Supplemental data
Figure S1, related to Figure 4

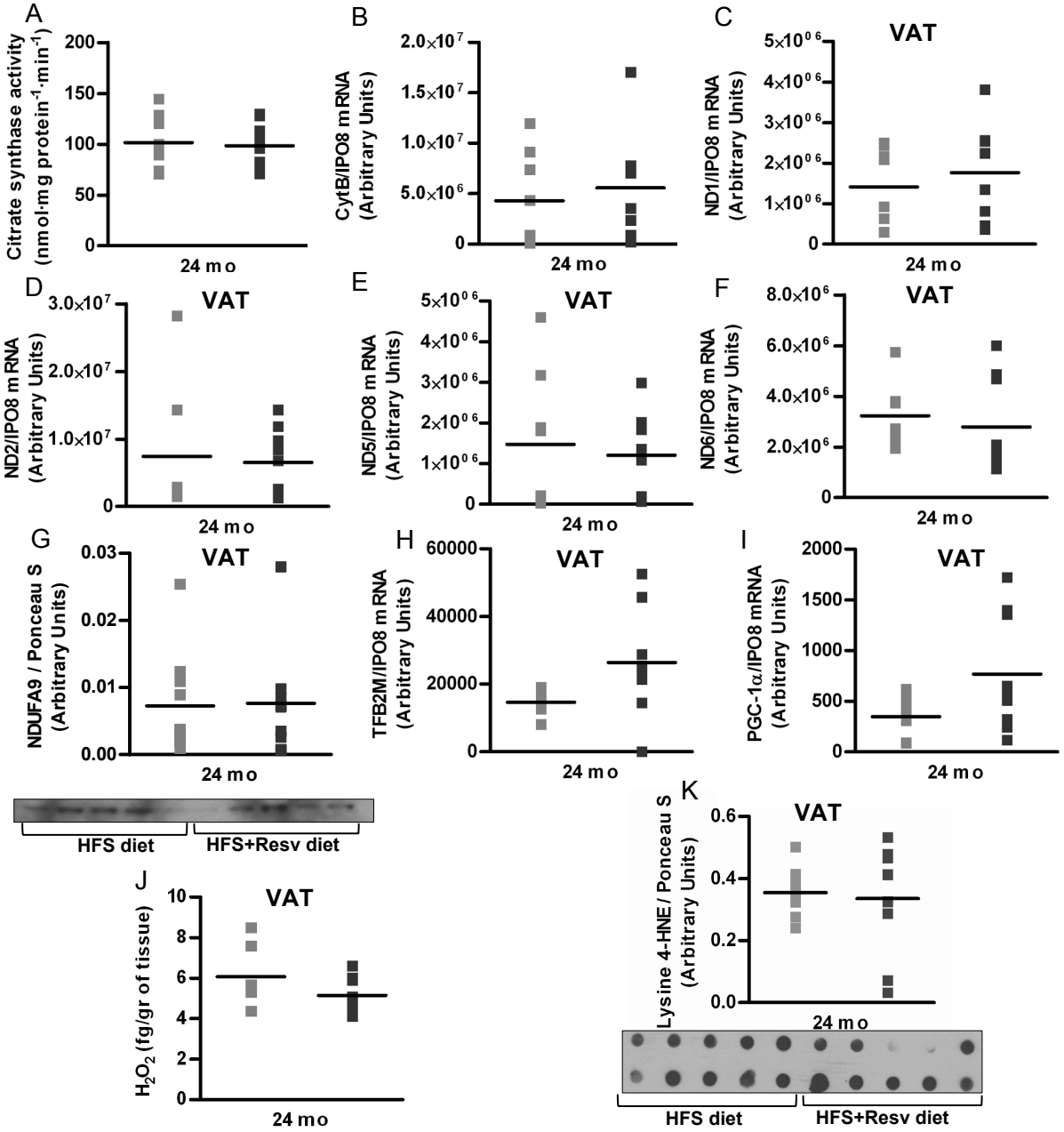

Figure S1, related to Figure 4. Mitochondrial content and biogenesis, and oxidative stress in visceral WAT of rhesus monkeys fed a HFS and HFS + Resv diet for 2 years. (A) Citrate synthase activity. (B) Cytochrome b mRNA expression. (C) NADH dehydrogenase (ND) 1 mRNA levels. (D) ND2 mRNA expression. (E) ND5 mRNA levels. (F) ND6 mRNA expression. (G) NADH dehydrogenase (ubiquinone) 1 alpha subcomplex, 9 protein levels. (H) Dimethyladenosinetransferase 2, mitochondrial mRNA expression. (I) PGC-1α mRNA levels. (J) H_2O_2 levels (K) Lysine 4-hydroxinonenal levels. (A to K) Results are expressed in a dot plot format, which represents the individual data and the mean. (A ,D, F, G, I, J and K) n=10 for each group. (B) n=8 (HFS diet); n=7 (HFS + Resv diet). (C, E and H) n=8 (HFS diet); n=8 (HFS + Resv diet). The data were analyzed using Independent-Samples *t* test to analyze statistical significance between HFS *vs.