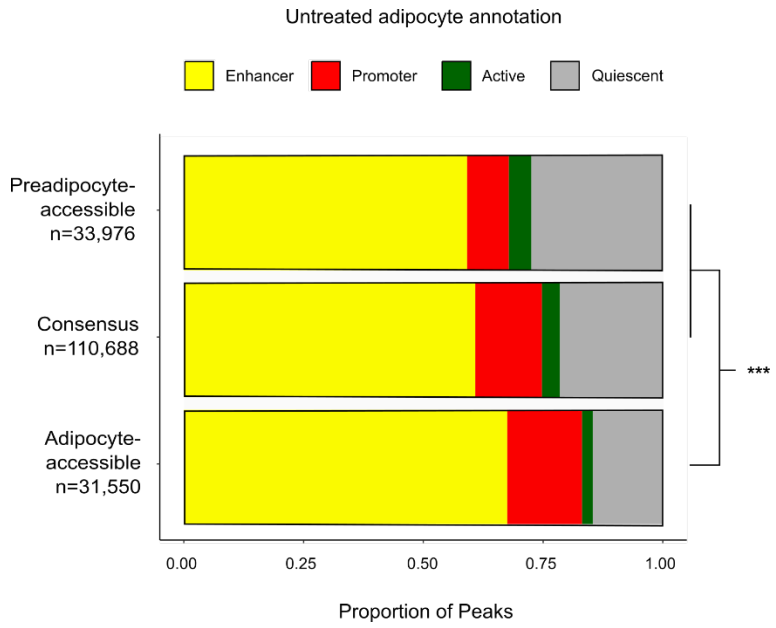
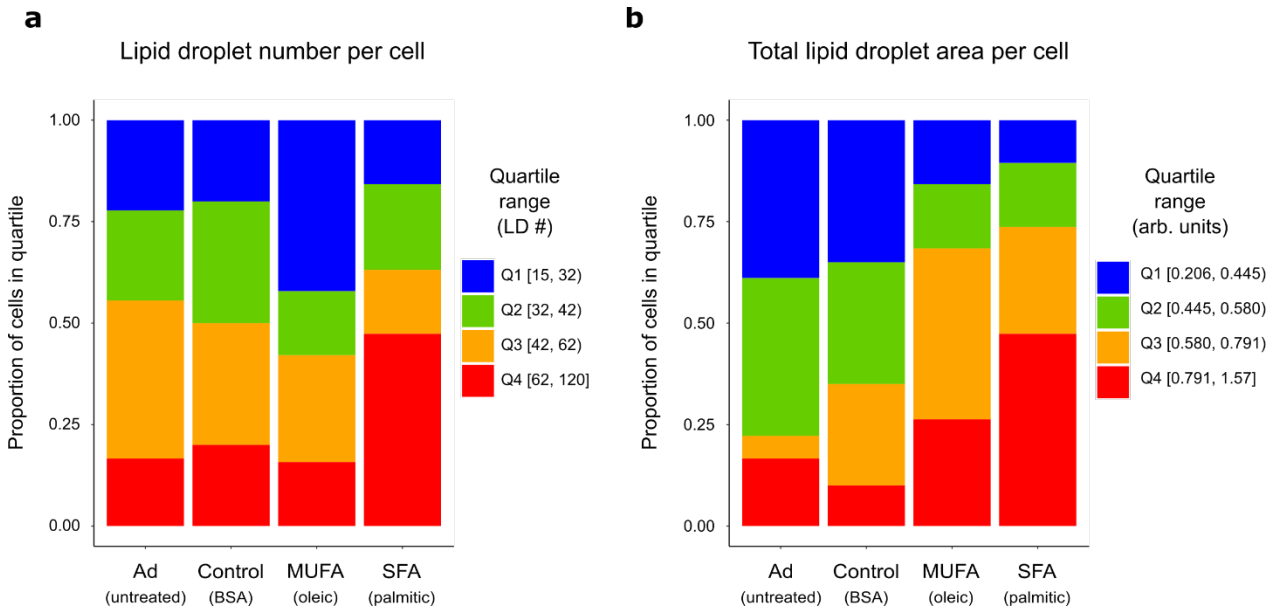
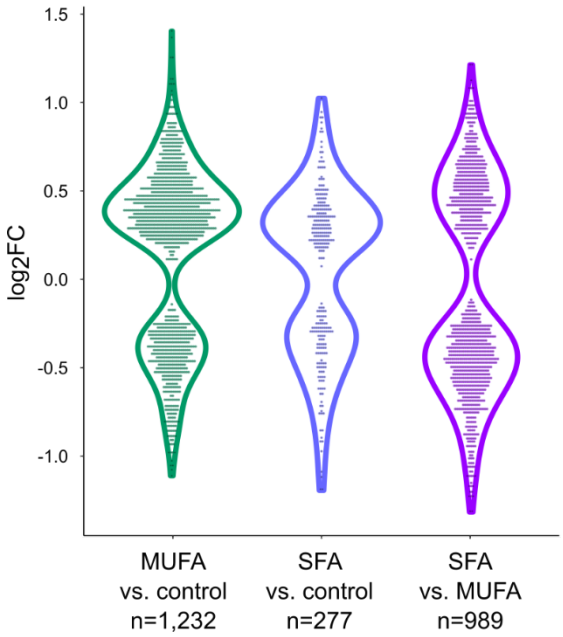


