Life Sciences and
Facility Management
ICBT Institute of Chemistry
and Biotechnology

New Product Showcase / Area 1435

Acoustic Bioactivation Of Autologous Blood: A Biophysical Platform For Advanced Wound Repair

Koeffel René, PhD*; Straumann Salome, BSc; Kern Stefanie, BSc; Bergmann Timothy, PhD (*contact: koff@zhaw.ch)

Background

Sound Induced Morphogenesis (SIM) is a novel acoustic biofabrication technology that uses precisely tuned sound signatures to orchestrate spatial organization and functional activation of biological systems. In this study we investigated the potential of SIM for bioactivating autologous blood in the process of blood clot formation for treatment of chronic and non-healing wounds.

Methods

Whole-blood samples were subjected to controlled acoustic signatures using SIM technology, while untreated clots served as negative controls (Figure 1). Activation profiles and structural metrics of SIM-treated and control samples were analysed by histological methods and serial image analysis. The acoustic field parameters were optimized to promote structural reorganization of blood components. Morphological characterization was performed via microscopy to assess fibrin density and spatial distribution of cellular components. This study focused on investigating the activation of thrombocytes and leukocytes by SIM. CD62P and CD83 represent reliable markers for evaluating cellular responses within haemostatic and thrombotic processes. To address this, expression of these markers was analysed, with CD62P serving as an indicator for thrombocytes and CD83 for leukocytes.

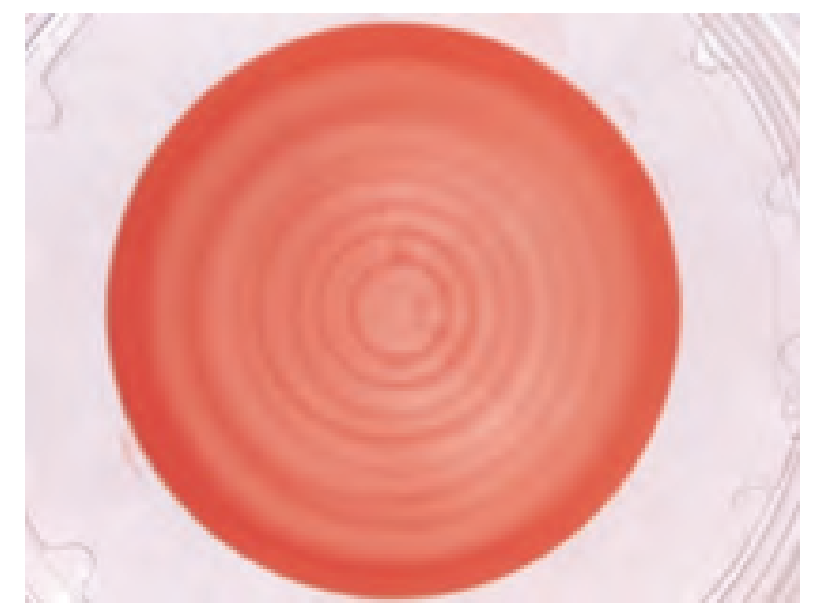


Figure 1: SIM induced bio-arrangement in autologous whole blood clot: Application of specific acoustic signature induce bioactive pattern formation. Figure shows circular cellular patterns forming bio-clusters creating concentrated regenerative hubs that can accelerate tissue repair

Results

SIM stimulation induced profound alterations in clot architecture relative to controls (Figure 2). Microscopic evaluation revealed a fibrin network interspersed with discrete clusters of leukocytes and platelets, forming localized regenerative hubs (Figure 3) In addition, a significantly enhanced blood cell activation was shown, i.e. by increased expression of CD83, after SIM stimulation (Figure 4).

