the upper portion of the abdomen (cephalad to the umbilicus). The superficial subcutaneous biopsy was taken ~1 cm below the skin and the deep subcutaneous biopsy was taken below Scarpa’s fascia ~1 cm from the fascia covering the abdominal muscles.

Protocol 2. Protocol 2 was modeled on a previously described study (18) to determine the fate of meal fatty acids. Three men and three women underwent the same dietary preparation and testing as previously described (18), with the exception that 133Xe measurements of adipose tissue blood flow were not performed, and only baseline indirect calorimetry measurements were obtained. Briefly, the diet was weight maintaining and provided 15% of calories as protein, 35% as fat, and 50% as carbohydrate (25% as simple carbohydrate and 25% as complex carbohydrate). The indirect calorimetry was performed after an overnight stay in the Mayo Clinic General Clinical Research Center, with the volunteers lying quietly in bed, and before the test meal. On the basis of the indirect calorimetry results, the volunteers were given a meal that provided 40% of resting energy expenditure. The meal consisted of Ensure Plus to which ~50 µCi of [3H]triolein had been added. The volunteers were also
provided solid food meals at 1300 and 1800 that provided the remainder of their usual daily energy intake and contained the same portions of nutrients as the diets provided during the week before the study. After volunteers consumed the meal, the test meal blood samples were obtained every 2 h for 12 h and every 4 h until the next morning. Urine was collected for 24 h after the test meal to assess $^{3}$H$_2$O losses and concentration. The volunteers were not confined to bed between blood samples, because measures of adipose tissue blood flow were not being conducted.

Twenty-four hours after the test meal consumption, subcutaneous adipose tissue biopsies from the abdominal (just lateral to the umbilicus), gluteal, and midthigh (anterior-lateral) regions were obtained using sterile technique with subjects under local anesthesia (17). The volunteers were then transferred to an operating room where they were given conscious sedation. A laparoscopy was performed, and a small sample (~1 g) of omental adipose tissue was obtained. Briefly, a 5-mm laparoscope and two additional 5-mm trocars were placed into the abdomen through subumbilical and suprapubic incisions, respectively. The distal tip of the omentum was grasped under direct visualization and then ligated with two separate 0-chromic endoloops before transection with endoscissors. The omental biopsy specimen was removed from the abdomen and immediately placed into a solution of ice-cold sterile saline. After recovery from the sedation, the participants were observed until they could be dismissed from the hospital. No subjects were allowed to drive on the day of the procedure per the Mayo Clinic Conscious Sedation policy. Two volunteers were able to return to work on the same day as the laparoscopy, and the other four returned to work on the following day. With the exception of mild symptoms of peritoneal irritation, there were no adverse events.

For protocol 1, the exact amount of $[^3]$H-triolein consumed was determined by assaying quadruplicate 50-µl samples of meal by use of liquid scintillation counting. The meal was weighed to the nearest milligram. Aliquots of the meal were also saved for measurement of meal lipid $[^3]$H SA. The lipid was extracted from adipose tissue, accurately weighed, and counted on a scintillation counter to <2% counting error. The adipose tissue ratio of TG $[^3]$H to dpm/mg lipid was calculated for each site.

Calculations and Statistical Analysis

To extrapolate from the amount of tracer in different adipose tissue depots to the fraction of meal uptake in adipose tissue, we divide total body adipose tissue into upper body subcutaneous, lower body subcutaneous, and intra-abdominal fat (18). The combination of CT- and DEXA-derived abdominal fat provides a measure of visceral fat (10). Upper body fat (DEXA) minus visceral fat is used to derive upper body subcutaneous fat mass. Lower body subcutaneous fat is measured with DEXA alone. The tracer concentration (dpm/g adipose tissue lipid) is multiplied by the depot-specific adipose tissue lipid content to derive disintegrations per minute per depot. The quantity of tracer in a depot is divided by the quantity of tracer administered to calculate the fractional storage of tracer in the depot. Because the half-life of fatty acids in adipose tissue depots is 6–18 mo (15), and because the adipose TG pool is well mixed (there is no first-in, first-out phenomenon), this calculation provides a good measure of adipose tissue fatty acid uptake. A tissue bed with a higher lipid SA may still account for a lesser fraction of meal fatty acid uptake, if it is a smaller depot (e.g., visceral fat).

