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Developmental endothelial locus–1 (Del-1), required for prevention of interleukin-17–mediated pathology, reduced by age

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Developmental endothelial locus-1 (Del-1) was recently identified as a novel endothelial-derived inhibitor of neutrophil extravasation. However, whether Del-1 regulates the local tissue-specific inflammatory response and controls chronic inflammatory diseases has not been addressed yet. Periodontitis, a prevalent chronic disease with an impact on systemic health, is critically dependent on neutrophils. Upon aging, normal mice displayed increased disease accompanied by diminished Del-1 expression. Consistent with a protective role for Del-1 in periodontitis, Del-1-/- mice developed spontaneous inflammatory periodontal bone loss characterized by excessive local neutrophil infiltration and interleukin (IL)-17 expressions. The disease was reversed in Del-1-/- mice with additional genetic deficiencies in the LFA-1 integrinor the IL-17 receptor. Strikingly, local administration of Del-1 suppressed neutrophil infiltration and IL-17 expression in the periodontal tissue. Therefore, Del-1 is required for tissue homeostasis by regulating LFA-1-dependent neutrophil trafficking, inhibiting IL-17-mediated pathology, and may be a promising novel therapeutic for the treatment of inflammatory diseases.

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