**Figure Legends**

**Figure 1. Phenotypic characterization of De-hMSCs.**De-hMSCs were prepared by stimulating undifferentiated hMSCs in adipogenic induction medium for 48 hours, washed with PBS, and then incubated in growth media without inducible factors. (a) FCM analysis of the expression of indicated cell surface markers for De-hMSCs. (b) The staining of Alizarin Red, Oil Red O and Alcian Blue for osteogenesis, adipogenesis and chondrogenesis of De-hMSCs, respectively (scale bar = 30 μm).

**Figure 2. Enhanced adipogenic differentiation in De-hMSCs.** hMSCs and De-hMSCs at passage 4 were stimulated in adipogenic induction media for a period of 21 days. (a) Microscopic visualization of ORO stained cells at 0 and 3 weeks after differentiation was induced. Scale bar: 30 μm. (b) Quantitative assessment of ORO staining in hMSCs and De-hMSCs. (c) RT-qPCR analysis for selected markers of adipocytic cells 21 days after adipogenic differentiation induction. The ΔΔCT method was used for relative quantification with *GAPDH* as reference gene. Statistical comparison between groups was performed with student’s t-test; \*\**p* < 0.01 vs. hMSCs. w: week.

**Figure 3. Comparative measurement of proliferation capacity of hMSCs and De-hMSCs.** (a) Cell proliferation assessed by CCK-8 colorimetric assay. hMSCs and De-hMSCs were seeded in 96-well plates and maintained in growth media over a 72-hour period. Absorbance was measured at 450 nm directly in the culture plates at various time schedules as indicated. \**p* < 0.05 vs. hMSCs; ns, no significance. (b) Representative cell cycle distribution of hMSCs and De-hMSCs. Data shown were from triplicate experiments.

**Figure 4. Improved cell survival of De-hMSCs under oxidative stress.** (a) CCK-8 assay of survival rates of hMSCs and De-hMSCs exposed to 0-400 µM t-BHP (\**p* < 0.05, \*\**p* < 0.01 vs. corresponding control). (b) Representative FCM analysis of Annexin V-FITC/PI-stained cells in hMSCs and De-hMSCs challenged with 300 μM t-BHP. (c) Quantificative analysis of primary FCM plots from three independent experiments (\**p* < 0.05, \*\**p* < 0.01; ns, no significance).

**Figure 5. Expression levels of apoptosis-related proteins in hMSCs and De-hMSCs treated with or without t-BHP.** Cells were treated with 300 μM t-BHP for 24 hours. (a) Western blot images for bcl-2, bcl-xL, caspase-3, and PARP expression under the indicated conditions. GAPDH was used as a loading control. (b) Quantification of band intensity via densitometric analysis. Values were calculated from three independent experiments. #, *p* < 0.05 vs. hMSCs; \*, *p* < 0.05 vs. hMSCs+t-BHP; \*\*, *p* < 0.01 vs. hMSCs+t-BHP.

**Figure 6. Autophagy analysis in hMSCs and De-hMSCs under oxidative stress.** (a) Western blot assessment for the expression level of autophagy related protein LC3. (b) Relative fold changes of LC3-II protein expression normalized to GAPDH. Data were from three independent experiments. \*\**p* < 0.01, \*\*\**p* < 0.001 vs. hMSCs; ns, no significance vs. De-hMSCs. (c) Confocal microscopic visualization of GFP-LC3-II puncta in hMSCs and De-hMSCs (scale bar = 30 μm).

**Figure S1. Changes in cell morphology during adipogenic differentiation and dedifferentiation.** Microscopic images were taken at the start of differentiation (indicated as hMSCs), after adipogenic induction for 48 hours (adipogenic hMSCs), and 48 hours after adipogenic medium withdrawal (De-hMSCs). (a) Monoclonal hMSCs were expanded and formed a monolayer of homogenous bipolar spindle-like cells with a whirlpool-like array when confluent. (b) When subjected to adipogenic induction medium, hMSCs lost their fibroblastic morphology and adopted a more spherical shape reminiscent of adipocytes. (c) Withdrawal of induction medium rapidly reverted adipogenic hMSCs to characteristic MSC morphology.