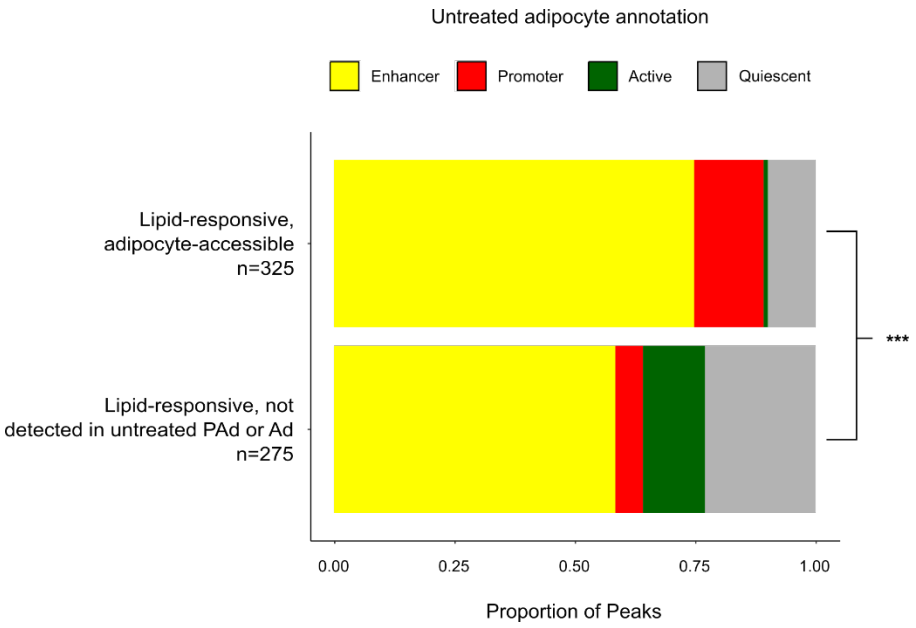
Supplementary Figure 1. Adipocyte-accessible peaks fall more into adipocyte enhancers and promoters than the preadipocyte-accessible peaks or the full peak set. ATAC-seq peaks from the indicated peak sets on the y-axis are distributed among four subsets of functional annotations from the 25-state imputed chromHMM[1] annotations from mesenchymal stem cell derived cultured adipocytes. Note that not all peaks were categorized into one of these 4 categories due to minimum peak proportion overlap (>50%) requirement not being met. ***depicts the *p*-value ($p < 1x10^{-5}$) for the chi-square test for independence between the distributions of peaks in the indicated annotations. Related to Figure 1.