*HFS + Resv diet at 24-mo of dietary intervention. CytB: cytochrome b; NDUFA9: NADH dehydrogenase (ubiquinone) 1 alpha subcomplex, 9; TFB2M: Dimethyladenosinetransferase 2, mitochondrial; PGC-1α: PPARγ coactivator 1-alpha; ND: NADH dehydrogenase; HNE: hydroxinonenal; HFS: high-fat, high-sugar; Resv: resveratrol; VAT: visceral adipose tissue.

Figure S2, related to Table 1

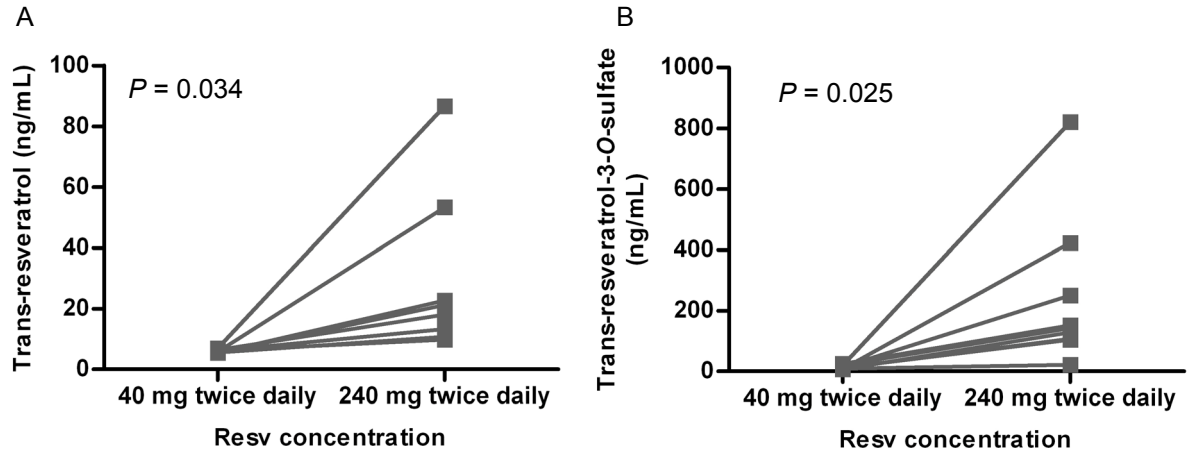

Figure S2, related to Table 1. Serum trans-resveratrol and trans-resveratrol-3-*O*-sulfate concentration from monkeys fed HFS diet supplemented with 40 mg or 240 mg of Resv twice daily. (A) Serum Resv concentration at 9 and 24 months of dietary intervention, (B) Resv-3-*O*-sulfate concentration at 9 and 24 months of dietary intervention. (A and B) Results are expressed in a dot plot format, n=9. The data were analyzed using RM-ANOVA to calculate the effect of dosage increments (40 *vs.* 240 mg twice daily). Resv: resveratrol.

