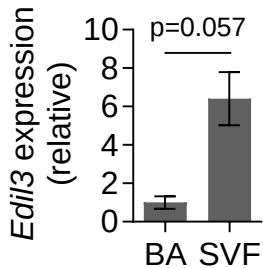
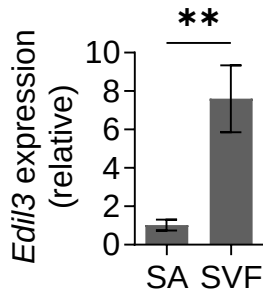


Supp figure 1

A

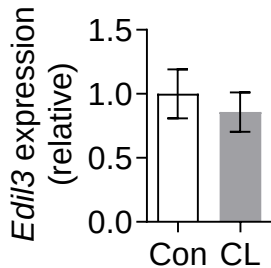


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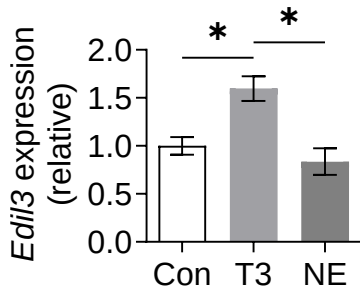


Supp figure 2

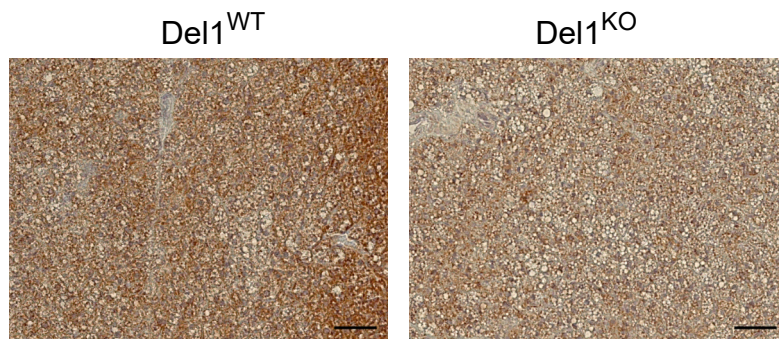
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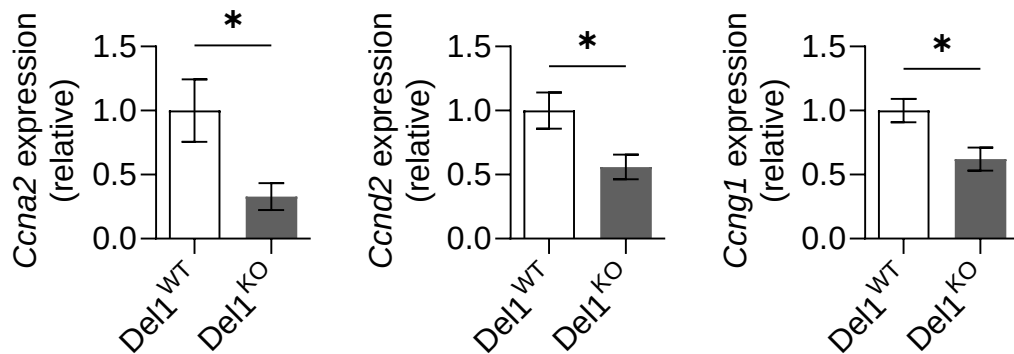
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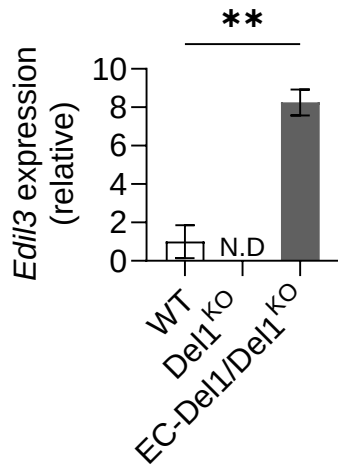
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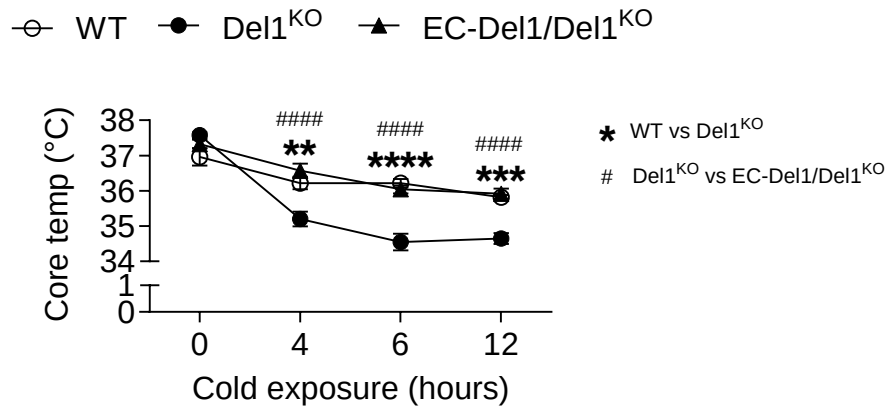
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A

