Supplementary Information

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TECHNIQUES AND ASSAYS:

a. Virus preparation. Ad-36 was obtained from American Type Culture collection (ATCC Cat# VR913) plaque purified and propagated in A549 cells (human lung cancer cell line) as described and used previously (8, 9). Ad-2 was also obtained from ATCC (Cat #VR846) and propagated in A549 cells. Viral titers were determined by plaque assay (9) and inoculations expressed as plaque forming units (PFU).

b. Biochemical assays:
Glucose: 2 μL of serum from each mouse was measured in a 96-well plate format using Raichem glucose Oxidase method (R80038). Absorbance was read at 500 nm. Insulin: Ultra-sensitive mouse insulin ELISA kit (Crystal Chem, # 90090) was used to determine insulin. Triglycerides were determined using Cardiochek Lipid panel test strips.

qRT-PCR: mRNA was extracted from the livers of chow-fed and HF-fed mice, using the RNeasy Mini kit as per the manufacturer's instructions (Qiagen, # 74101). Residual DNA was eliminated by using Amplification grade Deoxyribonuclease I (Invitrogen, # 18068-015). One μg of total RNA was reverse transcribed to cDNA using iscript^{TM} cDNA synthesis kit (BioRad, # 170-8890) as per the manufacturer's protocol. PCR core system II (Promega, # M7665) was used for the amplification of cDNA.
Quantitative RT-PCR was performed to examine the relative expression levels of the following genes
TNFα (Tumor Necrosis factor alpha, Applied Biosystems, # Mm00443259_g1),
Resistin (Applied Biosystems, # Mm00445641_m1),
IL6 (interleukin 6; Applied Biosystems, # Mm00446191_m1)
MCP-1(macrophage chemoattractant protein; Applied Biosystems, # Mm00441243_g1)
CD68 (Applied Biosystems, # Mm03047343_m1),
TLR4 (Toll like receptor 4; Applied Biosystems, # Mm00445274_m1),
MCSF (macrophage colony stimulating factor; Applied Biosystems # Mm00432688_m1),
Adipor1 (Adiponection Receptor 1; Applied Biosystems, # Mm01291334_mH),
Adipor2 (Adiponectin Receptor 2; Applied Biosystems, # Mm01184031_g1),
ApoB (Apolipoprotein B; Applied Biosystems, # Mm01545156_m1),
Cpt1a (Carnitine palmitoyltransferase 1a, liver; Applied Biosystems, # Mm00550438_m1),
Foxo1 (forkhead box O1; Applied Biosystems, # Mm00490672_m1),
Fas (Fatty acid synthase; Applied Biosystems, # Mm01253300_g1),
IL-10 (Interleukin 10; Applied Systems, # Mm00439615_g1),
INFγ (Interferon gamma; Applied Systems, # Mm99999071_m1),
LXR (Nuclear receptor subfamily 1, group H, member 3; Applied Biosystems, # Mm00443454_m1),
PPARα (Peroxisome proliferator activated receptor alpha; Applied Biosystems, # Mm00440939_m1),

MTP (Solute carrier family 39 (iron-regulated transporter), member 1; Applied Biosystems, # Mm00489837_m1),
SREBP1c (sterol regulatory element binding transcription factor 1; Applied Biosystems, # Mm01138344_m1)
GAPDH (glyceraldehydes 3 phosphate dehydrogenase; Applied Biosystems, # Mm99999915_g1) was used to normalize the expressions.
Means were compared by a T-test. Significance was set at $p \leq 0.05$.

c. Confirmation of infection:

Antibody titer:

Presence of neutralizing antibodies in serum was determined by the 'constant virus-decreasing serum' method - a sensitive, specific and gold standard assay for determining neutralizing antibodies as described in detail(3). Briefly, heat inactivated test sera were serially diluted (two-fold) from 1:2 to 1:512 in 96-well plates. A total of 100 TCID-50 (Tissue cultur infectivity dose 50) of the respective adenovirus work stock was added to each of the wells, followed by the addition of A549 cells after 1 h of incubation at 37^{o}C. Each test serum was run in duplicate. Serum control (serum and cells, but no virus), cell control (cells alone, no virus, no serum), and virus control (cells and virus, no serum) were included with each assay. Plates were incubated at 37^{o}C for 13 days and the presence of CPE (cytopathic effect) was noted. Serum samples without CPE in dilutions of 1:8 or higher were considered positive for the presence of neutralizing antibodies to the respective virus and evidence of prior infection with that virus. Samples with titers lower than 1:8 were considered negative for the presence of viral antibodies. A virus back-titration was conducted with each assay as a quality check.

Screening for viral DNA and RNA:

1. **Conventional PCR:**

DNA isolation: DNA was isolated using a QIAMP DNA mini kit (# 51306). Primers were designed to E4 gene of Ad36, Ad2 and also for mouse β-actin. DNA was amplified by PCR. The primer sequences were as follows:

Ad36 forward primer: 5'-GGCATACTAACCCAGTCCGATG-3',
Ad36 reverse primer:5'-TCACTCTCAGCAGCAGCAGG-3';
Ad2 forward primer: 5'-CCTAGGCAGGAGGGTTTTTC-3',
Ad2 reverse primer: 5'-ATAGCCCGGGGGAATACATA-3'
Mouse β-actin forward primer: 5'-GATCTTCATGGTGCTAGGAG-3',
Mouse β-actin reverse primer: 5'-ACGTTGACATCCGTAAAGAC-3'.

