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Subject and diet details

The procedures followed for this study were in accordance with the ethical standards of the institutional committees associated with the University of Vermont General Clinical Research Center, which approved the study, and in accordance with the Helsinki Declaration of 1975 as revised in 1983. The first volunteer began this study on August 30, 2007; therefore, the study was not registered as a clinical trial. The two cohorts, reported here, do not include a few subjects who did not complete the protocol or whose data were excluded (prior to breaking the diet code). Three female subjects did not complete the study because of family issues, and one female subject withdrew after the first experimental diet because of pain during a biopsy procedure. One male subject dropped out on day 3 of the control diet because of distaste for the food. The data of one male volunteer were not included because there was no evidence of insulin having been administered during one of the intravenous glucose tolerance tests.

As explained in the section on biostatistics below, the study was powered to determine effect of the diets on insulin sensitivity in 9 men and 9 women. However, as data emerged concerning this effect, we wanted to explore additional biological mechanisms, but we could not do this with the original cohort because there was insufficient muscle tissue left after extensive metabolomic analyses (and transcriptomics not described here). This was anticipated, and 10 additional subjects participated in the protocol.

As previously described (1), the foods, including chicken and turkey (the only sources of meat) were all very low in fat. Thus, fatty acids were mainly provided by vegetable oil blends appropriate to each diet (Natural Oils International, Inc., Simi Valley, California). The HPA and HOA diets otherwise contained the exact same foods with a three-day rotating menu. These oils, at room temperature, were mixed with food that had been warmed; thus, these oils were not used for cooking as in some other studies (e.g. (2)). The oil blend for the control diet consisted of palm oil - 36.9%, high Oleic Sunflower oil - 19.3%, and hazelnut oil - 43.8%. The HPA oil blend consisted of palm oil – 89%, peanut oil - 6.75%, and virgin olive oil - 4.25%, and the HOA "blend" consisted only of hazelnut oil. Except for the virgin olive oil, used only in the HPA diet, all natural oils were first extracted (e.g. centrifugation) and then refined (alkaline refining: crude oil treated with alkali to separate impurities from the triglycerides; filtering to remove impurities such as seed fragments). Thus, although we don't have specific analysis of compounds likely to have antioxidant effects, these oils, particularly the palm oil, may not have the same effect as less refined oils (3). The HOA and HPA diets had identical, low glycemic loads (10.7, average of the three days of menus)(4,5).

Subjects ate breakfast in the GCRC on Sunday – Friday, but most subjects chose to eat their two remaining meals each day at home. Each meal was packaged and ready to be reheated, using either an oven or microwave. Since the foods themselves were practically devoid of fat, the subjects also were given containers of oil to add to each meal, after it had been reheated. The subjects also were given instructions regarding convenient ways to add the oils to various food items on the menu. Each day, subjects completed and signed a questionnaire attesting to their having eaten all the food (and food oil) and to not having consumed any food or drink, except water, not on the menu. On Sunday, the volunteers completed questionnaires pertaining to Saturday as well as Friday. All food and oil containers were inspected each day to be sure all food and oil was consumed. Subjects were given instructions to use spatulas provided to help scrape all oil from its container but ultimately were instructed to lick the oil container to finally empty it. Occasionally, there was evidence of incomplete food/oil consumption or a subject would admit to ingesting, usually inadvertently, a cookie or mint etc. However, any consistent non-compliance was grounds for removal from this study. Fortunately, only one subject left the study on day 3 because of a dislike of fat-free cottage cheese. Because completely following the diet was a dichotomous issue and because the subjects were queried each day about this, we did not employ a detailed diet history during the study. Since the food was provided to the subjects, ready to eat except for reheating the non-fat component and since the food and oil containers were returned for daily inspection, there was no need to utilize photography, for example, to show what food was offered to the subject or was not eaten. For the original, n = 18, cohort, the average number of days when food was returned during the HPA and HOA diets was 1.33 and 1.67, and the average daily consumption of the oil for the HPA and HOA diets as a percentage of total oil administered (127.8 and 127.6 g/d) was 99.9% and 99.2%. For the n = 10 cohort used for molecular and ceramide studies in muscle,

the comparable data for the number of days when food was returned for the HPA and HOA diets was 0.8 and 1.4, and the analogous fractions of oil consumed was 99.4% and 100.00%.