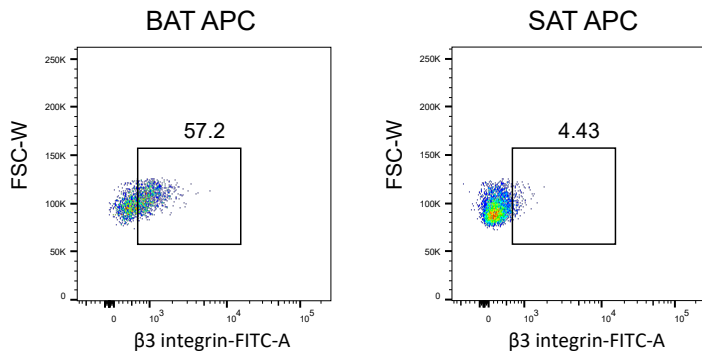


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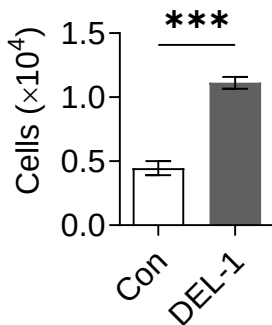


Supp figure 5

A



B



SUPPLEMENTARY FIGURE LEGENDS

Suppl. Figure 1. DEL-1 is mainly expressed in the SVF of different adipose tissues.

(A) *Edil3* mRNA expression in the adipocyte fraction containing brown adipocytes (BA) or the stromal vascular fraction (SVF) from the BAT, and (B) *Edil3* mRNA expression in the adipocyte fraction (SA) or the stromal vascular fraction (SVF) from the subcutaneous white adipose tissue from mice at room temperature (n=3-4). 18S expression was used for normalization and *Edil3* expression of BA or SA was set as 1. Data are mean $\pm$ SEM. **P < 0.01. Mann-Whitney U-test in (A), Student's t-test in (B).

Suppl. Figure 2. Regulation of DEL-1 expression in endothelial cells of the BAT.

(A) Wild-type mice received the β 3-adrenergic receptor agonist CL316243 (CL) or PBS (Con). *Edil3* mRNA expression in the BAT is shown (n=6 mice per group). 18S expression was used for normalization and *Edil3* expression of the PBS control group was set as 1. (B) Endothelial cells were isolated from BAT of wild-type mice and cultured cells were treated with 3,3',5-Triiodo-L-thyronine (T3, 10 nM) or L-Norepinephrine hydrochloride (NE, 1 μ M) or PBS as control (Con), as described in the Materials and Methods. *Edil3* expression was analyzed; 18S was used for normalization and gene expression of control-treated cells was set as 1 (n=3). Data are mean $\pm$ SEM. *P < 0.05. One-way ANOVA in (B).

Suppl. Figure 3. DEL-1 promotes thermogenic adaptation and APC proliferation in cold-stimulated BAT.

Del1^{KO} and Del1^{WT} mice were challenged with cold exposure (temperature of 4°C for 12 hours). (A) Representative images of UCP-1 staining from BAT of Del1^{WT} or Del1^{KO} mice. Scale bar, 50 μ m. (B) APCs were sorted as CD45⁻CD31⁻CD29⁺Sca1⁺PDGFR α ⁺ cells from the BAT of Del1^{WT} or Del1^{KO} mice (n=5-6) and expression of cell cycle-related genes was analyzed. 18S was used for normalization and gene expression of Del1^{WT} cells was set as 1. *Ccna2*, cyclin A2; *Ccng1*, cyclin G1; *Ccnd2*, cyclin D2. Data are mean $\pm$ SEM. *P < 0.05. Mann-Whitney U-test in (B) except for *Ccnd2* (Student's t-test).

Suppl. Figure 4. DEL-1 expression analysis.

Wild-type (WT; n=5), *Del1*^{KO} (n=4), and EC-*Del1*/*Del1*^{KO} (n=7) mice were challenged with a cold exposure (temperature of 4°C) and *Edil3* mRNA expression in the interscapular BAT was analyzed. 18S expression was used for normalization and *Edil3* expression of BAT from WT mice was set as 1. N.D., not detected. (B) Core body temperature during the cold exposure. Data are mean ± SEM. **P < 0.01, ***P < 0.001, ****P < 0.0001, #####P < 0.0001. Mann-Whitney *U*-test in (A), Two-way ANOVA in (B).

Suppl. Figure 5. Expression of β3 integrin on APCs and DEL-1 promotes proliferation of brown preadipocytes.

(A) Wild-type mice (n=5) were challenged with a cold exposure (temperature of 4°C for 12 hours) and β3 integrin expression in primary APCs (defined as CD45⁻CD31⁻CD29⁺Sca1⁺PDGFRα⁺ cells) from the BAT or SAT was analyzed by flow cytometry. Representative flow cytometry plots showing the percentage of β3 integrin-positive cells from APCs of BAT or SAT. (B) Proliferation of a brown preadipocyte cell line was studied in the absence (Con) or presence of DEL-1. Data are presented as the number of cells after 3 days of culture (n=3 independent experiments); the same cell number/well was seeded at the start of the assay, as described in the Materials and Methods. Data are mean ± SEM. ***P < 0.001. Student's t-test in (B).