Electronic Supplementary Material (ESI) for Soft Matter.
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Supporting Information

A Fiber-Reinforced Composite Vascular Graft Modulates Macrophage Polarization and Enhances Endothelial Cell Migration

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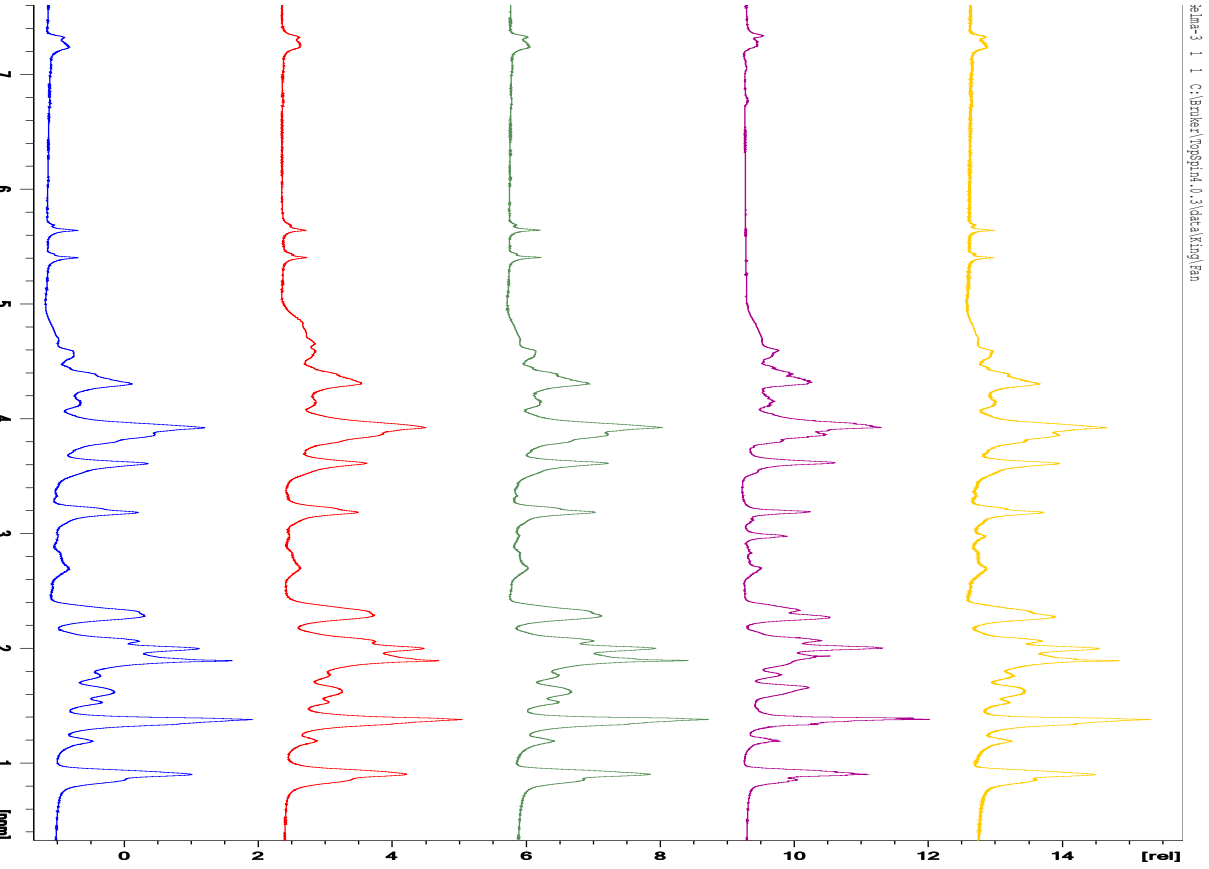

Figure S1. NMR spectra of different batches of GelMA. Purple: unmodified gelatin. Yellow, green, red and blue represent different batches of GelMA.