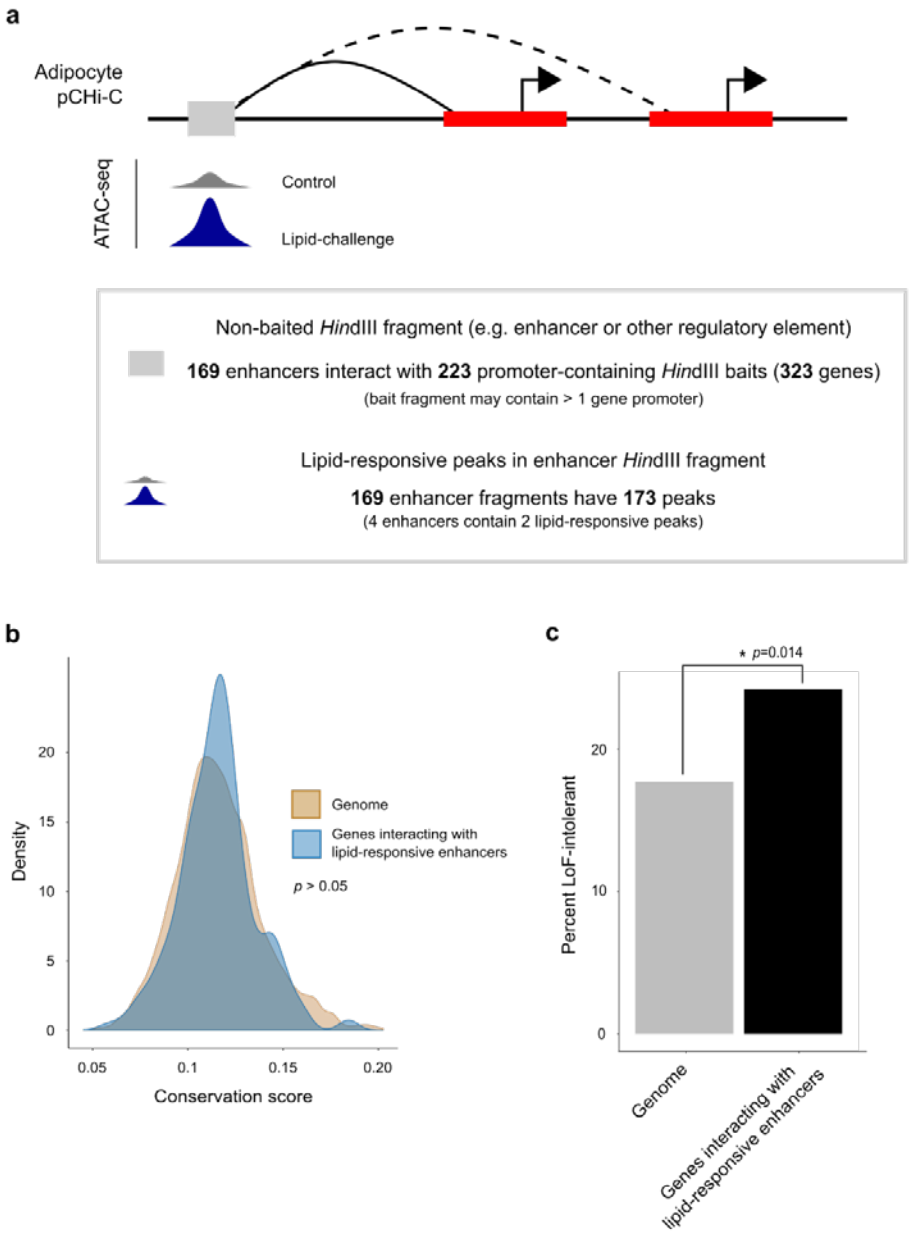
Supplementary Figure 2. Fatty acid lipid challenge in human adipocytes leads to increased storage of lipids in lipid droplets. (**a,b**) The proportion of cells in each of the indicated quartiles are reported for (**a**) lipid droplet (LD) number per cell, and (**b**) total LD area per cell, quantified from oil red o staining. Treatment with monounsaturated fatty acid (MUFA) leads to increased total area of LD but fewer total LDs (e.g. large LDs). Treatment with saturated fatty acid (SFA) leads to increased LD number and size. Data presented are from one representative experiment out of two independent experiments with similar results. Related to Figure 2.