Negative PCR control: water. Positive PCR control: DNA from Ad36 or Ad2 infected A549 cells. DNA was denatured for 2 min at 95^{0}C and subjected to 35 cycles of PCR (94^{0}C for 1 min, 55^{0}C for 1 min, 72^{0}C for 2 min followed by incubation at 72^{0}C for 5 mins. RNA was extracted using the RNeasy Mini kit as per the manufacturer's instructions (Qiagen, # 74101). Residual DNA was eliminated by using Amplification grade Deoxyribonuclease I (Invitrogen, # 18068-015). One μg of total RNA was reverse transcribed to cDNA using iscript^{TM} cDNA synthesis kit (BioRad, #170-8890) as per the manufacturer's protocol. PCR core system II (Promega, # M7665) was used for the amplification of cDNA.

2. Quantitative Real-Time PCR (qPCR):

Liver DNA isolated as described above was used for qPCR detection of viral DNA using SYBR green master mix from BioRad (Cat #170-8880). One hundred ng of DNA template per sample was amplified on the ABI prism 7900HT by qPCR using standard curve method by pooling samples of both DNA isolated from liver tissue from both infected and uninfected mice and from infected A549 cells. Standard curve was 100ng, 20ng, 4ng, 0.8ng, 0.16ng, 0.032ng, 0.0064ng, 0.00013ng, 0.00026ng DNA. The primer sequences listed above for Ad2 and Ad36 were used. Endogenous housekeeping gene primers used were as follows:

Mouse GAPDH Forward Primer: 5'-GATGCTAAATGGGCAGAAGC-3'

Mouse GAPDH Reverse Primer: 5'-CTGGCCCTCATAGCACACTT -3'.

Ad36 and Ad2 viral DNA in mice liver DNA were determined by both standard curve as well as the ΔΔ Ct methods. For both methods, quantities for Ad36 or Ad2 genes were normalized to the corresponding endogenous housekeeping gene (GAPDH). ΔΔ Ct for mice in Ad36 or Ad2 groups were calculated as fold viral DNA relative to the lowest amount in of viral DNA (Ct for viral gene - Ct for GAPDH) in the group. Value for viral DNA in one mouse was about 6000% greater - probably due to some artifact. This value was treated as an outlier after confirming by using Dixon's Q-test for detecting single outlier at $p<0.01$.

d. Glucose tolerance test:

Subsequent to a 16-h fast, conscious mice were injected with D-glucose (2.5 mg/g of body weight) intraperitoneally. Blood was collected from the tail vein prior to glucose injection (time 0) and at 10, 20, 30, 60, 120 and 150 min post-injection. Blood glucose was determined using a glucometer (Contour, Bayer).

e. Histochemistry:

Glycogen staining was performed on flash frozen liver samples of 3 mice each from Ad36, Ad2, and mock infected high fat fed mice, and one mock infected chow fed mouse as a control as reported (4). Tissue samples were embedded in OCT mounting medium and sliced at 8 um thickness. Glycogen was stained using periodic acid-schiff stain (PAS). Liver samples were fixed to enhance glycogen preservation and to help prevent streaming artifact in Carnoy's fixative (6 parts of ethanol, 3 parts of chloroform, and 1 part of glacial acetic acid) for 10 minutes at 4^{O} C. Upon fixation, sections were washed in distilled water with several rinses and then incubated in 1% period acid solution for 5 minutes at room temperature. After washing with distilled water, Schiff's reagent was added and incubated for 11 minutes. All slides were rinsed in cold running tap water for 10 minutes. Slides were air dried and a cover slip was applied using permount. Glycogen staining gives a magenta color to the section with a darker stain indicating more glycogen.

Lipid leaves the sample during fixation, and thus white blank area on the slide indicates lipid droplets (2). Images were made with a Zeiss Axioskop 40 FL. Three specimens per sample and three images per specimen were analyzed using Image J for quantification of glycogen and lipid.

Images were converted to 8 bit, and the threshold was determined where only glycogen specific staining was visible and the amount of glycogen was calculated as pixels^2 at this threshold. This number was subtracted from the total area to obtain the area of blank space for quantification of lipid as reported(2).

Experiment 3: Ad36 is associated with better glycemic control in humans:

A. HERITAGE (HEalth, RIsk factors, exercise Training And GEnetics) Family Study

The HERITAGE Family Study design, inclusion criteria, and protocol have been previously described(6). Serum neutralization assay was used to determine the presence of neutralizing antibodies to Ad36, as previously described(3) in 735 subjects. Of which, complete baseline Ad36 and IVGTT (intra-venous glucose tolerance test) data were available for 450 White subjects (220 men, 230 women) and 221 Black subjects (76 men, 145 women), which were included in statistical analyses. All subjects were healthy and sedentary at baseline. Sedentary was defined as no regular physical activity over the previous 6 months. The study protocol had been approved by the Institutional Review Boards at each of the five participating centers of the HERITAGE Family Study consortium. Written informed consent was obtained from each participant.

