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Efferocytosis, the clearance of dead and dying cells, is fundamental for tissue homeostasis and immune response (Boada-Romero et al. 2020). In the absence of efferocytosis, dead cells accumulate and cause tissue damage and inflammation. MEGF10 and CED-1 are key receptors involved in efferocytosis. MEGF10 is expressed on macrophages and is essential for the engulfment of apoptotic cells. CED-1 is a conserved efferocytosis receptor in *Caenorhabditis elegans*. The interaction between MEGF10 and CED-1 is critical for the recognition and engulfment of dead cells. Studies in *C. elegans* and mice have shown that MEGF10 and CED-1 are essential for the clearance of dead cells and for the resolution of inflammation. MEGF10 is also involved in the regulation of macrophage function and in the development of atherosclerosis. CED-1 is also involved in the regulation of macrophage function and in the development of atherosclerosis. The study of efferocytosis and the role of MEGF10 and CED-1 is important for understanding the mechanisms of tissue repair and the pathogenesis of inflammatory diseases.

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In the Williamson lab, we have been studying the role of MEGF10 in macrophage efferocytosis. We have identified several novel MEGF10 ligands and are currently studying their role in macrophage function and in the development of atherosclerosis. We have also identified several novel CED-1 ligands and are currently studying their role in macrophage function and in the development of atherosclerosis. Our research is focused on understanding the mechanisms of efferocytosis and the role of MEGF10 and CED-1 in the resolution of inflammation and in the development of atherosclerosis.

Efferocytosis is the engulfment of dead and dying cells. Dead and dying cells left uncleared will undergo secondary necrosis, which can cause damage to surrounding tissues (Boada-Romero et al. 2020). Lack of clearance is associated with early