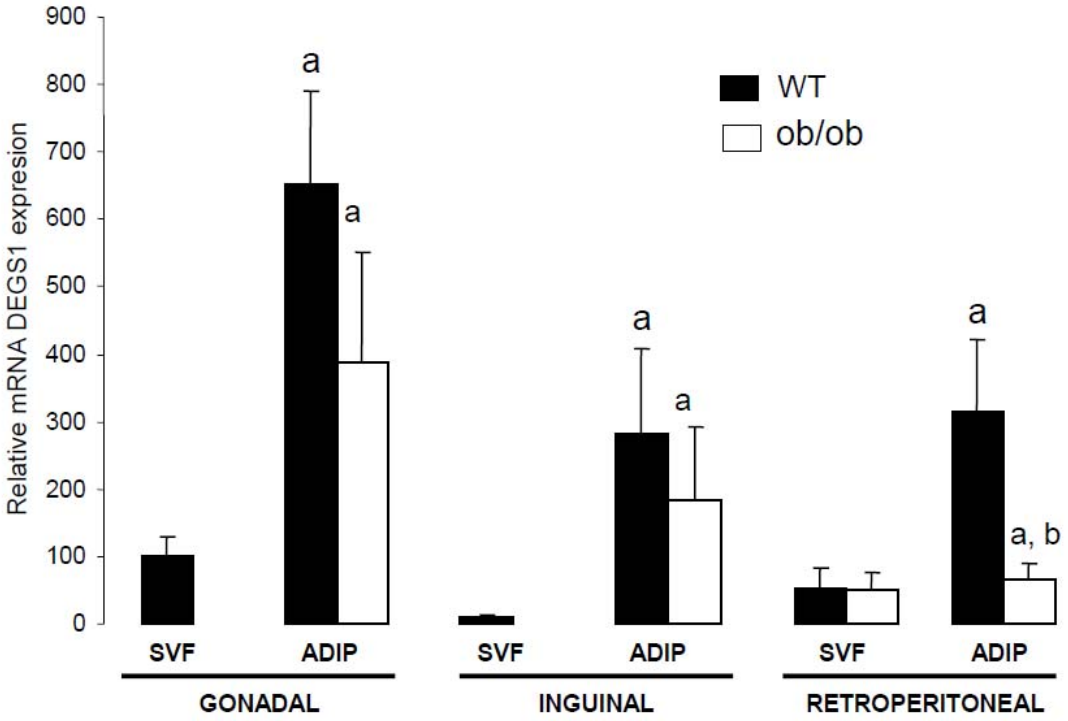
SUPPLEMENTARY DATA

Supplementary Figure 1. mRNA expression levels of DEGS1 in stromal vascular fraction and adipocytes of different fat depots in wild type and ob/ob mice (n=3). $p<0.05$: (a) versus svf. (b) versus lean WT



