

Myeloid phagocytes of the respiratory immune system, such as neutrophils, monocytes, and alveolar macrophages, are essential for immunity to *Aspergillus fumigatus*, the most common etiologic agent of mold pneumonia worldwide. Following the engulfment of *A. fumigatus* conidia, fusion of the phagosome with the lysosome is a critical process for killing conidia. TFEB and TFE3 are transcription factors that regulate lysosomal biogenesis under stress and are activated by inflammatory stimuli in macrophages, but it is unknown whether TFEB and TFE3 contribute to anti-*Aspergillus* immunity during infection. We found that lung neutrophils express TFEB and TFE3, and their target genes were upregulated during *A. fumigatus* lung infection. In addition, *A. fumigatus* infection induced nuclear accumulation of TFEB and TFE3 in macrophages in a process regulated by Dectin-1 and CARD9. Genetic deletion of *Tfeb* and *Tfe3* impaired macrophage killing of *A. fumigatus* conidia. However, in a murine immune-competent *Aspergillus* infection model with genetic deficiency of *Tfeb* and *Tfe3* in hematopoietic cells, we surprisingly found that lung myeloid phagocytes had no defects in conidial phagocytosis or killing. Loss of TFEB and TFE3 did not impact murine survival or clearance of *A. fumigatus* from the lungs. Our findings indicate that myeloid phagocytes activate TFEB and TFE3 in response to *A. fumigatus*, and while this pathway promotes macrophage fungicidal activity in vitro, genetic loss can be functionally compensated in the lung, resulting in no measurable defect in fungal control and host survival.