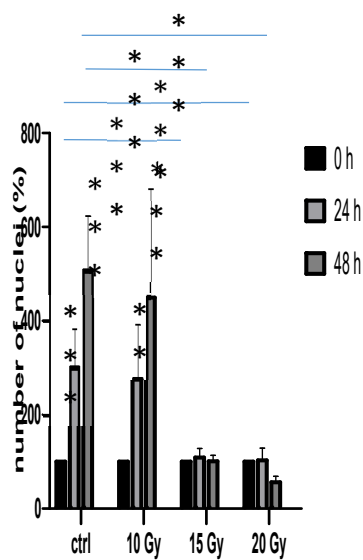
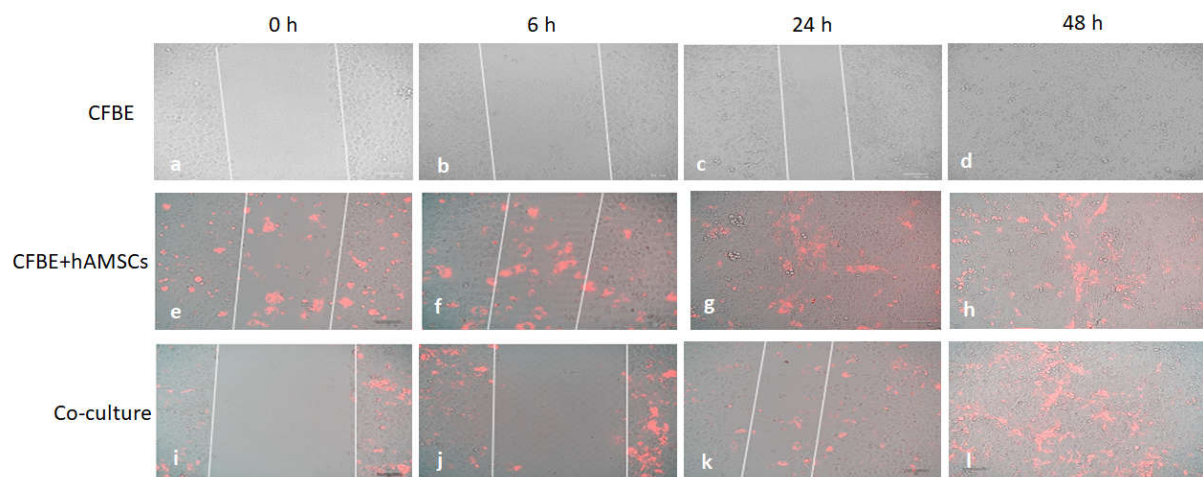
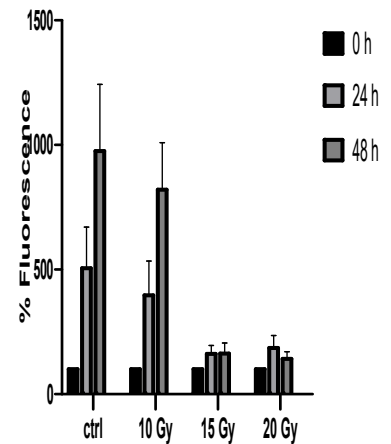
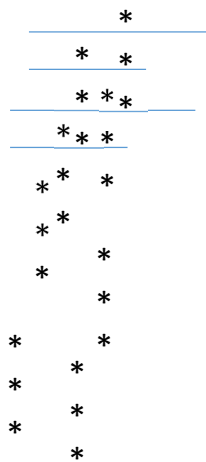
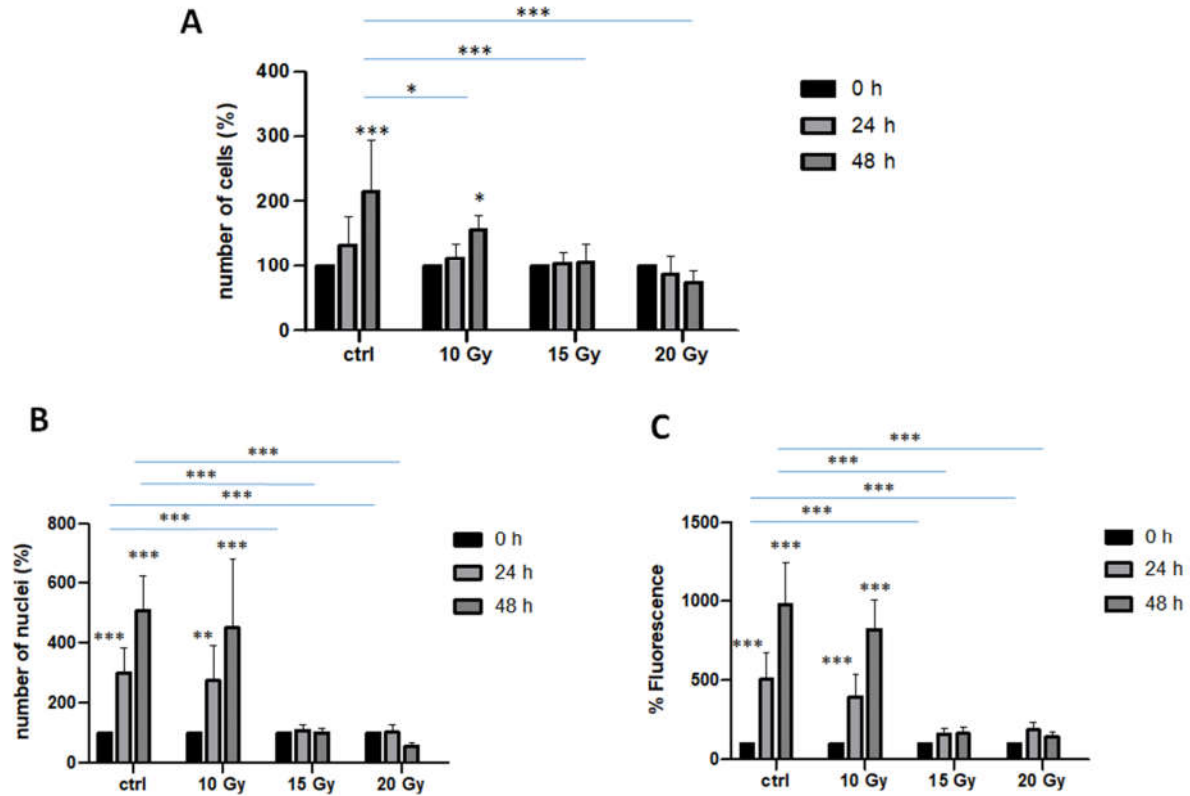