All data are presented as means ± SE unless otherwise stated. A repeated-measure ANOVA was performed to determine whether there were differences in $^{3}$H lipid SA between superficial and deep abdominal subcutaneous fat and between mesenteric and omental fat. If significant differences were present, a paired t-test was used for direct between-depot comparisons.

RESULTS

Subject Characteristics

The volunteers participating in protocol 1 were 52 ± 3 yr old with a height of 169 ± 2 cm, weight of 131 ± 11 kg, and body mass index of 45.9 ± 3.8 kg/m². The average fasting glucose of these volunteers was 7.6 ± 0.6 mmol/l, fasting serum TGs were 221 ± 37 mg/dl, and cholesterol was 203 ± 37 mg/dl. The majority of these volunteers were undergoing elective Roux-en-Y gastric bypass for treatment of severe obesity. The subject characteristics of the volunteers participating in protocol 2 are provided in Table 1. The mean (±SD) age of the volunteers in protocol 2 was 30 ± 3 yr, and their resting energy expenditure was 1,682 ± 54 kcal/day.

Protocol 1

The individual SA values of adipose tissue lipid for the subjects participating in protocol 1 are provided in Table 2. There were significant differences in the lipid SA values among the four tissue beds by repeated-measures ANOVA. By paired t-test, the adipose tissue lipid SA was not significantly different between mesenteric and omental adipose tissue ($P = 0.40$). Likewise, the adipose tissue lipid SA values of superficial subcutaneous and deep subcutaneous adipose tissue were not significantly different (403 ± 188 vs. 460 ± 182 dpm/g, respectively, $P = 0.17$). SA values of both intra-abdominal depots were significantly ($P < 0.05$) greater than those of both subcutaneous depots. To assess the degree of disagreement between the two visceral fat depots, the mesenteric lipid SA was subtracted from the mean of the omental and mesenteric lipid SA for each individual and expressed as a percentage. The SA of mesenteric fat lipid differed from the mean visceral fat lipid SA by an average of 15 ± 9%. The same approach was taken to examine differences between the two subcutaneous sites; the average superficial subcutaneous adipose tissue lipid SA was

<table>
<thead>
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<th>Table 1. Characteristics of subjects in protocol 2</th>
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<tr>
<td>Mean ± SD</td>
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<tr>
<td>Height, cm</td>
</tr>
<tr>
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<tr>
<td>Leg fat, kg</td>
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<tr>
<td>Upper body subcutaneous fat, kg</td>
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There were 6 subjects in protocol 2. BMI, body mass index.
From these analyses, we concluded that omental adipose tissue SA provides a good representation of visceral uptake of fat meal fatty acids, and that either superficial or deep subcutaneous adipose tissue is representative of the average subcutaneous uptake of meal fatty acids.

Protocol 2

Glucose, insulin, and TG responses. Figure 1 depicts the plasma glucose (top) and insulin (bottom) concentrations during the 24-h study period after the ingestion of the labeled meal. Figure 2 shows the SA (top) and concentration (bottom) of plasma chylomicron and nonchylomicron TGs from the volunteers in protocol 2. One of the men had fasting hypertriglyceridemia (5 mmol/l), which increased the average nonchylomicron TG concentration for the group.

Fate of meal fatty acids. As assessed by the generation of $\text{H}_2\text{O}$ from the $[^3\text{H}]\text{triolein}$ contained in the meal, 49.8% of meal fatty acids were oxidized in the 24 h after meal ingestion.

The individual SA values of adipose tissue lipid for the subjects participating in protocol 2 are provided in Table 3. The adipose tissue lipid SA was uniformly greater ($P = 0.007$) in omental than in abdominal subcutaneous fat, which was in turn greater ($P = 0.13$) than in thigh fat. The omental adipose tissue lipid SA was an average of 208 ± 56% greater than that of abdominal subcutaneous fat. The adipose tissue lipid SA of abdominal subcutaneous fat was 153 ± 46% greater than that found in leg fat. Intra-abdominal fat, although accounting for only 11 ± 7% of total body fat, took up 17 ± 8% of meal fatty acids that were not oxidized. Lower body adipose tissue accounted for 26 ± 11% of body fat and took up 16 ± 11% of meal fatty acids that were not oxidized. Upper body subcutaneous

<table>
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<tr>
<th>Subject no.</th>
<th>Omental</th>
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<th>Subcutaneous</th>
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<tr>
<td>1</td>
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<td>6</td>
<td>16,033</td>
<td>8,370</td>
<td>4,660</td>
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</table>

Mean ± SD 8,256 ± 4,338, 4,141 ± 2,409, 2,974 ± 1,827

SA values are in dpm/g of adipose tissue lipid.