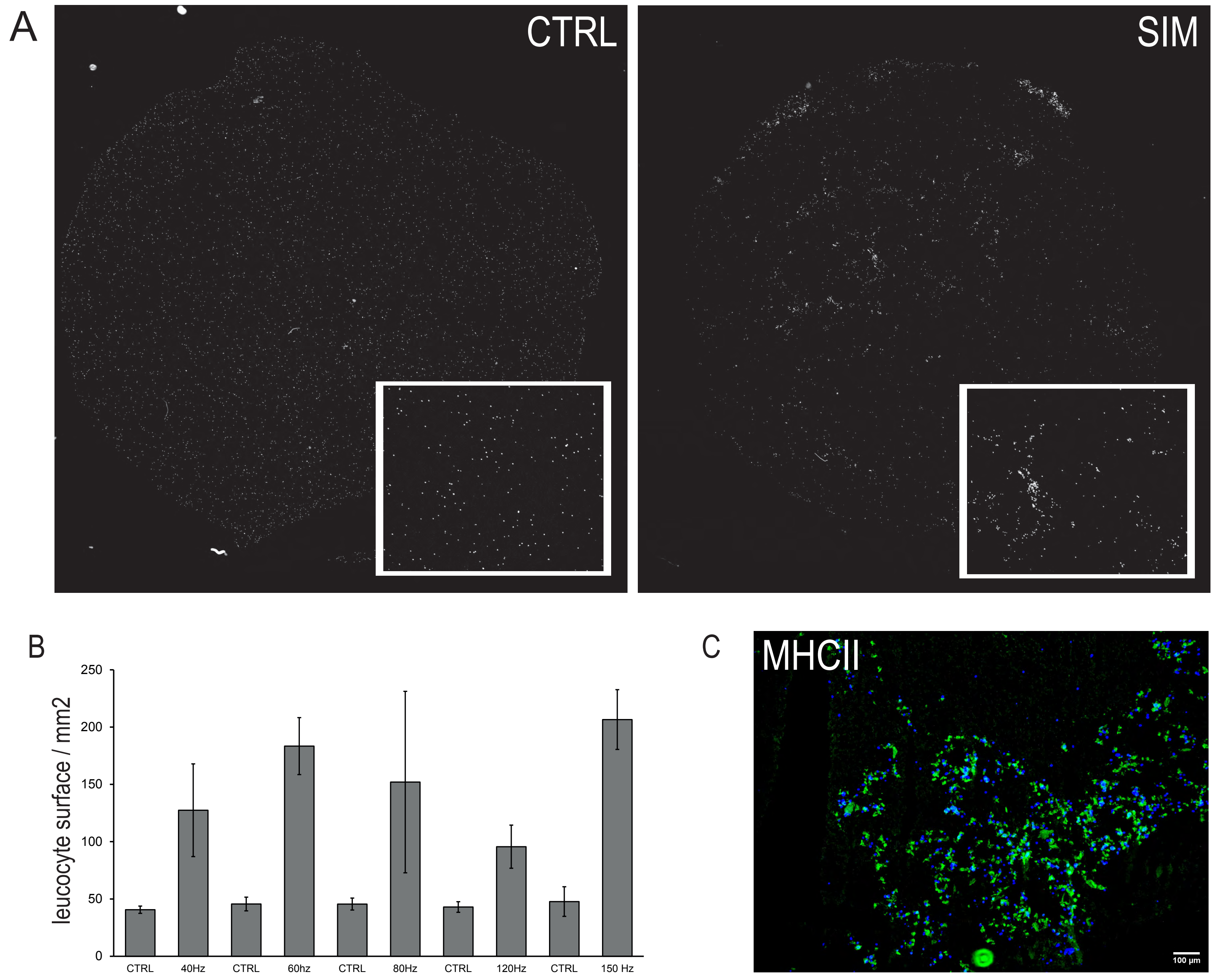