Figure S3, related to Figure 2

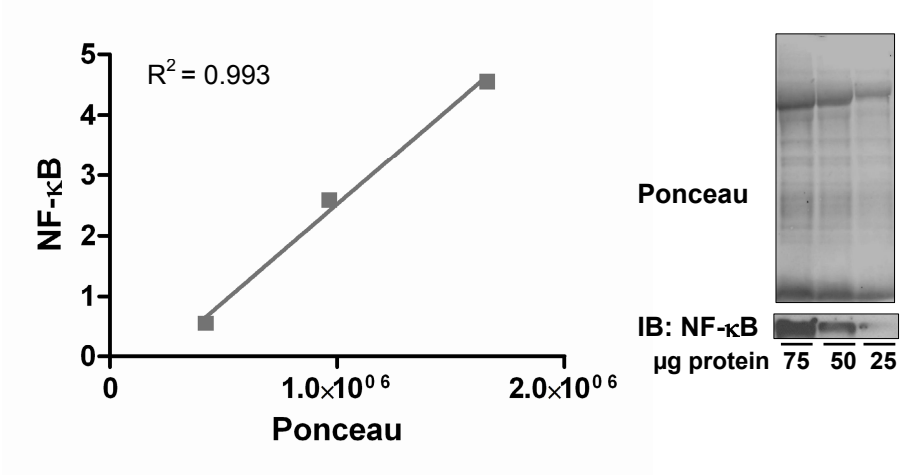

Figure S3, related to Figure 2. Correlation between Ponceau S staining and NF-κB densitometric signal. A visceral WAT sample was serially diluted and resolved by SDS-PAGE followed by electrotransfer onto a nitrocellulose membrane. The membrane was subjected to Ponceau S staining and then analyzed by Western blotting with anti-p65 Rel antibody. Note the strong correlation between the two methods.

Table S1, related to Table 1

Table S1, related to Table 1: Monkey characteristics at baseline[a,b].

	SD	HFS diet	HFS + Resv diet	P value[c]
Baseline (Pre-Intervention)				
N	4	10	10	
Age, *y*	9.76±0.84	11.03±0.60	10.34±0.54	0.461
Weight, *kg*	12.98±1.21	13.24±1.21	12.82±1.04	0.964
Waist, *cm*	42.00±4.16	40.40±3.39	41.35±3.37	0.960
Hip, *cm*	40.17±2.68	38.10±2.20	39.60±2.12	0.826
Total Cholesterol, *mg/dL*	139.75±5.33	135.90±8.75	148.70±11.48	0.636
LDL-C, *mg/dL*	55.00±3.94	57.50±4.22	58.60±4.46	0.898
HDL-C, *mg/dL*	72.00±6.72	66.2±5.86	77.00±8.19	0.540
Fasting Glucose, *mg/dL*	58.77±1.14	60.07±2.75	61.24±1.04	0.760
Fasting Insulin[d], *µU/mL*	27.34±6.61	31.44±16.03	35.78±13.24	0.673

[a] Values are presented as mean ± SEM.
[b] Blood samples were obtained from fasting animals.
[c] The data were analyzed using One-WayANOVA.
[d] Data were log-transformed before statistical analysis.
SD: standard diet; HFS: high-fat, high-sugar; Resv: resveratrol.

Table S2, related to Figure 1

Table S2, related to Figure 1. Ingenuity Pathway Analysis reveals diet-induced alterations in inflammation-related biological functions in subcutaneous fat depot.

Function'sname	P value[a]	# molec.	Comparison
Cancer	4.10E_02	40	HFS_SDb
HepaticSystemDisease	4.66E-02	38	
Gastrointestinal Disease	4.66E-02	61	
MetabolicDisease	4.66E-02	18	
InflammatoryDisease	9.74E-03	5	
Gastrointestinal Disease	4.63E-02	89	Resv_SDb
HepaticSystemDisease	3.99E-02	51	
InflammatoryDisease	1.13E-03	7	
Inflammatory Response	4.53E-02	28	
OrganismalInjury and Abnormalities	2.18E-02	42	
Cancer	4.13E-02	6	Resv_HFS
Inflammatory Response	1.43E-02	5	
Gastrointestinal Disease	3.95E-02	28	
HepaticSystemDisease	3.95E-02	4	
OrganismalInjury and Abnormalities	1.43E-02	2	

[a]The data were analyzed using Fisher´s exact test right-tailed.
HFS: high-fat, high-sugar, n=4; Resv: resveratrol, n=4; SDb: standard diet baseline, n=24.