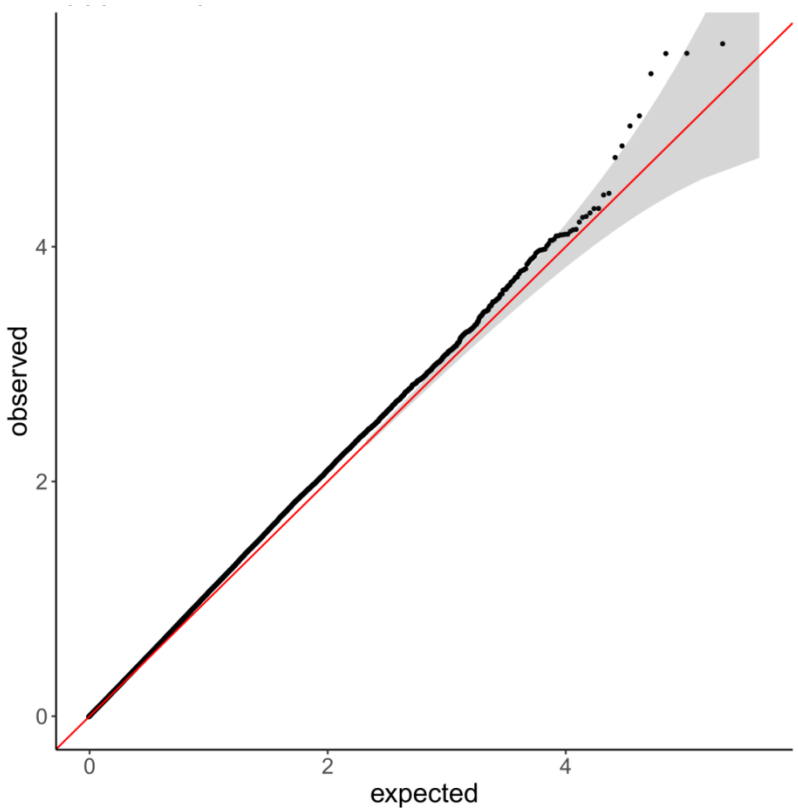
Supplementary Figure 3. Violin plots show the distribution of log2 fold-change (log2FC) for all differentially accessible peaks from the lipid challenge in adipocytes. Peaks were considered differentially accessible at a cutoff of FDR < 0.05. FDR was calculated (adjusting for n=122,252 ATAC-seq peaks) from the *p*-values of the QL F-test (see Methods) in the one-way ANOVA. For the *post hoc* test to determine which comparison was significant after the one-way ANOVA (OA vs. BSA, PA vs. BSA, or OA vs. PA), we determined the least significant difference. The violin plot characteristics are as follows: MUFA vs. ctrl (n=1,232) range: -1.11 – 1.40; median: 0.32; 25th percentile: -0.32; and 75th percentile: 0.47. SFA vs. ctrl (n=277) range: -1.19 – 1.02; median: 0.22; 25th percentile: -0.30; and 75th percentile: 0.37. SFA vs. MUFA (n=989) range: -1.31 – 1.21; median: -0.27; 25th percentile: -0.51; and 75th percentile: 0.47. MUFA indicates monounsaturated fatty acid; SFA, saturated fatty acid. Related to Figure 2.



Supplementary Figure 4. Lipid-responsive peaks in adipocyte-accessible regions fall more into adipocyte enhancers and promoters than lipid-responsive peaks in context-dependent regions. Lipid-responsive ATAC-seq peaks from the indicated peak sets on the y-axis are distributed among four subsets of functional annotations from the 25-state imputed chromHMM[1] annotations from mesenchymal stem cell derived culture adipocytes. Note that not all peaks were categorized into one of these 4 categories due to minimum peak proportion overlap (>50%) requirement not being met. ***depicts the *p*-value ($p < 1x10^{-5}$) for the chi-square test for independence between the distributions of peaks in the indicated annotations. Related to Figure 2.



Supplementary Figure 5. The 323 genes with promoters that interact with lipid-responsive enhancers exhibit constraints on loss-of-function mutations. (**a**) Schematic overview of the lipid-responsive sites in non-baited *Hin*dIII fragments from the adipocyte pCHi-C interactions. These data were integrated to identify the 323 gene promoters that interact with lipid-responsive enhancers in adipocytes. (**b**) Density plot shows the distribution of per-gene average conservation scores across placental mammals[2] for all protein-coding genes in the genome compared to all protein-coding genes in the set of 323 genes whose promoters interact with lipid-responsive enhancers. The two-sided Wilcoxon signed-rank test returned a non-significant *p*-value > 0.05. (**c**) Bar graph shows the proportion of protein-coding genes that are loss-of-function intolerant (i.e. are unlikely to have protein-truncating variants in humans)[3] in the whole genome (n=3,204/18,122; 17.7%) compared to the protein-coding genes among the 323 genes (n=50/207; 24.2%). LoF indicates loss-of-function; *depicts the *p*-value for the hypergeometric enrichment test. Compare with Figure 3 results for 154 genes with lipid-responsive promoters.



Supplementary Figure 6. Testing all SNPs genome-wide for gene-by-saturated fat intake effect on BMI does not show inflation or result in significant GxEs at the genome-wide significance threshold. We tested the SNPs that are not in the same LD block ($r^2 < 0.2$) genome-wide for a GxE between each SNP and saturated fat intake effect on BMI. There were a total of 211,187 SNPs and 167,908 individuals with no missing data available for study (see Equation 1 in the Methods). The Q-Q plot shows the observed *p*-values of β_{GE}, the expected p-values (red line) based on the multiple testing, and the 95% confidence interval (shaded area).

Supplementary Table 1. Sequencing, read processing, and QC metrics for untreated preadipocyte and adipocyte ATAC-seq.