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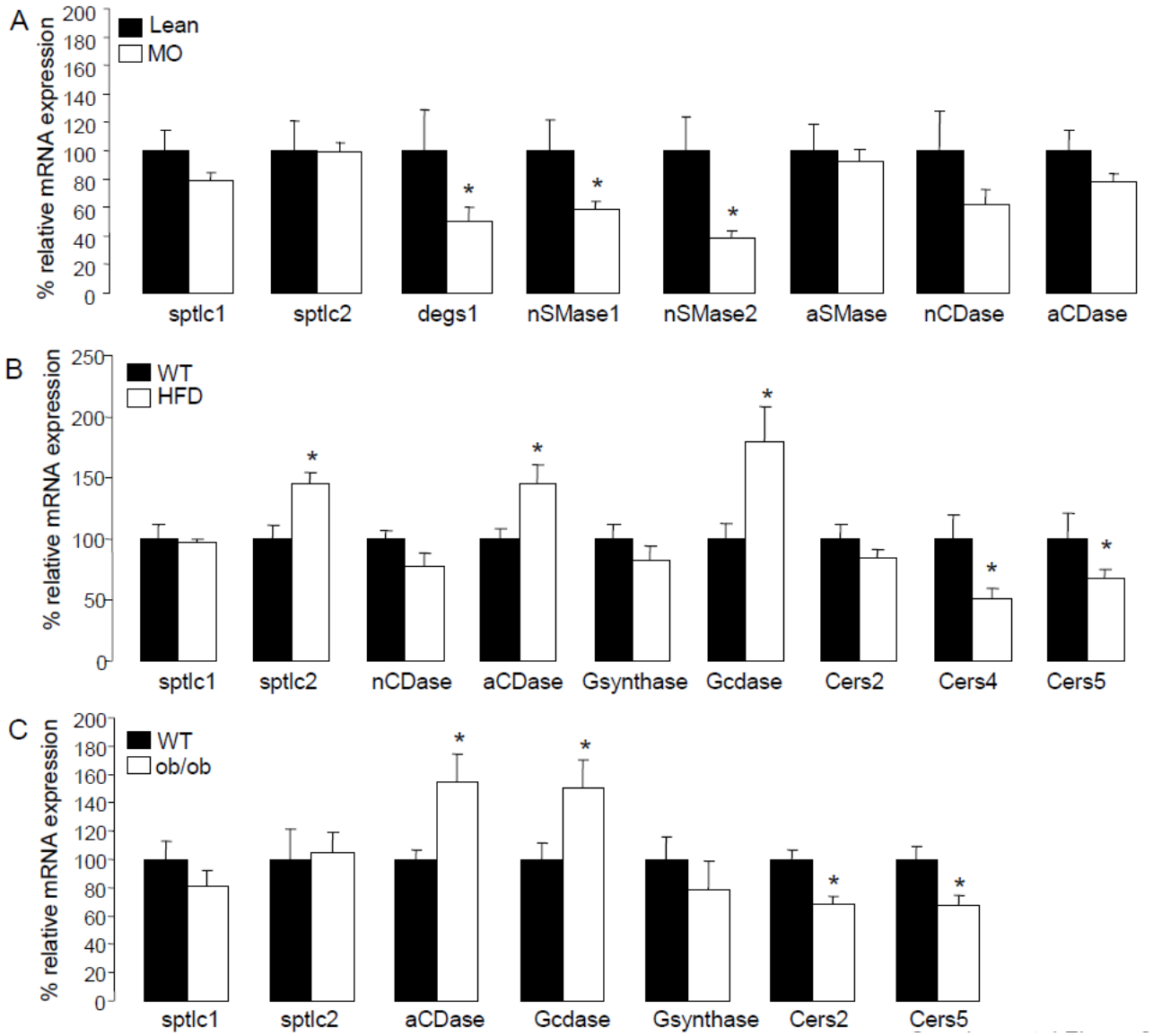
SUPPLEMENTARY DATA

Supplementary Figure 2. (A) Expression levels of DEGS1 and other ceramide related enzymes in visceral adipose tissue of morbidly obese patients (B) Expression levels ceramide related genes in response to HFD intervention in rodents(C) Expression levels ceramide related genes in ob/ob mice. The panels show means ± SEM of 28 morbidly obese patients and 6 lean controls for the human study and 6 to 8 animals per group in the rodent studies. Significant differences at $p<0.05$: (*) versus lean controls.