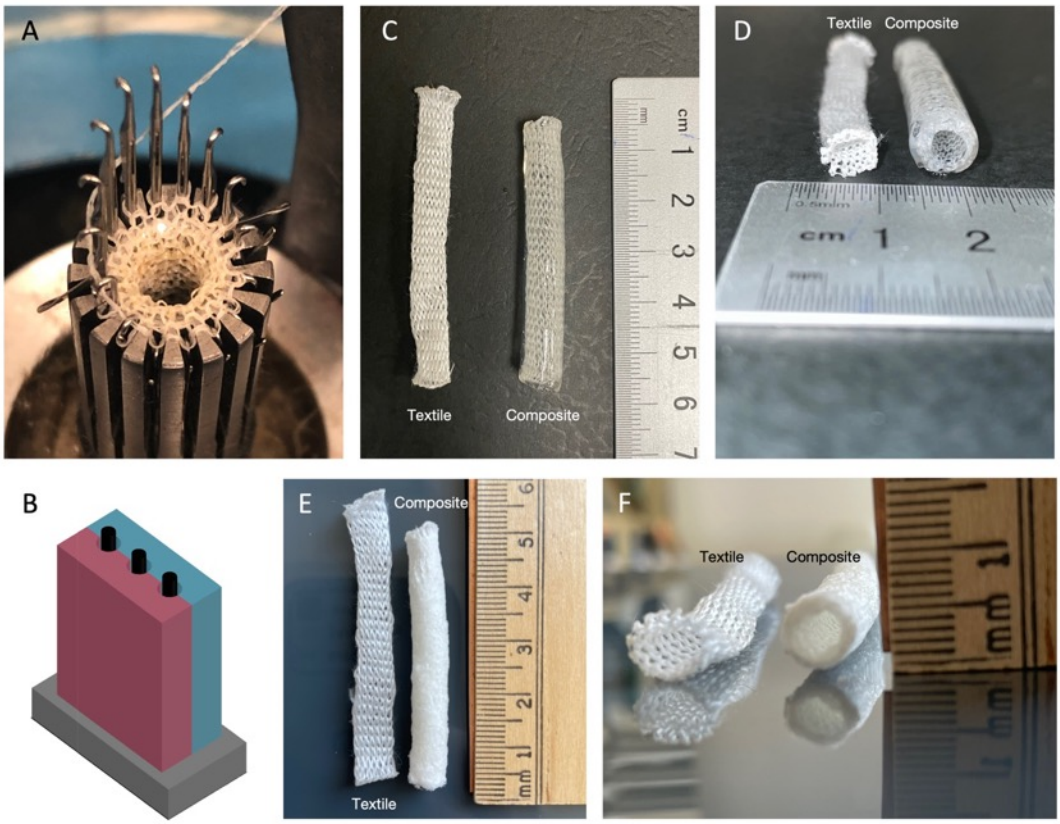

Figure S2. Fabrication of the textile-reinforced hydrogel composite vascular graft. (A) A polylactic acid multifilament yarn was fed into a Lamb circular knitting machine to knit the textile reinforcement. (B) A Teflon® mold was designed for hydrogel conduit formation. The plum, blue and grey parts of the mold can be separated and reassembled for inserting the reinforcing conduit onto the black rod. GelMA prepolymer solution was then added to the mold and cooled at -20˚C for 5 min to allow the hydrogel to form a temporary physical network to hold the shape of a conduit. Then the mold was disassembled, and the composite conduit was mounted on a circulating rotor and photo-crosslinked using ultraviolet light at a wavelength of 365 nm for 20 min while rotating to ensure even crosslinking. (C-F) Gross photographs of the textile reinforcement (left), the composite vascular graft (C&D) under hydrated conditions and the composite graft (E&F) after dehydration using lyophilization.

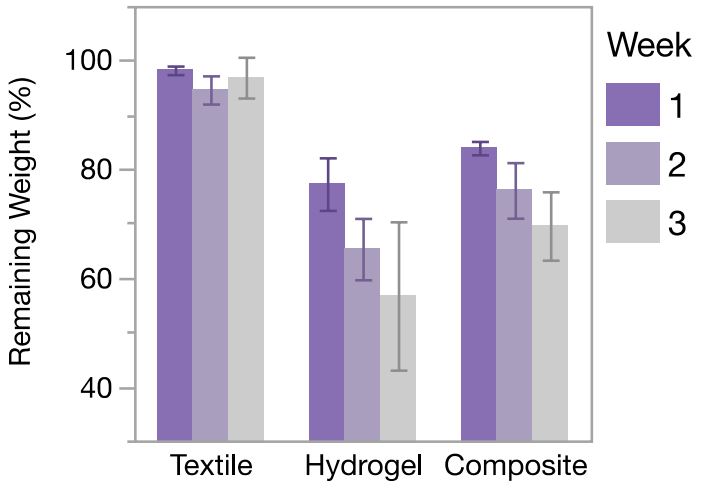

Figure S3. Degradation of composite and its component in collagenase I solution at 37 ˚C over 3 weeks.

