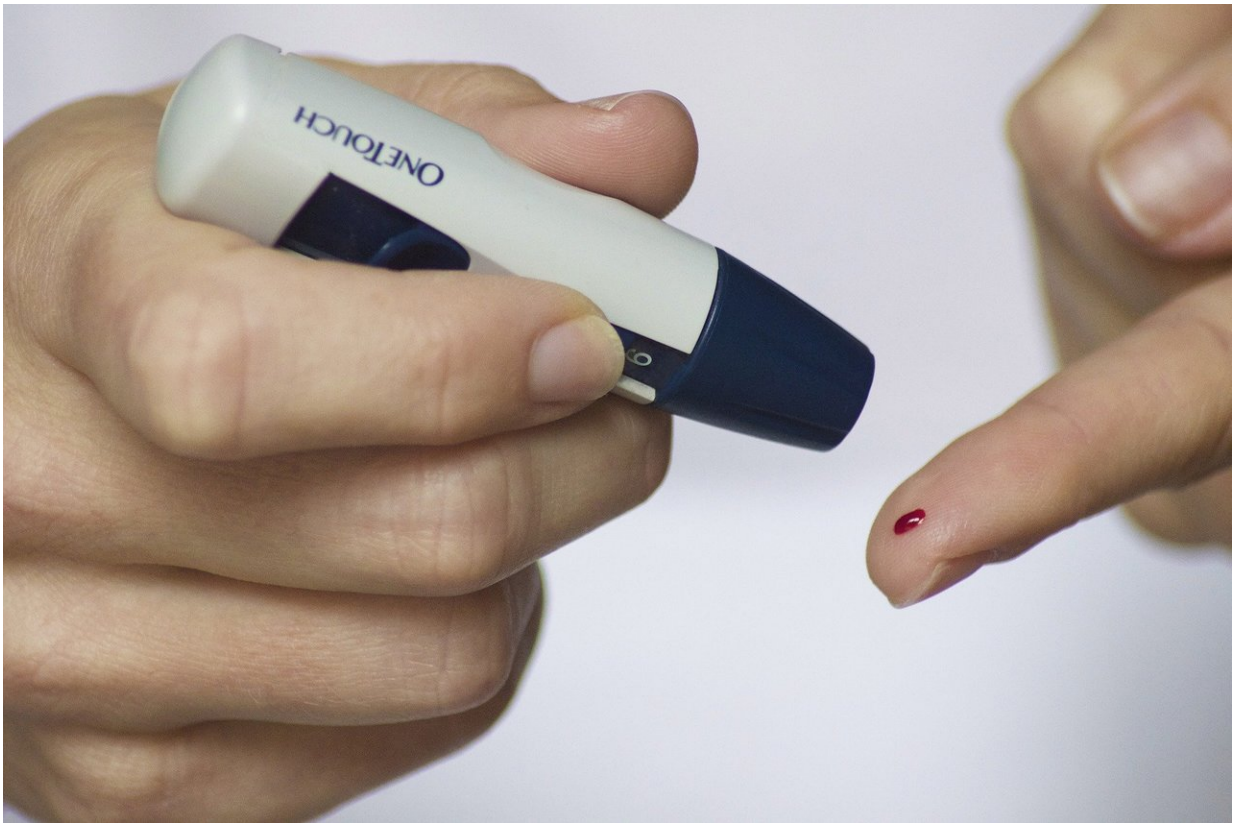
Parsing diabetic skin infections

December 3 2018, by Paul Govern



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People with diabetes are more susceptible to skin infection. According to a new study by C. Henrique Serezani, Ph.D., Stephanie Brandt, Ph.D., and colleagues, this susceptibility might be due to overabundance of a compound produced by phagocytes, called leukotriene B4 (LTB4).

The team found high levels of this inflammatory mediator in different mouse models of diabetes. High LTB4 in these mice was associated with larger nonhealing lesion areas and increased bacterial loads, as well as "dysregulated cytokine and chemokine production, excessive neutrophil migration but impaired abscess formation, and uncontrolled collagen deposition."

Shutting down LTB4's cellular receptor, BLT1, restored inflammatory response and abscess formation, leading to reductions in bacterial load and lesion size.

The authors conclude that "exaggerated LTB4/BLT1 responses mediate a derailed inflammatory milieu that underlies poor host defense in diabetes. Prevention of LTB4 production/actions could provide a new therapeutic strategy to restore host defense in diabetes."

The study appeared in the journal *JCI Insight.*

More information: Stephanie L. Brandt et al. Excessive localized leukotriene B4 levels dictate poor skin host defense in diabetic mice, *JCI Insight* (2018). DOI: 10.1172/jci.insight.120220

Provided by Vanderbilt University

Citation: Parsing diabetic skin infections (2018, December 3) retrieved 25 April 2024 from https://medicalxpress.com/news/2018-12-parsing-diabetic-skin-infections.html

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