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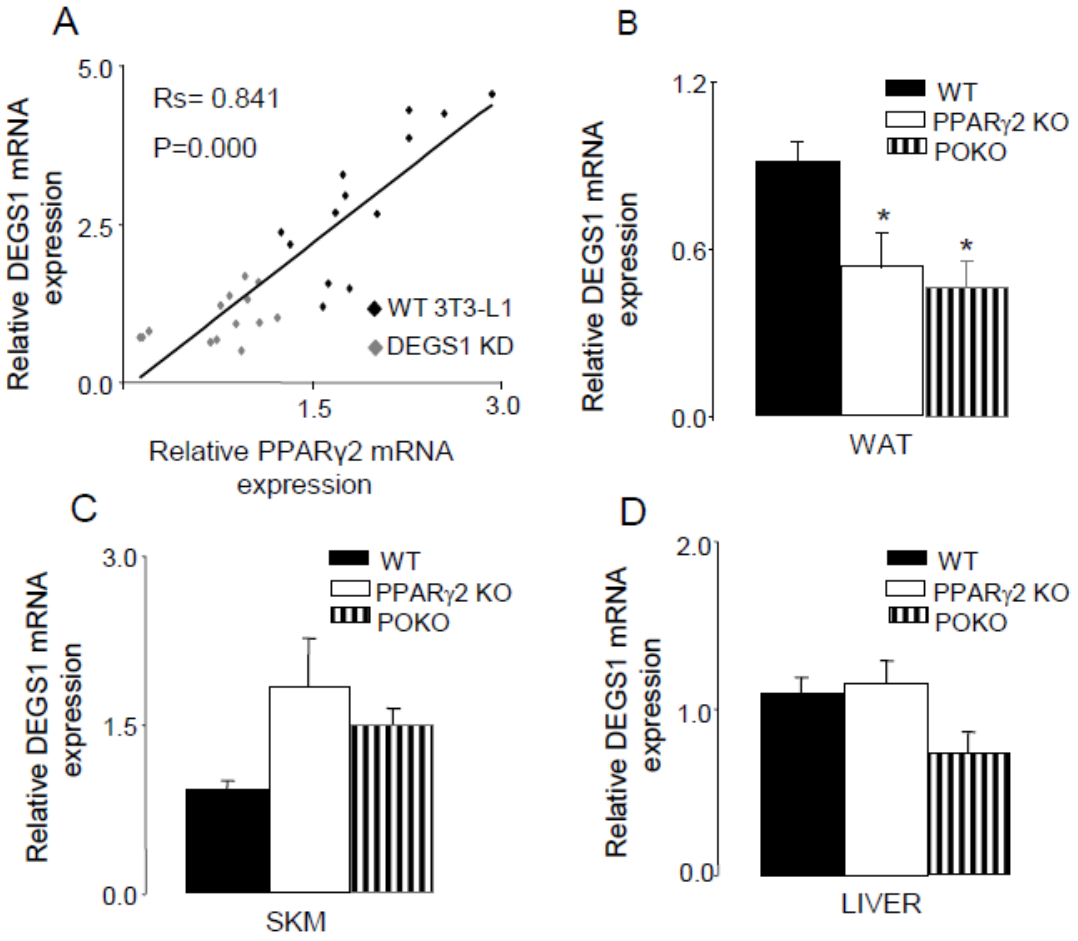
SUPPLEMENTARY DATA

Supplementary Figure 3. (A) Positive correlation between the mRNA expression levels of PPARγ2 and DEGS1 in WT and DEGS1 3T3-L1 knockdown cells with or without Rosiglitazone (0.1 µM) at 9 days of adipocyte differentiation. The Spearman correlation coefficients were calculated to estimate the linear correlations between variables. The rejection level for a null hypothesis was $p < 0.01$ (B, C and D) mRNA DESG1 expression in white adipose tissue (WAT), skeletal muscle (SKM) and liver of wild-type, PPARγ2 KO and POKO (PPARγ2 -/- and lep -/-) mice. Graphs show the mean ± SEM of a group of 6-8 animals. *p< 0.05 versus wild-type mice.