Measurement of glucose, insulin and intravenous glucose tolerance test (IVGTT) derived variables. A frequently sampled IVGTT (without intravenous insulin or tolbutamide injection) was administered in the morning after an overnight fast (12 hrs) as previously described(1). Insulin sensitivity (S_I), acute insulin response to glucose (AIR_g), disposition index (D_I) and glucose effectiveness (S_G) parameters were derived from the MINMOD Millennium software(5). Plasma glucose was measured enzymatically using a reagent kit (Diagnostic Chemicals Ltd., Oxford, Connecticut). Plasma insulin was determined using radioimmunoassay after polyethylene glycol separation.

Statistical analyses: The data distributions of for insulin sensitivity, acute insulin response to glucose and disposition index were skewed so the square root transformation was employed to achieve symmetry. Differences between Ad36 seropositivity and seronegativity with respect to the means of transformed insulin metabolism traits were tested using general linear models implemented in the SAS 9.1 software package. Age, sex, race, and body mass index were included in each model. The resulting least squares adjusted means were squared to transform back to the original scale. Two-sided 95% confidence limits were calculated in terms of the transformed data and also transformed back to the original scale where they were asymmetric about the estimated means. Significance was declared if $p \leq 0.05$ for one directional alternative hypotheses.

B. PBRC (Pennington Biomedical Research Center) Study.

Ad-36 seropositivity as an indicator for a natural viral infection, was determined post-hoc by serum neutralization assay as previously described(3) in baseline serum samples from 206 disease free normal, overweight or obese subjects participating in five different clinical trials conducted at PBRC. Each of the studies had been approved by the Institutional Review Board of

PBRC. Data and sample collection methods and assays conducted were consistent across all studies. Complete data for analyses were available from 206 subjects (males 47%, (Mean (SE) BMI: 29.5 (0.3) kg/m^2; Age: 34.3 (0.9) y). Height and weight were recorded to calculate BMI. Following a 12 hour overnight fast, subjects participated in a fasting blood draw, which was used for determining fasting glucose (Beckman DXC 600) and insulin (RIA, Immulite 2000) levels. Insulin resistance was determined by HOMA-IR and calculated as follows: (fasting glucose [mmol/L] x fasting insulin [mIU/L]) / 22.5. Body composition was determined by dual-energy X-ray absorptiometry (DXA, Hologic QDR 4500A whole-body scanner (Acclaim series, model number 010-0667, Hologic Inc., Bedford, MA), and intra-hepatic lipid (IHL) content was determined from liver density by multi-slice abdominal computed tomography of the abdomen at baseline (GE Light Speed, General Electric, Milwaukee, WI) as previously described(10). Briefly, CT measured hepatic density [Hounsfield units (HU)] was used as a direct measure of hepatic fat and was corrected for the spleen density by subtracting the spleen density from the liver density. Thus, higher HU value of liver density equates to lower lipid content. Prior studies comparing this method to hepatic quantitative proton magnetic resonance spectroscopy on a 1.5-T magnetic resonance imaging revealed a high concordance (R^2=0.81; unpublished observations, Larson-Myer, Smith and Newcomer).

Statistical analysis: The data distributions for fasting insulin, HOMA-IR and intra-hepatic lipid were skewed so the logarithmic transformation was employed to achieve symmetry. Differences between Ad36 seropositivity and seronegativity with respect to the means of fasting glucose, insulin, HOMA IR and intra-hepatic lipid were tested using general linear models implemented in the SAS 9.1 software package. Age, sex, race, and body fat were included in each model. For transformed data, the resulting least squares adjusted means were transformed back to the original scale. Two-sided 95% confidence limits were calculated in terms of the transformed data and also transformed back to the original scale where they were asymmetric about the estimated means. Significance was declared if $p \leq 0.05$ for one directional alternative hypotheses.

C. MET (Mechanisms of the Metabolic Syndrome in Prepubertal Youth) Study

The study's design, testing methodologies, data collection, exclusion and inclusion criteria, and recruitment methods were developed and tested for feasibility during a pilot study, "Study of Insulin Sensitivity in Louisiana Low, high or normal weight Youth (SILLY)" and the final protocols for this main trial were previously described (11, 12).

The protocol was previously approved by Institutional Review Boards at all study performance sites in New Orleans and Baton Rouge. Prior to enrollment in the study, legal guardians provided written informed consent and children provided written assent to participate. Baseline serum samples from 45 healthy, exclusively pre-pubertal children (32 Caucasian, 10 Black, 3 other races, 22 males, 23 females; age 7-9 years), (Mean (SE) BMI Z-score: 1.1 (0.1);) age: 8.2 (0.1)) were screened to determine the presence of neutralizing antibodies to Ad36, as previously described(3). Body composition was measured by DXA using a Hologic QDR 4500A (Bedford, MA). Following a 12 hour overnight fast, subjects participated in a fasting blood draw. Glucose was assayed via chemical reaction using an Ortho Clinical Diagnostics VITROS® 5,1 FS (Rochester, NY) and insulin using an EIA kit from ALPCO (Salem, NH). HOMA IR was calculated.