Table S3, related to Figure 1

Table S3, related to Figure1. Comparative analysis of select genesets between visceral and subcutaneous fat depots[a]

Visceral fatdepot		**HFS_SD**			**Resv_HFS**			**Resv_SD**		
Pathway	**n**	**Zscore[b]**	**P[c]**	**fdr**	**Zscore**	**P**	**fdr**	**Zscore**	**P**	**fdr**
Electron_Transport_Chain	82	6.61	4E-07	0.00	-4.14	0.000	0.01	0	0.511	0.74
Proteasome	16	4.57	2E-06	0.00	-2.93	0.000	0.00	0	0.663	0.84
Aged_Rhesus_Up	143	-2.82	0.042	0.22	4.42	0.002	0.03	0	0.068	0.27
TollPathway	28	-3.87	0.007	0.09	3.88	0.019	0.11	0	0.320	0.61
StressPathway	21	-4.36	0.005	0.08	4.26	0.031	0.16	0	0.369	0.65
Apoptosis_GENMAPP	37	-4.50	0.003	0.06	4.45	0.006	0.06	0	0.249	0.55
OxstressRPE_H2O2HNE_DN	27	-4.77	0.004	0.06	6.04	0.004	0.05	0	0.082	0.30
Subcutaneousfatdepot		**HFS_SDb**			**Resv_HFS**			**Resv_SDb**		
Pathway	**n**	**Zscore**	**P**	**fdr**	**Zscore**	**P**	**fdr**	**Zscore**	**P**	**Fdr**
Electron_Transport_Chain	82	5.86	0.000	0.03	-4.99	0.013	0.11	0	0.546	0.84
Proteasome	16	0	0.107	0.38	-3.56	0.005	0.06	-3.40	0.008	0.13
Aged_Rhesus_Up	143	0	0.374	0.67	0	0.312	0.61	0	0.927	0.97
TollPathway	28	-6.40	0.007	0.12	2.79	0.002	0.03	0	0.416	0.78
StressPathway	21	-4.66	0.018	0.18	0	0.228	0.52	0	0.207	0.62
Apoptosis_GENMAPP	37	-4.66	0.003	0.08	3.43	0.002	0.03	0	0.493	0.83
OxstressRPE_H2O2HNE_DN	27	0	0.238	0.54	0	0.259	0.55	0	0.144	0.57

[a]Gene Set Enrichment Analysis (GSEA) was carried out and select genesets were chosen for their known susceptibility to resveratrol supplementation.
[b]Zscore was given a value of '0' when a geneset was found to be nonsignificant.
[c]The data were analyzed using z-test between the tested pathway gene set and the whole array collection.
HFS: high-fat, high-sugar, n=4; Resv: resveratrol, n=4; SD: standard diet, n=2; SDb: standard diet baseline, n=24; fdr, false discovery rate.

Table S4, related to Figure 3

Table S4, related to Figure 3: Pro- and anti-inflammatory blood parameters of rhesus monkeys after dietary intervention[a,b].