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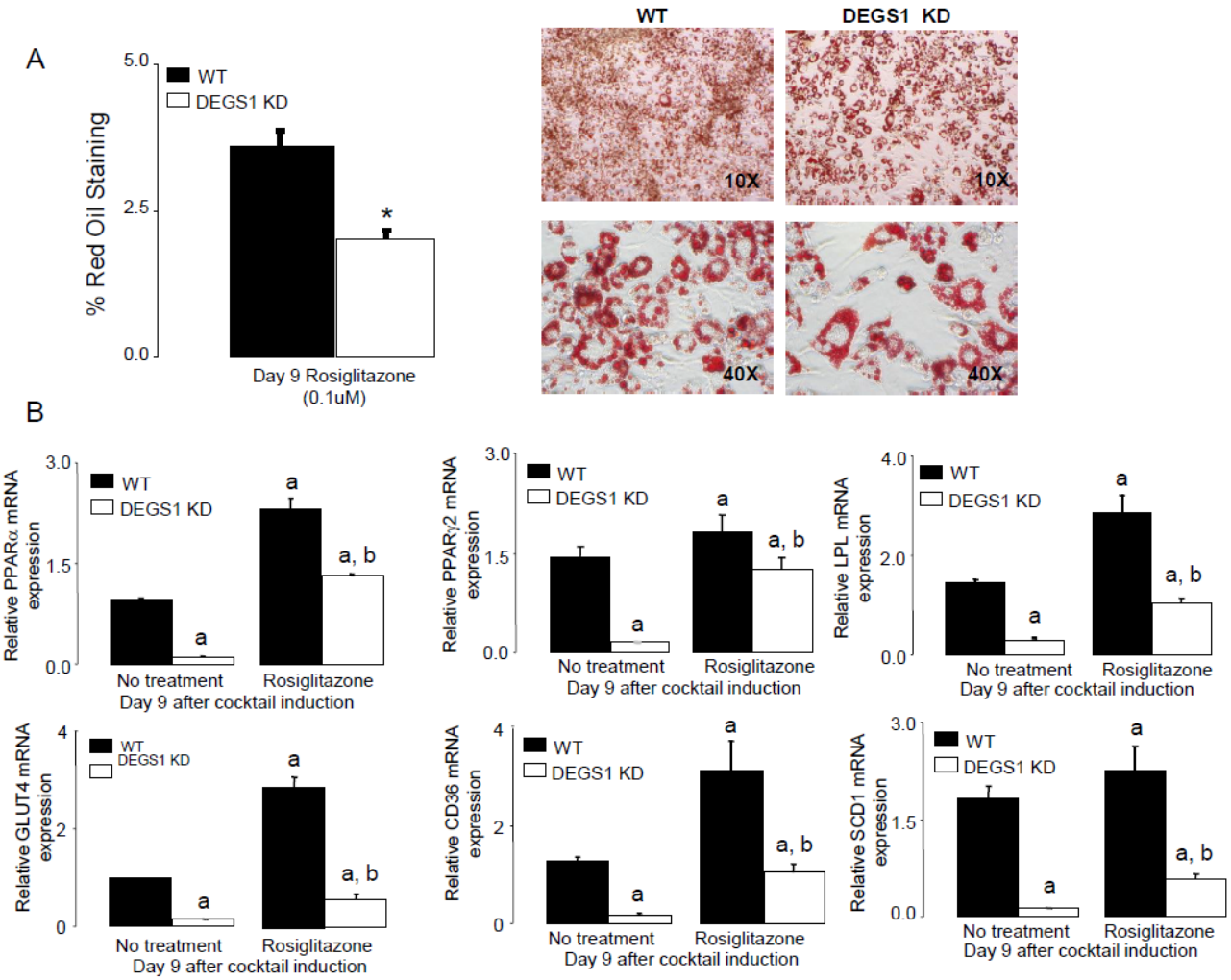
SUPPLEMENTARY DATA

Supplementary Figure 4. (A) Lipid accumulation in control and DEGS1 3T3-L1 knockdown cells treated with Rosiglitazone (0.1 µM) after 9 days of adipocyte differentiation. Lipid content was analyzed using O Red Oil staining. Graph shows the mean ± SEM of two separate experiments performed in triplicate; *p< 0.05 versus control cells. Panels show one representative experiment (B) mRNA expression of genes involved in adipocyte differentiation and lipid accumulation in controls and DEGS1 3T3-L1 knockdown cells with or without Rosiglitazone (0.1 µM) at day 9 of adipocyte differentiation. Graphs show the mean ± SEM of two separate experiments performed in triplicate; [a]p< 0.05 versus WT untreated cells and [b]p< 0.05 versus WT cells treated with Rosiglitazone.