	Reads	Uniquely aligned	Fraction uniquely aligned	Paired and filtered	De-duplicated	Fraction duplicates	Final Reads	Fraction mtDNA	Fraction reads in peaks
PAd rep1	23,240,008	16,960,464	0.73	20,403,898	16,707,418	0.18	15,810,810	0.019	0.48
PAd rep2	25,705,628	18,552,651	0.72	22,516,812	17,829,509	0.21	16,938,123	0.016	0.46
PAd rep3	27,548,038	19,008,502	0.69	23,815,866	19,142,102	0.20	17,984,628	0.025	0.47
Ad rep1	24,407,273	16,893,473	0.69	21,049,924	16,045,835	0.24	15,038,768	0.033	0.58
Ad rep2	18,190,931	12,109,272	0.67	15,537,582	11,344,211	0.27	10,459,715	0.049	0.53
Ad rep3	21,165,864	13,976,310	0.66	17,956,825	12,512,132	0.30	11,528,741	0.050	0.70

PAd indicates preadipocyte; Ad, adipocyte; mtDNA, mitochondrial DNA.

Supplementary Table 3. Sequencing, read processing, and QC metrics for adipocyte lipid-challenge ATAC-seq.

	Reads	Uniquely aligned	Fraction uniquely aligned	Paired and filtered	De-duplicated	Fraction duplicates	Final Reads	Fraction mtDNA	Fraction reads in peaks
BSA rep1	64,706,941	48,768,335	0.76	56,012,019	45,623,026	0.185	44,927,691	0.012	0.66
BSA rep2	24,133,180	18,644,383	0.77	21,149,557	17,972,138	0.150	17,655,287	0.015	0.66
BSA rep3	23,981,457	17,668,336	0.74	20,677,446	17,553,162	0.151	17,214,846	0.016	0.59
OA rep1	46,775,100	35,874,412	0.77	40,975,870	29,562,701	0.279	29,007,522	0.016	0.70
OA rep2	57,372,688	44,971,629	0.79	51,073,123	39,246,428	0.232	38,742,718	0.010	0.71
OA rep3	33,118,575	25,516,115	0.77	29,143,404	23,401,224	0.197	22,995,249	0.014	0.67
PA rep1	27,688,462	21,034,204	0.76	24,089,236	18,199,958	0.244	17,822,393	0.018	0.64
PA rep2	45,904,086	35,705,224	0.78	40,386,681	31,318,153	0.225	30,874,420	0.011	0.68
PA rep3	39,159,661	29,810,602	0.76	34,234,012	26,610,078	0.223	26,074,118	0.017	0.68

BSA indicates bovine serum albumin; OA, oleic acid; PA, palmitic acid; mtDNA, mitochondrial DNA.

Supplementary Table 5. Sequencing and read processing metrics for adipocyte lipid-challenge pCHi-C.

	Reads	Uniquely mapped and paired	Unique ditags	Cis-close	Cis-far	Trans
BSA rep1	156,781,294	115,665,713	69,664,503	10,560,429	49,905,501	9,198,573
BSA rep2	126,772,704	94,717,631	51,631,011	8,335,003	36,631,083	6,664,925
OA rep1	120,985,559	90,005,610	54,155,183	7,477,013	38,909,160	7,769,010
OA rep2	118,632,574	89,101,812	49,035,437	8,495,017	34,445,736	6,094,684
PA rep1	132,242,479	98,448,728	54,891,280	8,392,648	39,146,001	7,352,631
PA rep2	107,001,633	80,596,383	44,405,325	6,636,844	31,576,330	6,192,151

BSA indicates bovine serum albumin; OA, oleic acid; PA, palmitic acid.

Supplementary Table 6. The top 10 TF motifs enriched in adipocyte lipid-responsive open chromatin regions in chromosomal interactions.

Motif logo	Motif name	*p*-value	Adjusted *p*-value	Number of target sequences with motif (of 264)	Percent of target sequences with motif	Number of background sequences with motif (of 30,704)	Percent of background sequences with motif
	RUNX-AML(Runt)/CD4+-PolII-ChIP-Seq(Barski_et_al.)/Homer	$1.0x10^{-8}$	0	134.0	50.76%	10267.5	33.44%
	STAT4(Stat)/CD4-Stat4-ChIP-Seq(GSE22104)/Homer	$1.0x10^{-8}$	0	166.0	62.88%	13952.6	45.44%
	PPARE(NR),DR1/3T3L1-Pparg-ChIP-Seq(GSE13511)/Homer	$1.0x10^{-6}$	0	162.0	61.36%	14012.1	45.64%
	RXR(NR),DR1/3T3L1-RXR-ChIP-Seq(GSE13511)/Homer	$1.0x10^{-6}$	0	173.0	65.53%	15537.1	50.60%
	ZBTB18(Zf)/HEK293-ZBTB18.GFP-ChIP-Seq(GSE58341)/Homer	$1.0x10^{-6}$	$1.0x10^{-4}$	108.0	40.91%	8317.5	27.09%
	THRa(NR)/C17.2-THRa-ChIP-Seq(GSE38347)/Homer	$1.0x10^{-5}$	$1.0x10^{-4}$	105.0	39.77%	8090.0	26.35%
	KLF10(Zf)/HEK293-KLF10.GFP-ChIP-Seq(GSE58341)/Homer	$1.0x10^{-5}$	$1.0x10^{-4}$	108.0	40.91%	8567.6	27.90%
	Srebp1a(bHLH)/HepG2-Srebp1a-ChIP-Seq(GSE31477)/Homer	$1.0x10^{-5}$	$1.0x10^{-4}$	59.0	22.35%	3792.7	12.35%
	NF1-halfsite(CTF)/LNCaP-NF1-ChIP-Seq(Unpublished)/Homer	$1.0x10^{-4}$	$3.0x10^{-4}$	215.0	81.44%	21406.9	69.72%
	NPAS2(bHLH)/Liver-NPAS2-ChIP-Seq(GSE39860)/Homer	$1.0x10^{-4}$	$3.0x10^{-4}$	152.0	57.58%	13632.6	44.40%

