

Biofilm Disruption May Alter *Borrelia*–Immune Signaling

Clinical research findings from the study of BioDisrupt® and the rationale for addressing biofilm alongside antimicrobial therapy

Clinical research highlights:

- Metabolites from untreated *B. burgdorferi* biofilm triggered high IL-10 production, which may suppress phagocyte recruitment and phagocytosis.
- BioDisrupt® treatment decreased IL-10 and increased MIP-1α, shifting immune-cell signaling toward phagocyte recruitment.
- These findings provide a mechanistic rationale for addressing biofilm alongside antibiotic or botanical antimicrobial therapy.

Borrelia burgdorferi can organize into biofilms, communities in which bacteria are embedded in a self-produced extracellular matrix. The matrix creates a dynamic, protective environment that supports bacterial persistence by helping the organisms evade host immune surveillance and subsequent clearance.

Biofilms can also actively reshape the host immune response. In a 2024 in vitro study, metabolites released from established *B. burgdorferi* biofilms triggered high production of the regulatory cytokine IL-10 in human immune cells. IL-10 can reduce signals that recruit phagocytes and suppress their ability to engulf bacteria, weakening a response needed for bacterial clearance.

When researchers treated established biofilms with BioDisrupt®, a nutraceutical blend of water-soluble enzymes and botanical compounds formulated to disrupt biofilms, levels of IL-10 decreased. Additionally, levels of macrophage inflammatory protein-1 alpha (MIP-1α) increased.

Together, these findings suggest that **BioDisrupt® treatment shifts biofilm-associated immune signaling toward phagocyte recruitment. The observed immune effect adds to an existing rationale for pairing biofilm disruption with antimicrobial treatment: the biofilm matrix can limit antibiotic access to *B. burgdorferi*, and bacteria within it may become less metabolically active, reducing their sensitivity to antibiotics that target actively growing cells. These mechanisms support addressing biofilm alongside antibiotics and/or antimicrobial herbs in Lyme protocols.**

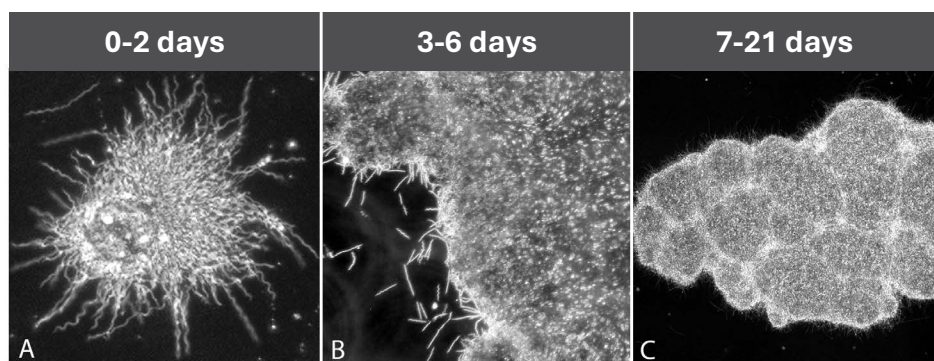
Two immune signals at the center of the findings

IL-10

An immune-regulating cytokine that helps regulate inflammation. During persistent infection, elevated IL-10 signaling may suppress phagocyte recruitment and phagocytosis, weakening immune activity needed to address the infection.

MIP-1α

A chemokine that helps recruit macrophages and other phagocytic immune cells to an affected area.



Biofilm formation and dispersal

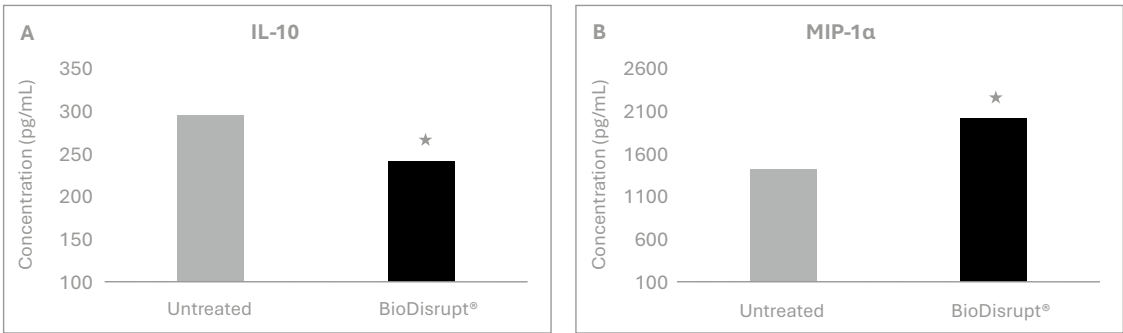
Free-floating planktonic bacteria attach to a surface, aggregate, and mature into a community embedded in a protective extracellular matrix. Epigenetic regulation helps the bacteria adapt to a sessile lifestyle within the biofilm. The mature biofilm releases metabolites that can influence host biology and immune signaling. Bacteria may later disperse from the biofilm and return to a planktonic state, allowing them to colonize new sites.