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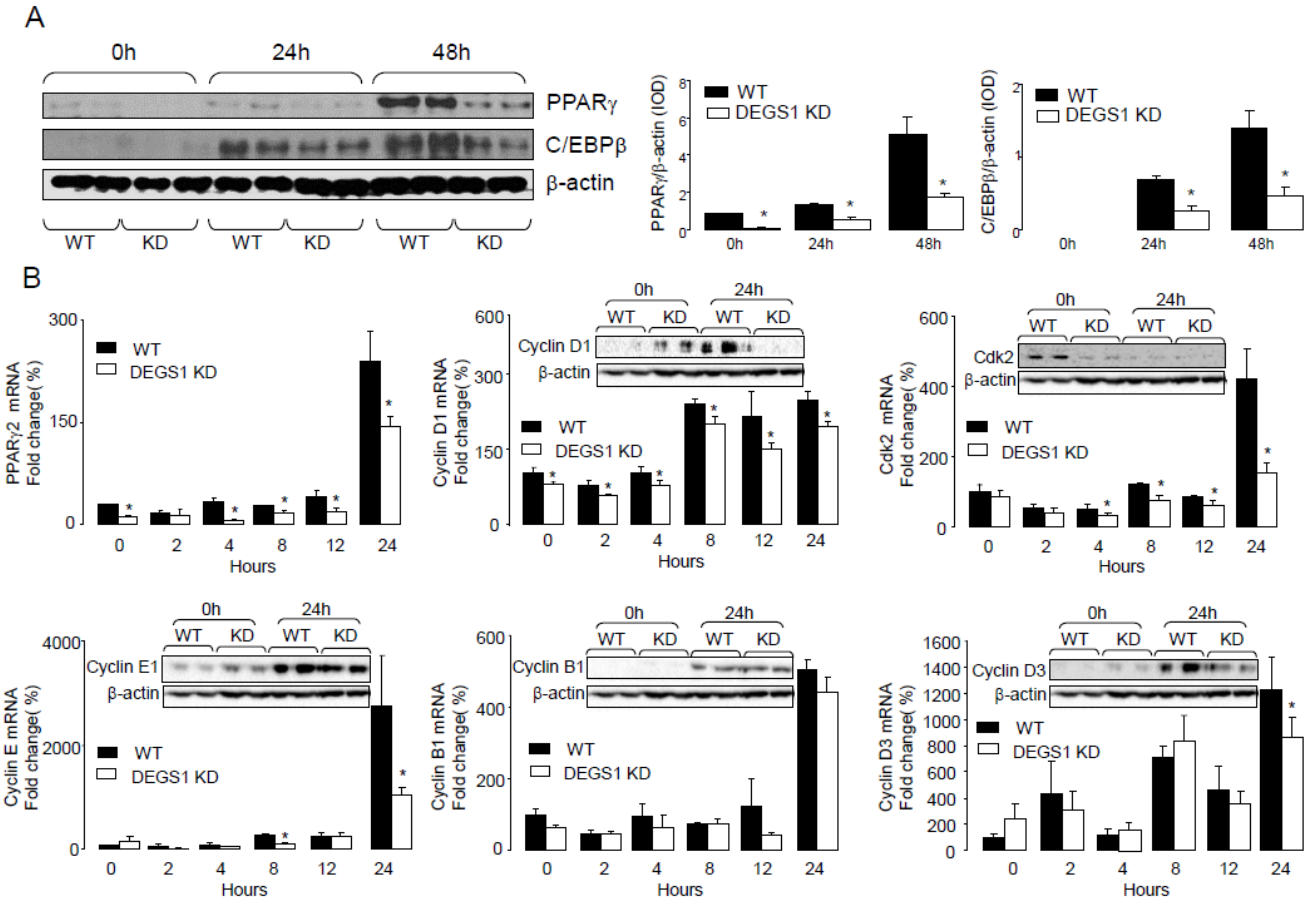
SUPPLEMENTARY DATA

Supplementary Figure 5. (A and B) mRNA and protein expression of PPARγ2, cEBPα, several cyclins and cdk2 in control and DEGS1 knock-down cells during early adipogenesis (at 0, 24 and 48 hours after adipocyte differentiation induction). Panels show one representative experiment of three different experiments performed separately. Graphs show the mean ± SEM of three separate experiments. *p< 0.05 vs. control cells. Rosi, Rosiglitazone