Fasting fatty acid composition and concentration of skeletal muscle diacylglycerol (DAG), triacylglycerol (TAG) and phospholipids

Muscle phospholipids were extracted and applied to TLC plates as described above, but using internal standards consisting of 15:0 phosphatidylcholine, 17:0 phosphatidylethanolamine, and 17:1 cardiolipin (all 1 mg/ml in chloroform). Phospholipid classes were separated using a solvent system of chloroform:ethanol:triethylamine:water (30:34:30:8, by vol) for the first development and hexane:diethyl ether (50:50, v/v) for the second development (6). Phospholipid samples were then transmethylated and analyzed by GC as described above. We used external standards to identify individual phospholipids: phosphatidylcholine, phosphatidylethanolamine, cardiolipin. For serum phospholipids, serum (300 µl) was extracted overnight and analyzed as above.

Bio-Plex analysis of signaling cascades active in muscle

Specimens of muscle tissue (5-10 mg), flash-frozen and stored at -70ºC, were thawed and rinsed in Bio-Rad Phosphoprotein Detection cell wash buffer supplemented with Factor 1, Factor 2, and PMSF. Tissue was transferred into a 2 ml glass tissue grinder into which was added 500µl of lysing solution per 10 mg tissue. The tissue was disrupted using 20 strokes on ice, placed into cryovials, and frozen at -70ºC for 30 min. Samples were thawed and briefly sonicated on ice with a HeatSystems Microson Ultrasonic Cell Disruptor (knob set at 14) for 18 pulses. Samples were transferred to Eppendorf tubes and centrifuged at 4,500 g for 4 min. The supernatant was collected, transferred to a new tube, centrifuged again at 4,500 g (7000 rpm) for 4 min, and 200-400 µl of supernatant was transferred to a new tube. Protein concentration was determined in the lysate using the Bio-Rad DC Protein Assay Kit and was normalized to 250 µg/ml. The same volume of assay buffer as lysate was added to each of the samples and mixed. The Bio-Plex total target assay was used to quantify the abundance of the target protein in one well, while the Bio-Plex phosphoprotein assay was used to quantify the level of target-specific protein phosphorylation in a separate well. Samples, positive control lysates, and negative control lysates were incubated overnight with beads coupled with antibodies recognizing total IκBα, total JNK, total Akt, phospho-NF-κB (Ser536), phospho- IκBα (Ser32/Ser36), phospho-JNK (Thr183/Tyr185), phospho-Akt (Ser473), and phospho-IRS-1 (Ser636/Ser639) in a 96-well filter plate. After washing using a Bio-Plex Pro II wash station, biotinylated detection antibodies were added to the appropriate wells for 30 minutes. After an additional wash, streptavidin-PE was added to all wells for 10 minutes, the wells were washed, and the beads were resuspended in sheath fluid. Data were acquired using the Bio-Plex suspension array system and Bio-Plex Manager 6.0 software. Depending upon commercially-available reagents, lysate input was normalized to total protein content or phospho/total protein levels were reported.

Serum concentration (pg/ml) of Interleukin-6 (IL-6), Interleukin-10 (IL-10), Tumor Necrosis Factor-□ (TNF-□), and ferritin

Briefly, 50 µl of coupled beads were added to each well of a pre-wet 96-well microtiter plate, washed twice using a Bio-Rad (Hercules, CA) Bio-Plex Pro II wash station with the magnetic plate carrier, and 100 µl of 1:4-diluted serum, standard, or assay buffer (background) was added to each well followed by 25 µl of conjugated magnetic beads. The plates were covered, shaken vigorously for 1 minute on an IKA (Wilmington, NC) MTS 2/4 digital microtiter plate shaker and then moderately shaken for 4 hours at room temperature. After washing, 25 µl of biotinylated detection antibodies were added to each well for 1 hour with shaking followed by addition of 50 µl of streptavidin-PE to all wells for 30 minutes with shaking. The wells were washed and the beads were resuspended in 125 µl sheath fluid and shaken to resuspend. Data were acquired at low PMT setting using the Bio-Rad Bio-Plex suspension array system and Bio-Plex Manager 6.0 software. Fluorescence intensity of the background was subtracted from the values for each sample and standard for each specific bead. Ten-point extended standard curves were generated from 4-fold dilutions of standards provided in the Bio-Plex kits, which