Main findings

The immune response to *B. burgdorferi* metabolites changed in response to BioDisrupt® treatment. The most clinically relevant shifts involved MIP-1α and IL-10.

	UNTREATED <i>BORRELIA</i> BIOFILM	AFTER BIODISRUPT® TREATMENT	CLINICAL SIGNIFICANCE
IL-10	Metabolites from untreated <i>Borrelia</i> triggered a pronounced increase in IL-10 production.	IL-10 decreased in BioDisrupt®-treated cells, a signal that can hold back phagocyte recruitment and phagocytosis.	Lower IL-10 suggests less biofilm-driven immune suppression. This supports addressing biofilm alongside antimicrobial treatment.
MIP-1α	Untreated <i>Borrelia</i> biofilm metabolites induced MIP-1α at baseline levels.	MIP-1α increased in BioDisrupt®-treated cells, suggesting stronger signaling for phagocyte recruitment.	Higher MIP-1α suggests a stronger cue for phagocytes to reach the site of infection. Read alongside the IL-10 decrease, the response moves toward conditions more favorable for clearance.
Other signals	<i>Borrelia</i> metabolites also induced pro-inflammatory and immune-regulating cytokines.	IL-1β, IL-6, and IL-1ra remained similar to untreated biofilm, indicating a selective rather than global cytokine shift.	The response reflected a selective change in immune signaling rather than a broad increase or decrease in cytokine production.

Metabolites from untreated *B. burgdorferi* biofilm may support immune evasion by driving IL-10 production. After BioDisrupt® treatment, IL-10 decreased and MIP-1α increased, shifting the response toward stronger signaling for phagocyte recruitment.



Immune-cell response to metabolites from untreated or BioDisrupt®-treated *B. burgdorferi* biofilms. Established biofilms were left untreated or treated with BioDisrupt® (0.5 mg/mL) for 24 hours. The collected, sterile-filtered fractions were then applied to human peripheral blood mononuclear cells for 24 hours. Compared with fractions from untreated biofilms, fractions from BioDisrupt®-treated biofilms elicited lower IL-10 (A) and higher MIP-1α (B) production (shown in pg/mL). *p < 0.01 versus untreated biofilm. Adapted from Cruickshank et al., *Microorganisms*. 2024;12(5):991.

What practitioners need to know

Pair biofilm disruption with antimicrobial treatment.

Borrelia’s ability to evade immune clearance via biofilm is a mechanistic reason to address biofilm alongside antibiotics or antimicrobial herbs.

Treat biofilm as biologically active.

Metabolites released from *B. burgdorferi* biofilm triggered high IL-10 production in human immune cells, even though no bacteria were present in the immune-cell culture.

Consider IL-10 and MIP-1α changes together.

BioDisrupt® treatment lowered IL-10 and increased MIP-1α. Together, these changes suggest less IL-10-mediated immune suppression and stronger signaling for phagocyte recruitment.

Taken together, these findings support biofilm disruption as a complementary component of a Lyme protocol that includes antimicrobial treatment.

Published studies

1. Cruickshank D, et al. Secreted Metabolites from *Pseudomonas*, *Staphylococcus*, and *Borrelia* Biofilm: Modulation of Immunogenicity by a Nutraceutical Enzyme and Botanical Blend. *Microorganisms*. 2024;12(5):991. doi:10.3390/microorganisms12050991

2. Jensen GS, et al. Disruption of Established Bacterial and Fungal Biofilms by a Blend of Enzymes and Botanical Extracts. *J Microbiol Biotechnol*. 2023;33(6):715-723. doi:10.4014/jmb.2212.12010.



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