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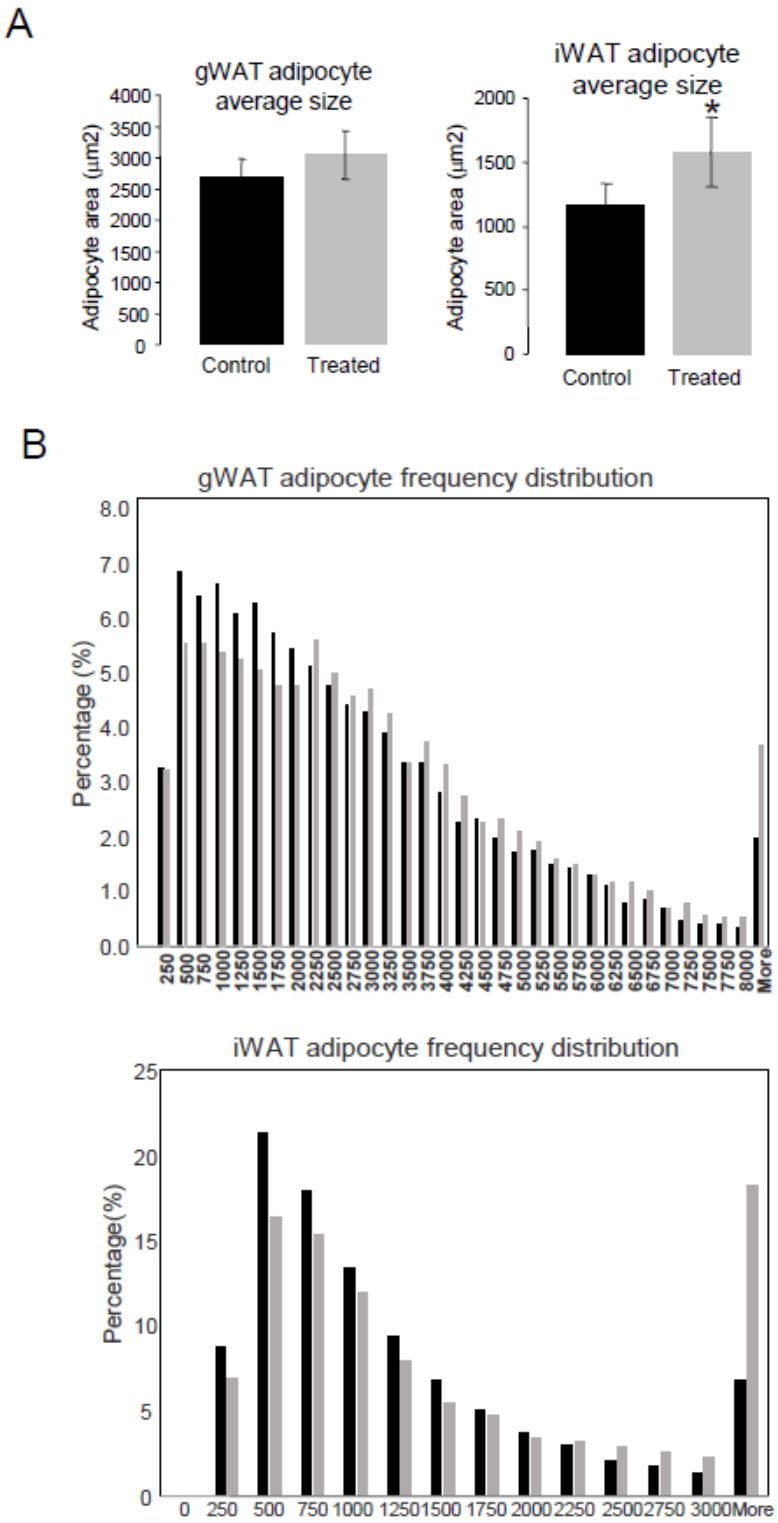
SUPPLEMENTARY DATA

Supplementary Figure 6. A) Adipocyte mean area and B) frequency of distribution of gonadal and inguinal WAT adipocytes (1000-3000 per mouse, n=8) from vehicle and C8-CPPC treated mice. $P < 0.05$