Enrichment *p*-values were derived from the hypergeometric enrichment test of proportion of the given TF motif in the peak set [lipid-responsive open chromatin regions in adipocyte chromosomal interactions (n=264)] compared with the background set of peaks [all non-lipid-responsive peaks in adipocyte chromosomal interactions (n=30,704)], adjusted (Benjamini-Hochberg) for the number of known motifs tested (n=364)[4]. The top 10 enriched TF motifs in include key TFs in lipid metabolism, such as the co-factors PPARG and RXR. Related to Figure 3.

Supplementary Table 8. KEGG pathway enrichment analysis of 154 genes with lipid-responsive promoters.

KEGG pathway	Ratio of enrichment	Number of genes	Genes in pathway	FDR
Glycine, serine and threonine metabolism	13.96	5	*AGXT2* *AOC2* *AOC3* *GLYCTK* *MAOA*	0.0072
Phenylalanine metabolism	23.46	3	*AOC2* *AOC3* *MAOA*	0.036

The 154 genes with lipid-responsive promoters in adipocyte chromosomal interactions were tested for KEGG pathway enrichment using WebGetstalt[5], using all genes that were involved in adipocyte chromosomal interactions (n=17,052) as the background set. The FDR is calculated from the *p*-values of the hypergeometric test, adjusted for the number of pathways tested through WebGestalt. Related to Figure 3.

Supplementary Table 10. Three lipid-responsive ATAC-peaks in interacting enhancers overlap with GWAS SNPs[6] for serum lipid traits.

peakChr	peakStart	peakEnd	intBaitGene[¶]	SNP in peak	MAF[†]	Associated trait[6]	*p*-value[6]	Index SNP[6]	LD with index SNP[†] (r^2)
7	73015109	73016308	Bait1: *WBSCR22* Bait2: *STX1A*	rs34346326	0.2	TG	1.31e-44	rs17145738	0.5
15	58591111	58592050	Bait1: *ADAM10* Bait2: *RP11-30K9.7/ U3.10*	rs12899879	0.14	HDL	3.55e-09	rs1532085	-
10	113902081	113908608	*ADRA2A*	rs2792744	0.28	TC	2.73e-09	rs2255141	0.77

[¶]The genes listed are the promoters in the baited *Hin*dIII fragment with which the lipid-responsive enhances interact. More than one bait is listed when the lipid-responsive enhancer is interacting with more than one bait in the adipocyte pCHi-C; [†]Minor allele frequency (MAF) is the European frequency from the 1000 Genomes Project. Linkage disequilibrium (LD) is calculated based on Europeans in the 1000 Genomes Project; LD calculations > 0.2 are reported. Lipid-responsive ATAC-seq peaks that land in enhancers within adipocyte chromosomal interactions (n=173) were assessed for whether they contain GWAS SNPs for serum lipid traits from the meta-GWAS performed in Willer et al.[6]

Supplementary Table 11. Lipid-responsive gene promoters with GWAS SNPs respond to SFA treatment.

peakChr	peakStart	peakEnd	gene-pCHi-C bait	log_2FC (MUFA/ctrl)	log_2FC (SFA/ctrl)	log_2FC (SFA/MUFA)
11	61594652	61596828	*FADS2/ FADS1*	n.s	n.s	**0.37**
7	73036880	73038991	*MLXIPL*	n.s.	n.s	**0.29**
2	27432323	27432971	*SLC5A6/ ATRAID*	n.s	n.s	**0.33**
16	68115758	68116375	*NFATC3*	n.s	n.s	**0.48**
19	10981139	10983631	*CARM1*	n.s	**0.24**	n.s

