Supporting Information

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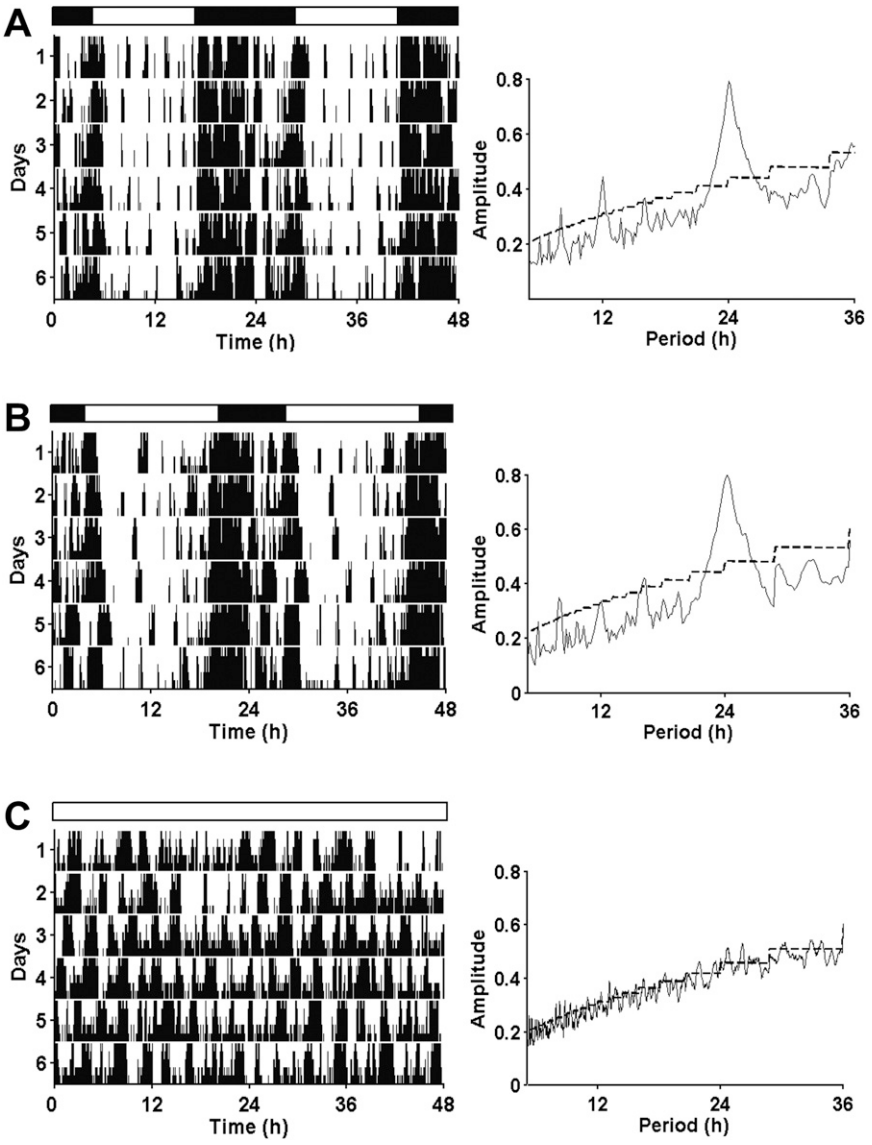

Fig. S1. Prolonged daily light exposure affects behavioral rhythms in mice. Mice were exposed to either 12-, 16-, or 24-h light ($n = 9$) for 5 wk, and behavioral activity was monitored by passive infrared detectors. Representative actograms (*Left*) and the pertaining F periodogram (*Right*) are shown of a mouse exposed to 12 (*A*), 16 (*B*), and 24 h (*C*) of light. The double-plotted actograms show consecutive days on successive lines and the vertical black upticks indicate behavioral activity measured by passive infrared detectors. The light regimes are plotted on top of the actograms; white areas represent light and black areas represent darkness. Periodogram analysis visualizes the strength of behavioral rhythmicity. The dotted lines in the periodograms indicate the 0.05 level of significance.

