

Supporting information

Table S1. Primer sequences

Genes	Primer	Sequence
GLUT4	Forward	GCTTTGTGGCCTTCTTTGAG
	Reverse	CAGGAGGACGGCAAATAGAA
CPT1	Forward	GCGCTCTTAGGACTACTTGCTAACC
	Reverse	ACTGGAGACCTGAGAGAGGAATGT
PPARgamma	Forward	GAAAGACAACGGACAAATCACC
	Reverse	GGGGGTGATATTTTGAACCTTG
Elovl3	Forward	GCCTCTCATCCTCTGGTCCT
	Reverse	TGCCATAAACTTCCACATCCT
CART	Forward	TGGATGATGCGTCCCATG
	Reverse	CGGAATGCGTTTACTCTTGAGC
NPY	Forward	CGCTCTGCGACACTACATCAA
	Reverse	GGGCTGGATCTCTTGCCAT
AgRP	Forward	GGTGCTAGATCCACAGAACCG
	Reverse	CCAAGCAGGACTCGTGCAG
POMC	Forward	CGAGGCCTTTCCCCTAGAGT
	Reverse	CCAGGACTTGCTCCAAGCC
leptin	Forward	GTGGTGGCTGGTGTGATGAT
	Reverse	TTGATGAGGTGACCAAGGTG
adiponectin	Forward	GGAGAGAAAGGAGATGCAGGT
	Reverse	CTTTCCTGCCAGGGGTTTC
18S	Forward	AGTCCCTGCCCTTTGTACACA
	Reverse	CGATCCGAGGGCCTCACTA
TFIIB	Forward	GTTCTGCTCCAACCTTTGCCT
	Reverse	TGTGTAGCTGCCATCTGCACTT

A		Wt	KO
<u>HS (control)</u>		87,02 ± 0,33	87,33 ± 0,24
<u>HS</u>		87,33 ± 0,38	87,56 ± 0,24
<u>HSDHA</u>		86,93 ± 0,14	87,37 ± 0,11
B		Wt	KO
<u>HS (control)</u>		87,19 ± 0,27	87,55 ± 0,16
<u>HS/HSHF</u>		87,31 ± 0,27	87,30 ± 0,06
<u>HSDHA/HSHF</u>		87,33 ± 0,06	87,55 ± 0,10

Table S2. Apparent food absorption efficiency

Apparent mass absorption efficiency calculated as the ratio of the ingested mass minus the excreted mass (feces mass) to mass ingested at two time points: at the shifting point from DHA635 enriched or deficient diet to high fat diet (**A**) and after 2 weeks of high fat diet treatment (at 636 week 6) (**B**). Values were normalized by arcsine transformation.

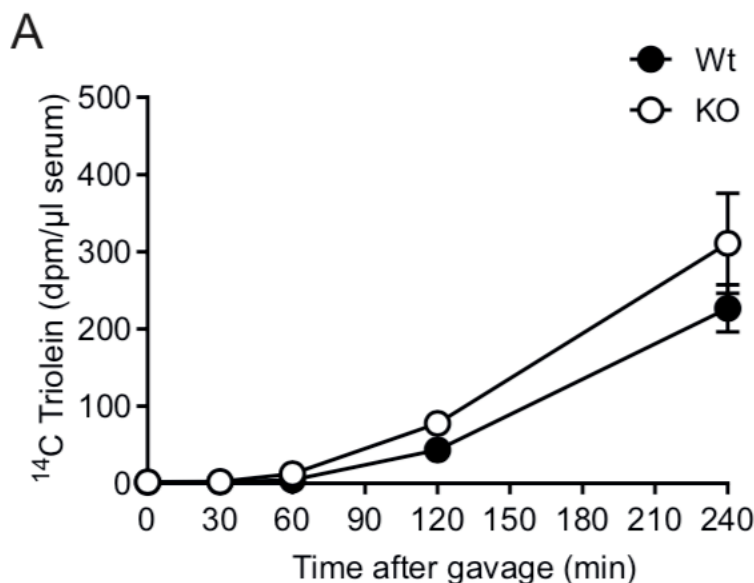


Figure S1. Intestinal lipid uptake test

18-20-week-old wild-type and *Elovl2*^{-/-} mice, maintained on low-sucrose diet, were fasted overnight and then injected with tyloxapol (Lipase inhibitor) 15 minutes prior to gavage of olive oil traced with ¹⁴C-triolein. Blood was collected 30, 60, 120 and 240 minutes post gavage to measure radioactive tracer recovery. Results shown are means of 6 mice ± SEM.