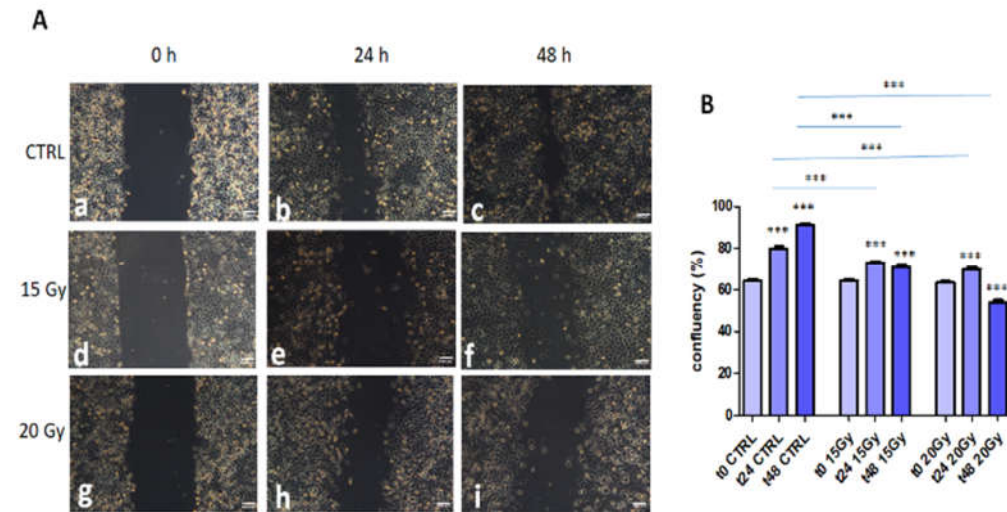
Supplementary Figure S1. Marker expression in hAMSCs. hAMSCs were evaluated for CD29, CD105, CD73, CD14, CD34, and CD 45 at isolation (p0) and p1-5 by flow cytometry. Results are expressed as mean \pm SD of triplicates for each isolate. * $p < 0.05$; ** $p < 0.01$, *** $p < 0.001$.

A**E**

Supplementary Figure S2. Wound closure in the presence of fluorescent hAMSCs. Injury was determined on CFBE (a-d), CFBE with the addition of CM-Dil-labeled hAMSCs (6×10^4) after wound (e-h), and CM-Dil-hAMSC:CFBE co-cultures (i-l). Wound closure was evaluated at 0 h (a, e, i), 6 h (b, f, j), 24 h (c, g, k), and 48 h (d, h, l). White lines denote wound edges. Bar = 100 μ m.



Supplementary Figure S3. CFBE proliferation is blocked by γ -rays. CFBE cells were irradiated at different doses of γ -rays (10, 15, or 20 Gy) and evaluated for cell numbers (A), numbers of DAPI-stained nuclei (B) or DAPI-associated fluorescence (C) as compared with untreated controls (ctrl). In each condition, time 0 is considered as 100%. Results are shown as mean \pm SD of three experiments. * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.



Supplementary Figure S4. Wound closure of CFBE monolayers after γ -irradiation. A) CFBE monolayers were γ -irradiated with 15 Gy (d, e, f) or 20 Gy (g, h, i) and then wounded. Control monolayers were not irradiated (a, b, c). Wound closure was evaluated at time 0 (a, d, g), 24 h (b, e, h) and 48 h (c, f, i). Bar = 100 μ m. B) Percentages of wound closure of non-irradiated CFBE, CFBE irradiated with 15 Gy, and CFBE irradiated with 20 Gy. *** $p < 0.001$.