

Corrigendum

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

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

The *JCI* regrets the error.