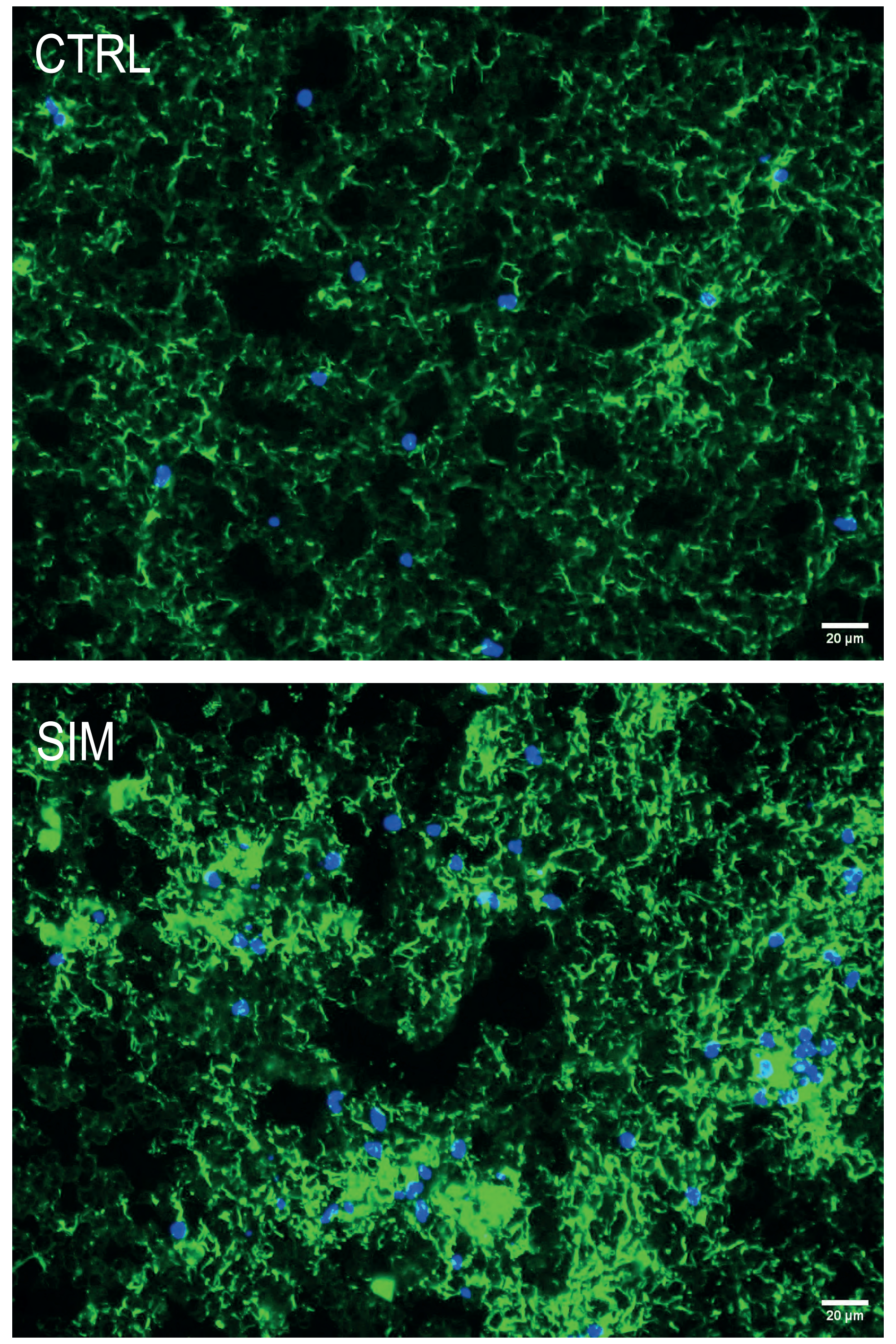