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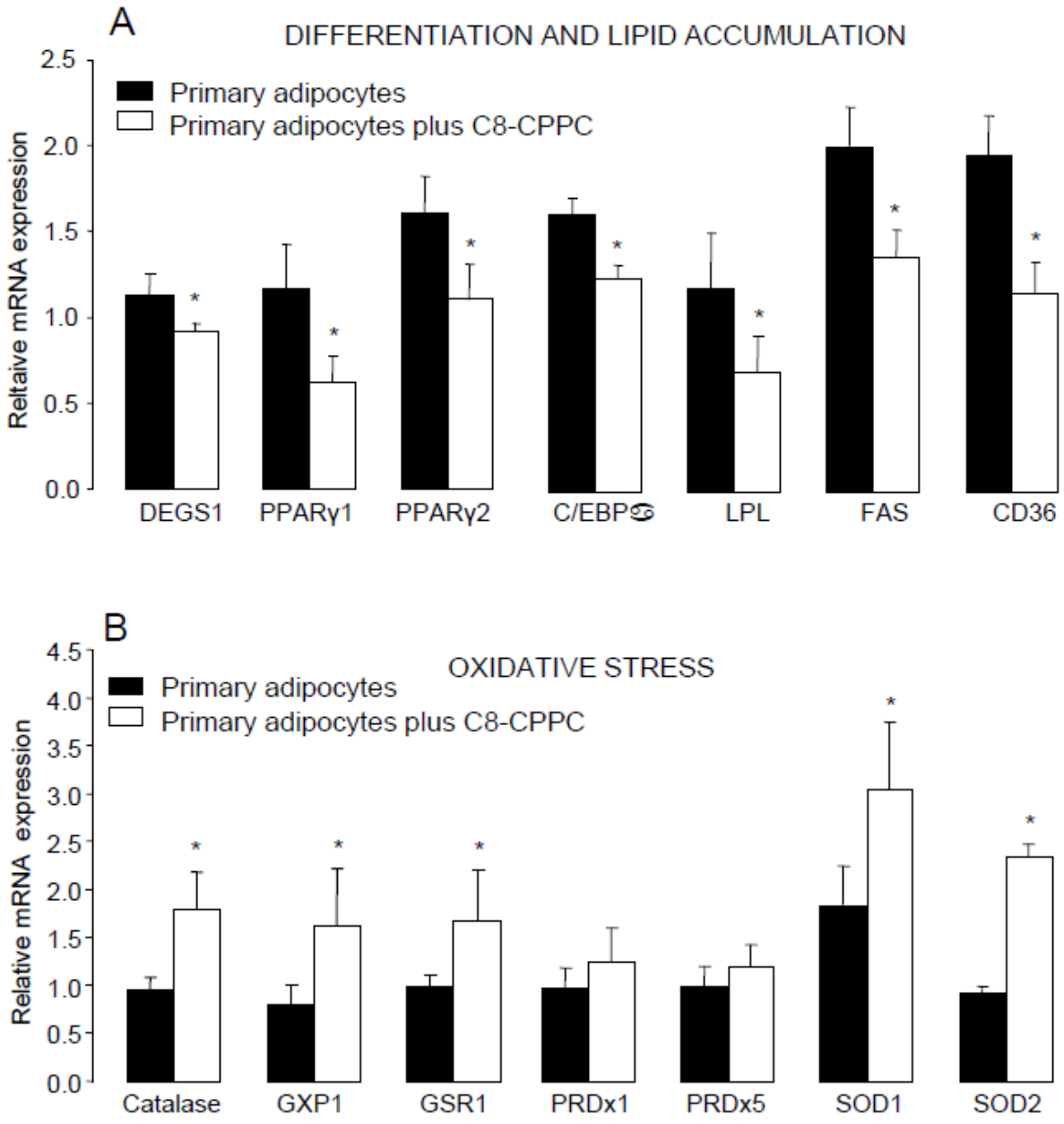
SUPPLEMENTARY DATA

Supplementary Figure 7. (A and B) mRNA expression levels of DEGS1, genes involved in differentiation (PPARγ1, PPARγ2 and cEBPα), lipid accumulation (LPL, FAS and CD36) and oxidative stress (Catalase, GXP1, GRS1, PRDx1, PRDx5, SOD1 and SOD2) in primary murine adipocytes treated with C8-CPPC for 20 hours. Panel shows the mean ± SEM of two separated experiments. *p< 0.05 versus untreated primary adipocytes.



