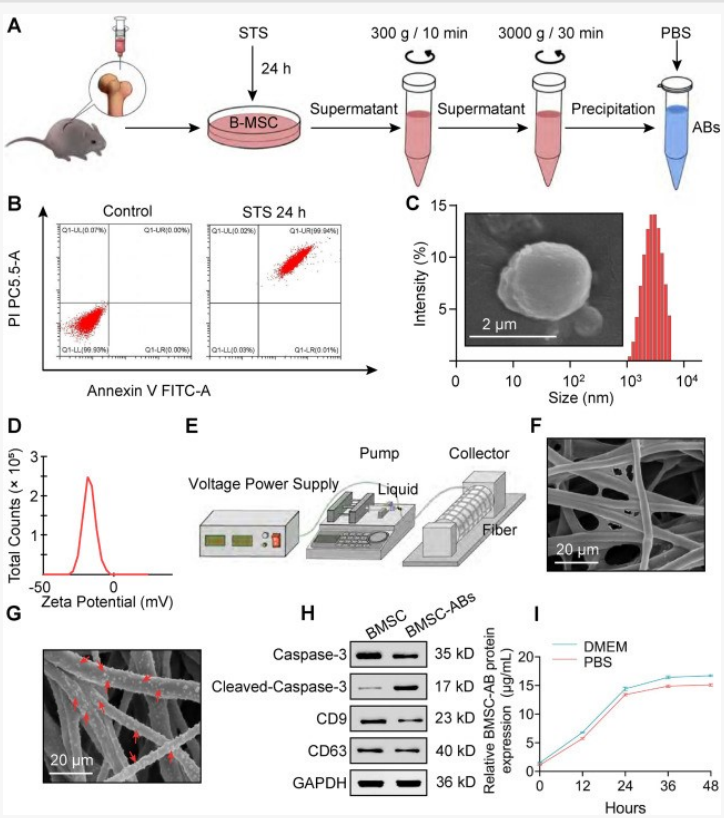
Apoptotic Vesicles Loaded Fibrous Scaffolds Promote Wound Healing

PCL fiber scaffolds loaded with BMSC-derived apoptotic vesicles regulate macrophage polarization via miR-21a-5p to promote wound healing

CHINA, March 6, 2025 /EINPresswire.com/ -- A recent study showed that paracrine delivery of miR-21a-5p by bone marrow mesenchymal stem cells-derived apoptotic bodies (BMSC-derived AB)-loaded polycaprolactone (PCL) scaffolds promote wound healing by regulating macrophage polarization to M2 phenotype.



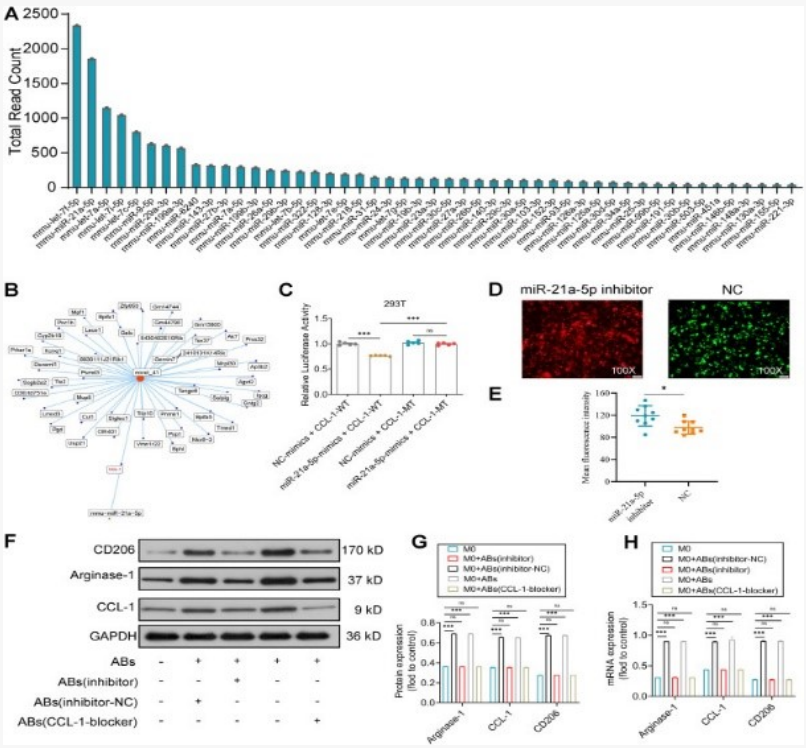
(A) Acquisition of BMSC-ABs. (B) Identification of apoptotic cells by flow cytometry. (C) Morphological images of ABs. Left: scanning electron microscopy (SEM) images of ABs (scale bar, 2 μm); right: particle size of ABs measured by dynamic light scatteri

Macrophages are critical players mediating wound healing, and the microenvironment of the wound influences macrophage polarization to either the M1 pro-inflammatory phenotype in the early stages or the M2 pro-healing phenotype in the later stages. Dysfunction of the macrophage polarization M2 phenotype, lower macrophage numbers, and reduced anti-inflammatory and angiogenic capacity are causatives of long-term non-healing.