Statistical Analyses: The data distributions for fasting insulin, HOMA IR and intra-hepatic lipid were skewed so the logarithmic transformation was employed to achieve symmetry. Differences between Ad36 seropositivity and seronegativity with respect to the means of fasting glucose, insulin, HOMA IR and intra-hepatic lipid were tested using general linear models implemented in the SAS 9.1 software package. Age, sex, race, and body fat were included in each model. Following statistical testing in terms of transformed data where appropriate, the resulting least squares adjusted means were transformed back to the original scale. Two-sided 95% confidence limits were calculated in terms of the transformed data and also transformed back to the original scale where they were asymmetric about the estimated means. Significance was declared if $p \leq 0.05$ for one directional alternative hypotheses.

D. VIVA LA FAMILIA Study:

Available baseline serum samples from the Viva La Familia Study of non-obese and obese Hispanic children in Texas(7) were screened for the presence of Ad36 antibodies as previously described(3). The study protocol was previously approved by the Institutional Review Board for Human Subject Research for Baylor College of Medicine and Affiliated Hospitals and the Southwest Foundation for Biomedical Research. BMI was calculated from measured height and weight and body composition was determined by DXA with the use of a Hologic Delphi-A whole-body scanner (Hologic Inc, Waltham, MA). Following a 12 hour overnight fast, subjects participated in a fasting blood draw. Fasting insulin levels were measured with radioimmunoassay kit (Linco Research Inc, St Charles, MO), and fasting glucose was measured by glucose oxidase method. Insulin resistance was determined by HOMA IR. Baseline serum samples from 588 subjects were screened to determine the presence of neutralizing antibodies to Ad36, as previously described(3). Of which, complete data were available for 585 pre-pubertal children (289 males, 296 females; age range 4-19.9 years), (Mean (SE) BMI Z-score: 1.42 (0.04);) age: 10.9 (0.16) which were used for statistical analyses.

Statistical Analyses: The analysis was performed in two steps: (1) an general linear model analysis of the means and (2) a logistic regression model analysis of the proportion of children with glucose, insulin and HOMA levels below the respective medians. The data distributions for fasting insulin, and HOMA IR were skewed so in the first step the logarithmic transformation was employed to achieve symmetry. Differences between Ad36 seropositivity and seronegativity with respect to the means of fasting glucose, insulin, and HOMA IR were tested using general linear models implemented in the SAS 9.1 software package. Initially, age, sex, body fat and family identification were included in each model. Following statistical testing in terms of transformed data where appropriate, the resulting least squares adjusted means were transformed back to the original scale. Two-sided 95% confidence limits were calculated in terms of the transformed data and also transformed back to the original scale where they were asymmetric about the estimated means. Significance was declared if $p \leq 0.05$ for one directional alternative hypotheses.

In step 2, logistic regression analysis was implemented using the logistic procedure in SAS 9.1 to estimate the odds ratio for the levels being below the median for Ad36 seropositivity relative to the odds for seronegativity adjusted for sex, adiposity and family. Significance was declared if $p \leq 0.05$ for one directional alternative hypotheses.

Supplementary figure legends:

Figure S1: Body weights of HF-fed mice
Body weight and body fat matched mice on HF-diet were infected with Ad36, Ad2 or mock infected (week 0) and individually housed in microisolator cages in the same room. Body weights were determined weekly for the next 20 wks.

Figure S2: Ad36 improves serum glucose of HF-fed mice
A: Fasting serum glucose of HF-fed mice infected with Ad36, Ad2 or mock infected, adjusted to baseline. Mean $\pm$ SE. $p<0.05$ compared to mock group.
B: Free-fed serum glucose 20 week post inoculation. The horizontal line indicates median.

Figure S3: Ad36 DNA load in the liver is negatively correlated to glucose levels.
Free-fed glucose levels of animals at 20 wk pi negatively correlate with Ad36 DNA in livers of mice. ($r=-0.94$, $p=0.001$).

Figure S4: Ad36 neutralizing antibody titer is not correlated to glucose levels.
Free-fed glucose levels of animals at 20 wk pi were not correlated with Ad36 antibody titer in Ad36 infected mice. Antibody titer was determined semi-quantitatively and expressed as the dilution that effectively neutralized the virus. Higher titer indicates greater amount of neutralizing antibodies.

Figure S5: mRNA expression of cytokines from adipose tissue of HF-fed mice.
mRNA was isolated from adipose tissue of mice killed 20 week post inoculation (n=10 per group). Gene expressions were determined (2 replicates / mouse) by qRT-PCR and normalized to GAPDH expression. Mean + SE. ** $p<0.005$; * $p<0.05$ or better, compared to mock group.
A: TNFα, **B:** MCP1, **C:** CD68. **D:** TLR4, **E:** IL6, **F:** MCSF **G:** Resistin.

