SUPPLEMENTARY FIGURES

A mechanomimetic model of skin fibrosis

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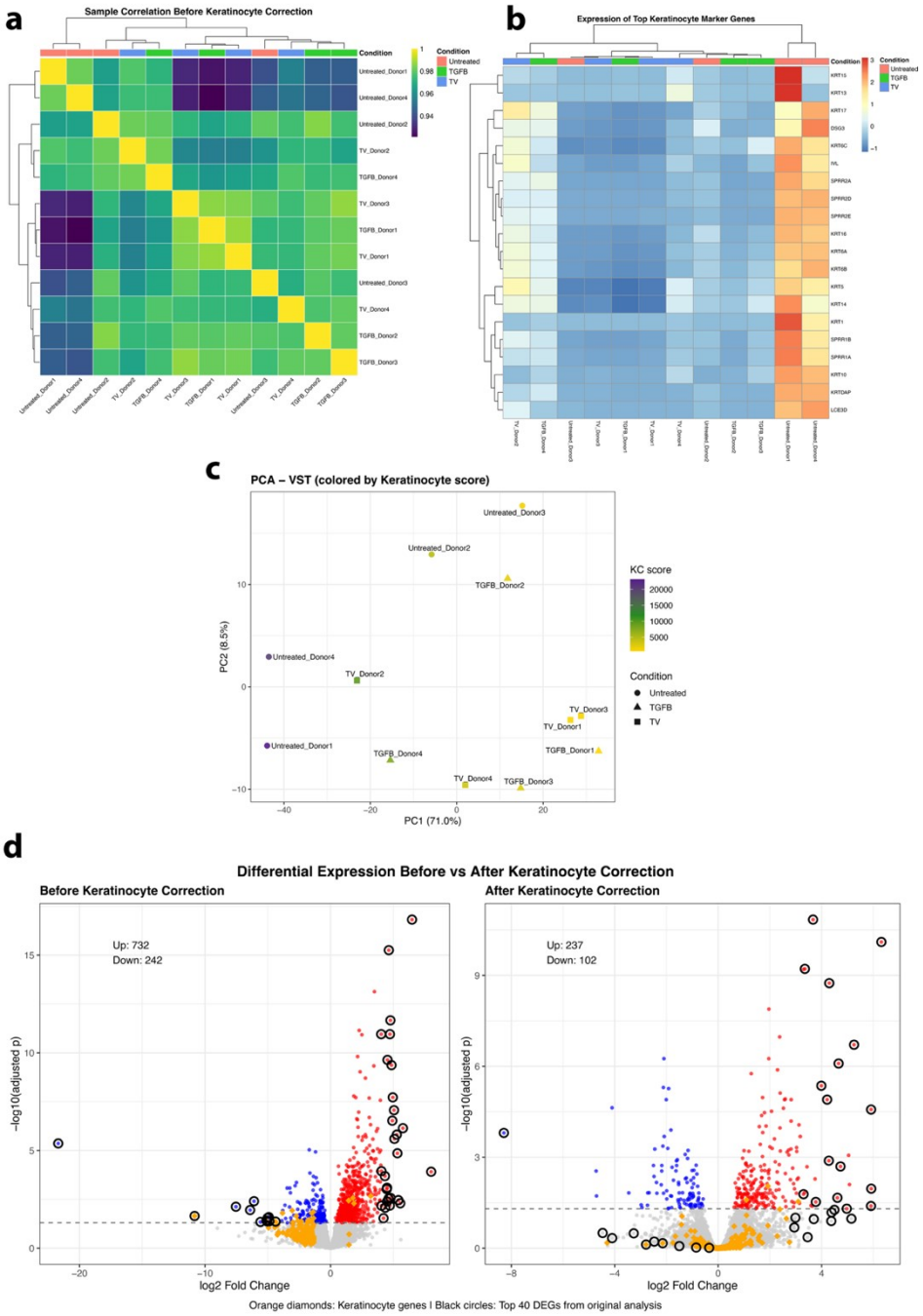
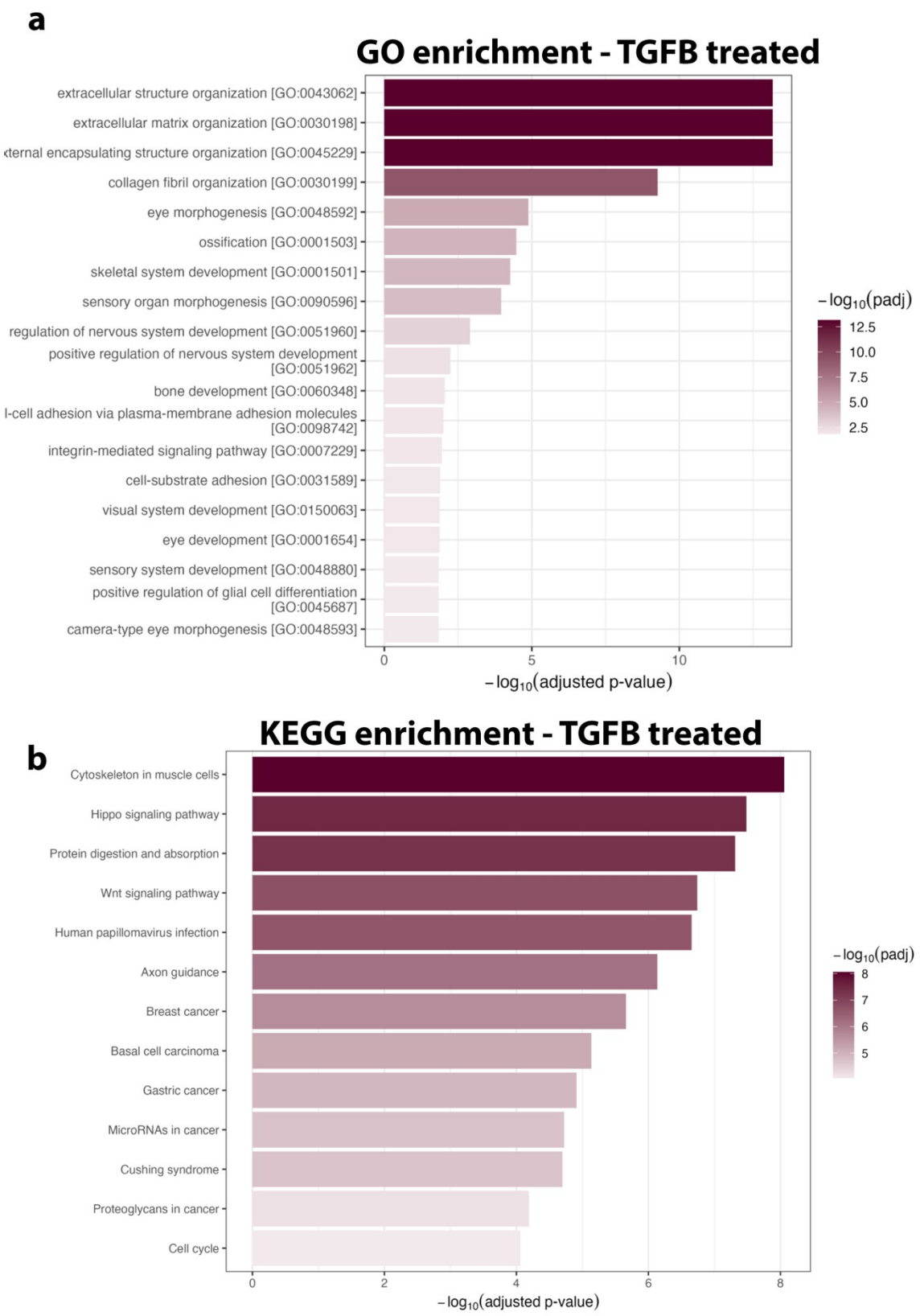
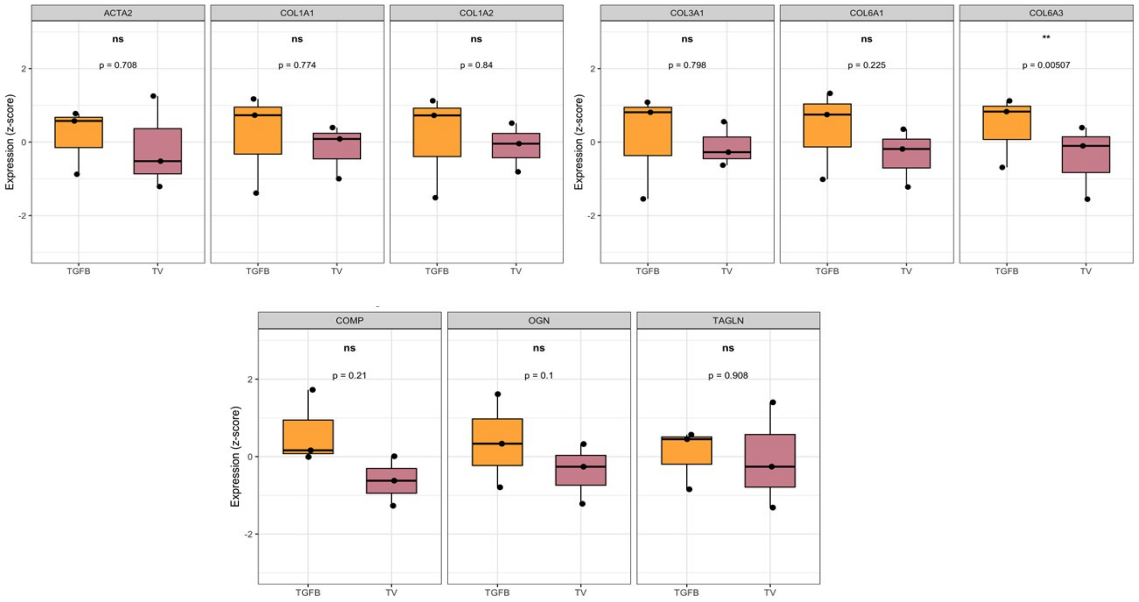


