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

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Erratum

Original citation: J Clin Invest. 2015;125(11):4196–4211. doi:10.1172/JCI81260. Citation for this erratum: J Clin Invest. 2016;126(2):795. doi:10.1172/JCI86020. Kristina I. Rother's middle initial was inadvertently omitted from the author list. The correct author list is above. The JCI regrets the error.

Corrigendum

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

Marcel Wüthrich, Benjamin Gern, Chiung Yu Hung, Karen Ersland, Nicole Rocco, John Pick-Jacobs, Kevin Galles, Hanna Filutowicz, Thomas Warner, Michael Evans, Garry Cole, and Bruce Klein

Original citation: *J Clin Invest*. 2011;121(2):554–568. doi:10.1172/JCI43984.

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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 authors regret the error.

Erratum

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

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