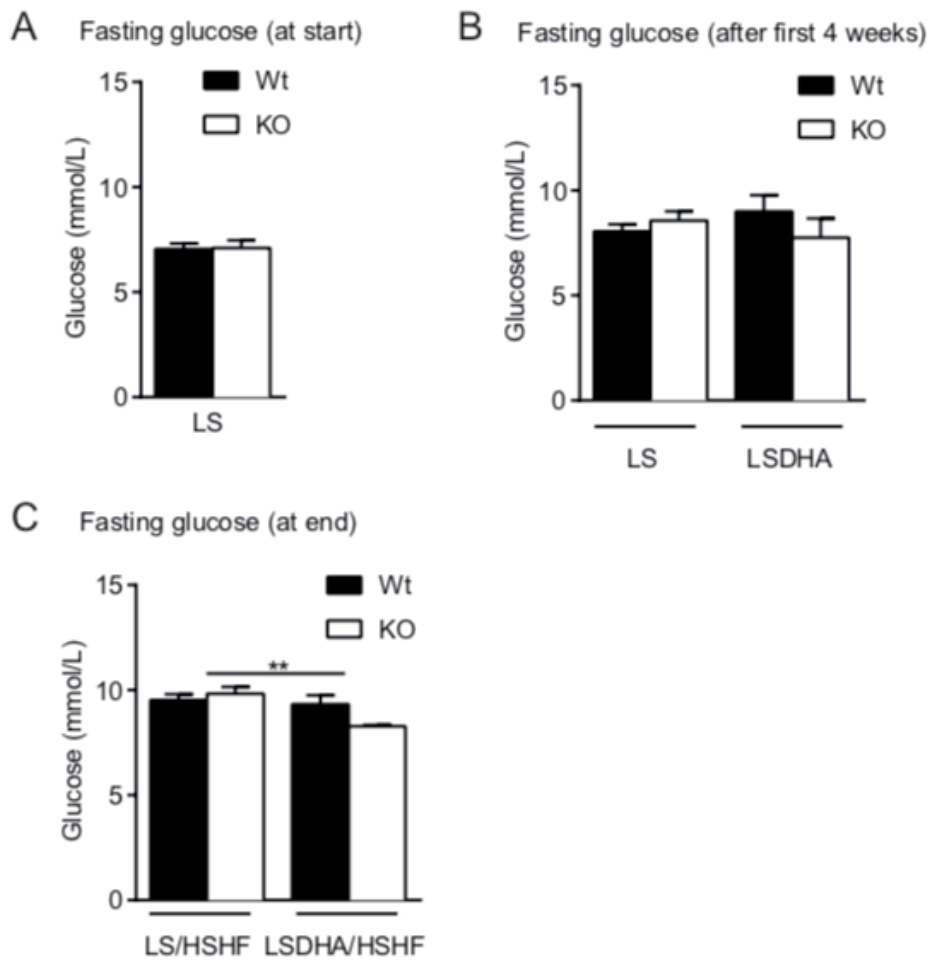


Figure S2. Fasting glucose concentration in blood.

Glucose levels in wild-type and *Elovl2*^{-/-} mice were measured at the beginning of the experiment, after 4 weeks of pre-treatment with low-sucrose or low-sucrose DHA-enriched diet (LSDHA), and at the end of 4 weeks of high-sucrose, high-fat diet (HSHF) feeding. Animals were fasted for 6 hours prior to the analysis. Results shown are means of 4 mice \pm SEM. Statistical significances are shown between groups $**P < 0.01$.

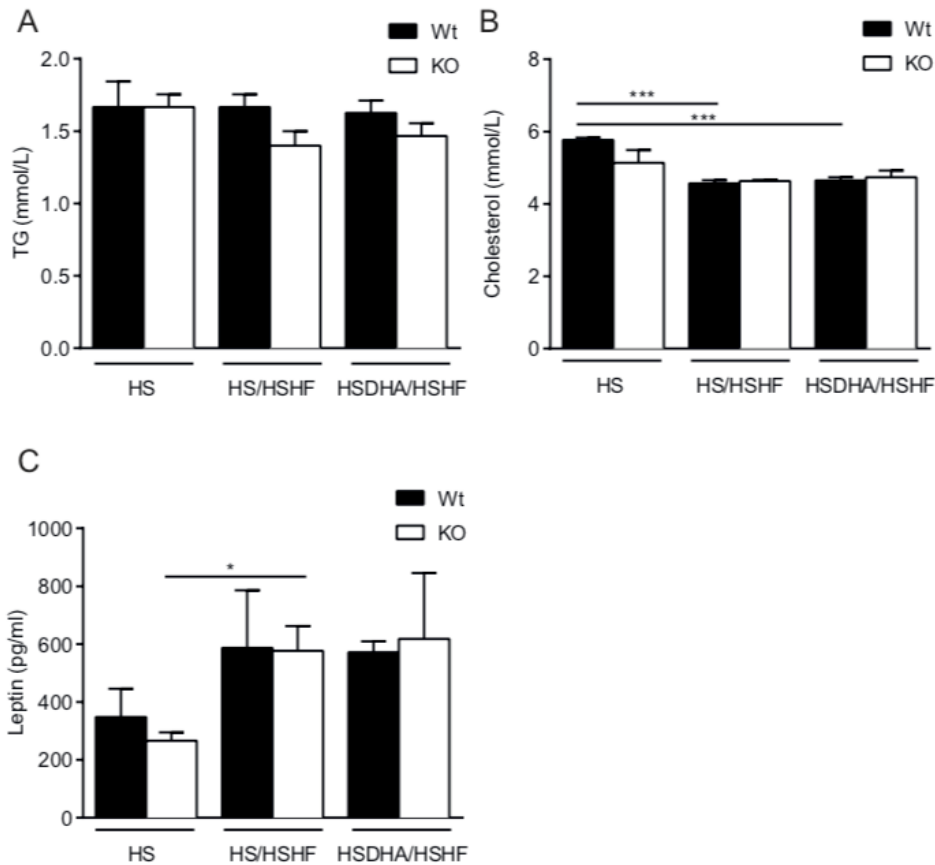


Figure S3. Parameters of lipid metabolism in the blood.

Triglyceride (TG), cholesterol and leptin levels were analyzed in blood and serum of wild-type and *Elov12*^{-/-} mice that were fed high-sucrose (HS) for 8 weeks and high-sucrose or high- sucrose DHA-enriched diet for 4 weeks followed by high-sucrose, high-fat diet (HS/HSHF and HSDHA/HSHF respectively) for 4 weeks. Serum samples for leptin and insulin measurements were collected at the end point of experiment; triglyceride and cholesterol levels were measured in after the GTT at 6 weeks of treatment (4 weeks of pre-treatment and 2 weeks of high-sucrose, high-fat diet (HSHF)). Results shown are means of 4 mice \pm SEM. Statistical significances are shown between groups * $P < 0.05$. *** $P < 0.001$