

Accepted Manuscript

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PII: S0022-202X(16)31138-1

DOI: [10.1016/j.jid.2016.04.017](https://doi.org/10.1016/j.jid.2016.04.017)

Reference: JID 312

To appear in: *The Journal of Investigative Dermatology*

Received Date: 19 October 2015

Revised Date: 14 April 2016

Accepted Date: 18 April 2016

Please cite this article as: Wang X, Hu C, Wu X, Wang S, Zhang A, Chen W, Shen Y, Tan R, Wu X, Sun Y, Xu Q, Roseotoxin B Improves Allergic Contact Dermatitis through a Unique Anti-inflammatory Mechanism Involving Excessive Activation of Autophagy in Activated T-Lymphocytes, *The Journal of Investigative Dermatology* (2016), doi: 10.1016/j.jid.2016.04.017.

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Unique Anti-inflammatory Mechanism Involving Excessive
Activation of Autophagy in Activated T-Lymphocytes**

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