	SDb	HFS diet	HFS + Resv diet	*P* value
N	24	10	10	
IL6[c], *pg/mL*	4.65±1.49	4.84±2.18	1.29±0.83	0.493
TNF-α, *pg/mL*	3.14±0.33	3.63±0.40	3.60±0.48	0.541
IL-1β, *pg/mL*	36.76±14.29[#]	94.27±33.06	92.69±19.74	<0.001
Adiponectin, *μg/mL*	20.25±2.36	19.49±3.42	12.45±1.88	0.191
CRP, *ng/mL*	10.13±4.55	9.02±1.60	14.70±5.77	0.934

[a]Values are presented as mean ± SEM. The data were analyzed using a linear mixed-effects model. [#], $P < 0.05$ (SDb *vs.* HFS ± Resv diet). SDb: Standard diet baseline; HFS: High-fat, high-sugar; Resv: Resveratrol. IL-6: n= 24 (SDb); n=10 (HFS diet); n=8 (HFS + Resv diet), all other parameters: n= 24 (SDb); n=10 (HFS diet); n=10 (HFS + Resv diet).
[b]Blood samples were obtained from fasting animals.
[c]Data were log-transformed before statistical analysis.

Table S5, related to Figure 3

Table S5, related to Figure 3: Summary of monkey and mouse primer sets for real-time reverse transcriptase-polymerase chain reaction (RT-PCR).

Gene	Genebanknumber	Species	Primers RT-PCR
IL-6	NM_001042733	Macaca Mulatta	Left: 5'-GGCAGAAAACAACCTGAACCT-3' Right: 5'-GCTTGCTCCTCACTACTCTCAAAC-3'
TNF-α	NM_001047149	Macaca Mulatta	Left: 5'- TTCCTCAGCCTCTTCTCCTTC-3' Right: 5'-GGTTTGCTACAACATGGGCTAC-3'
IL-1β	NM_001042756	Macaca Mulatta	Left: 5'-AGGCCGTGTCAGTTGTTGTAG-3' Right: 5'-GTTATTGCGTGTGTCGAGGAA-3'
Adiponectin	NM_001032871	Macaca Mulatta	Left: 5'-CGAGAAGGGTGAGAAAGGAGA-3' Right: 5'-CATGTTGGGGACAGTAACGTAG-3'
IPO8	XM_001082218	Macaca Mulatta	Left: 5'-GCTCCTTCCTGATTCCTCCTATT-3' Right: 5'-CATCCACGTTGTCATGGTTTG-3'
ND1	2846627	Macaca Mulatta	Left:5'- TCCATGCGGACTATTACAACC-3' Right:5'- ATAGGAGGAGGGCAATGAAGA-3'
ND2	2846626	Macaca Mulatta	Left:5'- CAGAAGTCACCCAAGGAACAC-3' Right:5'- TGGAGAGGGTTAGGAGGATGT-3'
ND5	2846631	Macaca Mulatta	Left:5'- CCTGAGCCCTATTCATCACTCT-3' Right:5'- GGTTTAGTAGAGCGGGGTTGT-3'
ND6	2846632	Macaca Mulatta	Left:5'- ACCACTTCTACCGCCTCATC-3' Right:5'- AGTATCCTGAGACATGGGGTTC-3'
CytB	2846625	Macaca Mulatta	Left:5'- GTCCAATGAATCTGAGGAGGA-3' Right:5'- TGGTTGTGTAGTAGGGGTGGA-3'
TFB2M	XM_001088795	Macaca Mulatta	Left:5'- GGTCCTGGAATCCTGACTGA-3' Right:5'- AGACATAGCGGGTGGCTTTA-3'
PGC-1α	XM_001105289	Macaca Mulatta	Left:5'- CGATGAAGCAGACAAGACCA-3' Right:5'- GGTGGTGACACGGAATCTCT-3'
IL-6	NM_031168	Mus Musculus	Left: 5'-TAGTCCTTCCTACCCCAATTTCC-3' Right: 5'-TTGGTCCTTAGCCACTCCTTC-3'
TNF-α	NM_013693	Mus Musculus	Left: 5'-CCCTCACACTCAGATCATCTTCT-3' Right: 5'-GCTACGACGTGGGCTACAG-3'
IL-1β	NM_008361	Mus Musculus	Left: 5'-GCAACTGTTCCTGAACTCAACT-3' Right: 5'-ATCTTTTGGGGTCCGTCAACT-3'
Adiponectin	NM_009605	Mus Musculus	Left: 5'-TCCTCTTAATCCTGCCCAGTC-3' Right: 5'-GAAGACCTGCATCTCCTTTCTC-3'
IPO8	NM_001081113	Mus Musculus	Left: 5'-CCTCCATTCTCTTCCTTTTCCT-3' Right: 5'-CCTCCTCACTTGAAATCTCCTC-3'