Supplementary Figure 1. Normalization of signal to exclude KCs. a) Sample correlation before KCs normalization. **b**) Principal component analysis indicating the KCs contamination score. **c**) Heatmap showing the differential expression level of KCs top marker genes across samples. Untreated donors 1 and 4 showed a substantial contamination. **d**) Volcano plot showing the differential gene expression of untreated control vs TGFb1-treated before and after normalization.

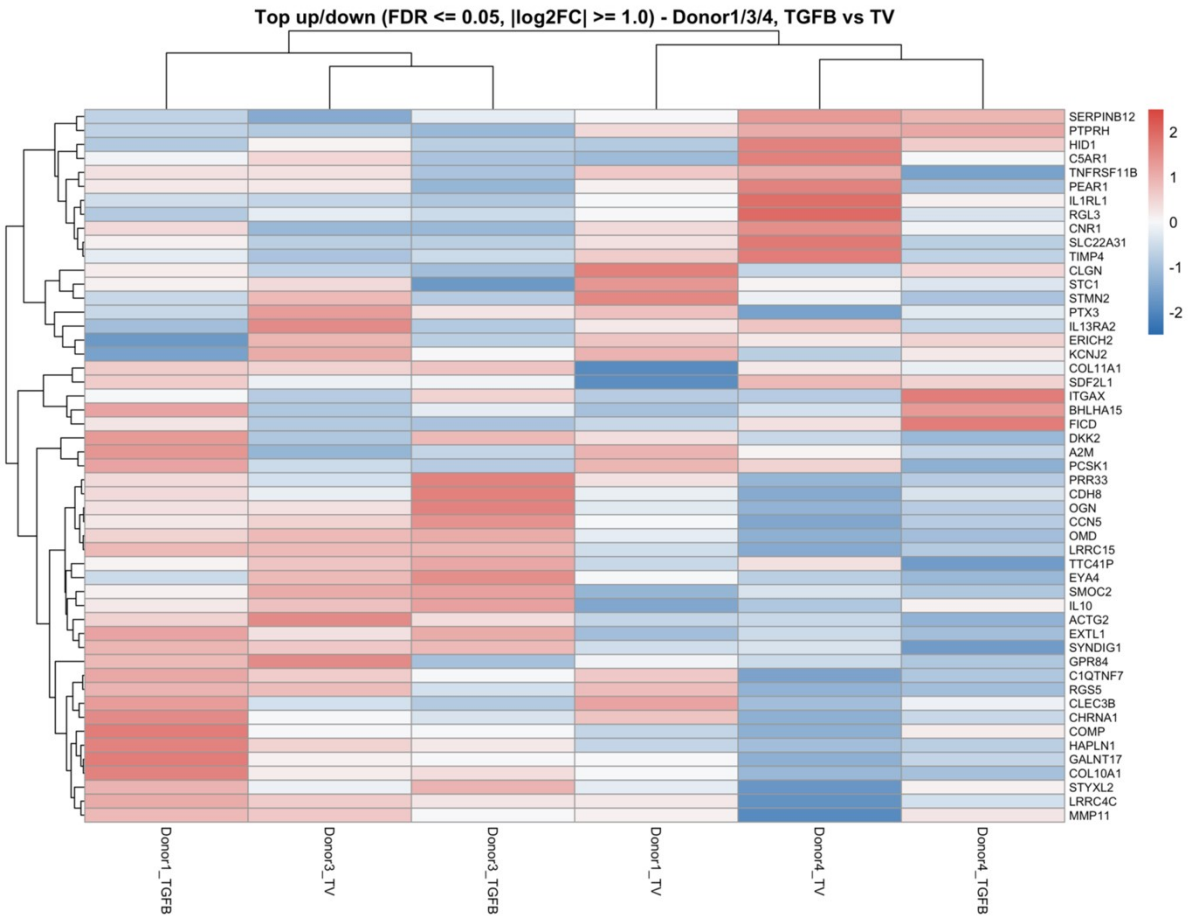


Supplementary Figure 2. Additional pathway analysis. GO (**a**) and KEGG (**b**) show similar pathway enrichment, mostly associated with extracellular organization, adhesion, collagen metabolism, integrin signaling and organ development.

