

SUPPLEMENTARY DATA

Dysregulation of Nrf2/Keap1 redox pathway in diabetes affects multipotency of stromal cells

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Supplemental Figures

Figure S1. Related to Figure 1.

Figure S2. Related to Figure 2.

Figure S3. Related to Figure 3.

Figure S4. Related to Figure 3.

Figure S5. Related to Figure 5.

SUPPLEMENTARY DATA

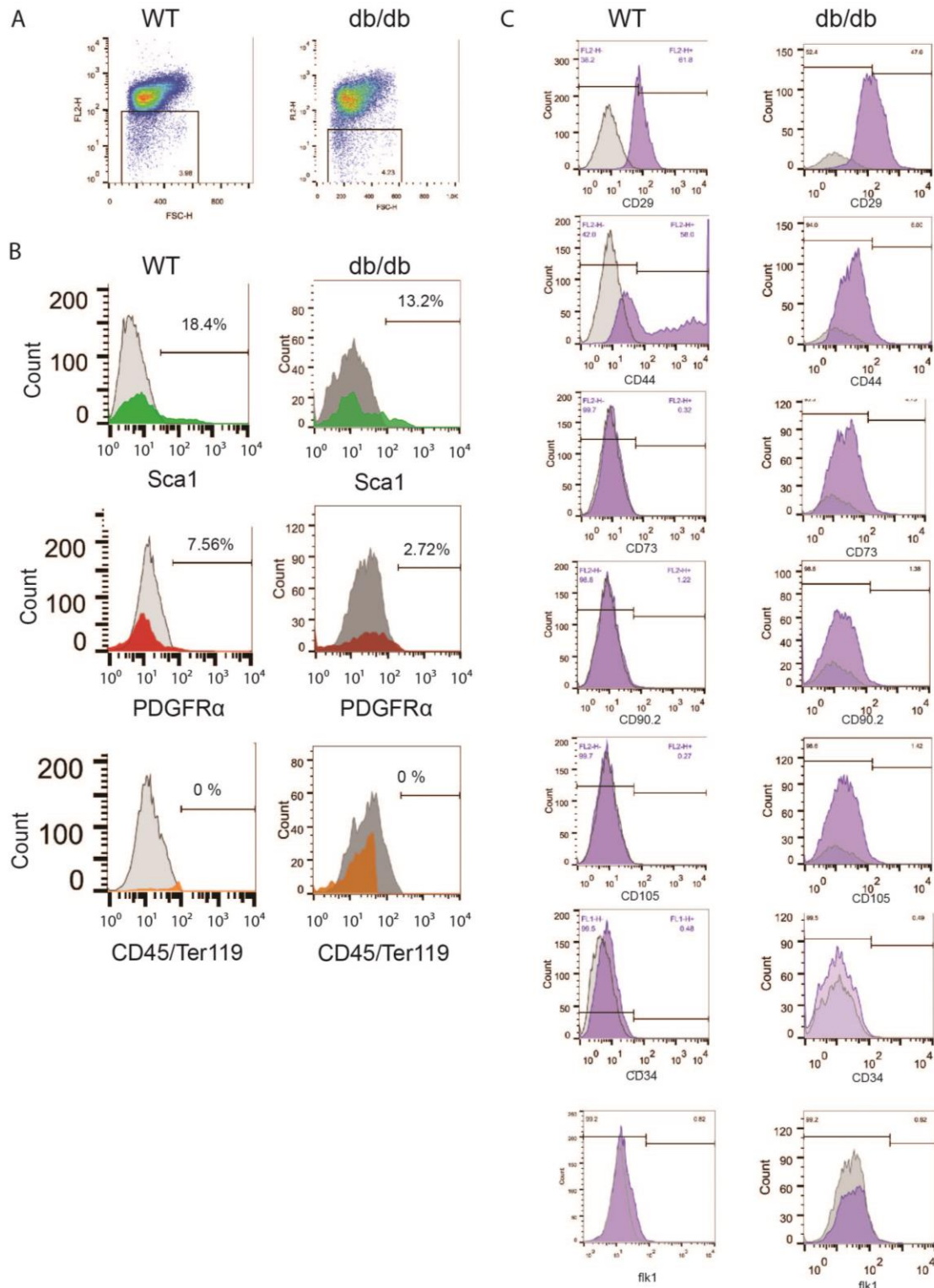


Figure S1. Related to Figure 1. Cell surface marker analysis of passage 3 BMSCs.

