The Journal of Immunology

IN BRIEF | APRIL 01 2023

Top Reads FREE

Online ISSN: 1550-6606
Print ISSN: 0022-1767

2023

Copyright © 2023 by The American Association of Immunologists, Inc.

J Immunol (2023) 210 (7): 855.
https://doi.org/10.4049/jimmunol.2390004

Related Content

IL-1/MyD88–Dependent G-CSF and IL-6 Secretion Mediates Postburn Anemia

J Immunol (February,2023)

Commensal Bacteria and the Lung Environment Are Responsible for Th2-Mediated Memory Yielding Natural IgE in MyD88-Deficient Mice

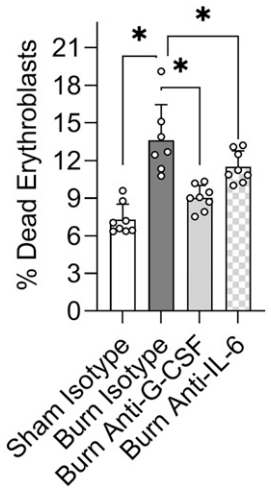
J Immunol (February,2023)

Top Reads

J Immunol (February,2022)

The Journal of Immunology

G-CSF and IL-6 Drive Postburn Anemia

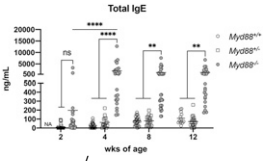


In this Top Read, Noel et al. (p. 972) showed that postburn anemia of critical illness (ACI) is enhanced by G-CSF and IL-6 secretion and is driven by IL-1/MyD88 signaling at the site of the burn wound. Using a previously described mouse model of ACI, G-CSF reduced erythroid composition in the bone marrow, whereas IL-6 reduced access of erythroid cells to iron via the transferrin receptor. MyD88, TLR2, and TLR4 individual knockout mice, and IL-1 neutralization, showed that G-CSF and IL-6 secretion was MyD88- and IL-1-dependent. IL-1α/β blockade improved serum iron levels and attenuated postburn anemia, thereby ameliorating ACI. Finally, skin samples from both the ACI mouse model and human eschar skin samples represented in a public RNA sequencing repository showed that high G-CSF and IL-6 protein secretion originates from burned skin. Altogether, the data provide a mechanism by which increased cytokines produced by severely burned skin drives ACI, and it suggests blocking IL-1 signaling as a future therapeutic.

SARS-CoV-2 Vaccine Responses in Persons with HIV

Persons with HIV (PWH) have high risk for severe COVID-19 disease, perhaps in part due to persistent immune dysfunction, even with effective antiretroviral therapy (ART). In this Top Read, Quinn et al. (p. 947) examined the magnitude and heterogeneity of immune responses following SARS-CoV-2 vaccination in PWH on ART. Of the 11 patients analyzed, 2 received mRNA vaccines and 9 received replication-incompetent vector vaccines. Spike-specific Ab was increased in all patients following vaccination; however, the highest Ab titers were in patients receiving the mRNA vaccine and in a patient with a previous SARS-CoV-2 infection. The neutralization potential also correlated with the magnitude of the spike-specific Ab response. The mRNA-vaccinated patients and the previously infected patient also showed the most robust virus-specific T and B cell responses. Virus-specific B cells were absent in only two patients, who also had the lowest Ab responses. Interestingly, spike-specific germinal centers (GC) were absent in all but one of the vector-vaccinated patients, although vector vaccination of an HIV-negative patient resulted in spike-specific GC formation. These data indicate that PWH on ART can mount an immune response to SARS-CoV-2 vaccination; however, these responses are varied and require additional investigation.

Lung Microbiota Induces IgE in *MyD88*$^{-/-}$ Mice



In this Top Read, Amano et al. (p. 959) demonstrated that memory B cells (MBC) spontaneously produce natural IgE in response to commensal lung *Streptococcus azizii* in MyD88-deficient (*MyD88*$^{-/-}$) mice. Plasma cells and B cells were differentially depleted in *MyD88*$^{-/-}$ mice, resulting in transient and sustained decreases in serum IgE, respectively, suggesting that MBC are responsible for IgE production. Dysbiosis in the lung was noted, wherein *MyD88*$^{-/-}$ mice had increased *S. azizii* colonization compared with separately housed *MyD88*$^{+/+}$ mice. Serum IgE levels decreased in the *MyD88*$^{-/-}$ mice following antibiotic treatment. Additionally, *S. azizii* infection increased serum IgE levels in *MyD88*$^{-/-}$ mice but had no effect on IgE levels in *MyD88*$^{+/+}$ mice. Lung exudate cells from *MyD88*$^{-/-}$ mice stimulated in vitro with *S. azizii* produced high levels of IL-4 and IL-13, suggesting that the bacteria primed a Th2 response supporting IgE production by MBC. Finally, excessive CSF1 produced by nonhematopoietic cells in the lung was responsible for the natural IgE production in *MyD88*$^{-/-}$ mice. Because spontaneously produced natural IgE can increase risk for allergic diseases, these data provide insight into mechanisms that may lead to high levels of serum IgE and provide potential targets for treatment.

www.jimmunol.org/cgi/doi/10.4049/jimmunol.2390004