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# Lung Icams and Vcam-1 in Innate and Adaptive Immunity to Influenza Infections: Implications for Vaccination Strategies

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**Abstract :** The b2 integrin ligands ICAM-1 ICAM-2 and the endothelial VLA-4 integrin ligand VCAM-1 are constitutively expressed on different lung vessels and on high endothelial venules (HEVs), the main portal for lymphocyte entry from the blood into lung draining lymph nodes. ICAMs are also ubiquitously expressed by many antigen-presenting leukocytes and have been traditionally suggested as critical for the various antigen-specific immune synapses generated by these distinct leukocytes and specific naïve and effector T cells. Loss of both ICAM-1 and ICAM-2 on the lung vasculature reduces the ability to patrol monocytes and Tregs to patrol the lung vasculature at a steady state. Our new findings suggest, however, that in terms of innate leukocyte trafficking into the lung lamina propria, both constitutively expressed and virus-induced vascular VCAM-1 can functionally compensate for the loss of these ICAMs. In a mouse model for influenza infection, neutrophil and NK cell recruitment and clearance of influenza remained normal in mice deficient in both ICAMs. Strikingly, mice deficient in both ICAMs also mounted normal influenza-specific CD8 proliferation and differentiation. In addition, these mice normally combated secondary influenza infection, indicating that the presence of ICAMs on conventional dendritic cells (cDCs) that present viral antigens are not required for immune synapse formation between these APCs and naïve CD8 T cells as previously suggested. Furthermore, long-lasting humoral responses critical for protection from a secondary homosubtypic influenza infection were also normal in mice deficient in both ICAM-1 and ICAM-2. Collectively, our results suggest that the expression of ICAM-1 and ICAM-2 on lung endothelial and epithelial cells, as well as on DCs and B cells, is not critical for the generation of innate or adaptive anti-viral immunity in the lungs. Our findings also suggest that endothelial VCAM-1 can substitute for the functions of vascular ICAMs in leukocyte trafficking into various lung compartments.

**Keywords :** emigration, ICAM-1, lymph nodes, VCAM-1
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