MUFA indicates monounsaturated fatty acid; SFA, saturated fatty acid; ctrl, control; n.s., non-significant (based on the *post hoc* test of the one-way ANOVA, see below). Lipid-responsive ATAC-seq peaks that land in promoters within adipocyte chromosomal interactions (n=91) were assessed for whether they contain GWAS SNPs for serum lipid traits from the meta-GWAS performed in Willer et al.[6] The direction of the ATAC-seq differential accessibility effect was then assessed based on the quality (e.g. SFA or MUFA) of the fatty acid. Differential accessibility was evaluated at an FDR cutoff of 0.05. FDR was calculated (adjusting for n=122,252 ATAC-seq peaks) from the *p*-values of the QL F-test (see Methods) in the one-way ANOVA. For the *post hoc* test to determine which comparison was significant after the one-way ANOVA (MUFA vs. ctrl, SFA vs. ctrl, or SFA vs. MUFA), we determined the least significant difference. The lipid-responsive gene promoters in chromosomal interactions that contain GWAS SNPs exhibit increased accessibility in palmitic acid (saturated fatty acid) lipid challenge. Related to Table 1.

Supplementary Table 12. Lipid-responsive enhancers with GWAS SNPs stratified by quality of fatty acid.

peakChr	peakStart	peakEnd	intBaitGene[¶]	log_2FC (MUFA/ctrl)	log_2FC (SFA/ctrl)	log_2FC (SFA/MUFA)
7	73015109	73016308	Bait1: *WBSCR22* Bait2: *STX1A*	n.s	**0.45**	**0.64**
15	58591111	58592050	Bait1: *ADAM10* Bait2: *RP11-30K9.7/ U3.101*	**0.45**	n.s	**-0.28**
10	113902081	113908608	*ADRA2A*	n.s	**0.50**	n.s.

[¶]The genes listed are the promoters in the baited *Hin*dIII fragment with which the lipid-responsive enhancers interact. More than one bait is listed when the lipid-responsive enhancer is interacting with more than one bait in the adipocyte pCHi-C; MUFA indicates monounsaturated fatty acid; SFA, saturated fatty acid; ctrl, control; n.s., non-significant (based on the *post hoc* test of the one-way ANOVA, see below). Lipid-responsive ATAC-seq peaks that land in enhancers within adipocyte chromosomal interactions (n=173) were assessed for whether they contain GWAS SNPs for serum lipid traits from the meta-GWAS performed in Willer et al.[6] The direction of the effect was then assessed based on the quality (e.g. SFA or MUFA) of the fatty acid. Differential accessibility was evaluated at an FDR cutoff of 0.05. FDR was calculated (adjusting for n=122,252 ATAC-seq peaks) from the *p*-values of the QL F-test (see Methods) in the one-way ANOVA. For the *post hoc* test to determine which comparison was significant after the one-way ANOVA (MUFA vs. ctrl, SFA vs. ctrl, or SFA vs. MUFA), we determined the least significant difference. The lipid-responsive enhancers in chromosomal interactions that contain GWAS SNPs are more often differentially accessible in palmitic acid (saturated fatty acid) lipid challenge. Related to Supplementary Table 10.

Supplementary Table 13. LDSC analysis[7] of SNPs in *cis* regions of the 154 lipid-responsive promoters.

Category	Proportion of SNPs	Proportion of h^2	Proportion of h^2 SE	Enrichment	Enrichment SE	Enrichment *p*-value
TC	0.029	0.086	0.021	2.95	0.74	0.0088
LDL-C	0.029	0.084	0.026	2.91	0.91	0.038
HDL-C	0.029	0.083	0.027	2.87	0.92	0.042
Serum TG	0.029	0.085	0.026	2.93	0.90	0.045

SE indicates standard error; h^2, heritability; TC, total cholesterol; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; TG, triglycerides. LD score regression (LDSC)[7] was performed using the SNPs in the *cis* regions (gene body +/- 500 kb) of the 154 genes with lipid-responsive promoters in adipocyte chromosomal interactions; and the serum lipid trait summary statistics from the meta-GWAS performed in Willer et al.[6] The enrichment *p*-value and the SE for the proportion of h^2 and enrichment were calculated from block jackknife resampling used in the LDSC method. The *p*-value reported is not adjusted for multiple tests as these serum lipid traits are highly correlated. The *cis* regions of the 154 genes with lipid-responsive promoters in adipocyte chromosomal interactions contribute significantly to the heritability of serum lipid traits in humans.

Supplementary Table 14. LDSC analysis[7] of SNPs in *cis* regions of genes with lipid-responsive enhancers.