were analyzed using 5-place logistic regression from standards within 70-130% of the expected values. Upper levels of quantitation and lower levels of quantitation were calculated by the Bio-Plex Manager 6.0 software. Reported concentrations are in pg/ml serum.

Statistics

At the inception of this study, pilot data from our own laboratory on insulin sensitivity that could be used to address statistical power considerations were unavailable; therefore, we conducted one interim analysis to determine the sample size to detect differences in SI. This allowed us to incorporate estimates of both the variance within each diet as well as the within-subject correlation of insulin sensitivity across the two diets in our laboratory, while maintaining assumptions regarding Type I and Type II errors. This analysis, in turn, led to the plan to analyze, in 9 men and 9 women, most outcome variables, including insulin sensitivity (SI) and those associated with the extensive lipidomic investigation.

This study examined a wide variety of both serum and muscle changes that might accompany the diet changes being studied. This included 329 variables measured in the fasting state and 277 measured in the fed state. PCA was used to organize the fasting and fed data, look for patterns among the variables, and identify similarities and differences among specific variables.

The variability of each individual variable contributes to an overall variability in the system as a whole. This variability can often be explained by a smaller number of variables, which we refer to as the principal components. These principal components are linear combinations of all of the fasted or fed variables, with the individual variables weighted in order to account for the greatest variability in the dataset. In addition, principal components are computed such that each component is not correlated with any of the other principal components. Variables with higher weighting on a particular component tend to be correlated with that component, as well as correlated with each other. Thus, interpretation of a given component allows identification of a group of variables that tend to vary in a similar manner. The interpretation of the principal components is aided by a linear transformation resulting in several of the variables having a high weighting or factor loading (in this case, greater than or equal to |0.40|) with the remaining variables having factor loadings near zero, a process referred to as rotation.

Once the principal components were identified, scores for each component were computed, and these scores were used to replace the individual variables in an effort to examine system-wide changes induced by the different diets. The SAS procedure PROC FACTOR, along with the VARIMAX rotation (to allow orthogonal rotation of the resulting components) was used to identify and compute the component scores.

In summary, for the principal components analysis, components with an eigenvalue $\geq$1.0 were retained and component scores for each subject were calculated using the standardized scoring coefficients (consisting of a weighted sum of the values of the standardized variables, weighted by the component loading calculated for each individual variable). Metabolites with a load of greater than |0.4| were identified as having a major contribution to a given component.

HOW INDIVIDUAL CHARACTERISTICS IN VERSUS WOMEN MAY HAVE IMPACTED THE DIET EFFECTS ON INSULIN SENSITIVITY (SI).

The ratio of SI on the HOA diet to that on the HPA diet averaged 1.04 in men (ranged 0.41 - 2.17, median 0.71), but in women, the average ratio was 1.60 (range 0.87 to 2.4, median 1.68). Change in SI correlated with physical activity on the HPA ($r = 0.68$, $P = 0.042$), but not the HOA diet ($r = 0.55$, $P = 0.12$). Aside from the correlations between change in SI and VO2 peak described in Figure 1, change in SI in women also correlated inversely with other indices of "metabolic fitness": BMI ($r = - 0.717$, $P = 0.03$), abdominal circumference ($r = - 0.817$, $P = 0.007$), and percent body fat ($r = - 0.800$, $P = 0.010$) measured at screening, as well as serum TAG concentration ($r = - 0.736$, $P = 0.024$) measured at the end of the baseline diet. In men, the relationships between metabolic fitness and SI change were in opposite direction: abdominal circumference, $r = 0.817$, $P = 0.007$; percent body fat, $r = 0.717$, $P = 0.030$.