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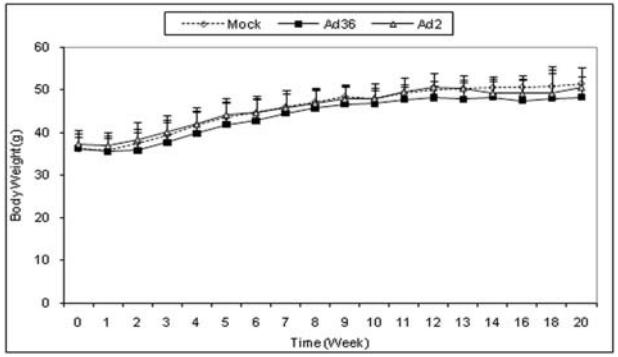
Table S1: Percent of the mice showing viral antibodies, viral DNA & RNA.

Group	Antibody Ad36 / Ad2	viral DNA / viral RNA			
		Liver	Adipose tissue	Lung	Kidney
Experiment 2: Chow-fed mice					
Mock		0/0		0/0	
Ad36		100 / 100		100 / 100	
Ad2		100 / 100		100 / 100	
Experiment 3: Mice on high fat diet					
Mock	0 / 0	0 / 0	0 / 0	0 / NA	0 / NA
Ad36	100 / 0	60 / 60	70 / 40	50 / NA	10 / NA
Ad2	0 / 40	0 / 0	0 / 0	100 / NA	50 / NA

NA: Viral RNA not determined in these samples.


Mock
Ad36
Ad2
Body Weight (g)
0
10
20
30
40
50
60
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 16 18 20
Time (Week)

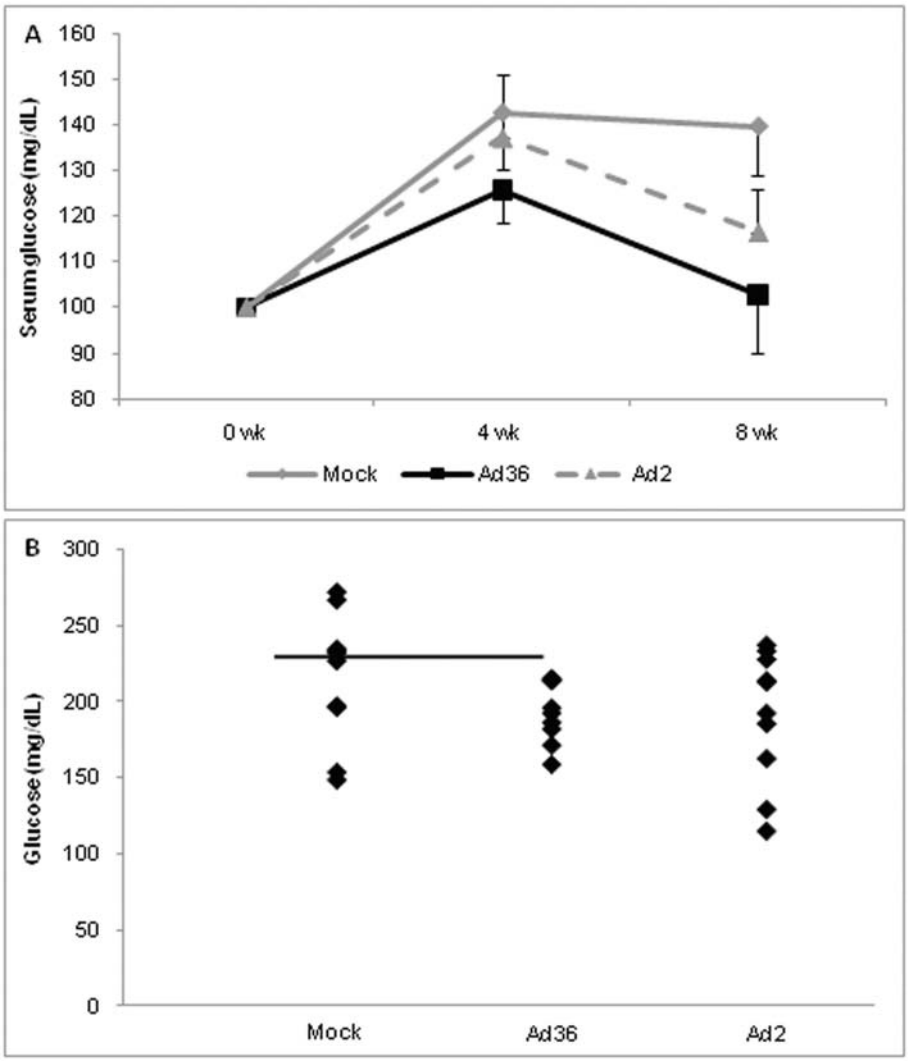

A
160
150
140
130
120
110
100
90
80
Serum glucose (mg/dL)
0 wk
4 wk
8 wk
Mock
Ad36
Ad2
B
300
250
200
150
100
50
0
Glucose (mg/dL)
Mock
Ad36
Ad2

Figure S3: Ad36 DNA load in the liver is negatively correlated to glucose levels.