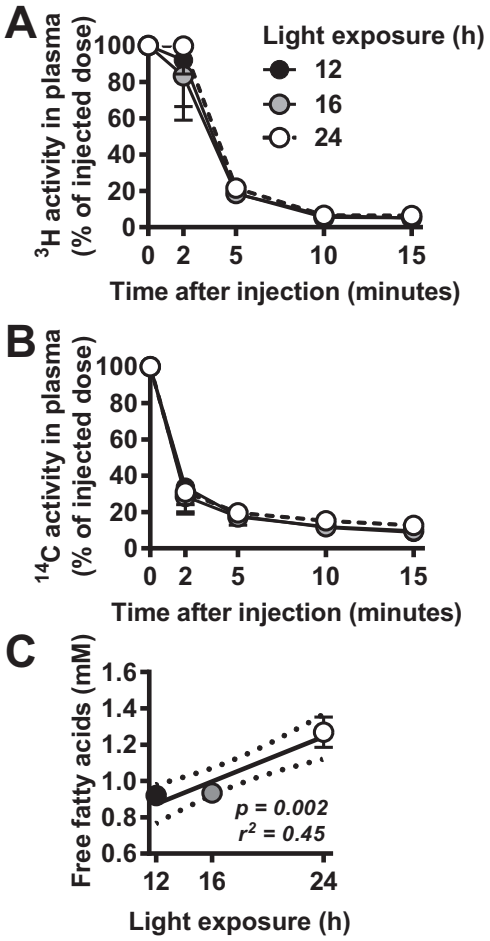

Fig. S2. Effect of light exposure on TG and glucose plasma clearance and FA plasma levels. Prolonged daily light exposure does not affect TG and glucose plasma clearance. Mice were exposed to either 12-, 16-, or 24-h light for 5 wk, and the VLDL-TG and glucose kinetics were assessed by injection of glycerol tri [^{3}H]oleate ([^{3}H]TO)-labeled emulsion particles and [^{14}C]deoxyglucose ([^{14}C]DG). Blood was drawn 2, 5, 10, and 15 min after injection. [^{3}H]TO (n = 8–9) (*A*) and [^{14}C]DG (n = 6–7) (*B*) derived activity was assessed in plasma samples by liquid oscillation counting. Basal plasma free fatty acid concentration (*C*) was determined using an enzymatic kit (NEFA-HR Wako diagnostics, Richmond, VA). Data are represented as means ± SEM. Statistical significance was determined by unpaired two-tailed Student's t test.

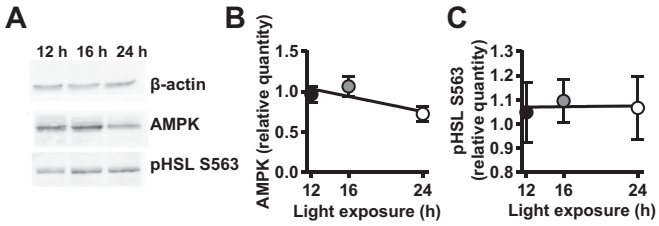

Fig. S3. Prolonged daily light exposure does not affect AMPK and pHSL S^{563} in BAT. Prolonged daily light exposure does not affect AMPK and pHSL S^{563} in BAT. Mice exposed to either 12-, 16-, or 24-h light (n = 9) for 5 wk, and iBAT was isolated for protein quantification. Representative Western blots are shown for β-actin, total AMPK and pHSL S^{563} (*A*). Correlation was determined between hours of light exposure and protein levels of AMPK (*B*) and pHSL S^{563} (*C*). Protein levels were normalized to β-actin levels. Data are represented as means ± SEM. Statistical significance between groups was determined by unpaired two-tailed Student's t test; linear regression analysis was performed to analyze the association of gene expression with light exposure.