Supplemental Experimental Procedures

Animals. Monkeys were housed individually in standard non-human primate cages on a 12h light/12h dark cycle, room temperature 25.5 ± 0.5 °C and humidity at 60 ± 20%. Monkeys received two meals per day at estimated *ad libitum* levels throughout the study. A controlled feeding regimen provided a closer control of food consumption and prevented obesity due to over eating, thus insuring that experimental effects were due to the treatment and not differences in food consumption. Food allotments were determined by calculating food consumption while monkeys had access to excess food. The preferred consumption during this time was then provided in two defined meals. Water was always available *ad libitum*. Monkeys were monitored at least 3 times daily by trained animal care staff and, during baseline assessment, all monkeys were maintained on a commercially available closed formula monkey chow (SD).

The Resv dose calculation for monkeys was made to get a target dose based on previous studies in rodents. This dose was derived from the protective dose reported in mice (22 mg/kg) (Baur et al., 2006) and adjusted by allometric scaling to an average monkey body weight of 12.1 kg. The monkey equivalent dose was determined to be 40.7 mg. To confirm the allometric calculation, HPLC was run on serum samples from Resv-fed mice; unmodified Resv was not detected but its 3-glucuronide metabolite was consistently found. A range of Resv doses was then given to the monkeys, serum was analyzed via HPLC, and the 40 mg dose of resveratrol was found to produce equivalent levels of 3-glucuronide as that seen in mice. Resv was used to formulate a flavored treat (Bio-Serve; Frenchtown, NJ), which was given to the monkeys prior to each meal.

Biochemical determinations. Trans-Resv and its *O*-sulfated metabolite in serum samples at 9- and 24-mo of dietary intervention were quantified by a chromatography and mass spectrometry method as follows:

Sample preparation. The extraction of trans-Resv and its metabolites were carried out from a previously described method (Boocock et al., 2007) with modifications. Briefly, 90 μl

of methanol and 10 μl of hexestrol (internal standard, Sigma-Aldrich, St-Louis, MO) were added to 100 μl of serum samples, which were vortex-mixed and centrifuged at 20 800 x *g* at 4 ^{o}C for 10 min. The supernatant was transferred to the autosampler vial for analysis. The Resv-3-*O*-sulfate standard was purchased from artmolecule (http://www.artmolecule.fr).

Separation of Resv and its O-sulfated metabolite by HPLC. The chromatographic experiments were carried out on a Shimadzu Prominence HPLC system (Shimadzu, Columbia, MD). The samples were introduced to the analytical column in 20 μl injections using Shimadzu SIL 20A autosampler and maintained at 4 ^{o}C in the autosampler tray. The separation of Resv and Resv-3-*O*-sulfate was accomplished using an Eclipse XDB-C_{18} guard column (4.6 mm x 12.5 mm) and an analytical column Discovery C18 (150x 4.6 mm ID, 5 μm; Supelco). The mobile phase consisted of water containing 0.1% acetic acid and 0.07% triethylamine as component A and acetonitrile as component B. A linear gradient was run as follows: 0-3 min 20% B; 3-25 min 20-60% B; 25-30 min 60-20% B at a flow rate of 1.0 ml/min. The total run time was 30 min per sample.

Mass spectrometry conditions. MS/MS analysis was performed using a triple quadrupole mass spectrometer model API 4000 system from Applied Biosystems/MDS Sciex equipped with Turbo Ion Spray® (TIS) (Applied Biosystems, Foster City, CA). The data was acquired and analyzed using Analyst version 1.4.2 (Applied Biosystems). Negative electrospray ionization data were acquired using multiple reaction monitoring (MRM), the standards were characterized using the following MRM transitions: Resv (227-185); Resv-3-*O*-sulfate (307-227) and hexestrol (IS) (269-134). The TIS instrumental source settings for temperature, curtain gas, ion source gas 1 (nebulizer), ion source gas 2 (turbo ion spray), entrance potential and ion spray voltage were 500 °C, 10 psi, 60 psi, 70 psi, -10V and -4500 V, respectively. The TIS compound parameter settings for declustering potential, collision energy, and collision cell exit potential were -70V, -25V, -7V for Resv; -50V, -28V and -9V for Resv-3-*O*-sulfate and -82V, -20V and -8V for hexesterol.