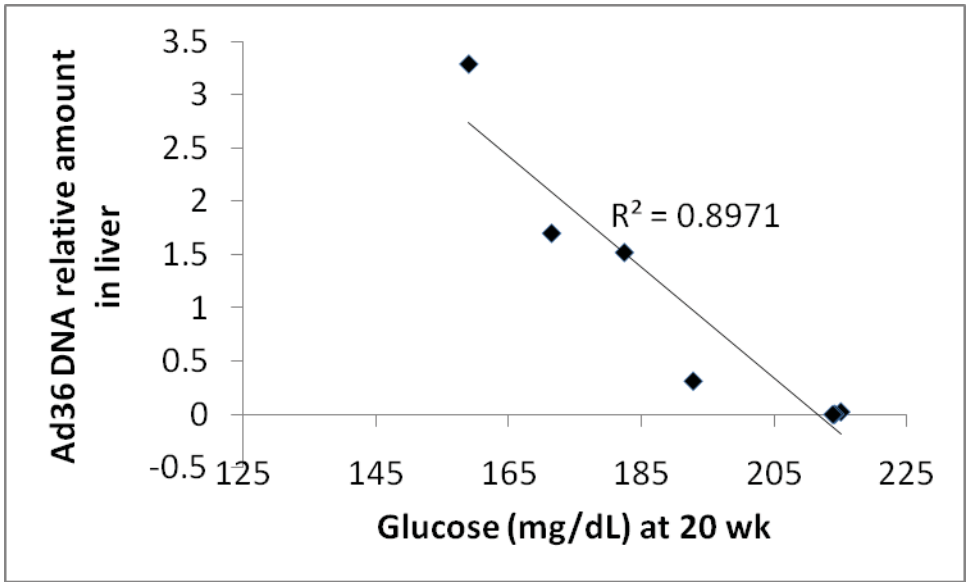


Figure S4: Ad36 antibody titer is not correlated to glucose levels.

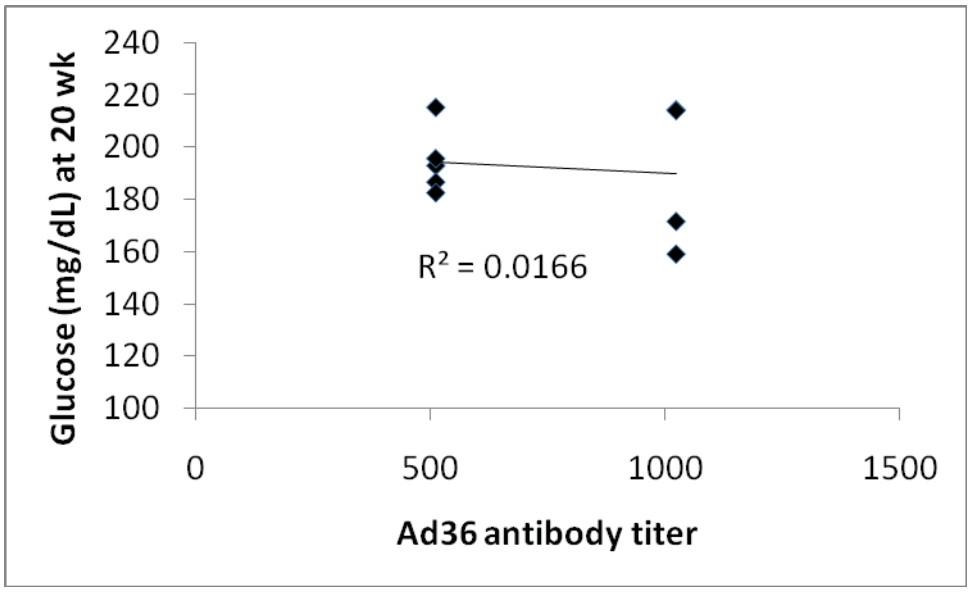


Figure S5A: Adipose tissue TNFα mRNA expression in HF-fed mice. Mean ± SE.

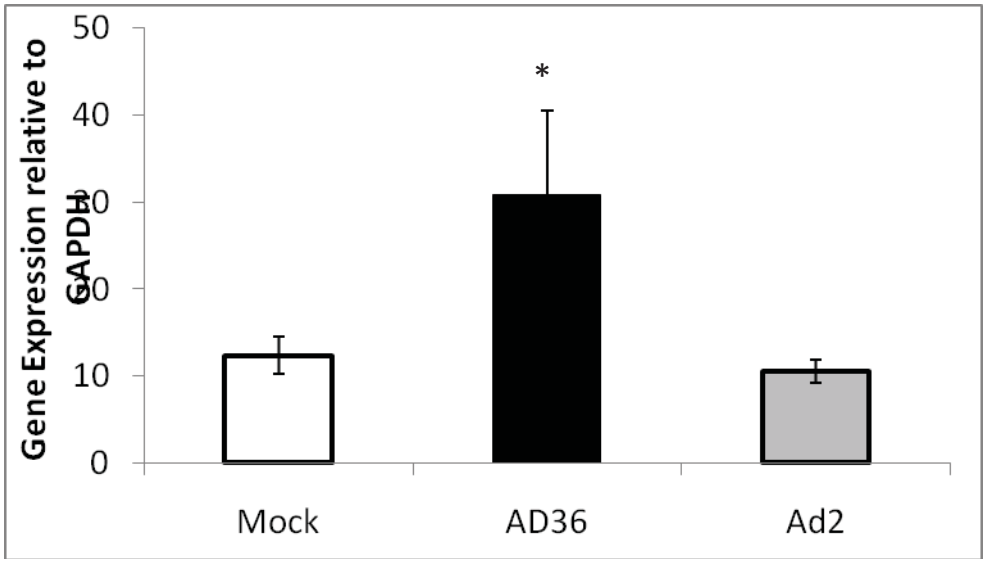


Figure S5B: Adipose tissue MCP1 mRNA expression in HF-fed mice. Mean ± SE.

