Phagocytosis of apoptotic cells and its physiological roles.

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**Abstract**

In our body, billions of cells die by apoptosis every day. The dying cells secrete “find me” signals, and expose “eat me” signals on cell surface for their clearance. In response to the “find me” signals, phagocytes approach the dying cells, and then recognize phosphatidylserine exposed on their surface as the “eat me” signal. Phagocytes recognize phosphatidylserine through specific receptors or via bridging molecules that link apoptotic cells to phagocytes. Phagocytes engulf the apoptotic cells in a Rac1-dependent manner probably through specific portals. When apoptotic cells are not engulfed efficiently, they undergo secondary necrosis and release intracellular components, which lead to local inflammation and SLE-type autoimmune diseases. Other examples of phosphatidylserine-dependent phagocytosis are the involution of mammary glands and the axon pruning that occurs during the development of neural circuits. In the involution of mammary glands, apoptotic mammary epithelial cells and milk fat globules are engulfed by living epithelial cells in a phosphatidylserine-dependent manner. The failure of this process induces mammary duct ectasia and periductal mastitis. In axon pruning, unnecessary or extra axons degenerate, expose phosphatidylserine, and are engulfed by glial phagocytes. Our latest findings and the future directions of the research will be discussed.