SUPPLEMENTAL INFORMATION

Hyperosmolar potassium inhibits myofibroblast conversion and reduces scar tissue formation

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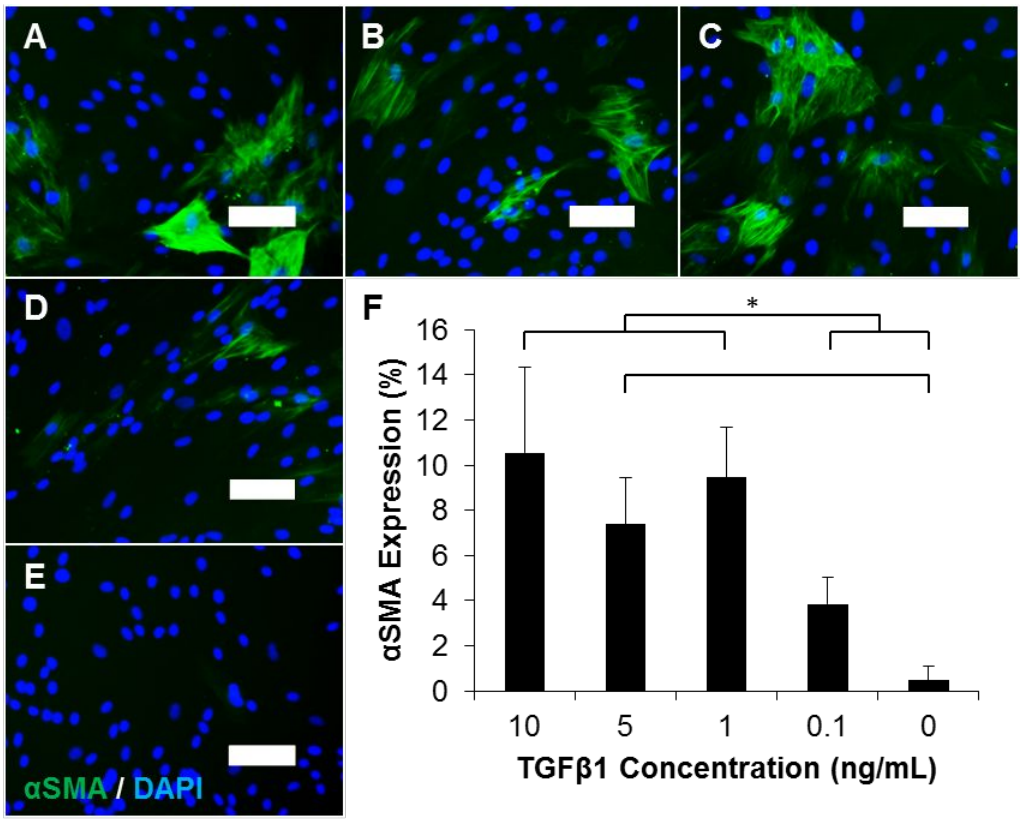

Figure S1. Titration of TGFβ1 to induce myofibroblast phenotype. (A-E) Representative images of fibroblasts cultured with (A) 10, (B) 5, (C) 1, (D) 0.1, and (E) 0 ng/mL TGFβ1. Myofibroblasts were identified via αSMA expression. Scale: 200 μm. (F) Quantification of αSMA positive area. * ($p<0.05$) and brackets indicate significance from other TGFβ1 concentrations as determined by one-way ANOVA with Holm-Sidak post hoc analysis (n=2 independent experiments).

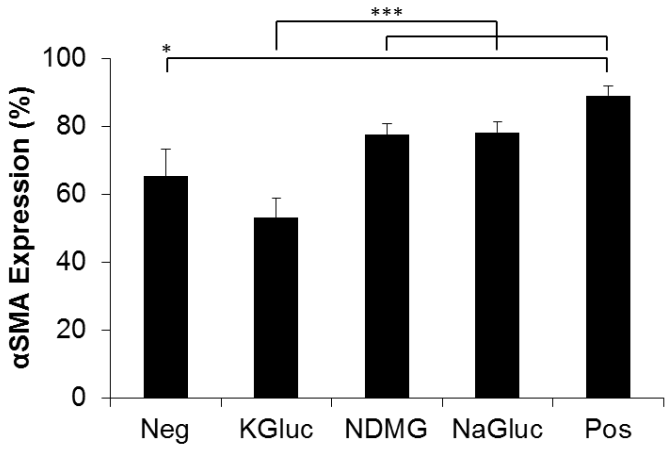

Figure S2. Determination of hyperosmolar contributions to KGluc-mediated inhibition of myofibroblast differentiation. Quantification of αSMA positive area of fibroblasts cultured in growth medium (negative control) or differentiation medium (positive control) supplemented with KGluc, NDMG (non-charged osmolar control), or NaGluc (charged osmolar control). * ($p<0.05$), *** ($p<0.001$), and brackets indicate significance from other TGFβ1 concentrations as determined by one-way ANOVA with Holm-Sidak post hoc analysis (n=2 independent experiments).

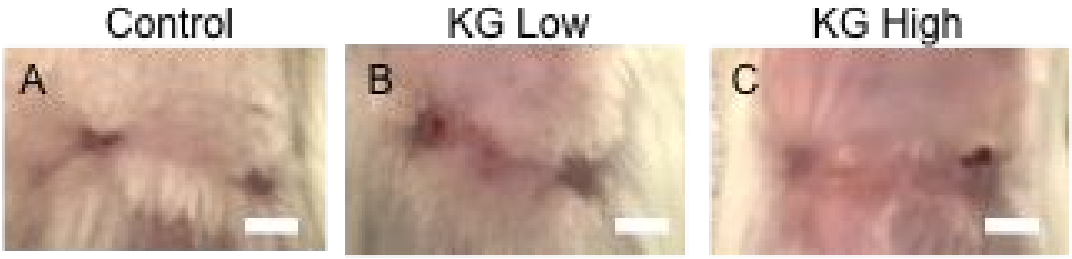

Figure S3. Representative images of full thickness skin defects after 14 days of healing that have been treated with (A) vehicle (Control), (B) hydrogel with low potassium gluconate (KG Low), or (C) hydrogel loaded with high potassium gluconate (KG High). For all images, the wound site on the left received treatment while the wound site on the right was a control that did not receive any intervention as an internal control. Scale: 0.5 mm.