Accepted Manuscript

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Clinical MMUNOLOGY
The Official journal of the Clinical Immunology Society

The Official Journal of the Official Immunology Society

The Official Immunology Society

PII: S1521-6616(17)30622-8

DOI: doi:10.1016/j.clim.2018.01.001

Reference: YCLIM 7990

To appear in: Clinical Immunology

Received date: 25 August 2017 Revised date: 30 October 2017 Accepted date: 2 January 2018

Please cite this article as: Tie Zheng Hou, Peter Olbrich, Jose Manuel Lucena Soto, Berta Sanchez, Paula Sanchez Moreno, Stephan Borte, Hans J. Stauss, Siobhan O. Burns, Lucy S.K. Walker, Qiang Pan-Hammarström, Lennart Hammarström, David M. Sansom, Olaf Neth, Study of an extended family with CTLA-4 deficiency suggests a CD28/CTLA-4 independent mechanism responsible for differences in disease manifestations and severity. The address for the corresponding author was captured as affiliation for all authors. Please check if appropriate. Yclim(2017), doi:10.1016/j.clim.2018.01.001

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ACCEPTED MANUSCRIPT

Study of an extended family with CTLA-4 deficiency suggests a CD28/CTLA-4 independent mechanism responsible for differences in disease manifestations and severity

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