SUPPLEMENTARY DATA

COMPREHENSIVE ANALYSES OF CIRCULATING AND CELLULAR LIPIDS (FIGURE 1, MEN AND WOMEN SEPARATELY).

Figure 1 C - F show the data for men and women combined, but for most variables, similar results were found for men and women separately . In reference to Figure 1 C, serum concentration (mM) of non-esterified PA was lower during the HOA diet in men ($P < 0.001$) but not women ($P = 0.84$); however, in women, only, OA trended upward on HOA ($P = 0.07$). Figure 1 D shows the serum concentration (mM) of total PA and OA (protein-bound plus non-esterified); in men and women separately, total PA was higher on HPA, and total OA was higher on HOA (P £ 0.002). Figure 1 E depicts the PA/OA ratio in serum NEFA, TFA, AC, and PC (P £ 0.001 for all bars, men or women separately). Figure 1 F shows the PA/OA ratio in muscle lipids: TAG, DAG, PC, and LCAC (P £ 0.03 for all bars in men and women separately, except, $P = 0.07$ for LCAC, women).

Supplementary Table 1. Composition of Experimental Diets (determined at Covance Laboratories, Madison, WI).

	HI PA	HI OA
% kcal		
Protein	16.8	16.89
Carbohydrate	42.85	43.51
Fat	40.45	40.13
Fatty Acid Profile g/100g		
Palmitic	40.29	4.59
Oleic	39.95	74.8
Linoleic	10.37	14.29
Stearic	4.22	2.81
α-Linolenic	0.18	0.14
Myristic	0.97	0
Palmitoleic	0.18	0.12
Eicosapentenoic	0	0
Docosahexanoic	0	0
Arachidonic	0	0
% kcal		
12:0	0	0
14:0	0.42	0.05
16:0	16.02	2.37
18:0	1.8	1.27
18:1	16.23	28.75
18:2	4.97	6.36
18:3	0.16	0.19

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Supplementary Table 2. Effects of diets in men and women on total and regional body fat.

	MEN	MEN	MEN	WOMEN	WOMEN	WOMEN
	BASELINE	HPA	HOA	BASELINE	HPA	HOA
Total Body Fat (kg)	14.41 ± 2.47	14.01 ± 2.77	13.73 ± 2.66	21.72 ± 3.43	20.95 ± 3.33	20.69 ± 3.38
Trunk Fat (kg)	7.15 ± 1.26	6.96 ± 1.42	6.73 ± 1.36	10.94 ± 2.15	10.43 ± 1.99	10.3 ± 2.14
Leg Fat (kg)	5.52 ± 0.93	5.35 ± 1.04	5.37 ± 0.99	8.2 ± 1.07	8.00 ± 1.08	7.87 ± 1.01
Android Fat (kg)	1.06 ± 0.24	0.97 ± 0.22	0.94 ± 0.22	1.64 ± 0.39	1.54 ± 0.37	1.49 ± 0.39
Gynoid Fat (kg)	3.00 ± 0.46	2.89 ± 0.5	2.88 ± 0.47	4.55 ± 0.53	4.44 ± 0.52	4.42 ± 0.53

Results are mean ± SEM. There are no significant diet group differences for either men or women or for men and women considered together.

Supplementary Table 3. Individual insulin sensitivity (SI) values*.

	HPA	HOA
MEN	3.056	4.384
	6.176	9.336
	2.110	1.494
	5.403	3.142
	6.000	8.526
	4.709	3.295
	4.666	10.141
	7.252	3.003
	9.969	4.450
WOMEN	2.0241	3.3568
	4.6235	7.7798
	2.3171	2.0182
	2.1345	2.4801
	6.9057	7.3667
	3.8861	6.7456
	3.2362	5.7909
	2.7921	6.7118
	7.7584	15.85

*SI x 10-4 (min-1/mU/ml). For each pair of values, HPA and HOA, the bolded value represents the diet ingested first.

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Supplementary Table 4. Variables derived from principal components analysis (PCA) with high loading scores (loading > 0.4) for PCF1-Fasted and PCF1-Fed.

