

In managing inflammation, controlling white blood cell flow may be key

Yale UniversityYale University

New research by Yale University scientists sets the stage for improved management of acute tissue inflammation related to wounds and chronic inflammatory diseases by advancing current understanding of inflammatory processes.

To exit blood vessels and reach injured tissue, white blood cells must pass through a series of natural barriers. Some aspects of blood vessel architecture facilitate white cell migration to a greater degree than others, a means of self-regulation: Excessive migration can result in extreme inflammation, turning otherwise helpful white cells into agents of disease.

Research by Yale bioengineers reported March 26 in the journal PLOS ONE sheds new light on the roles of specific layers of vasculature, suggesting ways of controlling inflammation.

“By understanding the regulatory mechanisms within the vascular wall, we hope we can identify potential treatments to ensure or restore the balance between protection and destruction of tissues,” said Anjelica L. Gonzalez, assistant professor of biomedical engineering at Yale and principal investigator of the research.

The work focuses in particular on the function of a less permeable (and little studied, researchers said) layer of cells within the blood vessel wall known as the pericyte layer.

Using a composite microvascular model that incorporates both the inner and outer layers of blood vessels, Gonzalez and colleagues showed that the outer pericyte layer helps restrict the number of exiting white blood cells. This helps prevent excessive inflammation, they said. In contrast, the inner (endothelial) layer primes white blood cells for passage through the pericyte layer by transforming them into a more versatile cell subpopulation. A malfunctioning pericyte layer could be responsible for excessive inflammation, they said.

“The results suggest that any disease or disorder that can be termed inflammatory — including wound healing, tissue fibrosis and cancer metastasis — may be exacerbated because of a poor pericyte barrier,” said Gonzalez. “White blood cell-mediated inflammation, in particular, is related to the progression of many inflammatory disorders. These findings give us targets on the white blood cell that will allow us to develop therapeutics aimed at inhibiting their contribution to disease progression.”

The paper’s lead author is Chantal E. Ayres-Sander. Co-authors are Holly Lauridsen, Cheryl L. Maier, Parid Sava, and Jordan S. Pober.

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