Differentiation of 3T3-L1 mouse fibroblasts into adipocytes.Mouse 3T3-L1 cells were grown and maintained in high glucose DMEM (4.5 g/L glucose, Biowhittaker, Lonza, Walkersville, MD) supplemented with 10% Newborn Calf Serum (Gibco, Invitrogen, Grand Island, NY), 4 mM glutamine and 1.5 g/L NaH_2CO_3 at 37 ºC and 5% CO_2. The cells were allowed to grow until 2 days post-confluence after which they were differentiated into adipocytes according to an established method (Student et al., 1980) with modifications. Briefly, confluent cells were incubated in high glucose DMEM containing 10% FBS (Gibco, Invitrogen), 4 mM glutamine and 1.5 g/L NaH_2CO_3 (base medium) with 0.5 mM isobutylmethylxanthine (IBMX; Sigma-Aldrich), 0.25 µM dexamethasone (Sigma-Aldrich) and 10 µg/ml insulin (Sigma-Aldrich) for 72 h (day 3). The medium was then replaced by base medium supplemented with 10 µg/ml insulin for an additional 72-h period (day 6) after which the medium was replaced by base medium alone every two days until day 10. All the experiments were performed on day 8 or 10 after differentiation.

Western blot. This was performed as previously described (Bernier et al., 2011). The visualization of immunoreactive bands was performed by chemiluminescence and/or infrared imaging using an Odyssey Imaging system (LI-COR Biosciences, Lincoln, NE). Band intensities were quantified using LI-COR and/or ImageJ software followed by normalization to Ponceau S staining. In this study, the primary antibodies were directed against SIRT1 and Akt1/2 (Santa Cruz Biotechnology, Inc., Santa Cruz, CA); IκBα, phospho-NF-κB p65 (Ser-536), IRS-1 and phospho-Akt (Ser-473) (Cell Signaling Technology, Inc., Danvers, MA); NF-κB (Epitomic, Inc., Burlingame, CA); NDUFA9 (Abcam, Cambrige, MA) and Lysine 4-HNE, which recognizes the 4-hydroxinonenal carbonylation residues in proteins (EDM-Calbiochem, La Jolla, CA), and were used generally at a dilution recommended by the manufacturer. To determine GLUT4 and TUG protein content, immunoblotting was performed using previously described antibodies directed to the C termini of GLUT4 and TUG (Yu et al., 2007).

Immunoprecipitation. Acetylated proteins were immunoprecipitated using a mouse anti-acetyl-lysine antibody (Millipore, Billerica, MA) overnight at 4 °C. Immunocomplexes were then incubated with protein-G-agarose beads for 4 h at 4 °C and solubilized in Laemmli SDS-sample buffer. Immunoblot analysis was carried out using a primary monoclonal antibody directed against p65RelA NF-κB (Epitomic). As negative control, immunoprecipitation was performed with mouse IgG (Santa Cruz Biotechnology).

Microarray data analysis. RNA was isolated from subcutaneous WAT at 0 (SDb, n=24) and 24-months and visceral WAT at 24-months of dietary intervention using Trizol Reagent (Invitrogen, Carlsbad, CA) following the manufacturer's instructions and further purified using RNeasy mini columns (Qiagen). RNA was then hybridized to Human6 Expression v3 and v4 beadchips (Illumina, San Diego, CA) following protocols listed on the Gene Expression and Genomics Unit of the NIA (http://www.grc.nia.nih.gov/branches/rrb/dna/index/protocols.htm) Raw data were subjected to Z normalization to ensure compatibility using the formula: z(raw data)=[ln (raw data) – avg(ln(raw data))]/[std dev(ln (raw data))], where ln is natural logarithm, avg is the average over all genes of an array, std dev is the standard deviation over all genes of an array (Cheadle et al., 2003). The Z ratio (between treatment A and B) is given by z(A)-z(B)/std dev. Individual genes with Z ratio > 1.5, *P* value < 0.05, and avg intensity > 0 were considered significantly changed.