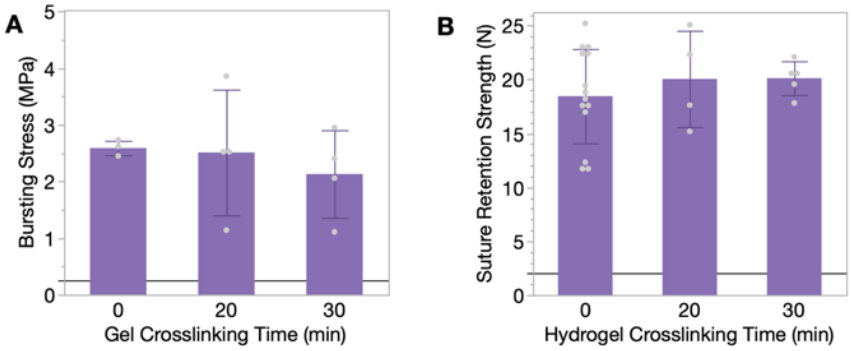

Figure S4. Hydrogel crosslinking time did not impact the (A) bursting strength and (B) suture retention strength of the textile/hydrogel composite vascular graft. Black lines indicate the minimum requirement for the vascular graft application.

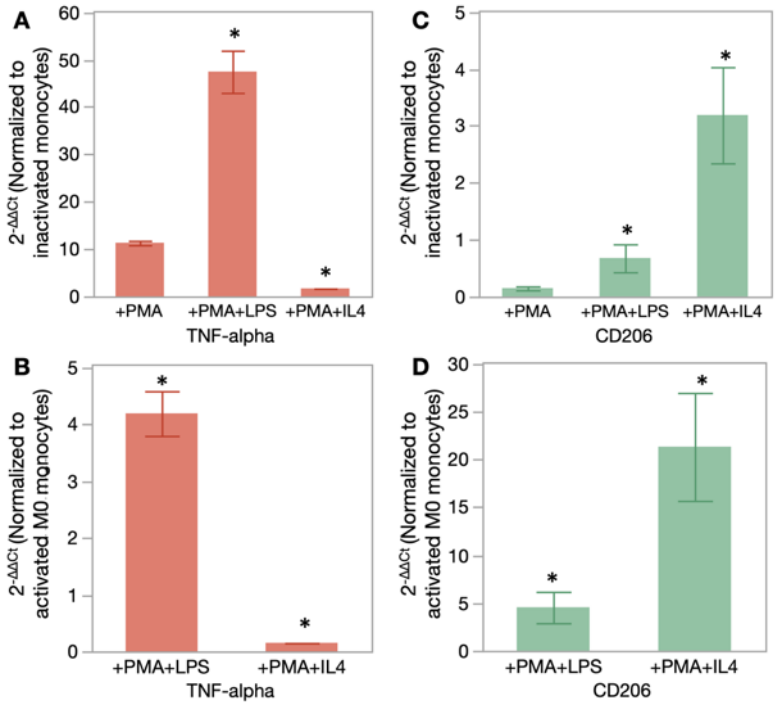

Figure S5. Gene expression of macrophages in response to chemical/cytokine treatments. THP-1 cells purchased from ATCC were stimulated by 200 ng/mL phorbol 12-myristate 13-acetate (PMA) to convert THP-1 monocytes into M0 macrophages. The M0 macrophages were then treated with either 100 ng/mL lipopolysaccharide (LPS) or 40 ng/mL recombinant human interleukin-4 (IL-4) to polarize them to an M1 or M2 phenotype respectively. The gene expression of (A-B) M1 macrophage marker tumor necrosis factor-alpha (TNF-alpha) and (C-D) M2 marker CD206 was measured. The $2^{-\Delta\Delta Ct}$ method was used to measure the relative expression of TNF-alpha and CD206. All gene expressions were first normalized to the endogenous control gene glyceraldehyde-3-phosphate dehydrogenase (GAPDH). In (A & C), the gene expression was further normalized to the untreated THP-1 monocytes. And In (B & D), they were normalized to the PMA treated M0 macrophages. (A & B) PMA activated M0 significantly upregulated the M1 marker, TNF-alpha, compared to the THP-1 monocytes. LPS treatment further upregulated THF-alpha. On the contrary, IL-4 treatment significantly downregulated THF-alpha. (C & D) The PMA and LPS treatments downregulated the M2 marker CD206 while the IL-4 treatment upregulated it. *$p<0.05$.