A) Passage 3 bone marrow was stained with CD45-PE and Ter119-PE to identify cells with and without these hematopoietic lineage markers. B) Single color stains on CD45-/Ter119- P3 cells. C) Single color stains as indicated.

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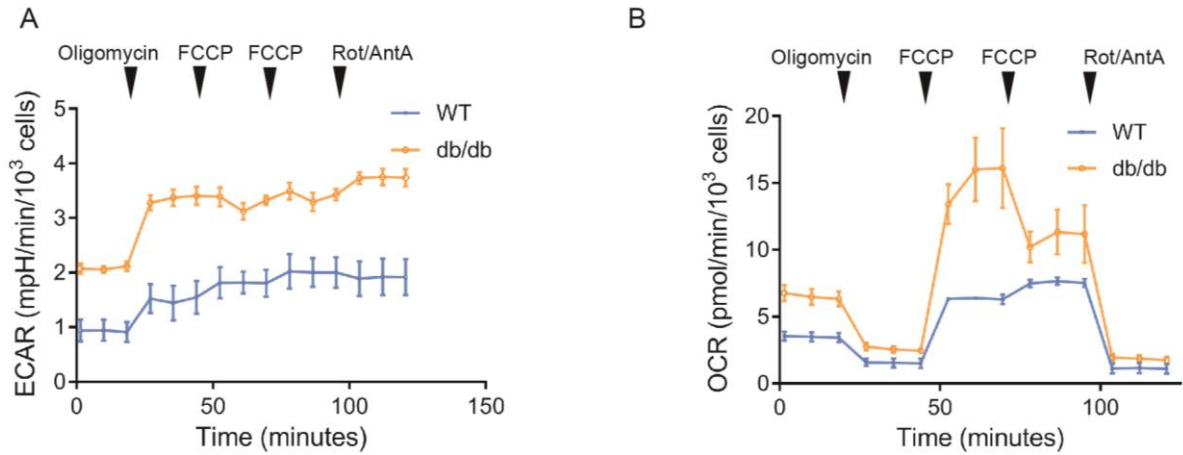


Figure S2. Related to Figure 2. Energy profiles of BMSCs with mitochondrial inhibitory analysis.

A) Extracellular acidification rate (ECAR) analysis, and (B) oxygen consumption rate (OCR) analysis in WT and dBMSCs. Baseline and measurements after addition of oligomycin, FCCP (2 separate injections to detect maximal mitochondrial respiration), and rotenone/antimycin A are shown.

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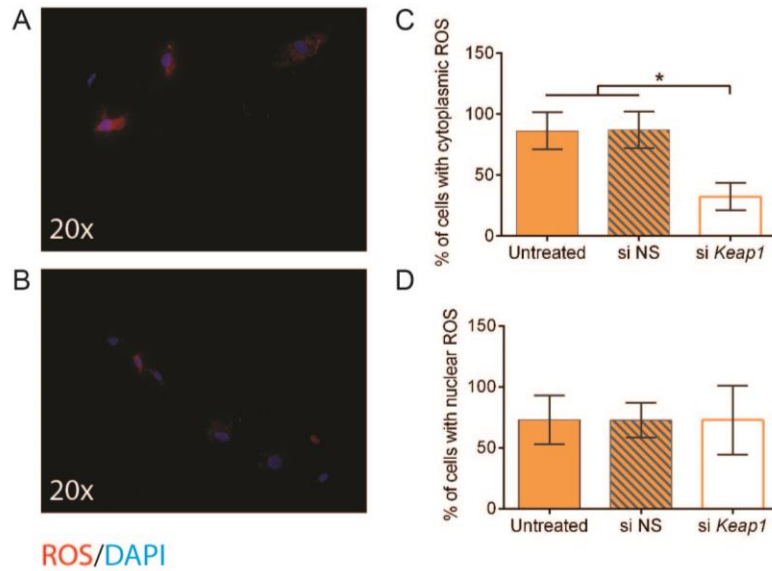


Figure S3. Related to Figure 3. ROS decreases in dBMSCs with *Keap1* inhibition.

