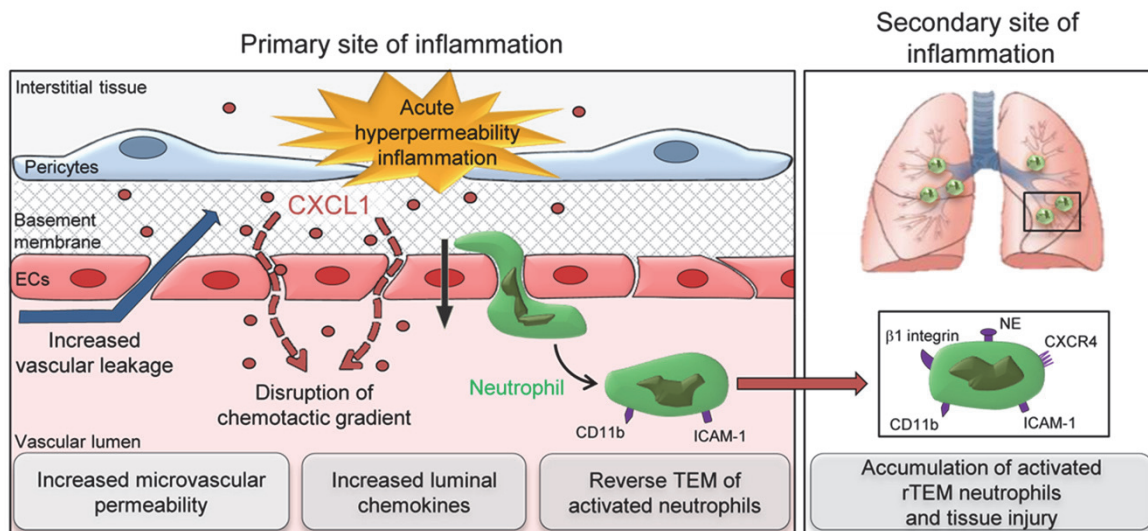


Local microvascular leakage promotes trafficking of activated neutrophils to remote organs

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Key findings

1. Enhanced microvascular permeability triggers retrograde motility of neutrophils within EC junctions, prompting neutrophil rTEM in vivo.
2. Mechanistically, increased vascular leakage aids movement of interstitial chemokines into the blood stream, disrupting their tissue localization.
3. Together, first experimental and mathematical evidence of bi-directional movement of molecules and cells across ECs at sites of inflammation.
4. In terms of pathophysiology, a novel in vivo cell labelling method reveals systemic dissemination, and an activated phenotype, of rTEM neutrophils.
5. Collectively, the present cascade of events offer a mechanism for the capacity of local hyperpermeability reactions to elicit remote organ damage.

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