

# Cytoskeletal dysregulation underlies Buruli ulcer formation

March 15 2013

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*Mycobacterium ulcerans* infects the skin and subcutaneous tissues and secretes a lipid toxin, mycolactone, which causes open skin lesions, known as Buruli ulcers.

In this issue of the *Journal of Clinical Investigation*, researchers led by Caroline Demangel at the Pasteur Institute in Paris investigated the molecular actions of mycolactone and found that it dysregulates the cellular skeleton (cytoskeleton) through activation of a protein known as N-WASP.

They found that excessive N-WASP activity caused defects in cell adhesion and migration that impaired the integrity of the skin.

Demangel and colleagues demonstrated that they could block the degradation process by administration of the N-WASP inhibitor wiskostatin.

These results reveal the [molecular pathogenesis](#) of *M. ulcerans* and suggest that drugs that disrupt mycolactone/N-WASP interaction could be used to treat Buruli ulcers.

**More information:** Mycolactone activation of Wiskott-Aldrich syndrome proteins underpins Buruli ulcer formation, *J Clin Invest.* [doi:10.1172/JCI66576](https://doi.org/10.1172/JCI66576)

Provided by Journal of Clinical Investigation

Citation: Cytoskeletal dysregulation underlies Buruli ulcer formation (2013, March 15) retrieved 28 April 2024 from

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