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Autore	Klaus Ley
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Sommario/riassunto	Arrest chemokines are a small group of chemokines that promote leukocyte arrest from rolling by triggering rapid integrin activation. Arrest chemokines have been described for neutrophils, monocytes, eosinophils, naïve lymphocytes and effector memory T cells. Most arrest chemokines are immobilized on the endothelial surface by binding to heparan sulfate proteoglycans. Whether soluble chemokines can promote integrin activation and arrest is controversial. Many aspects of the signaling pathway from the GPCR chemokine receptor to integrin activation are the subject of active investigation. Leukocyte adhesion deficiency III is a human disease in which chemokine-triggered integrin activation is defective because of a mutation in the cytoskeletal protein kindlin-3. About 10 different such mutations have been described. The defects seen in patients with LAD-III elucidate the importance of rapid integrin activation for host defense in humans. Here we present a series of ten reports that help clarify this crucial first step in the process of leukocyte transendothelial migration.

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