PCF1-Fasted	PCF1-Fed
DAG OA/SA[*,†]	DAG OA concentration
DAG OA/total FA	DAG OA/SA
DAG PA/OA	DAG OA/total FA
DAG PA/total FA	DAG PA concentration
TAG MA/ total FA	DAG PA/OA
TAG OA/SA	DAG PA/total FA
TAG OA/total FA	TAG OA/SA
TAG PA	TAG OA/total_FA
TAG PA/OA	TAG PA/OA
TAG PA/total FA	TAG PA/total FA
Mp PC OA/SA	TAG SA/total FA
Mp PC OA/total FA	PC OA/SA
Mp PC PA/OA	PC OA/total FA
Mp PC PA/total FA	PC PA/OA
Mp PC SA/PA	PC PA/total FA
Mp PC SA/total FA	PC SA/PA
Mp PE PA/OA	PC SA/total FA
Mp PC OA/SA	PE PA/OA
Mp PC OA/total FA	FA C16:0/C16:1
Mp PC PA/OA	FA C16:0/18:1
Mp PC PA/total FA	FA C18:0/18:1
Mp PC SA/PA	*Mp C16:0/18:0*
Mp PC SA/total FA	*Mp C16:0/C18:1*
Mp PE PA/OA	*Mp C18:0/C18:1*
S CL PA/total FA	Mp malate
S CL SA/PA	S C:14
S PC LA/ total FA	S C16:0
S PC OA/SA	S C10:3
S PC OA/ total FA	*S C16:0/C16:1*
S PC PA/OA	*S C16:0/C18:0*
S PC PA/ total FA	*S C16:0/C18:1*
S PC SA/PA	S C18:1
S PC SA/ total FA	*S C18:0/C18:1*
S PE OA/SA	S C8:1
S PE OA/total FA	S TFA C16:0
S PE PA/OA	S TFA C18:1

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S PE PA/total FA	S TFA C18:2
S PE SA/PA	TFA C16:0/C16:1
S PE concentration	TFA C16:0/C18:0
SFA C16:0/C16:1	TFA C16:0/C18:1
SFA C16:0/C18:0	TFA C18:0/C18:1
SFA C16:0/C18:1	Cerm C24:1
Mp AC 16:0/C16:1	Cerm C16:0
Mp AC 16:0/C18:0	Cerm C18:0
Mp AC 16:0/C18:1	Cerm C20:0
S AC16:0	Cerm C22:0
S AC 10:3	Cerm C24:0
S AC 16:0/C16:1	Cerm C25:0
S AC 16:0/C18:0	
S AC 16:0/C18:1	
S AC18:1	
S AC18:0/C18:1	
S AC C:18-OH/C16:0-DC	
S FA C16:0	
S TFA C16:0	
S TFA C18:1	
S TFA C18:2	
TFA C16:0/16:1	
TFA C16:0/C18:0	
TFA C16:0/C18:1	
TFA C18:0/C18:1	
Cerm C24:1	
Cerm C16:0	
Cerm C18:0	
Cerm C20:0	
Cerm C24:0	

* Bolded variables have negative loading scores.

†Non-standard Abbreviations: For lipids, other than acylcarnitine species contributing to PCF1-Fasted or PCF1-Fed, "/" refers to ratios of fatty acids of different chain lengths. For acylcarnitine species contributing to PCF1-Fasted or PCF1-Fed, the corresponding acylcarnitine ratios pertaining to different chain lengths are italicized. Otherwise, for other acylcarnitine species contributing to PCA Factor Fast, "/" refers to unresolved species. AA, arachidonic acid; AC, acylcarnitine; Ala, alanine; ALA, □-linolenic acid (18:3 n-3); Cerm, serum ceramide; Ci4. Isobutyrate; CL, cardiolipin; CPT LC, sum of the long chain acylcarnitines that are direct products of carnitine palmitoyltransferase I (CPT1); DAG, muscle diacylglycerol; DC, dicarboxyllic; DHA, docosahexanoic acid (22:6 n-3); EPA, eicosapentanoic acid (22:5 n-3); FA, serum non-esterified fatty acids; LA, linoleic acid; LCAC, total long-chain acylcarnitines; LC-OH, sum of all long-chain hydroxylated acylcarnitines; MA, myristic acid; AC MC, medium chain acylcarnitines; Mp, skeletal muscle (expressed per protein content); NEFA, total non-esterified FA in blood; OA, oleic acid; PA, palmitic acid; PC, phosphatidylcholine; PE,