Figure 2: Leukocyte distribution in whole clots (A) Controls (CTRL) show evenly distributed leukocytes within the clot and SIM (F = 80 Hz, A = 0.8) treated clots show focal accumulations of leukocytes at the central area and at the borders of the clot. Inserts show maginfications of the central area. Representative data from one donor is shown (n= 6). (B) Quantification of leukocyte area sizes show increasing leukocyte accumulation at different frequencies (F= 60 Hz - 150 Hz) as compared to untreated controls (F=0Hz; CTRL). Mean values +/- SEM of 3 donors each are shown. (C) SIM induced localization of MHCII$^+$ cells in hubs.

Figure 3: Effect of SIM on leukocyte distribution and fribrin accumulation. (A) Controls show a loose fibrin network with single dispersed leukocytes. (B) SIM (F = 80 Hz, A=0.8) treated clots show a more dense fibrin network with compact fibrin streaks and localized accumulations of leukocytes. Representative data from one donor is shown (n=6). (DAPI, blue; fibrin, green)



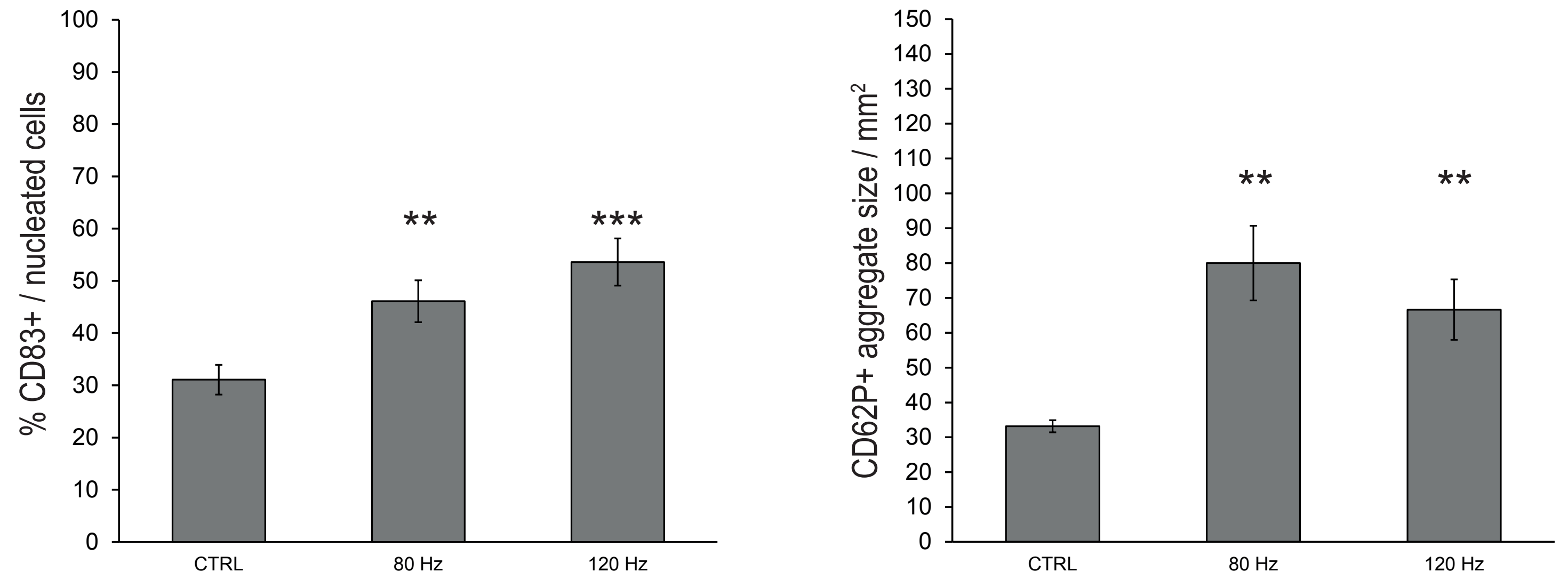


Figure 4: SIM induces an increase of immune cell and platelet activation markers in clots (A) Controls (CTRL) versus SIM treated clots (F= 80 Hz or 120 Hz). A significant increase of CD83+ leukocytes at F= 80 Hz and 120 Hz as compared to CTRL (F=0 Hz) is shown. Student t-test **p <0.01, *** p <0.001. (B) CD62P analysis in controls (F=0 Hz) versus SIM treated clots (F= 60 Hz - 150 Hz). An increase of CD62P signals at F= 80 Hz and 120 Hz as compared to CTRL (F=0 Hz) is shown. Student t-test **p <0.01. Mean values +/- SEM are shown (n=5).

Conclusions

Sound Induced Morphogenesis (SIM) induces marked remodeling of clot structure and cellular organization. Mechanical stimulation drives leukocytes from an even distribution into focal accumulations and increases leukocyte rich areas, including antigen presenting cells. At the same time, the fibrin network shifts from a loose mesh to a denser architecture with compact fibrin streaks aligned with leukocyte clusters. These structural changes coincide with enhanced immune and platelet activation, reflected by increased CD83 and CD62P expression at specific frequencies.
Overall, SIM generates a frequency dependent reorganization of the clot into a more bioactive matrix that may support angiogenesis and tissue repair. Future work will extend these findings to ex vivo wound models and preclinical systems to assess its regenerative potential.

This Work was supported by mimiX.

HEMATOLOGY &
IMMUNOLOGY