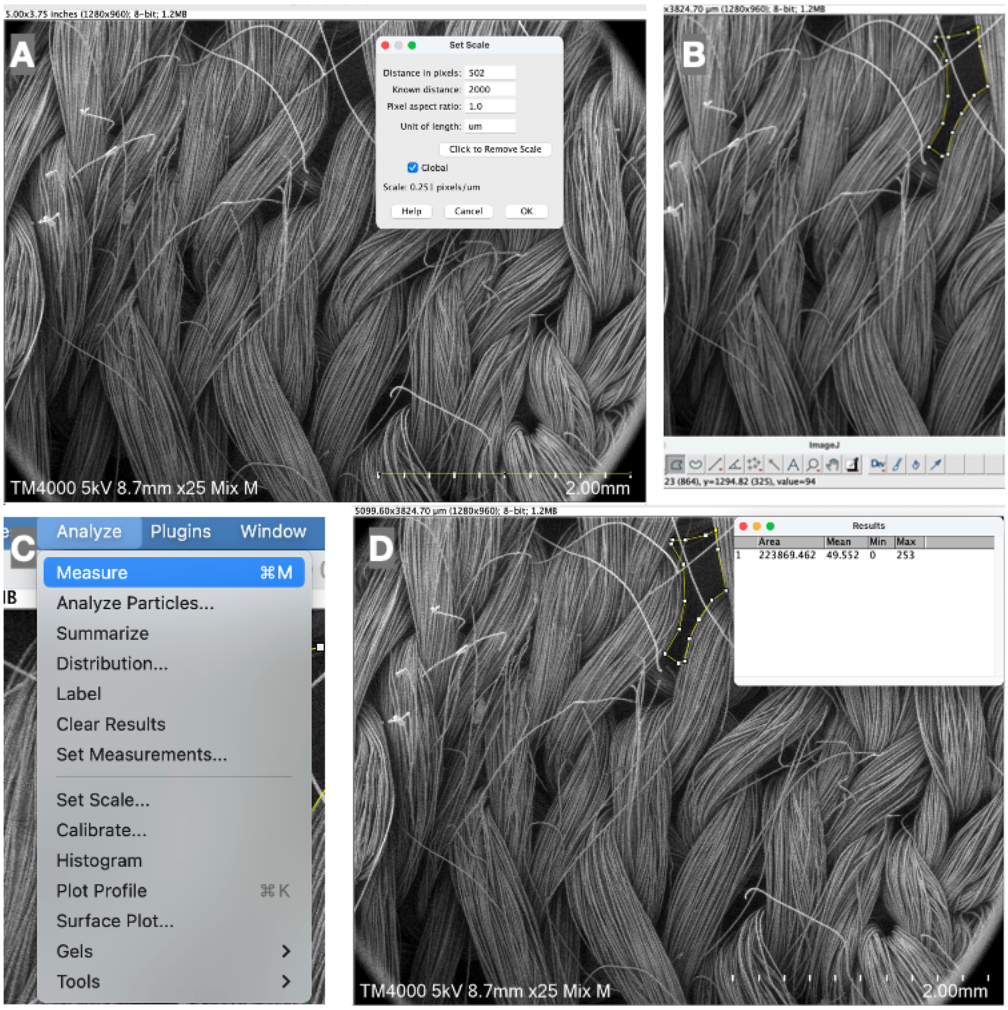

Figure S6. Step-by-step procedures for pore size measurement using Image J. (A) Set scale for the measurement. (B) Select pore area using "Polygon Selections" tool. (C) Using Analyze-Measure function to measure the selected area. (D) Obtain the Area result calculated by the software.