phosphatidylethanolamine; POA, palmitoleic acid; S, serum; SA, stearic acid; SC, serum acylcarnitine; TAG, muscle triacylglycerol; TFA, serum total FA; tot FA, total measured FA within a given species in blood or muscle. Total LC, sum of long-chain acylcarnitines.

Supplementary Table 5. Serum ceramide concentration.

CERAMIDE SPECIES		
M + F (n = 18) Fasting	HOA	HPA
C:14:0	0.023 ± 0.003*	0.021 ± 0.002
C:16:0	0.343 ± 0.022	0.460 ± 0.024†
C:18:0	0.070 ± 0.005	0.094 ± 0.009‡
C:20:0	0.078 ± 0.009	0.122 ± 0.007§
C:22:0	0.631 ± 0.055	0.796 ± 0.052§
C24:0	2.238 ± 0.198	3.245 ± 0.249†
C25:0	0.101 ± 0.016	0.166 ± 0.023§
C24:1	0.639 ± 0.058	0.821 ± 0.052§
total ceramide	4.162 ± 0.321	5.724 ± 0.381†
M + F (n = 18) Fed		
C:14:0	0.028 ± 0.003	0.022 ± 0.004
C:16:0	0.336 ± 0.016	0.522 ± 0.037†
C:18:0	0.063 ± 0.005	0.099 ± 0.010§
C:20:0	0.085 ± 0.007	0.139 ± 0.010†
C:22:0	0.628 ± 0.045	0.913 ± 0.069†
C24:0	2.188 ± 0.173	3.708 ± 0.313†
C25:0	0.108 ± 0.010	0.231 ± 0.026†
C24:1	0.644 ± 0.031	0.904 ± 0.074§
total ceramide	4.080 ± 0.262	6.539 ± 0.496†
F (n = 9) Fasting		
C:14:0	0.023 ± 0.004	
C:16:0	0.353 ± 0.038	
C:18:0	0.073 ± 0.009	
C:20:0	0.082 ± 0.011	
C:22:0	0.639 ± 0.087	
C24:0	2.109 ± 0.230	
C25:0	0.107 ± 0.030	
C24:1	0.634 ± 0.100	
total ceramide	4.100 ± 0.424	
F (n = 9) Fed		
C:14:0	0.028 ± 0.004	
C:16:0	0.347 ± 0.022	
C:18:0	0.067 ± 0.006	

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C:20:0	0.093 ± 0.013	
C:22:0	0.606 ± 0.052	
C24:0	1.982 ± 0.172	
C25:0	0.123 ± 0.009	
C24:1	0.648 ± 0.036	
total ceramide	3.894 ± 0.275	
M (n = 9) Fasting		
C:14:0	0.023 ± 0.005	
C:16:0	0.335 ± 0.027	
C:18:0	0.068 ± 0.006	
C:20:0	0.075 ± 0.014	
C:22:0	0.625 ± 0.075	
C24:0	2.353 ± 0.321	
C25:0	0.095 ± 0.017	
C24:1	0.644 ± 0.068	
total ceramide	4.218 ± 0.499	
M (n = 9) Fed		
C:14:0	0.028 ± 0.005	
C:16:0	0.326 ± 0.024	
C:18:0	0.059 ± 0.008	
C:20:0	0.078 ± 0.005	
C:22:0	0.650 ± 0.076	
C24:0	2.393 ± 0.295	
C25:0	0.093 ± 0.017	
C24:1	0.640 ± 0.053	
Total Ceramide	4.267 ± 0.455	

* mean ± S.E.M. (nmol/ml)
† $P < 0.001$
‡ $P < 0.05$
§ $P \leq 0.01$
¶ $P < 0.1 > 0.05$

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