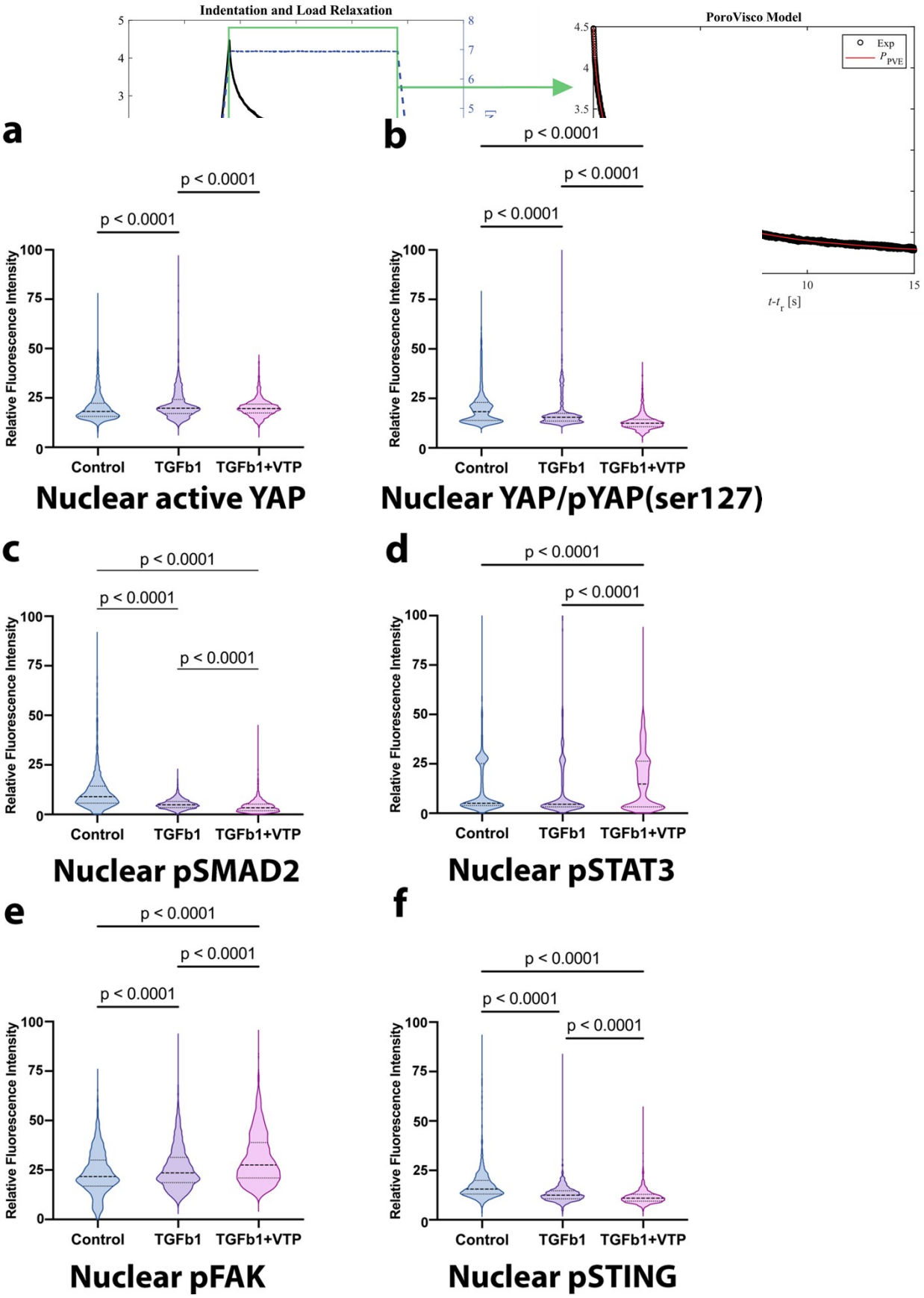
TGFB vs TV paired t test



Supplementary Figure 3. Paired t-test of fibrosis-associated genes (TGFB vs TV, donors 1, 3, and 4). COL6A3 was significantly downregulated under TV, while other genes showed non-significant downward trends. Donor 2 was excluded due to lack of response to TGFB.



Supplementary Figure 4. Bulk RNA-sequencing analysis comparing selected TGFb1-treated vs TGFb1+VTP-treated donors. Heatmap showing differentially expressed fibrosis-associated genes between TGFB and TV (verteporfin) treatment across three donors. The data highlight donor-specific transcriptional responses to TGFB-induced fibrosis and verteporfin treatment.



Supplementary Figure 5. Quantification of mechanosignaling mediators localized in the nucleus. IF was used to assess the nuclear localization of YAP, pYAP, pSMAD2, pSTAT3, pFAK, pSTING, and to calculate the YAP/pYAP ratio (**e-j**) (n>1700 per condition). The statistical analysis was performed using one-way ANOVA.