

Vaccine-induced protection against 3 systemic mycoses endemic to North America requires Th17 cells in mice

Marcel Wüthrich, ... , Garry Cole, Bruce Klein

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Corrigendum

Original citation: *J Clin Invest.* 2011;121(2):554–568. doi:10.1172/JCI43984. Citation for this corrigendum: *J Clin Invest.* 2016;126(2):795. doi:10.1172/JCI85788. The authors recently became aware that the IL-1R mice used for the original Supplemental Figure 7, A and B, were incorrectly genotyped and were heterozygous rather than homozygous knockout animals. The experiment with IL1r^{-/-} animals was repeated, and the correct Supplemental Figure 7 is now available online. The correct text describing the experiments in the Results and Discussion sections appears below. Results Lung CFUs also were reduced to the same extent in vaccinated IL18r^{-/-} and wild-type mice (Supplemental Figure 7B). In contrast, IL-17-producing T cells recruited to the lungs of IL1r^{-/-} mice were reduced, and the mice failed to acquire resistance in comparison with vaccinated wild-type controls. Thus, IL-18R, but not IL-1R, is dispensable in the development of T17 cells and vaccine resistance. Moreover, failed T17 differentiation of 1807 cells in Myd88^{-/-} mice is not due to impaired IL-18R signaling, but is likely due to impaired signaling via TLRs and IL-1R. Discussion The fact that adoptively transferred wild-type 1807 cells failed to recruit to the lung in Myd88^{-/-} mice and showed a deficit in IL1r^{-/-}, but not IL18r^{-/-}, mice indicates that the deficits in Myd88^{-/-} mice are not due to impaired IL-18R signaling, but are likely due to impaired signaling via TLRs and IL-1R. The [...]

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Vaccine-induced protection against 3 systemic mycoses endemic to North America requires Th17 cells in mice

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Results

Lung CFUs also were reduced to the same extent in vaccinated *Il18r^{-/-}* and wild-type mice (Supplemental Figure 7B). In contrast, IL-17-producing T cells recruited to the lungs of *IL1r^{-/-}* mice were reduced, and the mice failed to acquire resistance in comparison with vaccinated wild-type controls. Thus, IL-18R, but not IL-1R, is dispensable in the development of T17 cells and vaccine resistance. Moreover, failed T17 differentiation of 1807 cells in *Myd88^{-/-}* mice is not due to impaired IL-18R signaling, but is likely due to impaired signaling via TLRs and IL-1R.

Discussion

The fact that adoptively transferred wild-type 1807 cells failed to recruit to the lung in *Myd88^{-/-}* mice and showed a deficit in *IL1r^{-/-}*, but not *Il18r^{-/-}*, mice indicates that the deficits in *Myd88^{-/-}* mice are not due to impaired IL-18R signaling, but are likely due to impaired signaling via TLRs and IL-1R.

The authors regret the error.

Erratum

Additive loss-of-function proteasome subunit mutations in CANDLE/PRAAS patients promote type I IFN production

Anja Brehm, Yin Liu, Afzal Sheikh, Bernadette Marrero, Ebun Omoyinmi, Qing Zhou, Gina Montealegre, Angelique Biancotto, Adam Reinhardt, Adriana Almeida de Jesus, Martin Pelletier, Wanxia L. Tsai, Elaine F. Remmers, Lela Kardava, Suvimol Hill, Hanna Kim, Helen J. Lachmann, Andre Megarbane, Jae Jin Chae, Jilian Brady, Rhina D. Castillo, Diane Brown, Angel Vera Casano, Ling Gao, Dawn Chapelle, Yan Huang, Deborah Stone, Yongqing Chen, Franziska Sotzny, Chyi-Chia Richard Lee, Daniel L. Kastner, Antonio Torrelo, Abraham Zlotogorski, Susan Moir, Massimo Gadina, Phil McCoy, Robert Wesley, Kristina I. Rother, Peter W. Hildebrand, Paul Brogan, Elke Krüger, Ivona Aksentijevich, and Raphaela Goldbach-Mansky

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Kristina I. Rother's middle initial was inadvertently omitted from the author list. The correct author list is above.

The *JCI* regrets the error.