![Graph](image1.png)  
**Fig. 1.** Plasma glucose concentrations (top) and insulin concentrations (bottom) for the 6 volunteers participating in protocol 2 for 24 h. The test meal was administered at time 0, and standard meals were administered at 5 and 10 h.

![Graph](image2.png)  
**Fig. 2.** Specific activity (top) and log concentrations (bottom) of chylomicron and nonchylomicron triglycerides for the 6 volunteers participating in protocol 2. All of the $[^3\text{H}]\text{triolein}$ was contained within the breakfast meal consumed at time 0.
adipose tissue accounted for 63 ± 15% of body fat and took up 57 ± 30% of meal fatty acids that were not oxidized.

There remained some 3H-labeled fatty acids in plasma TGs on the following morning (an average of 350 dpm/ml of plasma). Using an estimate of plasma volume as 5% of body weight, we calculated that 0.9% of meal fatty acids remained in plasma 24 h after the labeled meal. We were able to account for 98 ± 6% of meal fatty acid disposal 24 h after ingestion of the labeled meal by summing the fatty acids oxidized, those remaining in plasma, and those stored in visceral, lower-body, and upper-body subcutaneous adipose tissue. The percentages of meal fatty acids oxidized, stored in the three fat depots, remaining in plasma, and unaccounted for are depicted in Fig. 3.

### DISCUSSION

The study designated protocol 1 was designed to determine whether meal fatty acid uptake is similar in the two visceral depots, omental and mesenteric, and in the two abdominal subcutaneous depots, deep and superficial. Patients undergoing elective intra-abdominal surgery consumed a meal supplement containing [3H]triolein on the day before surgery to allow between-depot comparisons. We found that omental fat provides good information regarding visceral fat and that superficial abdominal subcutaneous fat provides equally good information about abdominal subcutaneous fat in general. Although there were major differences in the uptake of meal fatty acids between visceral and subcutaneous depots, there were not major differences between the two sites within each depot. With these results in hand, protocol 2 was designed to measure the fate of meal fatty acids in healthy volunteers, including the uptake into visceral (omentum) fat. The more comprehensive study of meal fatty acid metabolism included an omental fat biopsy performed with laparoscopic techniques. Taking into account the uptake of meal fatty acids into visceral fat allowed us to identify the fate of virtually 100% of dietary fat after 24 h.

In protocol 1, we examined whether omental adipose tissue reflects meal fatty acid uptake into visceral (mesenteric + omental) fat. Collecting adequate amounts of omental fat during laparoscopic procedures is relatively easy, whereas similar amounts of mesenteric fat would be more difficult and hazardous to collect unless the laparoscopy involved an intestinal resection. Collecting mesenteric fat could potentially injure the blood vessels supplying that segment of bowel from which the sample was obtained. In our study, a small piece of jejunum with its mesentery is of necessity removed during the Roux-en-Y gastric bypass, eliminating the risk of injury for these patients. Fortunately, our results suggest that, for studies of meal fatty acid uptake, omental and mesenteric fat are sufficiently similar that omental fat can be used.

Protocol 1 also allowed us to compare whether differences in meal fatty acid uptake were present between deep and superficial abdominal subcutaneous fat. We observed an average 15% difference between the lipid SA values of superficial subcutaneous adipose and average subcutaneous adipose tissue in protocol 1. These variations are small compared with the differences between intra-abdominal fat, abdominal subcutaneous fat, and lower body fat that we found in the subjects participating in protocol 2. There appear to be no major metabolic differences in superficial vs. deep subcutaneous abdominal fat with respect to uptake or release of fatty acids; at steady state, the release of

**Fig. 3.** Disposal of meal fatty acids (means ± SD) 24 h after ingestion of [3H]triolein for the 6 volunteers participating in protocol 2. LB Sq and UB Sq, lower body and upper body subcutaneous, respectively. Values were determined as described in Calculations and Statistical Analysis.