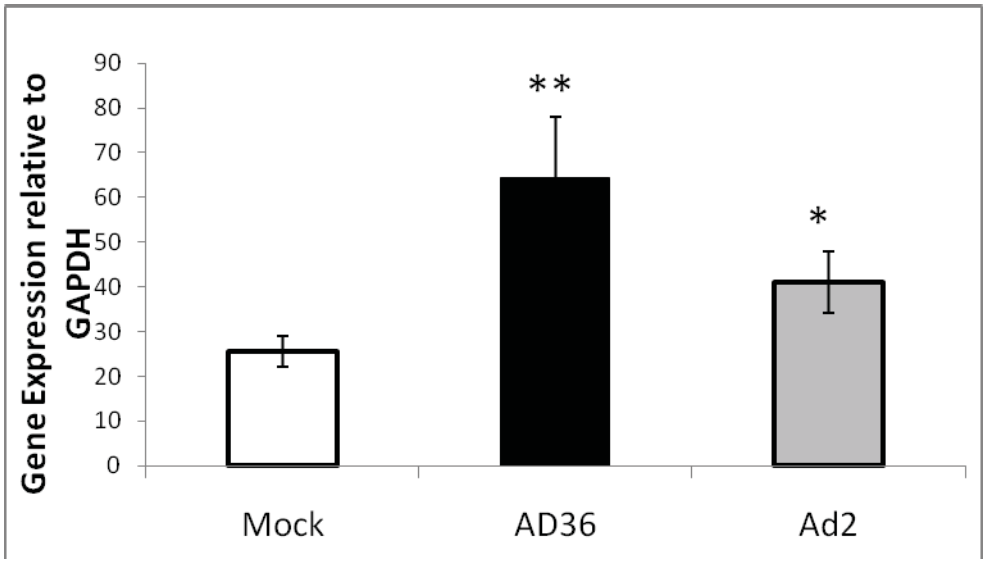


Figure S5C: Adipose tissue CD68 mRNA expression in HF-fed mice. Mean ± SE.

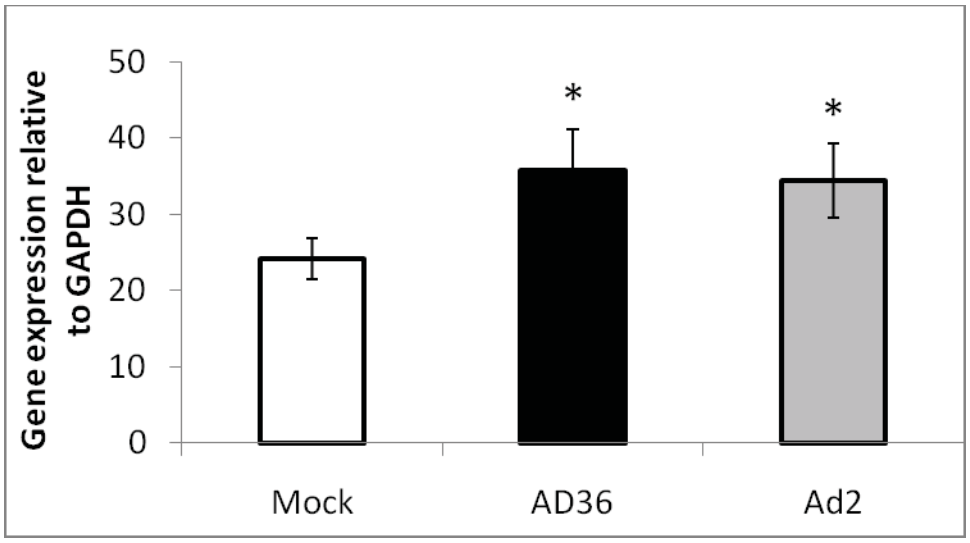


Figure S5D: Adipose tissue TLR4 mRNA expression in HF-fed mice. Mean ± SE.

