

LIPID MEDIATORS IN HOST-PATHOGEN INTERACTIONS DURING LEISHMANIA INFECTION:
AN INTEGRATIVE REVIEW

**TAINA LARISSA PIRES NASCIMENTO¹, DARLAINE ALVES DA SILVA^{1,2}, KAREN MARTINS
DORIA DOS SANTOS¹, MATHEUS OLIVEIRA SOUSA¹, BEATRIZ LORRANY DE ARAÚJO
CARVALHO¹, YASMIN MONARA FERREIRA DE SOUSA ANDRADE¹, THÉO
ARAÚJO-SANTOS^{1,2,3}**

¹LABORATÓRIO DE AGENTES INFECCIOSOS E VETORES (LAIVE), CENTRO DAS CIÊNCIAS
BIOLÓGICAS E DA SAÚDE (CCBS), UNIVERSIDADE FEDERAL DO OESTE DA BAHIA (UFOB),
BAHIA, BRASIL.

²PROGRAMA MULTICÊNTRICO DE BIOQUÍMICA E BIOLOGIA MOLECULAR (PMBqBM),
CENTRO DAS CIÊNCIAS BIOLÓGICAS E DA SAÚDE (CCBS), UNIVERSIDADE FEDERAL DO
OESTE DA BAHIA (UFOB), BAHIA, BRASIL.

Lipid mediators can control the inflammatory response in several infectious diseases, including leishmaniasis. However, these mediators may promote antagonistic roles in the *Leishmania*-host interaction depending on the *Leishmania* species involved in the infection. Herein, we analyzed the role of mediators in the *Leishmania*-host interaction in the experimental and clinical context, aiming to identify the main cell types studied, as well as the main eicosanoids and their influence during the infection. For this, we performed an integrative review including studies that evaluated the role lipid mediators in vivo and in vitro experimental models of *Leishmania* infection, as well as in clinical studies up to the present date. We identified 41 in vitro experimental articles, 13 in vivo experimental articles and 6 clinical studies. The main lipid mediators studied in the pathogen-host interaction were the eicosanoids LTB₄ and PGE₂, which are related to the inflammatory response in cutaneous and visceral leishmaniasis. In vitro models using macrophages and neutrophils infection reveal that LTB₄ plays a fundamental role in reducing the parasite load, while PGE₂ and PGF_{2α} promote the suppression of the cellular response, favoring the survival of the parasite in the host. In the in vivo infection, PGE₂ is related to the visceralization process of the disease and the persistence of tegumentary lesions. An emerging role in pathophysiology has been pointed out for mediators of the HETE class and for Resolvin D1, which act favoring *Leishmania* infection and are associated with more severe cases of the disease. Thus, it can be concluded that lipid mediators play crucial roles in the *Leishmania*-host interaction, modulating the inflammatory response and disease progression. Studies exploring the contribution of intervention in the production of lipid mediators during the course of the disease are still needed.

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