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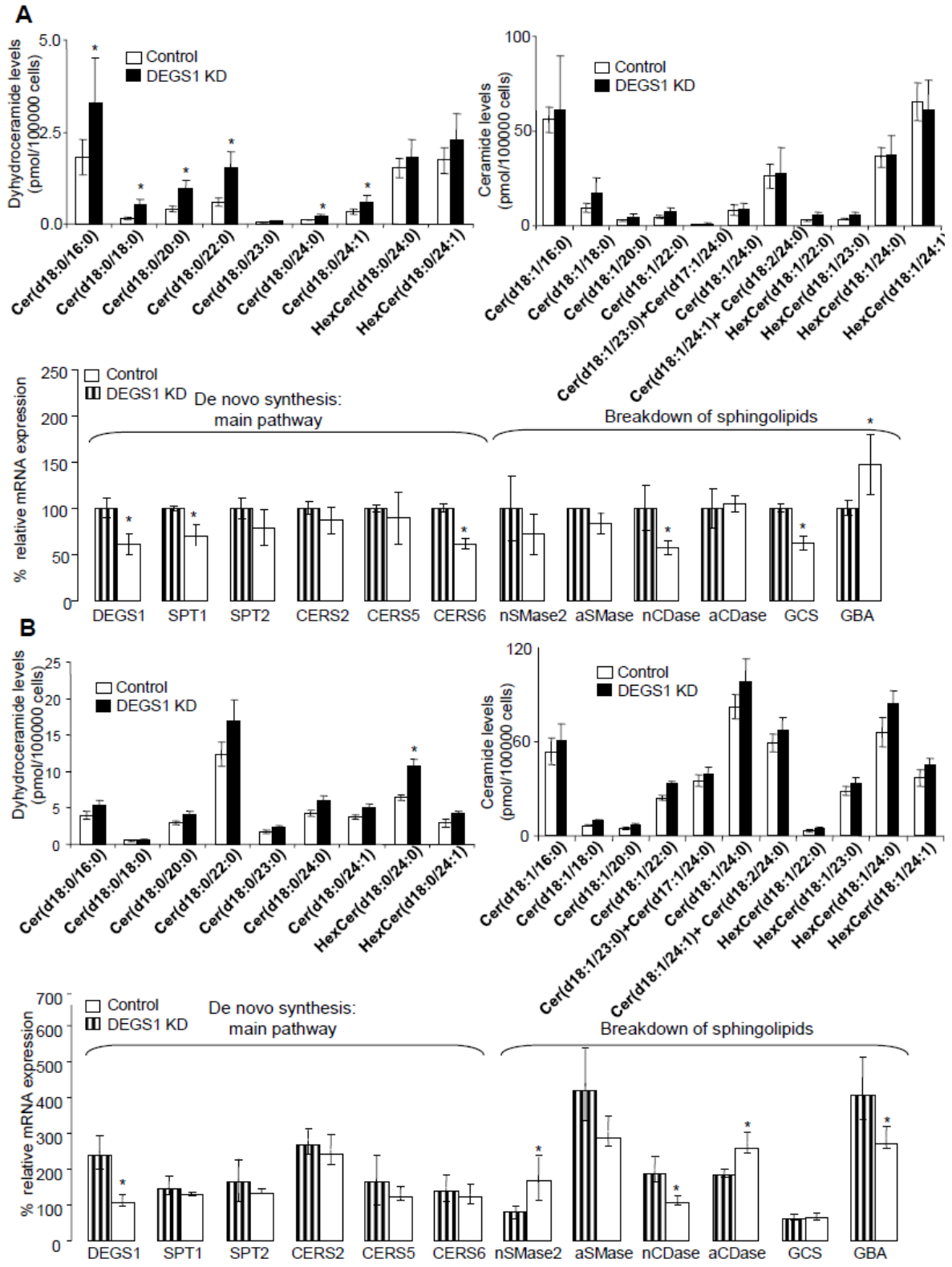
SUPPLEMENTARY DATA

Supplementary Figure 8. Total dihydroceramide and ceramide levels in controls and mRNA expression of enzymes involved in ceramide synthesis pathways in control and DEGS1 knockdown cells at 0 (A) and 9 (B) days of differentiation. Graphs show the mean ± SEM of two separate experiments *p< 0.05 vs. control cells for gene expression and p< 0.00 for lipidomic analysis.



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