Category	Proportion of SNPs	Proportion of h^2	Proportion of h^2 SE	Enrichment	Enrichment SE	Enrichment *p*-value
TC	0.055	0.078	0.015	1.41	0.28	0.12
LDL-C	0.055	0.057	0.0089	1.03	0.16	0.86
HDL-C	0.055	0.11	0.020	1.94	0.36	0.011
Serum TG	0.055	0.12	0.041	2.10	0.74	0.15

SE indicates standard error; h^2, heritability; TC, total cholesterol; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; TG, triglycerides. LD score regression (LDSC)[7] was performed using the SNPs in the *cis* regions (gene body +/- 500 kb) of the 323 genes with promoters that interact with lipid-responsive enhancers in adipocyte chromosomal interactions; and serum lipid trait summary statistics from the meta-GWAS performed in Willer et al.[6] The enrichment *p*-value and the SE for the proportion of h^2 and enrichment were calculated from block jackknife resampling used in the LDSC method. The *p*-value reported is not adjusted for multiple tests as these serum lipid traits are highly correlated. The *cis* regions of the 323 genes that interact with lipid-responsive enhancers in adipocytes contribute significantly to the heritability of HDL, but not the other serum lipid traits, in humans.

Supplementary Table 16. Significant GxE promoter SNPs with LD proxies.

SNP	*p*-g	*p*-g*e	β-g	β-g*e	Genes in Bait	*cis*-eQTL FDR[8†]	Target Gene[8]	log_2FC[8]
rs1974817	0.0089	0.0010	2.3	-0.089	*GLTSCR2/*	2.4E-31	*SEPW1*	0.73
rs2334290	0.22	0.0011	-1.65	0.088	*SNORD23*	1.2E-08	*SEPW1*	0.46
rs112438892[¶]	0.0017	0.0050	-0.50	0.012	*CARM1*	2.7E-05/ 0.015	*SMARCA4/ ICAM4*	-0.28/ 0.38
rs117569851[¶]	0.0037	0.016	0.461	-0.011		0.0014/ 0.046	*SMARCA4/ KRI1*	-0.21/ 0.18
rs35678764	0.028	0.025	-5.7	0.19				
rs66516040[±]	0.034	0.026	0.19	-0.0057	*RDH8/COL5A3*	0.012	*OLFM2*	-0.67
rs9797822	0.044	0.039	5.2	-0.17		0.047	*PIN1*	-0.24
rs10788522	0.013	0.027	3.9	-0.14	*LDB3*	6.1E-04	*LDB3*	0.42
rs2354358	0.033	0.042	-3.5	0.14		6.1E-04	*LDB3*	0.42
rs867773	0.31	0.033	-2.0	0.11	*PLIN2*	0.048	*PLIN2*	-0.39
rs12379376	0.19	0.034	2.1	-0.11		0.048	*PLIN2*	-0.39

[†]The *cis*-eQTLs were identified in the adipose tissue from the METSIM cohort[8,9]. [±]This SNP is the only genome-wide significant *cis*-eQTL from the set of GxE SNPs with LD $r^2 > 0.2$ in the lipid-responsive peak. [¶]These GxE SNPs are *cis*-eQTLs for more than one gene. For 5 of the significant promoter GxE SNPs listed in Table 2, SNPs with LD $r^2 > 0.2$ in the lipid-responsive region that also exhibited a significant GxE effect of saturated fat intake on BMI are listed. Redundant SNPs are listed together in order of more to less significant. The reported *p*-values are from the βs in the multi-variable linear model (see Equation 2 in the Methods), where g is the number of minor alleles of the genotype and e is saturated fat intake. Here *p*-g indicates the *p*-value for the genotype effect; *p*-g*e, the *p*-value for the GxE effect; beta values follow the same notation. For the multi-variable linear model, there were a total of 290 SNPs and 38,394 individuals with no missing data available for study. Related to Table 2.

Supplementary Table 21. EMSA oligo probes used for analysis of GxE SNP rs10788522.

Probe Name	Probe Sequence
rs10788522 FWD labeled	biotin - 5'- TCTGGGGAGAGGAAG**G/A**GGGACAGGCTGAGAC - 3'
rs10788522 FWD unlabeled	5'- TCTGGGGAGAGGAAG**G/A**GGGACAGGCTGAGAC - 3'
rs10788522 REV unlabeled	5' - GTCTCAGCCTGTCCC**C/T**CTTCCTCTCCCCAGA - 3'

Oligonucleotides were designed to target the GxE SNP rs10788522 in the *LDB3* promoter *Hin*dIII fragment (+/- 15 bp).

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