For parametric analysis of gene set enrichment (PAGE), a list of pathways was obtained from http://www.broad.mit.edu/gsea/msigdb/msigdb_index.html (C2 collection). Our expression data was tested for gene set enrichment using the PAGE method as previously described (Kim and Volsky, 2005). Briefly, for each pathway a Z score was computed as Z (pathway) = (sm mu)*pow(m,0.5)/delta, where mu = mean Z score of all gene symbols on the microarray, delta = standard deviation of Z scores of all gene symbols on the microarray, sm = mean Z score of gene symbols comprising one pathway present on the microarray, and m = no of gene symbols in a pathway present on the microarray. For each Z (pathway) a *P* value was also computed in JMP 6.0 to test for the significance of the Z score obtained.

These tools are part of DIANE 6.0 and are available at http://www.grc.nia.nih.gov/branches/rrb/dna/diane_software.pdf.
Gene expression data was deposited in NCBI Gene Expression Omnibus (http://www.ncbi.nlm.nih.gov/geo/) under the accesion number GSE50005.

Total RNA isolation and Real-Time PCR. Samples of frozen visceral adipose tissue from rhesus monkeys were mechanically disrupted in TRIzol® reagent (Applied Biosystems/Ambion, Austin, TX). Total RNA was extracted using the RiboPure™ kit (Applied Biosystems/Ambion) following the manufacturer's instructions. Total cellular RNA from frozen 3T3-L1 samples was extracted using the TRIzol method (Tri® Reagent, Sigma-Aldrich). Subsequently, total RNA was converted to cDNA with the High Capacity cDNA reverse transcription kit (Applied Biosystems, Foster City, CA), and real-time PCR was carried out with SYBR® Green PCR master mix to quantitate the expression levels of IL-1β, TNF-α, IL-6, adiponectin, ND1, ND2, ND5, ND6, CytB, TFB2M and PGC-1α on an ABI Prism 7300 sequence detection system (Applied Biosystems). Importin 8 (IPO8) was selected as a housekeeping gene (Hurtado del Pozo et al., 2010), as its expression was not significantly different between samples. Real-time PCR data were analyzed by the standard curve-based method. The expression of target genes was normalized to that of the housekeeping gene. All measurements were performed in duplicate. Controls consisting of reaction mixture without cDNA were negative in all runs. Fidelity of the PCR was determined by melting temperature analysis.

Statistical analysis. The normal distribution of variables was assessed using the Kolmogorov-Smirnov test and log–transformed if appropriate. A linear mixed-effects model was used to analyze the repeated measures blood parameters (Verbeke et al., 2000). Experimental group (SDb, HFS, HFS + Resv and SD) was included as a categorical variable. First group (SDb) are all animals at baseline and they were compared to the SD-fed subjects for 24 months to analyze the aging effect. Then, all baseline animals (SDb) were compared to the HFS +/- Resv diet-fed subjects for 24 months. Post-hoc comparisons were

made to compare the three groups (SDb, HFS and HFS + Resv) using Bonferroni adjuted p-values to account for multiple comparisons. The rest of parameters were analyzed using Fisher´s exact test right-tailed, Z-test, Independent-Samples *t* test, One-Way ANOVA and repeated measures ANOVA (RM-ANOVA) as appropriate. In this last analysis, we studied: the effect of time alone on the change in the variable, independently of diet; the effect of diet, independently of time; and the interaction of both factors -diet and time-, which is indicative of the magnitude of the dietary response among diet groups. A post-hoc statistical analysis using the Bonferroni's test was needed to identify significant differences between dietary treatments. The contrast statistic used when the sphericity assumption was not satisfied was Huynh-Feldt.

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