Whereas T cells are generally thought of as mediators of tissue damage in chronic tissue inflammation, the cellular infiltrate is always dominated by myeloid cells. The granulocyte-macrophage colony-stimulating factor (GM-CSF) was initially classified as a hematopoietic growth factor. However, unlike its close relatives macrophage CSF (M-CSF) and granulocyte CSF (G-CSF), the majority of myeloid cells do not require GM-CSF for steady-state myelopoiesis. Instead, in inflammation, GM-CSF serves as a communication conduit between tissue-invading lymphocytes and myeloid cells. Even though lymphocytes are in all likelihood the instigators of chronic inflammatory disease, GM-CSF-activated phagocytes are well equipped to cause tissue damage. The pivotal role of GM-CSF at the T cell: myeloid cell interface might shift our attention toward studying the function of the myeloid compartment in immunopathology and targeting specifically the crosstalk between T cells and myeloid cells through GM-CSF holds promise for the development of therapeutics to combat chronic tissue inflammation. I will discuss how GM-CSF licenses phagocytes to initiate tissue damage in chronic inflammatory diseases.

**ABSTRACT**

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