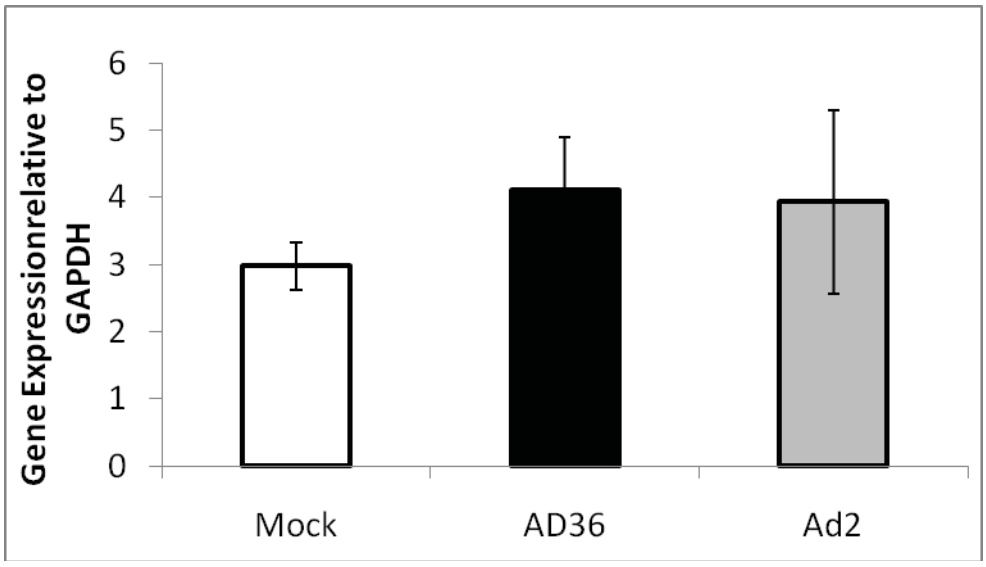


Figure S5E: Adipose tissue IL6 mRNA expression in HF-fed mice. Mean ± SE.

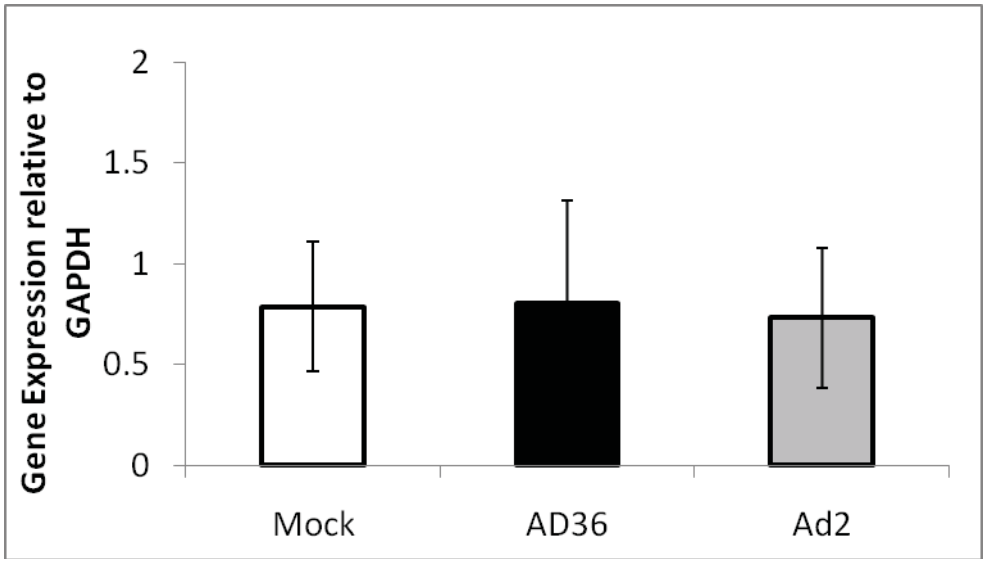


Figure S5F: Adipose tissue MCSF mRNA expression in HF-fed mice. Mean ± SE.

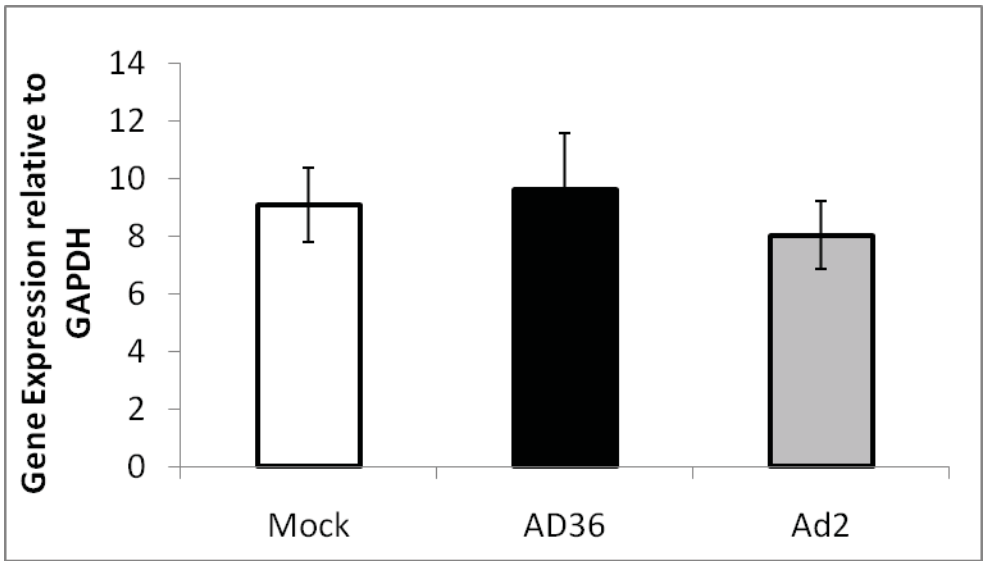


Figure S5G: Adipose tissue Resistin mRNA expression in HF-fed mice. Mean ± SE.