A) Cytoplasmic and (B) nuclear ROS imaging following si*Keap1* transfection in dBMSCs. C, D) Quantification of cells displaying ROS. At least 3 fields at 20x were used for counting DAPI⁺ROS⁺ and DAPI⁺ROS⁻ cells.

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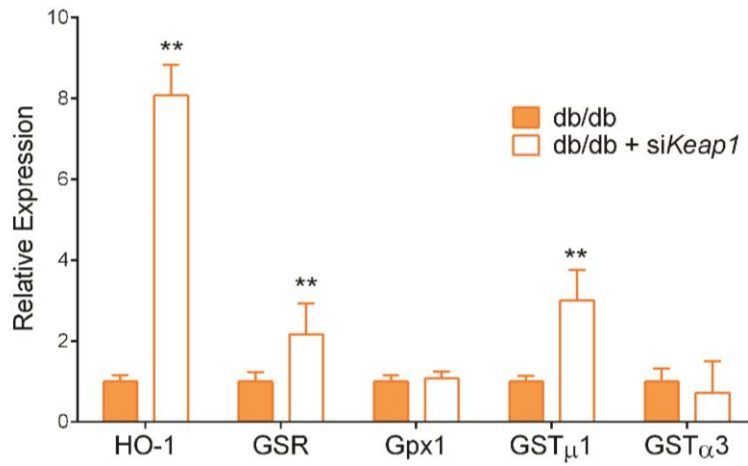


Figure S4. Related to Figure 3. Antioxidant gene expression changes in db/db BMSCs following silencing *Keap1*.

Relative expression of antioxidant genes, downstream of Nrf2, 24 hours after *siKeap1* transfection in db/db BMSCs.

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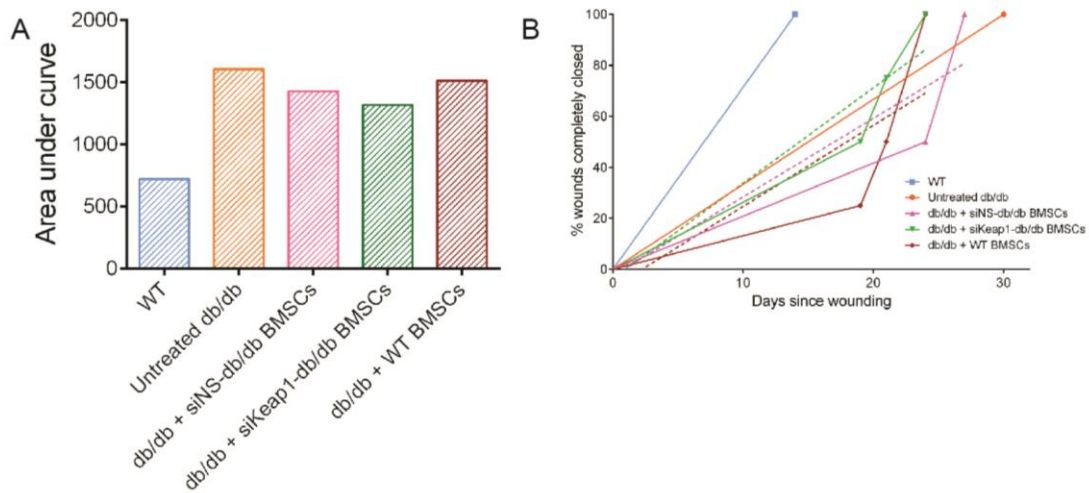


Figure S5. Related to Figure 5. *siKeep1*-db/db BMSCs accelerate diabetic wound healing and lower pathologic wound burden.

A) Wound burden (area under curve of Figure 5D). B) Wound healing pace of diabetic wounds with seeded BMSC as indicated. Color of dashed lines correspond to BMSC treatments in key, and slope indicates rate of closure.