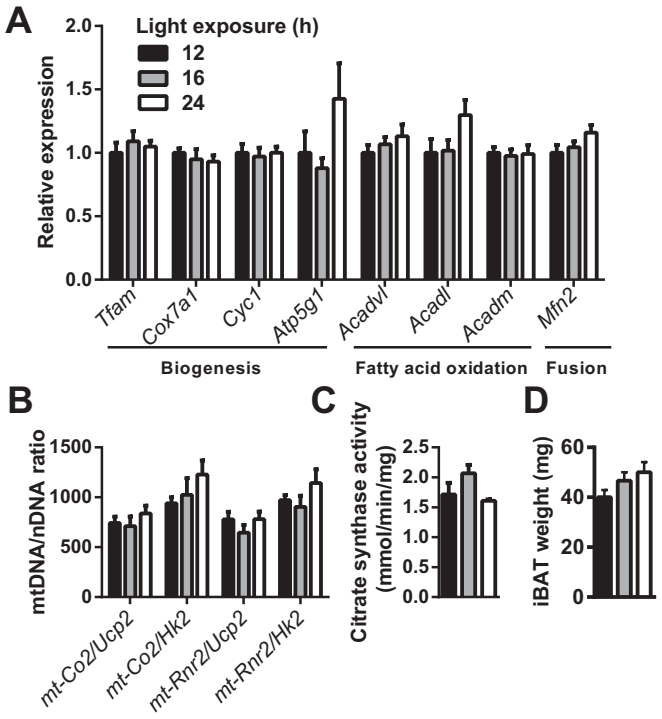

Fig. S4. Effects of prolonged light exposure on mitochondrial function. Prolonged daily light exposure does not decrease structural mitochondrial function in BAT and quantity of BAT. Mice were exposed to either 12-, 16-, or 24-h light ($n = 9$) for 5 wk, and interscapular and subscapular BAT was isolated for gene expression and mitochondrial function analysis. Expression of genes involved in mitochondrial biogenesis, fatty acid oxidation and fusion genes did not change upon prolonged light exposure (*A*). The same is true for relative mitochondrial DNA quantity (*B*) and mitochondrial citrate synthase activity (*C*). iBAT was removed quantitatively and weighed (*D*). Gene-expression levels were normalized to *36B4* levels. Mitochondrial DNA quantity is expressed as ratio of mitochondrial gene expression relative to nuclear gene expression (genes indicated below) Data are represented as means ± SEM. Abbreviations: *Acadl*, acyl-CoA dehydrogenase, long-chain; *Acadm*, acyl-CoA dehydrogenase, medium chain; *Acadvl*, acyl-CoA dehydrogenase, very long chain; *Atp5g1*, ATP synthase, H+ transporting, mitochondrial F0 complex, subunit C1; *Cox7a1*, cytochrome *c* oxidase subunit VIIa 1; *Cyc1*, cytochrome C-1; *Hk2*, hexokinase 2; *Mfn2*, mitofusin 2; *mtCo2*, cytochrome *c* oxidase II, mitochondrial; *mt-Rnr2*, 16S rRNA, mitochondrial; *Tfam*, transcription factor A, mitochondrial; *Ucp2*, uncoupling protein 2.

Table S1. Primer sequences used for quantitative PCR on BAT

Gene	Forward primer sequence	Reverse primer sequence
BAT gene primers		
Ucp1	TCAGGATTGGCCTCTACGAC	TGCATTCTGACCTTCACGAC
Pgc1α	TGCTAGCGGTTCTCACAGAG	AGTGCTAAGACCGCTGCATT
36B4	GGACCCGAGAAGACCTCCTT	GCACATCACTCAGAATTTCAATGG
Mitochondrial gene primers		
Tfam	AGTTCCCACGCTGGTAGTGT	GGATAGCTACCCATGCTGGA
Cox7a1	AAAACCGTGTGGCAGAGAAG	CCAGCCCAAGCAGTATAAGC
Cyc1	ACCTGGTGGGAGTGTGCTAC	GAGGTCAGGGGGTAAAGCTC
Acadvl	ATGGCTTCAAGGTTGCTGTC	GAATTTTGTCCCCAAACTGG
Acadl	GCCAAAAGATCTGGGAGTGA	TTCCGTTTTCCACCAAAAAG
Acadm	GCTAGTGGAGCACCAAGGAG	CCAGGCTGCTCTCTGGTAAC
Atp5g1	GCTGCTTGAGAGATGGGTTC	AGTTGGTGTGGCTGGATCA
Mfn2	ACGTCAAAGGGTACCTGTCCA	CAATCCCAGATGGCAGAACTT
36B4	AGATTCGGGATATGCTGTTGG	AAAGCCTGGAAGAAGGAGGTC
Genomic DNA primers		
mtCox	GTTGATAACCGAGTCGTTCTGC	CCTGGGATGGCATCAGTTTT
mt-Rnr2	CCGCAAGGGAAAGATGAAAGAC	TCGTTTGGTTTCGGGGTTTC
Ucp2	CTACAGATGTGGTAAAGGTCCGC	GCAATGGTCTTGTAGGCTTCG
Hk2	TCTGGCTCTGAGATCCATCTTCA	CCGGCCTCTTAACCACATTCC