**Fig. 4.** Adipose tissue lipid specific activity (SA, dpm/g) in mesenteric vs. omental fat for volunteers participating in protocol 1. ▲, obese nondiabetic volunteers; ■, obese type 2 diabetic volunteers; ○, nonobese volunteers.
fatty acids from these tissue beds will equal their uptake.

To our knowledge, this is the first study to use the approach of laparoscopy to collect omental fat in normal healthy volunteers for research purposes. The laparoscopic technique employed is routinely used for various minor gynecological procedures and has an excellent record of safety. In this regard, laparoscopy was performed, with subjects under conscious sedation, by well-trained clinicians in fully equipped surgical facilities, making the procedure safe with a major complication rate (defined as any event in which either laparotomy or relaparotomy was required) of <4/1,000 for clinical procedures involving instrumentation of visceral structures (21). Previous studies that examined the fate of meal fatty acids have collected intra-abdominal fat from individuals scheduled for elective surgery (14). Although this is a useful and necessary approach, the underlying medical condition for which the patient is undergoing surgery could influence the results of the study. The volunteers in protocol 2 were not undergoing the procedure for a known medical condition, and thus we can be confident that the results of this study represent truly normal metabolic function.

In protocol 2, we found that almost 50% of dietary fatty acids were oxidized in the 24 h after the meal. In a previous study (18), only −30% of dietary fatty acids were oxidized. The difference between that study (18) and protocol 2 may be related to the level of physical activity allowed. The volunteers in the current protocol 2 did not have measures of adipose tissue blood flow (which requires bed rest) and thus were ambulatory between blood samples. This difference in activity may be important, because physical activity increases meal fatty acid oxidation rates (22).

Some limitations of these studies must be acknowledged. We included only six volunteers in protocol 2, which was approved primarily as a feasibility study to use laparoscopy to collect intra-abdominal fat. Although we were able to account for essentially all meal fatty acids after 24 h, it is possible that with a larger number of volunteers we might have detected small amounts of unaccounted for meal fatty acids. Muscle and liver would appear to be the most likely potential sites if some exist. Another limitation is that most of the volunteers participating in protocol 1 were undergoing bariatric surgery. An advantage of working with this latter population is that it is easy to define the deep and superficial abdominal subcutaneous adipose tissue beds. We might have obtained different results if less severely obese individuals had been studied; however, the three volunteers not undergoing bariatric surgery did not differ from the 11 who were. We plotted the omental vs. mesenteric adipose lipid SA for these volunteers to look for obvious between-group differences. Although there were not large numbers in the different groups, the results from lean and obese, diabetic and nondiabetic volunteers do not appear to differ (Fig. 4).

When sampling abdominal subcutaneous adipose tissue in our ongoing studies, we use a 14-gauge needle attached to suction to aspirate fat that is pinched up. This approach seems likely to aspirate both deep and superficial adipose tissue except in severely obese persons. In contrast, blunt-tipped liposuction needles may less readily aspirate the deep abdominal subcutaneous adipose tissue if the fascial plane is resilient.

Our experience with laparoscopy in the six volunteers was positive from the volunteer acceptance perspective, and no volunteer had an unexpected adverse event. Most volunteers noted symptoms of mild peritoneal irritation for 1–2 days after the procedure; however, acetaminophen provided adequate analgesia. The skill of a highly trained laparoscopist and anesthesia staff was key to the acceptability of the procedure. One of the volunteers had previously participated in hepatic vein catheterization and muscle biopsy studies; that individual reported that the laparoscopy with conscious sedation was less uncomfortable than those procedures. That said, laparoscopy is expensive, and the potential for serious adverse events is equivalent to that of other invasive procedures, such as hepatic vein catheterization. This approach should not be taken without careful scrutiny of the risk-to-benefit ratio and meticulous attention to informed consent.

In summary, omental adipose tissue is a good representation of meal fatty acid uptake into visceral fat, and deep and superficial abdominal subcutaneous fat depots do not differ substantially with respect to their uptake of meal fatty acids. Direct measures of meal fatty acid uptake into visceral fat allowed us to account for virtually 100% of meal fatty acid disposal 24 h after the ingestion of a labeled meal. This observation suggests that the unaccounted for meal fatty acids we observed in a previous study (18) were stored in visceral fat, but additional studies will be needed to confirm this hypothesis.

We acknowledge the technical assistance of Carol Siverling, the support of the staff of the Mayo Clinic General Clinical Research Center, and the editorial assistance of Monica Davis.

DISCLOSURES

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