Mesenchymal stem cells (MSCs) aid tissue repair and regeneration by delivering endogenous regulatory factors via paracrine extracellular vesicles. Previous studies have shown that BMSC-derived ABs induce macrophage polarization to the M2 phenotype, which mitigates inflammation and enhances the migration and proliferation of fibroblasts.

In this study, published in the Genes and Diseases journal, researchers at Chongqing Medical University and Sichuan Provincial Orthopedic Hospital investigate the wound healing efficacy and macrophage polarization potential of electrospun PCL scaffolds loaded with BMSC-ABs, in vitro

and in vivo.
The results showed that the BMSC-AB loaded PCL scaffolds could prevent or reduce delayed wound healing by reprogramming the macrophages into the M2 phenotype and reducing inflammatory infiltration, thereby potentiating anti-inflammatory and angiogenic effects. Furthermore, the paracrine delivery of miR-21a-5p through the slow-release of BMSC-AB by the PCL scaffolds reprograms macrophages to the M2 phenotype by regulating the expression of the CCL-1 gene, promotes fibroblast migration, and initiates angiogenesis by secreting anti-inflammatory cytokines and the angiogenesis-related factors, VEGF and vWF.



(A) The top 50 known miRNAs detected in BMSC-ABs. (B) miRNA-mRNA regulatory network. (C) The binding of miR-21a-5p to the target gene CCL-1 in 293T cells validated by dual luciferase assay. (D) Colocalization of miR-21a-5p with M□s. (E) Fluorescence inten

In conclusion, the BMSC-AB-loaded PCL scaffolds, through the targeted and sustained delivery of miR-21a-5p, drive macrophage polarization to the M2 phenotype, which exerts a dual effect by regulating inflammation and angiogenesis, thereby synergistically promoting wound healing.

Reference

Title of the original paper: Fibrous scaffolds loaded with BMSC-derived apoptotic vesicles promote wound healing by inducing macrophage polarization.

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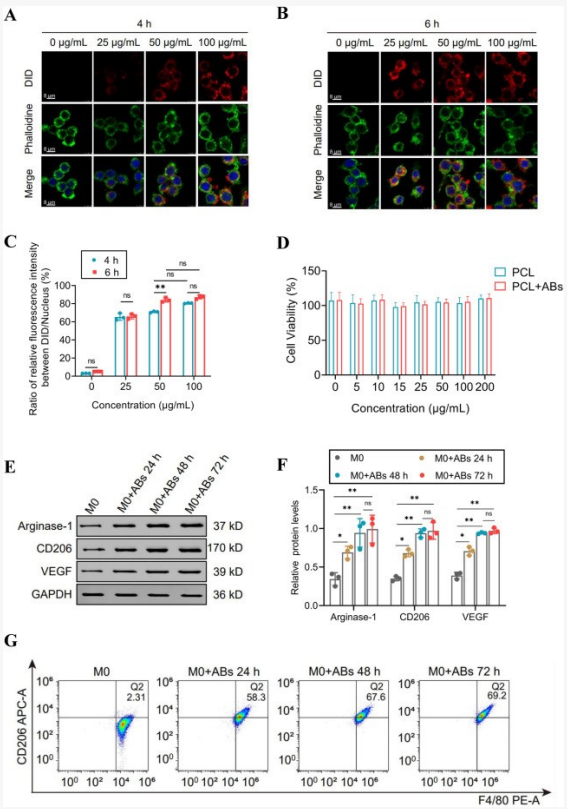

(A) Immunostaining of 0–100 μg/mL PCL-BMSC-ABs incubated for 4 h. Green: cytoplasm; blue: nucleus; red: ABs. (B) Immunostaining of 0–100 μg/mL PCL-ABs incubated for 6 h. Green: cytoplasm; blue: nucleus; red: ABs. (C) Relative fluorescence intensity of 0–1

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