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Titel des Beitrags:
The triggering receptor expressed on myeloid cells 2 inhibits complement component 1q effector mechanisms and exerts detrimental effects during pneumococcal pneumonia.

Abstract:
Phagocytosis and inflammation within the lungs is crucial for host defense during bacterial pneumonia. Triggering receptor expressed on myeloid cells (TREM)-2 was proposed to negatively regulate TLR-mediated responses and enhance phagocytosis by macrophages, but the role of TREM-2 in respiratory tract infections is unknown. Here, we established the presence of TREM-2 on alveolar macrophages (AM) and explored the function of TREM-2 in the innate immune response to pneumococcal infection in vivo. Unexpectedly, we found Trem-2/-/- AM to display augmented bacterial phagocytosis in vitro and in vivo compared to WT AM. Mechanistically, we detected that in the absence of TREM-2, pulmonary macrophages selectively produced elevated complement component 1q (C1q) levels. We found that these increased C1q levels depended on peroxisome proliferator-activated receptor-? (PPAR-?) activity and were responsible for the enhanced phagocytosis of bacteria. Upon infection with S. pneumoniae, Trem-2/-/- mice exhibited an augmented bacterial clearance from
lungs, decreased bacteremia and improved survival compared to their WT counterparts. This work is the first to disclose a role for TREM-2 in clinically relevant respiratory tract infections and demonstrates a previously unknown link between TREM-2 and opsonin production within the lungs.